REICHEL'S

Care of the Elderly

Clinical Aspects of Aging







EIGHTH EDITION

EDITED BY
Jan Busby-Whitehead
Samuel C. Durso
Christine Arenson
Mary H. Palmer
Rebecca Elon
William Reichel

CAMBRIDGE Medicine

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In Memoriam for Dr. Reichel

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Chapter 1

Essential Principles in the Care of the Elderly

Jan Busby-Whitehead, Samuel C. Durso, and William Reichel

The world is aging. In 2020, the US Bureau of the Census reported that 54.1 million citizens (16.5%) were 65 years or older.[1] The first baby boomers turned 65 in 2011, and 80.8 million adults will be over the age of 65 by 2040. The fastest-growing segment of the population, those aged 85 to 99, will reach 14.4 million in 2040. Centenarians numbered 100,322 in 2020; this group will continue to grow. Further, 23% of older adults have diverse racial and ethnic backgrounds. This percentage is projected to increase to 34% (27.7 million) of older adults in 2040. Nearly 1 in 10 people aged 65 and older (8.9% or 4.9 million) in 2020 lived below the poverty level. Another 2.6 million (4.4%) were classified as "near-poor." The poverty threshold in 2018 was $12,261 for householders aged 65 and older who lived alone.[1]

Advances in chronic disease management, new medical therapies, diffusion of "best practices," and increased attention to maintaining physical, cognitive, and psychological function are likely to extend average life expectancy and years of active life. However, it must be noted that 19% of adults aged 65 and older reported they could not function at all or had a lot of difficulty with at least one of six functioning domains (vision, hearing, mobility, communication, cognition, and/or self-care).[1] Other forces that impede progress toward healthy aging include persistent health equity issues in our health-care system, a decreasing ratio of workers to support older adults, and an increasing burden on family caregivers.

Everyone wants good health care in the "golden years." But what is good care? In the care of the elderly patient, 11 essential principles should be considered: (1) the role of the physician as the integrator of the biopsychosocial-spiritual model; (2) continuity of care; (3) bolstering the family and home; (4) good communication skills; (5) building a sound doctor–patient relationship; (6) the need for appropriate evaluation and assessment; (7) prevention and health maintenance; (8) intelligent treatment with attention to ethical decision-making; (9) interprofessional collaboration; (10) respect for the usefulness and value of the aged individual; and (11) compassionate care.

These 11 principles are consistent with the new framework for creating Age-Friendly Health Systems, a movement led by the John A. Hartford Foundation and the Institute for Healthcare Improvement in partnership with the American Hospitals Association and the Catholic Health Association of the United States.[2] The 4Ms are the essential initial elements that health-care systems need in order to provide older adults with the best care possible: What Matters, Medication, Mentation, and Mobility.[2] This framework is a new way to organize care for older adults to ensure consistency in all health-care settings. Following the 11 principles, in concert with the 4Ms, will enable health-care practitioners to provide optimal care.

The Physician as Integrator of the Biopsychosocial-Spiritual Model

The past 50 years have witnessed enormous growth in technology and options to cure acute illness and manage chronic conditions. However, one result of this trend has been increasingly complex, specialized care. Good care requires having a physician who provides leadership in the integration and coordination of the health care of the elderly patient. The current generation of older adults has witnessed amazing advances in research and diagnostic and curative medicine, but it has also seen that a reductionist approach to human disease can result in fragmented and poorly coordinated medical care. It is imperative that the health-care professional responsible for the care of older adults keeps the "big picture" firmly in mind – never forgetting that the patient is much more than the sum of his/her organ systems.[3,4]

Society is calling for physicians with a commitment to the person, not just specific disease states or mechanisms. The person is part of a family and a larger community, or sadly, without family and/or community. An essential role for the physician is to act as integrator for the elder

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Society is calling for physicians with a commitment to the person, not just specific disease states or mechanisms. The person is part of a family and a larger community, or sadly, without family and/or community. An essential role for the physician is to act as integrator for the elder

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within a biopsychosocial and spiritual model. To accomplish this, the physician must know the patient thoroughly. This does not diminish the importance of specialties and subspecialties within this model. But ideally, holistic health care not only considers the contribution of specialists to elder health care but also includes full consideration of the individual's physiological, functional, emotional, and psychosocial state, informed by continuity of experience with the patient over time.

In recent years, medical specialty organizations, purchasers of care, and third-party payers have recognized the need to reintegrate coordinated, person-centered, primary care as the foundation of an effective healthcare system. Further, the palliative care and hospice movement, public calls to support dignity in aging and during the dying process, and an increased focus on shared decision-making all depend on enhanced integration of person-centered care. The 4Ms framework provides a way to achieve these goals.[2]

In addition, effective primary care must recognize and incorporate social determinants of health into the overall care for individuals and populations.[5] The Patient-Centered Medical Home is designed to achieve this integration and has been embraced by family medicine, internal medicine, and geriatric medicine as a framework for "continuous, caring relationships" and to restore a robust primary care infrastructure in the USA. Other developed nations such as Canada, the United Kingdom, and Australia have already made significant strides in reinvigorating primary care and generalist practice.[6] No population will benefit more from effective, coordinated, well-resourced primary care than older adults.

The primary care provider must also ensure the coordination, supervision, and integration of care that is vital for the older patient to navigate a complex system that often provides conflicting recommendations and is not always organized to meet the needs of the patient. The primary physician, then, acts as advocate to obtain needed services, and as adviser. At times, the best advice is to avoid tests or treatments that have little potential benefit, but significant potential to harm. Perhaps most importantly, the physician will come to know the patient as an individual, within a family and a community, with particular values, beliefs, and priorities. Thus, the physician comes to serve as interpreter, integrator, and advocate, helping patients to obtain health care that is most consistent with their preferences and needs. This role will most often be a family physician, general internist, geriatrician, or nurse practitioner. However, for some patients, it may be a trusted oncologist, cardiologist, or other specialist. The key factors are the interest and ability to see the patient as a whole person and the commitment in time and expertise to serve in this critical role.

In the coming years, research will hopefully clarify the interaction of biological, psychological, social, and spiritual components to health. For example, clinical distrust, chronic stress, and depression have been linked with increased inflammatory markers that may result in higher rates of cardiovascular disease.[7] There is now overwhelming evidence that depression coexisting with diabetes leads to poor outcomes, including increased mortality.[8] One study has demonstrated that social support may play a protective effect with respect to interleukin-6 (IL-6) elevation, and thus might result in a survival benefit in ovarian cancer patients.[9] There is much to learn about the dynamic relationships between wellness and disease, psychosocial factors, and the spiritual state. The clinician in practice is aware of the higher mortality in the first year after widowhood, more pronounced in the surviving widower than in the widow, and the higher morbidity and mortality seen in elderly persons upon relocation.[10]

Continuity of Care

The ideal longitudinal clinical relationship is often one that is warm and supportive, with the same personal physician serving as adviser, advocate, and friend. However, the realities of today's complex medical environment, with the patient moving between clinic, home, hospital, specialized care units (coronary care units, intensive care units [ICUs], stroke units, or oncology centers), nursing home, and hospice care, often make this ideal impossible. In fact, patients often receive the best care from physicians and other health professionals who focus their practice in these specialized environments. The medical intensivist provides the most skilled care in the ICU; the physician in regular nursing home practice will be more available to patients, staff, and families than one who has a few nursing home patients scattered among several facilities.

The failure of physicians to make visits as necessary in the home and long-term care facilities is related to several factors in the United States, including training, physician attitudes, and reimbursement systems. Our medical schools and residencies for generalist physicians continue to struggle with incorporating meaningful house call and nursing home care as part of their training. Although reimbursement for visits to the home and nursing home has improved in recent years, high office overhead and productivity expectations continue to limit the ability of

physicians to practice in these relatively time-inefficient sites. Physician attitudes are also problematic, in that doctors of recent decades have been more interested in the acute aspects of care than in chronic and long-term care. These attitudes are reinforced by the educational environments and reimbursement systems. Fortunately, there is an increased push for research and educational initiatives designed to address this gap in chronic care knowledge and these attitudes of our students and residents.[11,12]

Nevertheless, continuity of care remains an essential principle in the care of the older patient.[13] A wealth of literature documenting the critical importance of adequate communication among health professionals around transitions in care lends support to the notion that safe, effective, efficient, and patient-centered care can only occur when the in-depth knowledge and understanding of the personal physician is communicated to and incorporated by the specialized teams in the ICU, general hospital, long-term care, and even hospice settings.[14,15,16] While electronic health records offer the promise of more effective and efficient communication within and across care teams and settings, that vision is not yet fully realized.

The physician caring for older adults must recognize that optimal health care often requires a team of professionals, including primary care and specialty physicians, hospitalists, nurses, pharmacists, therapists, and social workers. This does not abrogate, but rather highlights, the need for continuity in care. Physicians, nurse practitioners, and others with a long-term relationship with a patient may remain active advocates and sounding boards, even when they are not the "provider of record" at any point in time. Equally important to patient safety is attention to continuity at transition points in the care of the older patient – from home to hospital, and from hospital to rehabilitation unit or nursing home. The physician responsible for the care of patients at each juncture must communicate fully and accurately with the patient, family, and receiving health-care team, to ensure that the patient's treatment plan, values, expectations, and preferences are known and honored.

Bolstering Family and Home

Every physician should enlist those means that assist elderly persons, whenever possible and consistent with their goals of care, to maintain their independence, either in their own home or other setting. The physician should use the prescription for a nursing home as specifically as a prescription for any other intervention, with consideration of all of the potential benefits and harms.

A number of forces have resulted in patients going to institutional settings when other alternatives might have been possible. Between 1960 and 1975, a massive push toward institutionalization took place, creating hundreds of thousands of nursing home beds. What forces have contributed to overutilization of institutional care? One factor is funding that has disproportionately directed reimbursement for institutional care away from other alternatives. Another is increased mobility of families, smaller families, and increased numbers of women moving into the workforce, therefore limiting the number of family members able to participate in the care of their elders. In spite of these forces, rates of institutionalization have declined slightly in recent years, as older adults and their families have chosen to overcome obstacles to keep loved ones at home. Additionally, a rapid growth in largely privately funded assisted living facilities has provided an option for older adults with less extensive care needs and the financial resources to pay for lower-acuity, more home-like living environments.[17]

What alternative can the physician recommend? The list includes home health aides, other types of homecare, daycare, aftercare, specialized housing settings, visiting nurses, friendly visitors, foster homecare, chore services, home renovation and repair services, congregate and home-delivered meal programs, transportation programs, and shopping services.[18] Personal physicians should also understand and utilize legal and protective services for older patients whenever indicated.

Publicly financed programs such as the Program of All-Inclusive Care for the Elderly (PACE) and home-based Medicaid waiver programs that support nursing home-eligible elders to remain in their homes have grown in recent years, as federal and state governments have recognized that supporting seniors' desire to stay in their own homes is not only better, but actually less expensive care.[19] States have explored options to provide services in the homes of nursing home-eligible patients through a combination of Medical Assistance waivers and other programs. In addition, a growing body of research demonstrates the benefits of home-based interventions that target patient and caregiver priorities and teach problem-solving skills to maintain physically frail and demented individuals in their homes.[20]

In spite of the pressure to contain institutional long-term care costs, funds have not been available for adequate expansion of publicly funded programs to support frail older adults in their own homes. Further, many of

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the evidence-based interventions that might provide cost-effective strategies for supporting older persons in the community are not reimbursed by insurance. Thus, resources remain limited and disjointed. The role of health-care providers is to facilitate referrals, coordinate services, and become knowledgeable about general resources available and appropriate referral sources (i.e., care manager, area agency on aging) with expertise to help patients and families navigate the system effectively.

Who are the caregivers in American society? Data from the American Association of Retired Persons Caregiving in the U.S. 2020 report cites that 41.2 million Americans are providing care to someone who is 50-plus years old.[21] In 2018, the percentage of older adults over age 85 requiring personal care assistance (21%) was five times the percentage of adults aged 65–74 (4%).[1]

More women than men are caregivers, and women continue to provide more of the most difficult personal care tasks such as bathing. A majority of caregivers have spent an average of 20.4 hours per week on caregiving tasks for three or more years. This average increases to 39.3 hours per week for live-in caregivers. The average age of a caregiver is 63 years, and one third of all caregivers rate their own health as fair to poor: 40–70% of caregivers experience depression. Caring for a person with dementia may negatively impact the caregiver's immune function for up to three years after the experience ends.[22] Despite the very real burdens of caregiving, however, at least five population-based studies have found reduced mortality and extended longevity for caregivers compared with non-caregivers. Many caregivers in fact report benefits from their role.[23]

Family/informal caregiver services were valued at $470 billion in 2013, exceeding the value of paid homecare and total Medicaid spending in that year, and almost matching the value of the sales of Walmart ($477 billion).[24] Caregivers may experience adverse economic impacts, with many having to reduce working hours or even stop working. Those caring for older adults lost an estimated $13 trillion in wages, pensions, retirement funds, and benefits. Employers of caregivers experience an average of 6.6 lost work days and an 18.5% reduction in work productivity among employees who are caregivers.[25]

Many families feel the burden of caregiving, sandwiched between the demands of their parents and of their children and grandchildren. It has been said that the empty nest syndrome has been replaced by a crowded nest syndrome. In 2019, approximately 1.1 million grandparents aged 60 and older were responsible for the basic needs of one or more grandchildren under age 18 living with them.[1] The physician will often see a caregiver who is in more distress than the patient, and who develops serious physical and emotional problems as a result of the burden and stress encountered.[22]

Communication Skills

Specific communication skills are critical in good management of the elderly patient. Most important is listening and allowing patients to express themselves. Ideally, the physician should employ open-ended questions, both interpreting what the patient says and "reading between the lines." The physician might rely on intuition to interpret the patient's meaning. What motivated the patient's visit? For example, the elderly patient may speak about somatic symptoms that originate from grief or depression. Often nonverbal clues such as posture, grooming, or sighing communicate as much as or more than verbal content.

Leaving the door open for other questions or comments by the patient, both at the conclusion of the visit and for the future, is important. It is worth saying: "Are there other questions or concerns that you have at this time?" A physician that anticipates a specific problem can make it easier for the patient to discuss the issue. For example, "You are doing well, but I know that you are concerned about your arthritis and whether or not you will be able to climb the stairs in your home. At some point, we may want to discuss options that are open to you."

An important feature of American demography is the increasing diversity of older adults.[1] In the past, white English-speaking individuals have comprised the vast majority of older adults. However, increasingly, health-care providers care for a racially, ethnically, and linguistically diverse population of elders. Physicians and other professionals caring for older patients must provide culturally sensitive care, recognizing the unique and varied cultural contexts of their patients. Further, groups including the federal government have recognized the critical role of appropriate health translators in order to provide appropriate care to patients who are not proficient in English. All of these issues may be magnified in the care of older patients with sensory or cognitive disabilities.

Frequently, physicians providing care to elderly patients must include the considerations of adult children in their parents' care. These children may play a vital role in decision-making and support, and the physician must,

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therefore, possess skill in communicating with them and in dealing with their emotional reactions, such as guilt or grief. Respecting the older adult's independence, autonomy, and capacity for decisions is paramount. Managing parent-child relationships in the context of the patient's care requires skill and knowledge of ethical and legal considerations.

In this age of increasing technology and subspecialization, the patient's recovery and coping with illness may still depend on the physician's ability to reduce panic and fear, and to raise the prospect of hope. Cousins describes the "quality beyond pure medical competence that patients need and look for in their physicians. They want reassurance. They want to be looked after and not just over. They want to be listened to. They want to feel that it makes a difference to the physician, a very big difference, whether they live or die. They want to feel that they are in the physician's thoughts." [26] For example, picking up the phone and calling the patient to say: "I was thinking about your problem. How are you doing?" can be a powerful step in cementing the relationship between the doctor and patient.

Jules Pfeiffer's cartoon character, the "modern Diogenes," carries on the following discourse upon meeting an inquisitive fellow traveler through the sands of time:

"What are you doing with the lantern?" asks the traveler.

"I'm searching," replies Diogenes.

"For an honest man?" he asks.

"I gave that up long ago!" exclaims Diogenes.

"For hope?"

"Lots of luck."

"For love?"

"Forget it!"

"For tranquility?"

"No way."

"For happiness?"

"Fat chance."

"For justice?"

"Are you kidding?"

"Then what are you looking for?" he implores of Diogenes.

"Someone to talk to."

Help comes from feeling that one has been heard and understood.[27]

Knowing the Patient

Several steps are recommended in building a sound doctor-patient relationship, particularly applicable to the elderly patient.[28] The first rule is that the physician should know the patient thoroughly. The interested physician performs this step by gathering a complete history, including personal and social history, and performing a complete physical. The physician should be a good listener, warm and sensitive, providing patients ample opportunity to express their concerns, and to prioritize what matters most to them. This approach embodies the 4Ms framework.[2] But forces in contemporary health care oftentimes prevent the physician from being a good and engaged listener. The physician cannot be attentive while at the same time entering information into an electronic health record. Understandably, patients often feel that physicians who are facing a computer and typing on a keyboard are not present for them.[29]

Family and friends represent the principal support system for the elderly and usually call for nursing home placement only as a last resort, after all alternatives have failed. However, the physician must be able to recognize the dysfunctional family. There are elderly people who have been rejected by their children. There are elderly people who have rejected a child for a variety of reasons. There are families with members estranged from each other for many years. The patient may have had a stable and supportive marriage or other relationship, but increasingly, older adults have had multiple marriages, or may be divorced, or partnered in a same-sex relationship. It is critical for the practitioner to understand family or other social dynamics when engaging family or friends in support of an elder, and also to recognize when dysfunctional family or friends are harming the patient.

Creating a Partnership with the Patient

In most instances, the physician should be honest and share information truthfully with the patient, which is the foundation for building an effective partnership. The doctor should first review his/her perception of the patient's problems. Then, for each problem, alternative choices are considered, and decision-making is shared with the patient. Frankness is essential to creating and maintaining trust with patients, although there are situations in which this approach is counterproductive. There are also situations in which the elderly patient does not want to share in decision-making, but prefers to surrender autonomy to a relative such as a spouse or adult child, or to the physician.

The experience of dying in America is too often dehumanizing, and there is broad recognition that end-of-life care can be improved. Isolation, unrelieved pain, and

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anxiety during the dying process are too common and compel some to call for assisted suicide. But there are alternatives to assisted suicide that can address these shortcomings in the care of dying patients. The greatest danger posed by assisted suicide or euthanasia is a slippery slope that places unwarranted judgment as to the value of human life, and puts frail, debilitated elderly persons in jeopardy.

Some consider physician-assisted suicide as the ultimate act of patient autonomy – the opportunity to define the conditions and time of one's own death. However, it is critical that discussions with the patient or family members be presented in a positive manner that addresses pain and fear.

The physician should be cautious that discussions with family members be held with the patient's consent. If the patient lacks decision-making capacity, then it might be appropriate to deal with the closest relative or identified surrogate decision-maker. Complex ethical and legal questions can arise in the matter of confidentiality and decision-making when the presence or absence of capacity is not absolute. As with other clinical decisions, judgment and consultation with ethicist and legal counsel is advisable.

Need for Thorough Evaluation and Assessment

The physician must avoid prejudging the patient. One must not allow preconceived notions of common patterns of illness to preclude the most careful individualized assessment of each patient. Conscientious history and physical examination are essential. Treatment choices should be considered only following a thorough evaluation. Judicious consideration of all factors may result in a decision to treat or not to treat certain problems in certain patients. Attention to lesser problems may be postponed according to the priorities of the moment, rather than complicating an already complex therapeutic plan.

Physicians must avoid "wastebasket" diagnoses. The past concept of "senile dementia" is one such example. Not all mental disturbance represents dementia, and not all dementias in older people are Alzheimer's disease. Neuropsychiatric disturbance in older adults might be casually accepted as both inevitable and untreatable when, in reality, a very treatable cause may be present. The physician must consider and seek out treatable disease.

For example, neuropsychiatric disturbance, including a dementia syndrome, may be caused by severe depression that is a treatable disorder. Neuropsychiatric disturbance may also include delirium secondary to medical illness or drug toxicity. Delirium can resolve if the primary disorder is recognized and treated; failure to do so may lead to the hastened death of the patient.

It is often difficult to disentangle the physical from the emotional. Emotional disorder such as depression may present primarily as somatic symptoms. Conversely, physical disease might present as a mental disorder with confusion, disorientation, or delirium being the first sign of common medical ailments, including myocardial infarction, pulmonary embolism, occult cancer, pneumonia, urosepsis, or dehydration. For this reason, it cannot be emphasized enough that proper diagnosis is essential in order to make specific treatment plans.

Knowing the organic, anatomic, or psychiatric diagnosis is often not sufficient; rather, the physician should seek a more complete understanding of the elderly patient. Many times, precise assessment of the older patient's functional status contributes more to the patient's care than the diagnostic or anatomic label. For example, in the case of a cerebrovascular accident, knowing whether the patient can walk or climb stairs; handle his/her own bathing, eating, and dressing; get out of bed and sit in a chair; handle a wheelchair; or whether he/she requires a cane or walker conveys more information relevant to the patient's care than identifying the anatomic lesion via MRI angiography. All these functional concerns must be considered in evaluating an elderly patient.

Polypharmacy is a major problem in the care of the elderly patient. Many medications considered benign in younger individuals may cause significant side effects in elders. Changes in body-mass composition and altered renal, cardiac, and hepatic functions can affect drug distribution and elimination. In general, older individuals demonstrate greater variability and idiosyncrasy in drug response in comparison to younger persons. Prudence, therefore, is extremely important in prescribing drugs for the older individual. The physician must determine if the patient's overall function will be enhanced or harmed with pharmacologic treatment. Is this medication absolutely necessary? Has its efficacy been reassessed? Might a new or chronic symptom in fact be an adverse drug reaction? Does the absolute benefit over a time interval that is meaningful to the patient outweigh the absolute risk of harm – remembering that the benefit-to-risk ratio is often less for older patients than for younger people? The physician must attempt to keep the total number of medications as low as possible. The Beers Criteria for

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Potentially Inappropriate Medication Use in Older Adults identify medications that typically should not be prescribed in older patients. Of note, adverse events are common even with appropriately used medications such as diuretics, anticoagulants, and hypoglycemic agents.[30]

Signs and symptoms of disease in elders may be slight or nonspecific (e.g., delirium in patients with myocardial infarction). Pain, white blood cell response, and fever and chills are examples of defense mechanisms that may be diminished in older persons. The aged person may have pneumonia or pyelonephritis without chills or a rise in temperature.[31] Myocardial infarction, ruptured abdominal aorta, perforated appendix, or mesenteric infarction may be present without pain in the elderly patient.[32]

Multiple clinical, psychologic, and social problems are characteristic of older people. Clinically and pathologically, an elderly patient may have 10 or 15 problems. Geriatric patients should benefit from the use of a problem-oriented approach to medical records. Medical records should not only include medical conditions but also record functional, psychologic, social, and family problems. The key feature of the problem-oriented record is the problem list, which serves as a table of contents of the patient's total medical history. Current electronic health records provide structured formats for the problem list, but it falls to the clinical care team to develop a comprehensive list of current and past conditions and concerns. Without a detailed problem list, one can easily lose track of past problems that remain relevant, such as a psychiatric hospitalization a decade or more ago. In addition, an up-to-date medication list is crucial to patient care and safety.

Prevention and Health Maintenance

In the United States there is growing emphasis on prevention, health maintenance, and wellness. Unfortunately, strong evidence supporting most primary and secondary preventive care recommendations is lacking for adults over 75 who have multiple chronic illnesses or who are frail.[33] For example, few studies of primary and secondary prevention for heart disease and stroke include patients over 75. Still, clinicians caring for these patients should be prepared to consider preventive and screening recommendations with older adults in light of their healthcare goals and preferences, and assist them in interpreting potential risks and benefits in the context of their function and overall health.

There is much that clinicians can achieve by focusing on health maintenance and wellness in their practice and in their community educational programs. The personal physician has an opportunity to encourage preventive medicine and health maintenance at every age level and at each level of functional ability or disability.

Research has revealed the important role of nutrition, exercise, and strength training in the prevention or reversibility of frailty, physiologic decline, and recovery from surgery or trauma.[34] The health of many elders is improved by regular prescriptions of exercise and physical activity. Continued advances in nutrition and exercise are likely to reduce the risks of disease, improve function, and compress years spent with disability.

Intelligent Treatment with Attention to Ethical Decision-Making: Choosing Wisely

The aphorism "First, do no harm" paraphrases the Hippocratic Oath and provides a guidepost to the practice of medicine. It is particularly important in the care of older adults, where interventions may easily disturb a delicate homeostasis maintaining physiology and function. A similar concept was articulated over 50 years ago by Seegal as the "principle of minimal interference" in the management of the elderly patient.[35] "First, do no harm" and the principle of minimal interference should be remembered when one considers the multiple potential paths to iatrogenic injury.[36,37]

The principle of minimal interference can be applied not only to diagnostic and treatment decisions but also in regard to hospitalization or placement in a long-term care facility. It may result in recommendations that are both humanistic and cost-effective; for example, it may be prudent for patients to remain in their own homes where family and friends can more easily visit and food preferences are more easily accommodated, in contrast to moving into a long-term care facility where nursing care is available. Similarly, the recommendation to forgo a gastrointestinal workup to evaluate anemia may be wise when, in the physician's judgment, the findings are unlikely to change management based on frailty or competing illness.

However, medical nihilism is never justified; there are times when aggressive intervention is needed. Certainly, the patient with dementia caused by myxedema deserves skillful replacement of thyroid hormone. The elderly patient with depression deserves specific intervention for this very treatable disorder. Also, more and more elderly patients are benefiting from minimally invasive

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surgery, including complex procedures such as cardiac valve replacement.

Increasingly, national attention has been focused on these challenging decisions at the interface of clinical and ethical decision-making. The Triple Aim (improving the experience of care, improving the health of populations, and reducing the per capita cost of care) has become widely accepted as a definition of successful health system redesign.[38] The Choosing Wisely initiative of the American Board of Internal Medicine (ABIM) Foundation has leveraged the concept of the Triple Aim and encouraged specialty societies to define key opportunities "to promote conversations between providers and patients by helping patients choose care that is supported by evidence, not duplicative of other tests or procedures already received, free from harm, and truly necessary."[39] To date, over 80 organizations have adopted and publicized Choosing Wisely guidelines that specifically address issues of importance to older adults. For instance, the American Geriatrics Society advises hand-feeding patients with advanced dementia rather than using feeding tubes, which are associated with significant potential harms. Another recommendation is to avoid a hemoglobin A1c goal of less than 7.5% with medication other than metformin in most adults aged 65 and older because of the increased risk of hypoglycemia with tight control.[40] Many national medical and nursing organizations have responded to the ABIM Foundation's call to adopt more evidence-based, cost-effective, and patient-centered practice for all patients, including elders.

In the future, we will be faced with more and more difficult decisions of an ethical nature. For example, an 80-year-old gentleman may present with a past history of resection of an abdominal aneurysm 15 years ago, multiple myocardial infarctions, and multiple strokes causing severe dementia. His main problem in the current hospitalization is pneumonia, causing a worsening of his confused state. Because of periods of sinus arrest, a pacemaker is considered. Should a pacemaker be utilized in patients with significant dementia? Should pneumonia be treated in patients with severe dementia or terminal carcinoma? Difficult and ambiguous clinical problems such as these will face the personal physician with increasing frequency. The physician in the future will be called upon to make complex decisions according to the accepted traditions and values of the specific society, culture, religion, or nation, with major guidance from the patient's stated wishes that were affirmed at a time when the patient was fully competent. In regard to all therapeutic decisions, a personal physician is at an advantage if his/her understanding of the patient is based on continuity of care.

Interprofessional Collaboration

The physician must understand when to call upon other health professionals, including nutritionists, pharmacists, visiting home nurses, social workers, psychologists, and representatives of community agencies. One must know when to recommend legal or financial counseling, and when and how to consult with the patient's or family's clergy or spiritual adviser.

The physician should know when to recommend specific rehabilitative therapies. Specific use of physical, occupational, recreational, and speech therapies is vital for the proper care of certain problems. For example, the elderly patient with diabetic neuropathy and foot drop might benefit from bilateral leg braces. Another patient recovering from stroke might benefit from occupational therapy that should be used as a reintroduction of the patient to the activities of normal daily living, and not simply as a recreational or diversionary therapy.

The improvement of health care of the chronically ill elderly patient requires that health professionals work together for the best interests of the patient. A genuine collaborative effort is required to bring about a coordinated approach that best meets the needs of frail elders. There is increasing evidence that effective collaborative practice improves patient outcomes.[41] Competencies for effective collaborative practice have been defined and are actively being promoted in health professions education.[42]

Respect for the Usefulness and Value of the Aged Individual

Much in nontraditional societies devalues older adults. In modern culture, where so much marketing is youth-oriented, the physician must guard against unwarranted judgment that an elderly patient is less than capable and autonomous (ageism). Lack of respect for and devaluation of older adults are frequently communicated by society at large, in the workplace, in the family, and in entertainment media, but it should not occur in the doctor's office or other clinical settings. Fortunately, in some societies older adults continue to have valued roles within the family and community structure.

Most countries are witnessing an unprecedented growth in their aging populations, especially in the cohort

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over age 85. Accompanying social and economic changes may allow elders to function as a continuing resource to society. It is possible that reduced restrictions on older workers may occur, with particular reference to mandatory retirement. More educational programs may be enacted that provide skilled training, job counseling, and placement for older men and women in order to initiate, enhance, and continue their voluntary participation in the workforce. Hopefully, the breakdown of stereotypes and greater recognition of the value of the elderly person as a human resource will occur.

Already, more and more older adults are choosing to remain in the workforce. In 1930, 54% of males aged 65 and over were in the workforce. By 1985, only 15.8% of older men were working. Compare this to 2018, when 24% of men and 16% of women were working, representing 5% of the US labor force.[43] Interestingly, participation in the workforce by men aged 65 and over declined steadily from 1900 through the 1980s. After remaining level for nearly two decades, participation of older adults in the workforce has been steadily increasing since 2002.[1]

Evaluation of workers aged 51–56 in 1992 and 2004 as part of the Health and Retirement Study suggests that lower rates of retiree health insurance from employers, higher levels of educational attainment, and lower rates of defined benefit pension coverage have led significantly more workers from the 2004 cohort to expect to work past age 65, compared to the 1992 cohort.[44] Many older workers indicate that they would prefer phasing down, and continuing to do some paid work when they retire. Others approaching or in retirement opt for a retirement career. There are many in good health and who have financial stability or a satisfactory pension who would prefer to pursue a part-time or full-time retirement career with passion. The person retiring today at age 65 or younger may enjoy a retirement career that might span 10–20 years. Society must allow elders to fulfill such roles, and to retain the wisdom that has accumulated with time. At the same time, there are those approaching retirement who would not want or be able to continue employment, whether in their former role or a new one. All of these variations need to be considered in counseling our patients.

Compassionate Care

Care and compassion mean that the physician must spend sufficient time with elderly patients. One study reported that physicians spent less time with elderly patients than with younger ones, even before current pressures to increase clinical productivity.[45] Fifteen to twenty minutes may be the minimal time needed to carry out a visit in the office, home, hospital, or long-term care facility. One and a half hours, not necessarily in one sitting, may be required to complete an examination of a new patient, particularly in the presence of multiple complex problems. More time will be required in each encounter or more frequent encounters scheduled if the various functions of counseling, psychologic support, health maintenance, and prevention are to be carried out, in addition to making decisions about treatment and possible rehabilitation.

The physician should be a good listener. Often, by nonverbal means, the physician can express warmth, understanding, or empathy. Staying close to the patient and maintaining eye contact is helpful. Sitting adjacent to the patient's bed or sitting on the edge of the bed in the hospital or long-term care facility brings the doctor right into the patient's personal universe. The physician might put a hand on the patient's shoulder and pat or touch the patient or hold hands at appropriate points during the visit. In primary care practices that transition to Patient-Centered Medical Homes, the expanded team can contribute to the care of older patients and their families, bringing not only additional skills and expertise but also additional time to get to know the patient and understand the context and priorities for their care.

Changing Times in Health Care

Although the organization of health-care delivery will undoubtedly change, society will ultimately demand a quality of care that we would each want for ourselves. Social pressure will enforce the maintenance of quality of care, patient satisfaction, and the fulfillment of the professional ethics of medicine and other health-care professions. Health systems that embrace the 4Ms framework to become Age-Friendly Health Systems will emerge as leaders in providing outstanding health care for all older adults.

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Chapter 2

Biology of Aging

Allyson K. Palmer and James L. Kirkland*

Introduction to Interventions to Target Aging

Aging is the leading risk factor for diabetes, cancer, atherosclerosis, stroke, dementias, arthritis, and other chronic diseases that together account for the bulk of health care spending worldwide.[1] Older adults make up a rapidly expanding segment of the population in which chronic disease is nearly ubiquitous.[2] In order to face an increasingly aged and complex patient population, modern medicine may benefit from considering strategies to target fundamental aging mechanisms. Substantial proof now exists to support that aging is not fixed. On the contrary, interventions that target fundamental aging mechanisms can extend lifespan and health span in lower organisms and mammals.[3] These mechanisms, including cellular senescence, chronic low-grade inflammation, macromolecular damage accumulation, and progenitor cell dysfunction, are also pathogenic mechanisms associated with the major chronic diseases of aging.[4–7] Targeting these fundamental aging mechanisms might delay, prevent, alleviate, or reverse age-related chronic diseases as a group, rather than targeting one disease at a time as they arise.[4,8] In this way, modifying aging mechanisms could have a significant impact on the overall health of individuals and far-reaching effects on population health and the economy.[1] In order to translate these findings into reality, significant coordination and effort will be necessary to properly design, conduct, and analyze the preclinical, early-phase clinical trials and subsequent formal clinical trials necessary to prove the viability of this strategy, which has the potential to revolutionize health care.

Health Span and the Longevity Dividend: Goals of Translational Aging Research

Aging places a significant economic and social burden on society.[9] Increased life expectancy coupled with a rising prevalence of chronic disease is creating a monumental burden on health-care systems and social programs of developed as well as developing nations. To live a long life, individuals must often accept the presence of one or more chronic diseases, most of which lead to years of suffering, loss of vitality, or debilitation. Therefore, although lifespan has increased in the past century, health span has not. Health span is the period of one's life spent without disease or disability, and is characterized by independence, productivity, and well-being. Older adults are more interested in extending health span than increasing lifespan.[4] The ability to age in good health would harness the opportunity that lifespan extension presents. Over the past decade, the field of basic aging biology has shifted its focus toward health span, rather than focusing solely on extending lifespan. This shift in priorities has led the field to think seriously about the feasibility of clinical translation, and to begin to devise initial clinical trials.

Chronic disease is a threat to human health span whose prevalence is increasing, with individuals over 65 more likely than not to have multiple chronic diseases.[2,10] Past and current efforts in medicine have traditionally focused on the treatment of already-established disease, addressing one disease at a time as they arise. Chronic disease is usually evident only when symptomatic because of the limitations of screening and biomarkers. Therefore, dissecting the mechanisms of chronic disease pathogenesis in humans is challenging, since it is not feasible to collect biologic specimens from healthy humans during early phases of disease

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development. These challenges may be circumvented by targeting the basic mechanisms of aging that may lie upstream of these disease processes (Fig. 2.1).[4]

Based on the current landscape and recent advances in the basic biology of aging, the "geroscience hypothesis" has been proposed.[11] It posits that by targeting fundamental aging processes, it may be possible to delay, prevent, alleviate, or reverse age-related chronic diseases as a group, rather than individually. Therefore, targeting fundamental aging mechanisms could provide a pathway to improve health span on a population scale. Even if we were able to eradicate a single chronic disease, such as atherosclerosis, life expectancy would only increase by two or three years.[12,13] Focusing on health span and the prevention of chronic diseases as a group, rather than individually, could have significantly greater impact, and would be a revolutionary change in medical practice.

Frailty increases with aging, and in turn predisposes to the development of chronic disease, loss of independence, and institutionalization, eventually leading to death.[14] Frailty can also cause impaired efficacy of interventions or therapies such as surgery, stem cell therapy, organ transplantation, or chemotherapy. The degree of multisystem dysfunction is a predictor of frailty in a nonlinear fashion, indicating that at some threshold level of dysfunction in multiple systems, homeostasis is no longer maintained, and frailty develops.[12] In this scenario, it may not be enough to improve the function of one system or to treat one disease in a person approaching frailty or who is already frail.[12] The approach of targeting aging mechanisms that underlie dysfunction in multiple systems may be more efficient in improving overall health in such an individual, or to prevent the development of frailty. A more complete discussion of frailty, its definitions, and its consequences is provided in Chapter 8.

Centenarians often exhibit a "compression of morbidity," or a shortened period of disease just before the end of life.[15] Many would consider this to be a goal of aging research: to compress morbidity by pushing back the onset of chronic diseases as a group, instead of one at a time (Fig. 2.1). In fact, in rodents with delayed aging phenotypes or in calorie-restricted rodents, the prevalence of disease (e.g., cancer or kidney disease) is decreased, even at the time of death, compared to normally aging rodents at their end of life.[16] Therefore, the life extension seen in these models is not just a "stretching" of the normal life course, but seems to actually reduce morbidity and dysfunction at the end of life, improving health span.[17]

Development and progression of chronic disease incurs medical costs, reduces productivity, and hinders

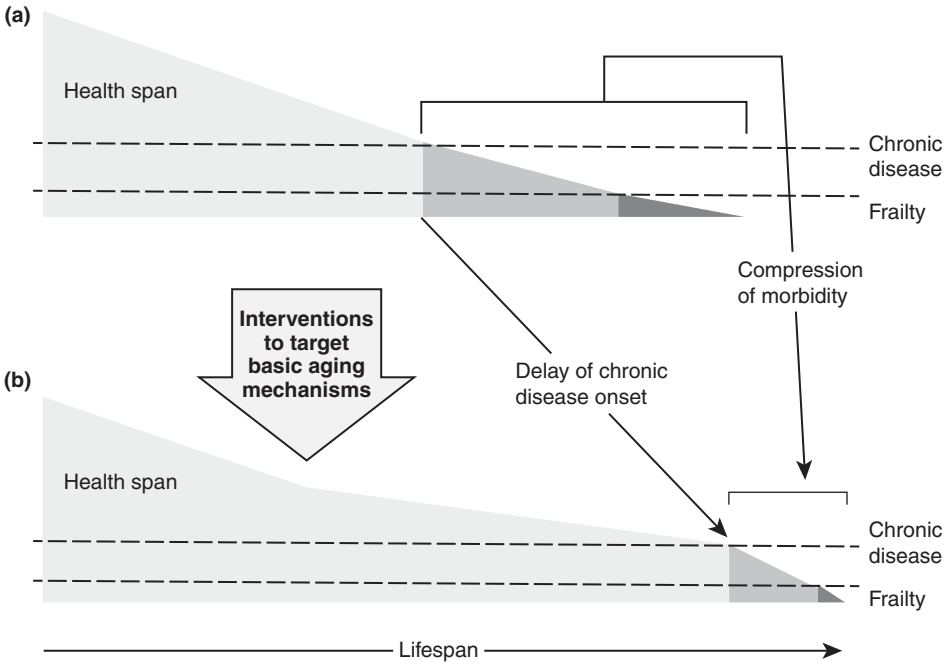

Figure 2.1 Potential effects of interventions to target basic aging mechanisms. (a) Typical trajectory of chronic disease (represented by medium-gray-shaded area) and frailty (dark-gray-shaded area) development during the lifespan. (b) Interventions that target basic aging mechanisms have the potential to increase health span (light gray), delay the onset of chronic disease and frailty, and lead to a compression of morbidity.

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quality of life for the remainder of an individual's lifespan. Pushing the onset of chronic disease until much later in the lifespan would allow individuals to remain more active, prosperous, and independent for longer. This would increase the ability of elderly individuals to contribute to society, with reduced dependence on costly programs such as Medicare and reduced utilization of hospitals, rehabilitation centers, and skilled nursing facilities. For example, it has been proposed that delaying aging would increase the number of healthy individuals over the age of 65, but would not increase the number of disabled individuals over 65.[1] The economic, health, and societal benefits that would be realized by delaying disease through targeting basic aging mechanisms has been referred to as the "longevity dividend." This longevity dividend of health-span extension would benefit the generation in which it was achieved, and would continue to benefit all subsequent generations.[18]

Advances in the Biology of Aging: Moving toward Translation

There is increasing evidence to support the idea that aging is not entirely fixed, but is a modifiable process. Leveraging knowledge about the fundamental mechanisms of aging, researchers have identified strategies to extend lifespan and health span in worms, flies, mice, and primates.[3,19] In addition, mutations that modulate longevity and knockout models with extended lifespans have been identified.[20] These models provide clues to pathways that would be amenable to interventions that impact aging processes. The possibility to intervene in mechanisms of aging has expanded the field of basic aging biology from a focus mainly on descriptive and theoretical work, to include efforts to identify mechanisms and interventions that could have a major impact on human health.

Interventions that have been shown to modulate lifespan in mice include caloric restriction, acarbose, metformin, 17α-estradiol, rapamycin and related compounds ("rapalogs"), aspirin, and flavonoids related to resveratrol.[21] To be amenable to clinical translation, a pharmacologic intervention should have low toxicity and few side effects, and would need to show efficacy in mid- or later life, when pathology or symptoms have already developed.[4] This is because an intervention meant to be started in early life would not only be impractical to implement but would also take decades to show efficacy in clinical trials. Discussed below are three interventions with potential to extend health span: caloric restriction, which is perhaps the most studied intervention to extend lifespan in a variety of species but may not be directly translatable to humans; senolytics, which are a recently discovered class of pharmacologic agents that target a fundamental mechanism of aging, cellular senescence; and metformin, which may target fundamental aging mechanisms and has immense potential for translation because of its widespread and safe use in humans. In addition, many other interventions are under development.

Caloric restriction has long provided one of the fundamental lines of evidence to suggest that a single intervention can prevent age-related diseases as a group. There is robust evidence in yeast, flies, worms, and mice that restriction of calorie intake can extend lifespan.[22] In mice, the lifespan extension can approach 50%. In addition, calorie-restricted mice reach the end of life with less disease, showing an impact on health span.[16,19] A small number of studies have indicated that caloric restriction also improves health span in primates, but were less conclusive with respect to lifespan.[23,24] Periodic or intermittent fasting, protein restriction, ketogenic diets, and methionine restriction are additional interventions related to caloric restriction that have shown beneficial health or lifespan effects in mice.[25–29] Acute caloric restriction has also been studied in humans as a preoperative or prechemotherapy intervention.[30,31] Although caloric restriction itself is unlikely to gain traction in the general population, especially in the face of an obesity epidemic, strategies to target pathways that are activated by caloric restriction might prove beneficial.[8]

Cellular senescence is another basic aging mechanism that, when targeted, can impact aging and chronic disease. Cellular senescence is a growth arrest that cells undergo in the face of significant stress. Senescent cells are characterized by altered morphology, chromatin structural changes, and the production of a host of cytokines, chemokines, and matrix remodeling factors known as the senescence-associated secretory phenotype (SASP). Senescent cells accumulate with aging in a variety of tissues, and have also been identified in areas of focal disease. Through the SASP, they impact a variety of processes that are altered during aging, including progenitor cell function, insulin resistance, tumor growth, and vascular function, and can spread senescence to previously normal cells, both locally and at a distance.[32–35] Therapies to eliminate senescent cells ("senolytics") or to prevent senescent cells from releasing SASP factors ("SASP inhibitors") are beginning to emerge and show promise in preclinical models. For example, senolytics

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prevent the progression of osteoporosis and cardiovascular aging, alleviate radiation-induced damage, prevent age- and high-fat-diet-induced vascular calcification and hyporeactivity, and alleviate age-related and obesity-induced metabolic dysregulation and renal disease. They also reduce neuroinflammation, enhance brain microvascular circulation, and improve cognitive function in mouse models of dementia and neuropsychiatric disease, and delay age-related diseases, including cancers, as a group.[33–42][38,39] SASP inhibitors also have beneficial effects including delaying frailty, sarcopenia, osteoporosis, and metabolic dysfunction in naturally aged mice.[34,35,43] Translation of senolytic therapies to clinical trials is underway. The first such trial of senolytics was in a small group of elderly patients with idiopathic pulmonary fibrosis, a relentlessly progressive disease associated with cellular senescence, in which a short course of senolytics improved physical function.[44] A preliminary report from another trial of senolytics in patients with diabetic kidney disease showed that a brief course of senolytics decreases senescent cell abundance in adipose tissue and reduces circulating SASP factors in humans.[45]

Mice treated with the standard-of-care diabetes drug metformin have modest, yet significant lifespan extension.[46] In humans, metformin has been associated with a decreased risk of cancer in diabetic individuals.[47] Another study showed a surprising increase in the survival of diabetic patients treated with metformin when compared with age-matched, non-diabetic controls.[48] This survival data should be interpreted with the understanding that the "healthy control" group over 65 likely contains individuals with undiagnosed type 2 diabetes who would have benefited from glucose-modulating treatment.[48,49] However, the evidence is sufficient to pursue trials of metformin in healthspan extension and disease prevention, an effort that is gaining traction. In fact, metformin will be used in a landmark clinical trial hoping to show proof of principle that chronic diseases can be treated as a group by targeting fundamental aging processes.[50]

The continued investment of time, funding, and training into the identification of new therapeutic targets and strategies for aging is crucial. One such innovation pipeline is the Interventions Testing Program (ITP), funded by the United States National Institutes of Health (NIH), which has standardized the study of longevity in genetically heterogeneous mice in order to test novel compounds suggested by the aging research community at large. Several aforementioned pharmacologic agents that extend lifespan were validated using the ITP framework. Other efforts to identify novel targets for aging research lie within the sphere of "omics." For example, genomic studies are being undertaken to determine the genetic signatures of supercentenarians and other individuals with exceptional longevity.[51] Proteomic and transcriptomic analyses of senescent cells are aiding in the development of novel senolytics.[36] Parabiosis and tissue transplantation experiments represent other platforms that have been used in an attempt to identify "youthful" or regenerative factors in the circulation that might improve the health of an aged individual as well as harmful factors in the aged individual that impair function in young individuals.[52, 53] Parabiosis is also being used to identify "aging" factors that are detrimental to the younger parabiosis partner, aiming to discover useful targets to alleviate age-related dysfunction and chronic diseases.

Preclinical Models

Along with a push toward clinical translation and maintained investment of funding and time into preclinical, mechanistic, model system-based aging, basic biological research must remain a priority. This will allow a continued pipeline of innovation that will identify novel therapeutic targets. Information gained from preclinical models will be indispensable in the design of clinical trials for age-modifying therapies. For example, preclinical models of specific diseases might be used to determine which age-modulating therapies have an impact on a particular pathological process. The response of a particular pathology might differ depending on the strategy used to target fundamental aging mechanisms (e.g., caloric restriction versus senolytic therapy). These studies would be useful to tailor therapeutic strategies to those patients in whom they might be most effective. Drug absorption, distribution, metabolism, excretion, and activity also need to be studied in preclinical models. The information gleaned from such studies will be important for determining optimal dosing of new drugs, such as senolytics.

Studying animal models of natural aging is time-consuming and expensive. Models of accelerated aging, such as progeroid mice, may not fully recapitulate the development of age-related disease, although they provide a convenient medium in which to test strategies targeting aging mechanisms.[54] Some age-related diseases occur only in humans or a few other species. Single-gene mutants have been developed to mimic age-related disease such as Alzheimer's disease; however, these

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models are still often studied at young ages, which does not recapitulate the aging microenvironment. Such studies may gain clinical relevance by using conditional mutants that can be activated later in the lifespan, or by using multiple models of the same disease in order to show general applicability. Standardized measures and phenotypes that accurately model human diseases or syndromes are needed in order to assess therapeutic efficacy. Moving forward in this regard will require the combined efforts of basic biologists, geriatricians, veterinarians, and others.[55]

Another possibility for preclinical studies is to induce damage with external agents, such as chemotherapeutic drugs, irradiation, skin wounding, inhaled toxins, or high-fat feeding in order to model resilience, or the ability to return to homeostasis after an insult. These models are valuable individually if the tissue response to the insult mirrors aging mechanisms that lead to the same pathology. If used together, these models could be used as a sort of high-throughput screen for new compounds that target basic aging processes to determine in which disorders they might best be used.

Clinical Trial Design for Aging Interventions

Studying the efficacy of novel therapies that target basic aging processes on lifespan extension is impractical in humans, and even studies on health span may be laborious and decades long. However, the effectiveness of therapies that target fundamental aging mechanisms cannot be proven without clinical data. The path to the translation of strategies that target fundamental aging mechanisms is still under development. Several interventions that have shown lifespan extension in rodents have been used in humans, but not in studies of lifespan. Instead, these studies have focused on specific age-related pathologies, for example using rapalogs to improve the immunization response of elderly subjects,[56] or studying the effects of rapamycin on Alzheimer's disease.[57]

Careful selection of study populations is extremely important for early clinical trials that will constitute first-in-human studies to target aging. Strategic study design must be employed to ensure that meaningful data is acquired in a timely fashion in order to move forward the study of therapeutics to target fundamental aging mechanisms. For example, it may be feasible to complete a prospective study of an elderly cohort or a cohort with one particular disorder, such as diabetes, and monitor for a delay in the onset of additional chronic diseases. Another scenario might be to target several age-related diseases within the same subjects, to test the ability of a single drug to alleviate multiple comorbidities. Within a cohort of patients with a single disease, disease progression or the development of complications could be monitored. For example, potential outcomes in a study of diabetic patients could be the development of complications such as nephropathy and peripheral neuropathy, or change in hemoglobin A1c values over time, or accumulation of additional chronic diseases such as Alzheimer's, hypertension, osteoporosis, or atherosclerosis. Localized age-related disease processes, for example osteoarthritis or impaired wound healing, could also be studied.

Resilience is another possible outcome of clinical trials to target aging mechanisms. For example, recovery time after surgery, after chemotherapy, or after a myocardial infarction could be studied. Related to this is the progression of geriatric syndromes, for example frailty or mild cognitive impairment. Progeria patients could also be monitored for symptom appearance. Another potential population would be patients with otherwise fatal conditions that share pathogenic mechanisms with aging, such as cellular senescence in primary sclerosing cholangitis, idiopathic pulmonary fibrosis, frailty, dementias, certain cancers, or the accelerated aging-like state that can develop after cancer treatment, when other treatment options have been exhausted.[6,58,59]

Identifying the proper treatment regimens for interventions that target basic aging mechanisms will be crucial. Little is known about the optimal age at which such interventions would be best utilized, for example in middle age, early old age, before or after the development of disease, during a specific stage such as puberty or menopause, and so on.[17] In addition, the proper frequency at which these therapies might be administered is unknown. Taking cellular senescence as an example, SASP protectors that block the release of damaging secreted factors by senescent cells may need to be given continuously, while senolytics, or drugs that selectively kill senescent cells, have shown benefits after a single dose.[35,36] There may be some age-modifying therapies that are effective to delay disease onset generally, and others that are most effective for certain disease states (e.g., therapies that target a specific cell type). Monitoring for side effects will also be an important component of trials of interventions to target aging processes, as these trials are likely to include novel classes or combinations of drugs, for example senolytics, or novel target populations for well-studied drugs, such as metformin.

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Biomarkers of aging are needed in order to track the efficacy of clinical interventions. These can include markers of molecular and cellular mechanisms, such as cellular senescence or oxidative damage, or surrogate endpoints related to aging in a particular system, such as hemoglobin A1c or blood pressure. The latter types of biomarkers have not yet been established for health span or lifespan. However, efforts are underway to validate biomarkers for sarcopenia and frailty, for example muscle strength or cytokine levels.[4]

The possible lack of generalizability will need to be considered when conducting clinical trials of aging, as will the inherent heterogeneity of results that can be expected based on target study populations. It is important that the outcomes of early trials are not generalized to all age-related pathologies, all aging-targeting therapies, or all aging populations. It is likely that therapies targeting age-related pathologies will need to be tailored to the individual and the pathology being treated, much like current individualized medicine strategies that aim to treat patients with the right dose of the right drug at the right time. The nature of translational aging research may necessitate the use of a heterogeneous patient population with similarly heterogeneous disease trajectory. It is known that populations become more heterogeneous with age. Therefore, it will be necessary to accept complexity, for example the presence of chronic disease, in the research of aging individuals in order to arrive at practical, valid conclusions that can impact the aging population at large.[60] Investigators, funding agencies, review panels, journal editors, regulators, and the scientific community more broadly will need to take this inherent variability into account when assessing such trials.[60] Despite the heterogeneity in the study population, it is possible that by targeting basic aging mechanisms, medicine could become more simplified rather than more complex. This has yet to be seen.

Challenges Remain for Translational Aging Research

In medicine, the focus has traditionally been to alleviate existing disease, especially those diseases that are imminently life-threatening. Additionally, biomedical research is divided into segments that study one or the other disease, combating them individually. Indeed, even funding agencies such as the NIH are organized in this way. Targeting aging mechanisms is akin to preventive medicine.[61] This strategy is a way to circumvent the problem of solving the pathogenesis of each individual chronic disease, by preventing their occurrence though targeting a shared upstream mechanism: aging.

Funding for the biology of aging has been very restricted until recently, with the majority of research dollars funding disease-specific investigation. The notion of allocating funds to extending lifespan (a likely side effect of improving health span by targeting basic aging mechanisms) has been politically unpopular for a variety of reasons.[62] Improved health span and the longevity dividend, however, comprise a compelling argument that is beginning to gain traction. Importantly, the NIH and other funding agencies around the world have recently been expanding resources available for geroscience research. For example, the NIH established an internal cross-institute Geroscience group, and an NIH-supported Translational Geroscience Network including multiple institutions across the USA has been initiated to move interventions that could enhance health span from bench to bedside.

The current regulatory framework is not amenable to studies of aging. Aging is not currently an indication for investigational new drugs. Therefore, it has not been feasible or practical for pharmaceutical companies to invest in R&D for aging. In addition, the trials necessary to test therapies to target aging do not fit into the rapid-translation model used by most pharmaceutical companies.

More investigators are needed for clinical aging research. There are few individuals trained in clinical geriatrics as well as basic aging research. Because of the priorities of their day-to-day responsibilities, clinicians are not accustomed to thinking of aging as a risk factor for disease and generally focus on established disease or imminently life-threatening conditions. More geriatricians need training in basic science research, and more basic scientists need training in clinical issues of aging populations and translational research.[55,63] The formation of interdisciplinary teams to bridge basic science and clinical geriatrics is crucial.

Societal perceptions of aging research are also a challenge. The public at large has concerns about extending lifespan for its own sake. These concerns are economic, philosophical, and religious.[18,62] Extrapolating results from the most successful rodent studies might suggest that with a successful strategy to prolong human lifespan, we might expect a 40% increase, which would correlate to an average lifespan of around 112 years for Caucasian American and Japanese women.[62] However, more dramatic increases in health span may be expected. It remains to be determined how interventions that add, for example, three months to the health span of mice scale to humans.

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Three months in mice roughly translates to 10 years in humans. Could interventions that add three months of health span to mice add 10 years to humans (relative scaling), only add three months (absolute scaling), or something in between?

Conclusion

Advances in the basic research of aging have the potential to fundamentally change the practice of medicine. Strategies have been identified that target fundamental aging mechanisms and extend health span and lifespan in model organisms. These novel approaches are already the subject of initial studies in humans. To facilitate a path to clinical translation, efforts are underway to shift regulatory, funding, and clinical paradigms toward supporting clinical trials to target fundamental aging mechanisms. Clinical research will represent a novel subdiscipline within the aging research community. This should be pursued in a way that does not detract from continued investment of research efforts in the basic biology of aging that are so critical for sustaining and expanding the pipeline of innovation from the bench to clinical translation. If translation of strategies to target basic aging mechanisms can be achieved, this could transform medical practice and put the longevity dividend within reach.

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Chapter 3

Comprehensive Geriatric Assessment

Serena P. Wong, Kristin Hlebowitsh, and Mitchell T. Heflin

Introduction

Older adults are prone to declines in physical function and cognition that can threaten their independence and quality of life. Traditional biomedical assessment focused on diagnosing and treating individual diseases can miss early manifestations of declines in health and ability that, when recognized and treated, can prevent adverse outcomes and improve quality of life.[1] Detecting and intervening upon these "geriatric syndromes" requires a multidimensional assessment including measures of physical, cognitive, affective, social, financial, and environmental domains. Comprehensive geriatric assessment (CGA) offers a holistic approach to care that accounts for these domains by developing care plans that aim to improve health outcomes, including function, safety, and quality of life. Traditionally, CGA is performed by a multidisciplinary, interprofessional team working collaboratively to collect data and develop care plans. The approach and outcomes, however, are highly dependent on the care setting and available resources and professions. Models include those focused on care of older adults in hospitals, long-term care, ambulatory practice, and home care. Alternatively, condition-focused versions provide CGA for patients with cancer or dementia, or for those having surgery. While CGA has proliferated in many forms with the growth of the older adult population, it also suffers from a number of practical challenges when implemented in a busy clinical practice.[2] In this chapter, we provide an overview of the evidence for CGA, a guide to its key components, and a practical vision for its implementation in primary care practice.

The form, function, and evidence of benefit of CGA vary dramatically by target population and location. The most robust data and best outcomes exist for inpatient units focused on geriatric assessment and care. These units provide geographically focused care with trained multidisciplinary expertise onsite. These include acute care of the elderly (ACE) units providing short-term care focused on acute illness and prevention of complications. Geriatric Evaluation and Management Units (GEMUs) offer more subacute services and restorative care over a longer period. Systematic reviews revealed improved function and survival and a higher likelihood of discharge to home among those assigned to ACE units or GEMUs.[3] While efficacious, inpatient care units can present significant administrative and financial challenges.[4] Inpatient geriatrics consultation service models that aim to provide a virtual ACE unit effect have demonstrated improvements in hospital complications and processes of care.[5,6] Inpatient consults, however, are hampered by problems with efficiency and lack of continuity across different services.

Models that aim to improve transitions of care from hospital to home, particularly those with an element of home care, have demonstrated reduced readmissions and emergency department visits, but little effect on mortality. These programs focus on patient-centered approaches addressing common issues with transitions, including medications, personal care needs, and communication and follow-up with providers.[7]

Outpatient consultative CGA in a geriatrics practice has mixed evidence for benefit, which, like inpatient consultation, depends on creating mechanisms for continuity and adherence to recommendations.[8,9] Additional models with established benefit have focused on older adults at critical health transitions, including those with a new diagnosis of cancer or dementia.[10] Additionally, older adults scheduled to undergo elective surgery benefit from pre-op and postop coordinated geriatric care services. This is particularly true of co-management of those having orthopedic surgery and/or hip fractures.[11,12]

Across the many forms of CGA, key elements emerge that determine effectiveness, including a team-based approach and mechanisms for maintaining continuity and enhancing adherence with focused recommendations. Depending on available resources and expertise, primary care practice may provide an ideal setting for addressing issues with continuity and adherence for frail

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older adults. Before describing the ways in which CGA may be adapted to primary care settings, however, it is important to first examine the specific components of the assessment.

What Does CGA Entail?

CGA is a multidimensional, multidisciplinary process including a wide array of assessments across domains and systems. The components can be organized into three categories: functional, medical, and social. Collection and synthesis of data from these areas allows for decision-making and development of a coordinated, patient-centered care plan. Table 3.1 summarizes the main CGA process and relevant team members.

Functional Assessment

Identifying and addressing functional deficits is the foundation of CGA, to preserve function and quality of

Table 3.1 Summary of CGA components

Domain	Suggested tools	Possible care plan components and team members
Gait and balance	Timed Get Up and Go POMA	Physical therapy Supervised exercise
Functional mobility	Shoulder and neck range of motion	Occupational therapy
Cognitive impairment	Mini-cog MMSE MOCA SLUMS RUDAS	Specialist evaluation Neuropsychiatric testing Anticipatory guidance
Vision impairment	Snellen chart	Optometry Low-vision rehabilitation
Hearing impairment	Audiometry Subjective hearing loss question	Audiology Hearing aids
Oral health	Visual inspection of dentition	Dentist Social work
Falls	STEADI screening tool	Focused physical exam Medication review Physical therapy Home safety evaluation
Urinary incontinence	Two questions: – Do you experience involuntary urine leakage? – If so, does it affect your daily activities?	Avoiding bladder irritants in diet Bladder training Pharmacologic therapy Urogynecology
Malnutrition	MNA-SF SNAQ	Increase protein intake Exercise program Dietitian
Mood	PHQ-9 Geriatric Depression Screen	Psychology or psychiatry Consideration of antidepressant Community partners
Polypharmacy	Beers Criteria	Pharmacist
Social isolation	Lack of a partner as proxy screening question	Social work Community partners
Elder abuse	Three questions: – Is anybody hurting you? – Are you afraid of anybody? – Is anybody using your money without your permission?	Adult protective services Social work
Spirituality	FICA	Community partners
Social determinants of health	Leads Screening Questionnaire	Social work Community partners

life. Twelve percent of Americans over the age of 75 report difficulty performing at least one basic activity of daily living (ADL), and nearly 20% need assistance with an instrumental activity of daily living (IADL).[13] Functional decline in late life predicts increased morbidity and mortality and threatens the ability to live independently. Examples of comprehensive self-reported measures of functional status include the Older Americans Resources and Services (OARS) Multidimensional Functional Assessment Questionnaire and the Katz ADL and IADL indices.[14,15] These measures provide critical information informing the development of person-centered care plans.

Gait Instability and Falls

The Timed Get Up and Go test, gait speed assessment, and functional reach are ways to identify impaired mobility.[16] Observing gait as a patient walks to the examination room can also provide valuable information. The Performance-Oriented Mobility Assessment (POMA) provides guidance for what gait deficits are significant: hesitancy in initiation of gait, whether the swing foot leaves the floor and passes the standing foot, and whether steps seem symmetrical and continuous.[17] If a patient already uses a mobility aid such as a cane or a walker, it is important to ascertain whether the device was properly fitted to them. Many older adults obtain assistive devices from friends or family without adjusting them, increasing the risk of falls. Patients with impaired gait or ill-fitting assistive devices may benefit from evaluation and treatment by a physical therapist.

One in three older adults living in the community fall each year.[18] Even falls that do not cause direct injury may lead to a fear of falling and inappropriate self-restriction of movement, potentially initiating a downward spiral of functional decline. Screening for falls may be performed with the Centers for Disease Control and Prevention's (CDC's) STEADI tool.[19] A positive fall screen may warrant a separate visit specifically to evaluate for underlying medical causes including undiagnosed neurologic or musculoskeletal disorders, cardiac disease, seizures, uncontrolled chronic conditions, or medication side effects. The STEADI toolkit provides fall risk intervention guidance, such as home safety evaluation, physical therapy referral, and medication review.[20] Evaluation and treatment for osteoporosis and fracture risk should also be included in fall assessment.

Functional Mobility

Ambulation is not the only form of mobility important for daily functioning. The upper extremity range of motion can affect a patient's ability to bathe or groom. Shoulder dysfunction is highly correlated with frailty in older adults, but often goes unrecognized, even by the patient themselves.[21] Decreased neck mobility can make driving dangerous even if cognition is fully intact. Impairments in coordination, dexterity, and reaction time can present difficulty with performance of ADLs and IADLs that can lead to loss of independence and safety concerns. Occupational therapists provide assessments and adaptive interventions for a range of conditions affecting functional impairment.

Sensory Impairment

Vision loss is common among older adults, and is associated with falling, functional decline, and depression. Optometry and occupational therapy can provide guidance on adaptive interventions like technology for magnification and assessments of personal safety. Although the US Preventive Services Task Force in 2016 did not find sufficient evidence to support universal screening for adults over 65 years of age,[22] experts in ophthalmology and geriatrics recommend assessing older adults for vision loss every one to two years to facilitate early identification and treatment of ocular diseases.[23] Office-based questionnaires do not appear to have sufficient accuracy,[24] and screening is best performed with a Snellen chart or optometry referral. Specialty-based screening also allows testing of other visual domains besides visual acuity, such as visual field and contrast sensitivity testing.

Age-associated hearing loss is present in up to a third of adults over 65. Older adults with hearing impairment are twice as likely to develop dementia.[25] Early identification and correction of hearing loss is essential to prevent morbidity related to cognitive impairment and social isolation. Many older adults are not aware of the harms of untreated hearing loss and do not spontaneously report symptoms. The simplest screening method is to ask the patient or a family member if the patient has subjective difficulty with hearing. This has a sensitivity of 78% for mild hearing loss and 93% for moderate hearing loss,[26] comparable to the sensitivity of more time-consuming tests such as the Whisper Test (whispering words at a set distance from the patient and assessing for understanding) and the National Health and Nutrition Examination Survey (NHANES) audiometric battery

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questionnaire.[16] Specificity of the single-question test is only 67% but can be confirmed by referring to an audiologist for formal testing. The most accurate office-based method is to use an audiometer.

Oral Health

One in five seniors no longer has any natural teeth, and over half of older adults have moderate or severe periodontal disease.[27] Untreated dental disease can significantly affect physical health; absent or painful teeth can limit oral intake and are associated with malnutrition.[28] About 70% of older Americans lack dental insurance, resulting in low rates of preventive dental care. Thus, the physician's office may be the only source of oral health evaluation. An examination of the patient's mouth for absent teeth, gum erythema or swelling, and large caries may identify the cause of weight loss or pain. If the patient lacks access to routine dental care, a referral to social work may be of assistance.

Medical Assessment

Medical assessment, as part of the CGA, goes beyond a typical history and physical to include evaluation for common syndromes in older adults. Many older adults may consider these problems part of normal aging and may not bring them to medical attention without explicit screening.

Urinary Incontinence

Incontinence of urine is a common problem among older adults of both sexes, with significant impact on quality of life, including increased falls risk, diminished social engagement, and poor sleep. It often goes unreported because of embarrassment or the assumption that it is normal or untreatable. In fact, behavioral interventions have excellent success rates, and safe medications exist that can improve quality of life. Screening should evaluate whether patients experience urinary incontinence, and if so, whether it affects their activities and quality of life. Three screening instruments for women that are applicable to primary care settings include the Actionable Bladder Symptom Screening Tool, Michigan Incontinence Symptom Index, and Overactive Bladder Awareness Tool.[29]

For men, incontinence is most frequently the result of benign prostatic hyperplasia. Many males benefit from medication therapy using an alpha antagonist or 5-alpha reductase inhibitor. Those with a component of urge incontinence may benefit from the beta-3 agonist mirabegron, which has fewer side effects than anticholinergics.

For women who screen positive, further questioning is required to determine the type of incontinence (stress, urge, overflow, mixed). Acute causes should be ruled out (including urinary tract infection and medication side effects) and lifestyle factors assessed (including alcohol and caffeine intake). A voiding diary including frequency of voiding may be helpful in assessing symptoms. Referral to urogynecology and measurement of postvoid residuals may be helpful in assessment and choosing management options.

Malnutrition

Malnutrition may encompass both excesses and insufficiencies in nutrition, including obesity, vitamin deficiencies, and unintended weight loss. The European Society for Clinical Nutrition and Metabolism (ESPEN) 2018 guidelines recommend screening older adults for malnutrition annually. Screening tools for such purposes include the Short Form of the Mini Nutritional Assessment (MNA-SF) and the Simplified Nutritional Appetite Questionnaire (SNAQ).[30,31] Partnering with a registered dietician can be invaluable in performing a detailed assessment of malnutrition and providing targeted education and treatment plans for older adults.

Obesity is the most common form of malnutrition among older Americans and is associated with comorbidities such as diabetes and osteoarthritis, as well as functional decline. Sarcopenic obesity, characterized by low muscle mass and elevated body mass index, is of special concern, doubling the risk for disability and tripling the risk of knee osteoarthritis compared to obese adults without sarcopenia. Consideration of intentional weight loss should be individualized with a goal to prioritize quality of life and functional independence. While unintentional weight loss is associated with increased mortality, intentional weight loss for obese adults in their 60s and early 70s can reduce mortality and improve function.[32] For patients with sarcopenic obesity, a combination of caloric restriction by 500–1,000 kcal/day and protein supplementation of 1 g/kg/day may show benefit.[33] Protein supplementation should be split throughout the day to prevent loss of insulin sensitivity in skeletal muscle. Combined resistance and aerobic exercise in addition to weight loss results in greater improvements in function compared to each intervention alone.[34]

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Unintended weight loss is a cause for concern in an older adult. Weight loss of more than 5% over six to twelve months is more likely to include loss of muscle mass, compared to a younger adult with weight loss.[35] This can lead to sarcopenia, frailty, and functional decline. A careful history can delineate whether the cause of the weight loss is medical, social, psychological, physical, or a combination. Social factors are important to exclude; 5–10% of older adults cannot consistently afford food.[36]

Cognition

Neurocognitive disorders affect up to 40% of adults over 85. Cognitive evaluation is discussed in more detail in Chapter 20. During CGA, the emphasis is on early identification of treatable causes of cognitive impairment, as well as early diagnosis of dementia in order to provide more robust anticipatory guidance, plans for ADL/IADL support, personal safety, caregiver support, and advance care planning. The Mini-Cog, a rapid screening tool, includes only a three-item recall and a clock drawing and may be ideal as part of the Annual Wellness Visit. While quite specific, it lacks sensitivity among those with mild dementia.[37] The Mini-Mental State Examination (MMSE), Montreal Cognitive Assessment (MOCA), Saint Louis University Mental Status (SLUMS) exam, and Rowland Universal Dementia Assessment Scale (RUDAS) are all office-based questionnaires that are comparable in their ability to identify mild Alzheimer's dementia.[38] With appropriate training, they can be performed reliably by most team members, including nursing and medical assistants. All of these tools have shortcomings, including imperfect sensitivity and specificity and measurement biases. Further, the MMSE and MOCA have copyright limitations and require payment for use. Differences in education, culture, first language, and prior experience with similar tests can all affect a patient's score. All should be viewed as case-finding or screening tools to lead to a more detailed evaluation if abnormal. Other causes of cognitive decline, including untreated depression or delirium, should be ruled out. Referral for complete assessment by geriatrics, neurology, or geropsychiatry, possibly including neuropsychiatric testing, provides a definitive way to diagnose dementia, especially if the clinical picture is unclear or complicated.

Mood Disorders

Up to 25% of older adults with chronic medical conditions suffer from depression.[39] Symptoms can differ from younger populations; older adults with depression may show a more significant functional loss than younger people. The widely used Patient Health Questionnaire (PHQ-9) is a reliable method to screen for and track symptoms of depression in older adults.[40,41] The Geriatric Depression Scale, consisting of 15 yes/no questions, was designed for and validated in older adult populations.[42] A score of >5 suggests depression. Suspicion of depression should prompt evaluation of other social factors (reviewed in more detail later), including isolation and loneliness.

Polypharmacy

Many older adults suffer from polypharmacy, which is often defined as regular use of seven or more systemic medications. Although each of these drugs may have an individual indication, polypharmacy is associated with negative outcomes.[43] Deprescribing is a patient-centered practice by which drugs are identified and discontinued when existing or potential harms outweigh existing or potential benefits. The process is guided by each patient's goals, values, preferences, functional level, and life expectancy.[44]

The first step of deprescribing involves accurately reconciling all of a patient's drugs and their indications. Then, each drug should be evaluated for its appropriateness. Reasons to consider discontinuation include the absence of a valid indication, risks of adverse events that outweigh potential benefits, or undue cost or complexity of the regimen.[44] As is the case with starting new medications, deprescribing should involve close monitoring for unintended adverse effects.

Partnering with a pharmacist can be especially helpful in identifying deprescribing targets and safer alternatives. The American Geriatrics Society's Beers Criteria are updated every few years, and provide evidence-based guidance on potentially inappropriate medications to consider deprescribing in older adults.[45] https://deprescribing.org (accessed on 6/21/20) is an online resource maintained by the Canadian Deprescribing Network that provides patient-facing educational resources as well as decision aids that can support providers discussing deprescribing with their patients.

Social Assessment

Without assessing and accommodating for the social factors impacting each patient's life, interventions to improve functional and medical issues will not be optimal. Social disadvantage is correlated with poor health; likewise, improving social factors can positively impact health.[46]

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Social Isolation

Social isolation is an epidemic among older adults and is independently associated with mortality, nursing home admission, frailty, and functional decline.[47,48] Lack of a partner may be used as a screening proxy measure for social connection;[46] however, this assessment is complex and warrants further evaluation. Those who live alone may have robust local support networks. Conversely, housemates may be present but not able to provide any kind of assistance, such as disabled adult children or a cognitively impaired spouse. The assistance of a social worker to explore a person's social connections and insurance status is invaluable to plan for future functional decline and identify possible sources of ADL/IADL assistance.

Elder Abuse

Elder abuse is common among older adults, especially those with cognitive impairment. This topic is addressed in detail in Chapter 54, but remains an important CGA component. Experts in this area advise use of three questions for routine screening: (1) Is anybody hurting you? (2) Are you afraid of anybody? and (3) Is anybody using your money without your permission?[49] Social workers, home health professionals, and nurses can help identify abuse.

Spiritual Assessment

Spirituality is an important part of many people's lives and often plays a central role in developing a person-centered care plan. For some older adults, the term "spirituality" is less stigmatizing than "mental health" and can be an important avenue to address mood disorders, loneliness, and grief.[50] The FICA spiritual assessment tool provides four questions that can efficiently assess a patient's spiritual history: (1) What is your Faith or belief? (2) Is it Important to your life? (3) Are you part of a spiritual or religious Community, and (4) How would you like your provider to Address these issues in your health care? Performing a spiritual assessment can enhance patients' trust in their providers,[51] making goals-of-care discussions more fruitful.

Social Determinants of Health

Defined by the World Health Organization as "the conditions in which people are born, grow, work, live and age, and the wider set of forces and systems shaping the conditions of daily life"[52], social determinants of health (SDOH) can perpetuate health disparities despite good medical care. SDOH factors include finances, housing, personal safety, literacy/educational attainment, isolation, food insecurity, use of alcohol or tobacco products, and transportation. Including an SDOH questionnaire in CGA can identify barriers and enablers as a care plan is being developed. For example, creating an exercise plan that involves walking outside may not be feasible if a patient does not feel safe in their neighborhood. Questionnaires assessing SDOH, such as the Health Leads Screening Tool,[53] can be incorporated into the CGA.

Although identifying SDOH can be straightforward, addressing these factors can be resource intensive. Utilizing members of an interprofessional team is critical, especially social workers and community liaisons. The Essential Care Model includes an interprofessional team to address SDOH.[54] It encourages engagement with direct service workers (e.g., home health aides) and community-based organizations, to provide holistic, integrated social and medical care. It also advocates for racial and ethnic diversity among team members, which is associated with improved quality of care for minority populations.

Medical Decision-Making and Advance Care Planning

The information delineated in the preceding sections need not be obtained by a physician. Synchronous or asynchronous evaluation by interdisciplinary team members can identify and treat most deficits in function, physical health, and social well-being. Table 3.1 summarizes the components of CGA. The provider's role is to prioritize deficits in order to generate an appropriate, evidence-based plan. Any plan of care for an older adult must acknowledge the prognosis and preferences of the patient. This "current care planning" naturally leads into advance care planning for future illness or end of life.

One care plan framework involves development of short-term (<1 year), midrange (1–5 years), and long-term (>5 years) goals.[55] For a healthy, independent older adult, short-term goals may include addressing pressing issues such as falls, bone health, sensory impairment, and vaccinations. Midrange issues may include depression, blood pressure, lipid screenings, social support, and maintenance of functional independence. Long-term goals include advance care planning and monitoring the appropriateness of the patient's living situation.

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When identifying goals, it is crucial to consider the patient's prognosis. The above suggested time frames would not be appropriate for someone with a life-limiting illness such as cancer, advanced heart failure, or dementia, and expected health trajectories need to be reassessed regularly. The University of California at San Francisco's online prognosis tool, https://eprognosis.ucsf.edu, provides validated prognosis calculators that take into account patient factors, including whether they reside in the community or conjugal living, medical comorbidities, and functional status.[56] There is always a factor of uncertainty in estimating life expectancy, but measuring prognosis can guide discussions about treatment options, disease prevention, and advance care planning, all critical factors in care plan development.

CGA Models That Apply to the Primary Care Setting

Although CGA is traditionally performed by an interprofessional geriatrician-led team, it can be adapted to meet the resources available to primary care providers (PCPs) and communities. Outpatient CGA models are heterogeneous in many aspects, including which health professions are involved, and how they are utilized.[57] Most primary care-based CGA teams include a PCP and nurse, with the nurse coordinating care. Other team members may include social workers, physical and occupational therapists, speech language pathologists, dietitians, pharmacists, mental health providers, and geriatricians. Other medical subspecialists, welfare consultants, and community-based service liaisons may also be involved. Some models engage multiple interprofessional team members to perform their own parts of the assessment, while others have one person perform the entire CGA, with input from other specialists when necessary. Models also vary in their inclusion/enrollment criteria and assessment tools. As such, we describe a variety of mechanisms for incorporating CGA into primary care practices.

Geriatrician-Led CGA

One method of incorporating CGA into primary care engages a geriatrics-trained provider and emphasizes sharing resources within the primary care clinic. In the Geriatric Resources for Assessment and Care of Elders (GRACE) model, the PCP employs a geriatrics-trained nurse practitioner (NP) and social worker (SW), who perform an in-home CGA. They meet with a full GRACE interdisciplinary team (comprising a geriatrician, mental health worker, pharmacist, physical therapist [PT], and community services liaison) to create a personalized care plan, and discuss this with the PCP. The NP and SW then implement the plan and engage in follow-up. Although there were mixed outcomes on health-related quality of life and physical function, this model demonstrated improved quality of care and reduced acute care utilization.[58] In another model, the geriatrician evaluated a referred patient within the PCP's office, and recommendations were implemented by the PCP's interprofessional team.[59] Recommended changes included medication adjustments, physical or occupational therapy referrals, and increased assistance in the home. Enrolled patients had a decreased number of PCP visits compared to a historical comparison group, suggesting that CGA led to fewer medical needs. In these examples, CGA with a geriatric provider leveraged resources and expertise available within the PCP's office, such as nursing and social work. Communication between the geriatrician and PCP was key in ensuring patient adherence and follow-up.

PCP-Led CGA

A limitation in implementation of the above models is the availability of a geriatrician. Some models are led by the patient's PCP, with staff assisting in assessment and follow-up. The Assessing Care of Vulnerable Elders (ACOVE-2) project aimed to improve care for vulnerable older adults within PCP offices without adding personnel or administrative changes.[60] This model used pre-visit screening to target three specific geriatric syndromes: falls, urinary incontinence, and cognitive impairment. Syndrome-specific assessments were performed by clinical staff, and this data was provided to the PCP. Program analysis suggested that intervening on high-priority areas for patients could improve quality of care and prolong survival for older adults with complex comorbidities. A different program, the Community Actions and Resources Empowering Seniors (CARES) Model, used a goal-oriented multidisciplinary PCP plan to improve care for older adults.[61] Their five priorities were: (1) identifying patients who were vulnerable but not severely frail, and who were motivated to participate; (2) assembling a team of staff to perform a CGA and frailty scale; (3) creating a plan encouraging exercise, socialization, and nutrition; (4) pairing a telephone-based health coach with each patient to support them and track progress; and (5) performing repeat CGA and frailty assessment at the six-month timepoint. On average, the patients showed an 11% reduction in frailty score at

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follow-up. This study intentionally left the composition of the wellness plan up to the PCP to create in partnership with the patient, to allow for flexibility in resources used and to build upon the patient–provider relationship.

Nurse-Led CGA

In addition to geriatrician and PCP-led CGA, other programs are led by other health professionals such as nurses. Guided Care utilized a nurse to perform an in-home CGA and create a care plan in concert with the patient, caregiver, and physician.[62] A Care Guide, documenting the patient's care preferences, health status, and plans, was shared with all of their health-care professionals. Guided Care increased patient-rated quality of care and reduced usage of home care services.[63] Primary care Assessment Tool for Elderly (PASTEL) was another nurse-led CGA program that involved telephone assessment and in-clinic evaluation to identify areas of need.[64] After review of the CGA results between the PCP and nurse, a plan was formed with recommendations and follow-up. Participating PCPs and nurses noted that using PASTEL led to more patient-centered care and preventive actions that they may otherwise not have implemented.

CGA Tools That Can Be Adapted to Primary Care

Several tools have been developed to incorporate CGA assessments into a primary care setting. One example is the Multidimensional Assessment of Older People in Primary Care (AMPI-AB).[65] Taking less than 6 minutes to administer, it assesses 17 dimensions of CGA and classifies patients into low, intermediate, or high complexity of care ratings. These correlate with mortality, worsening functional status, falls, and health-care utilization. Another tool is the Rapid Geriatric Assessment (RGA), which compiles short screening questionnaires (FRAIL scale, SARC-F, SNAQ, and Rapid Cognitive Screen) to evaluate for frailty, sarcopenia, anorexia of aging, and cognitive impairment.[66] The RGA takes 5 to 10 minutes to administer, can be performed by any trained staff member, and is available at https://aging.slu.edu.[67]

Although incorporating CGA can seem like a major undertaking in already busy primary care practices, it can be adapted to fit available resources and priorities. One way is by incorporating a geriatric review of systems into the visit (e.g., on a pre-visit questionnaire or note template). Formal screening tools like the RGA, AMPI-AB, and PASTEL can support completion of the Medicare Annual Wellness Visit. Regardless of which tools are used, patient motivation to comply with CGA recommendations, support and follow-up from the PCP and staff, and implementation of actionable, cost-effective interventions are key to improving outcomes. Table 3.2 summarizes important components of CGA to consider in the primary care setting.

Table 3.2 Key components for CGA within primary care

Recommended component	Discussion
Assembly of CGA team	• Usually includes provider and nurse • Other members based on available resources, patient needs, and specific goals
Identification of patients	• Not too frail, not too independent • Willing to engage in interventions

Performance of CGA	• Tools:
	  ○ Geriatric review of systems
	  ○ CGA questionnaire
	• Can be done by different team members
	• Can be done synchronously or asynchronously

Creation of patient-centered care plan	• Incorporating input from CGA • If possible, document and share with others who provide care for the patient • Advance care planning
Follow-up and support	• Buy-in from all team members necessary for success • Patients benefit from regular check-ins
Regular reevaluation	• Adapt plan to adjust for changes in goals of care, social situation, independence

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Conclusion

Significant barriers to implementing CGA in primary care exist. Logistically, it can be labor and resource intensive, and financial reimbursement may be limited. Results in the literature have been mixed, depending on methods, location, and measured outcomes. Many questions remain regarding who is the right patient and what is the right timing for CGA. Very frail patients with limited life expectancy may have fewer opportunities for intervention to improve function or remain living in the community. On the other hand, the worried well may utilize time and resources better allocated for patients with multimorbidity and higher risks of decline.

Despite these barriers, CGA presents an opportunity to improve the quality of life of older adults by identifying needs that may otherwise be missed in a disease-focused evaluation system. Geriatric syndromes are multifactorial, underreported, and underrecognized;[68,69] CGA tools and an interprofessional team can help PCPs identify important problems, and create and implement goal-based, patient-centered care plans. Managed care settings and population health models, whose goals align with CGA, may lead to improved reimbursement.[70]

Older adults present with a wide range of physical, mental, emotional, and spiritual health needs. Traditional disease-focused assessment and management may not adequately address multifactorial geriatric syndromes or other complex issues prevalent in this population. Integrating a CGA model of care within the primary care office provides an opportunity to improve quality of life through an interprofessional, holistic approach.

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Chapter 4

Screening and Prevention

Mariah Robertson and Jessica L. Colburn

Introduction

Preventive health is a broad term encompassing screening tests (e.g., for cancer), healthy lifestyle counseling (e.g., nutrition and physical activity), immunizations, and safety considerations (e.g., falls, driving). These discussions become more important with age as a clinician considers an individual patient's goals and values, prognosis and life expectancy, and whether a patient is likely to benefit. The 4Ms (What Matters Most, Medications, Mentation, and Mobility) provide a useful framework for thinking about how to prioritize and frame discussions with older adults in the primary care setting.[1] The goal should be to follow evidence-based practice, do no harm, and align with patient priorities.

The Medicare Annual Wellness Visit

Though discussions of preventive health and consideration of the 4Ms should happen at every visit, the Medicare Annual Wellness Visit (AWV) is an ideal opportunity to review a patient's preventive health and screening goals. While a component of the AWV is discussion of screening tests, perhaps more important are the assessments of function (activities of daily living [ADLs], instrumental activities of daily living [IADLs]), medications, cognition, mental health, sexual health, vision, hearing, fall risk, social determinants of health, and social support systems.[2] At all stages of elderhood it is critical to understand and address these factors because of their impact on an individual's health outcomes and quality of life. This is especially true for patients with limited life expectancy where the time to benefit from specific screening tests is too far in the future.

Cancer Screening

The goal of cancer screening is to identify a cancer that would lead to harm or death if not treated. It is important that decisions to screen account for an individual's values, life expectancy, the evidence base as it applies to older adults, and potential harms of testing.[3] Current guidelines typically apply age-based recommendations on when to stop cancer screening, though there can be wide heterogeneity in life expectancy for older adults of the same age. As a result, geriatric guidelines are beginning to move toward prognosis-based rather than age-based recommendations.[4] Despite the evidence that prognosis should be key to guiding decisions surrounding screening, many patients still receive screening despite limited life expectancy.[4,5] Tools such as ePrognosis are available to assist with estimating an individual patient's prognosis as well as the time to benefit from a particular screening test: https://eprognosis.ucsf.edu. See Table 4.1 for Choosing Wisely recommendations for cancer screening in older adults.

It is important that we recognize that for some patients, particularly those who have been subject to systemic racism, they may have distrust about recommendations not to screen. These concerns must be explored, discussed, and respected. Approaching screening discussions utilizing a framework can help eliminate the implicit biases clinicians may unknowingly bring to decision-making and discussions with patients.[6]

Breast Cancer Screening

Mammography has been shown to reduce mortality from breast cancer in women who are likely to live an additional 10 years. Based on this evidence, in average-risk individuals, the United States Preventive Services Task Force (USPSTF) recommends biennial screening mammography with cessation at age 75.[7] The American College of Gynecology (ACOG) supports this same recommendation, with the plan for cessation at age 75 years taking both health status and longevity into account.[8] American Cancer Society is less prescriptive, suggesting that women over age 55 screen every other year as long as life expectancy is at least 10 years.[9]

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Table 4.1 Choosing Wisely recommendations for cancer screening in older adults (choosingwisely.org)

Organization	Recommendation
American Geriatrics Society	Don't recommend screening for breast, colorectal, prostate, or lung cancer without considering life expectancy and the risks of testing, overdiagnosis, and overtreatment.
Society for General Internal Medicine	Don't recommend cancer screening in adults with life expectancy of less than 10 years.
American College of Preventive Medicine	Don't perform screening for cervical cancer in low-risk women aged 65 years or older and in women who have had a total hysterectomy for benign disease.
American College of Surgeons	Avoid colorectal cancer screening tests on asymptomatic patients with a life expectancy of less than 10 years and no family or personal history of colorectal neoplasia.
AMDA – The Society for Post-Acute and Long-Term Care Medicine	Don't recommend screening for breast, colorectal, or prostate cancer if life expectancy is estimated to be less than 10 years.
American Academy of Family Physicians	Don't screen women older than 65 years for cervical cancer who have had adequate prior screening and are not otherwise at high risk of cervical cancer.
American Society of Oncology	Don't perform prostate-specific antigen testing for prostate cancer screening in men with no symptoms of the disease when they are expected to live less than 10 years.

Cervical Cancer Screening

In average-risk individuals, the USPSTF, ACOG and American College of Physicians (ACP) agree on the recommendation to screen every three years with cervical cytology alone and every five years with high-risk human papillomavirus testing or in co-testing with cytology. All guidelines recommend stopping cervical cancer screening after age 65 if patients have had normal recent screening – specifically three negative PAP tests in a row or two negative co-test results in a row in the past 10 years, with the most recent test within the past five years.[10–12]

Colorectal Cancer Screening

In average-risk individuals, the USPSTF, American Cancer Society (ACS), and ACP recommend continued screening until age 75 with shared decision-making between ages 75 and 86. There are seven options outlined by the USPSTF for colorectal cancer screening: annual fecal immunochemical testing (FIT); colonoscopy every 10 years; FIT plus fecal DNA (Cologuard) every one to three years; computed tomographic colonography every five years; the combination of flexible sigmoidoscopy and FIT (i.e., flexible sigmoidoscopy every 10 years plus FIT every year); flexible sigmoidoscopy alone every five years; or annual guaiac-based fecal occult blood testing. Adults between 75 and 86 who have never been screened for colorectal cancer are more likely to benefit from screening than adults who were previously screened.[14–16]

Lung Cancer Screening

In high-risk individuals (30-pack-year smoking history and currently smoking or quit within the past 15 years), the USPSTF recommends annual low-dose computed tomography in adults starting at age 55. Individuals should cease screening at age 80 or if the individual has a health problem that substantially limits life expectancy or ability or willingness to have curative lung surgery.[17] In contrast, the ACS recommends cessation at age 75.[18]

Prostate Cancer Screening

The USPSTF, American Urologic Association (AUA), and ACS all recommend that baseline prostate-specific antigen (PSA) testing occur with a specific focus on periodic testing in patients in the 55–69-year-old age range, based on shared decision-making with their clinician.[19–21] The recommendation is to cease testing at age 70.

Other Screening Tests and Preventive Measures

Hypertension

Hypertension is common in older adults, and is a leading risk factor for heart disease and cerebrovascular disease.[22] The USPSTF recommends screening all adults for hypertension with annual screening recommended for adults over the age of 40. The USPSTF guidelines also recommend correlating elevated blood pressure readings with readings obtained outside of the clinical setting before initiating treatment.[23]

Though screening is recommended for older adults, decisions about treatment should be made considering an older adult's goals of care, functional status, and comorbidities. Older adults with limited life expectancy may be less likely to benefit from, and may even be harmed by, tight blood pressure control. Older adults who are falling frequently may choose to prioritize reducing their fall risk

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over blood pressure control, leading a clinician to choose a slightly higher target blood pressure goal.[24]

Type 2 Diabetes Mellitus

Screening for abnormal blood glucose is recommended as part of a cardiovascular risk assessment for adults between the ages of 40 and 70 who are overweight or obese (Grade B).[25] There are no recommendations for screening of adults over age 70 for diabetes. Though diabetes and prediabetes are both common in older adults, screening in the older adult population has not been shown to improve life expectancy or quality of life. Screening may be considered based on goals and life expectancy, while balanced against the risk of overtreatment in older adults who are less likely to benefit.[26,27]

Abdominal Aortic Aneurysm

The USPSTF, American College of Cardiology, and American Heart Association all recommend a one-time screening with abdominal ultrasound for abdominal aortic aneurysm (AAA) in men between 65 and 75 with a prior smoking history (Grade B).[28] One-time screening in men at risk has been shown to reduce AAA-related mortality, AAA-rupture, and all-cause mortality at long-term follow-up.[29,30] The net benefit of screening in men without a prior history of smoking is relatively small, and the evidence is insufficient to support screening women with a prior smoking history. The USPSTF recommends against screening women without a smoking history or family history.

Osteoporosis

Osteoporotic fractures have a significant impact on older adults and mobility, function, independence, and quality of life. Additionally, 21–30% of older adults who sustain a hip fracture die within one year.[31] The USPSTF recommends osteoporosis screening for all women over age 65 with bone mineral density testing (Grade B), and for women under age 65 if they are identified to be at increased risk of osteoporosis as evaluated by a validated risk assessment tool (Grade B).[32] One commonly used risk assessment tool is the Fracture Risk Assessment Tool (FRAX), which provides a prediction of 10-year overall fracture risk and 10-year hip fracture risk based on a patient's individual risk factors. Typically treatment is recommended for patients with a 10-year overall fracture risk greater than 20% or a 10-year hip fracture risk greater than 3%.[33]

Hyperlipidemia

The USPSTF recommends screening for and treating lipid disorders in adults between 40 and 75 years old who have a 10-year cardiovascular risk that is greater than 10% (Grade B). Additionally, they recommend selectively treating adults between 40 and 75 years with a 10-year cardiovascular risk that is between 7.5 and 10% (Grade C). In the 2015 guidelines, the USPSTF updated their prior guidelines to state that screening for lipid disorder is a necessary, but not sufficient, step in determining who should be treated with a statin for cardiovascular risk reduction, as determination for statin therapy may be made on additional cardiovascular risk factors.[34,35] The USPSTF has concluded that evidence is insufficient to recommend statin therapy for older adults after the age of 75.[34,36]

Hypothyroidism

The USPSTF has concluded that the current evidence is insufficient (Grade I) to recommend screening for thyroid dysfunction in nonpregnant asymptomatic adults.[37] Though the evidence is clear that screening of asymptomatic adults will detect an abnormal thyroid-stimulating hormone (TSH), there is variation in what would be considered a normal or abnormal range for TSH, especially in older adults. Additionally, studies have shown that treating subclinical hypothyroidism in older adults does not improve clinical outcomes, including cognitive outcomes.[38,39]

Hepatitis B and C

The USPSTF recommends screening for adults at increased risk of hepatitis B infection, including adults who were born in a country with a high prevalence of hepatitis B, patients who are HIV positive, patients with a history of intravenous drug use, men who have sex with men, and adults who are household contacts of or sexual partners with people who have chronic hepatitis B (Grade B).[40]

The USPSTF recommends a one-time screening of all adults between age 18 and 79 for hepatitis C. They also suggest screening adults older than 79 if they are determined to be at high risk, such as adults with prior history of intravenous drug use, as well as consideration of periodic screening of adults who are at ongoing risk, such as due to ongoing intravenous drug use or need for hemodialysis. This guideline was updated in 2020, in part because of the high efficacy of antiviral medications in treating hepatitis C to prevent future development of

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cirrhosis and liver failure in patients who are found to screen positive.[41]

Common Geriatric Health Issues

Mobility and Falls

Falls are very common in community-dwelling older adults, with annual fall rates nearing 50% in patients as they approach their eighth and ninth decades of life.[42] Increasing age, decline in cognitive function, and increase in sensory deficits all contribute to falls. External factors such as the home environment (poor lighting, limited or narrow spaces to navigate with assistive devices, throw rugs, etc.) and inadequate footwear further increase risk.[43] Following the 4Ms framework, it is important to ask about mobility and falls at each primary care visit. The AWV provides an opportunity to screen for fall risk with a validated screening tool, with questions such as: (1) Have you fallen in the past year? (If yes, how many times and were you injured?) and (2) Do you feel unsteady on your feet when standing or walking? If yes to either question, it would be important to evaluate gait such as with the Timed Get Up and Go test.[44,45]

Cognition

Over 5 million US adults are living with dementia, and one third of older adults will die with dementia.[46] The most common form of dementia is Alzheimer's disease (AD), which accounts for nearly 60–80% of cases. Other causes of dementia such as vascular dementia, Lewy body dementia, and frontotemporal dementia are also seen, though less commonly. A patient may also have mixed disease, particularly AD and vascular dementia.

The USPSTF states that there is insufficient evidence to support screening for dementia in asymptomatic, community-dwelling older adults aged 65 and older.[47] The predominant reason for screening asymptomatic older adults would be to capture mild cognitive impairment (MCI), or impairment that has not impacted function. Approximately 10–15% of individuals with MCI go on to develop dementia each year, but data supporting the benefits of identifying MCI early is lacking. Though universal screening is not recommended, an understanding of dementia signs and symptoms is important in order to know when to pursue additional evaluation in an older adult.

Cognitive testing is a component of the AWV and part of the 4Ms framework (mentation, along with depression). It is recommended that clinicians screen annually for memory loss during the AWV. For a quick screening test, the Mini-Cog Assessment Instrument for Dementia is one option, which combines the clock-drawing test with a three-item recall. It takes less than 3 minutes and is relatively unaffected by language barriers and education levels. Longer testing options, including the Folstein Mini-Mental Status Examination (MMSE), the Montreal Cognitive Assessment (MoCA), and the Saint Louis University Mental Status (SLUMS) exam, are also used to assess memory in the primary care setting. The MMSE has several limitations, including issues with cost to administer the test (now proprietary) and inherent biases based upon age, race, education, and socioeconomic status. Alternative options include the MoCA or SLUMS – both of which take between 7 and 10 minutes to complete. It is thought that the MoCA may better detect MCI in patients with higher education levels. It has been adapted for visual impairment (MoCA Blind), and the paper form of the MoCA has been translated into nearly 100 different languages. All cognitive testing should be interpreted in the context of a patient's education level, literacy level, and potential language barriers.[48]

Depression

Depression is quite common, with prevalence as high as 40% in older adults with comorbidities such as stroke, myocardial infarction, and cancer, and is associated with increased morbidity and mortality in older adults.[49] Despite the prevalence, late-life depression is often underdiagnosed, particularly in older adult men and Black and Latinx older adults.

The USPSTF recommends screening adults for depression as long as a provider has systems in place to ensure accurate diagnosis, effective treatment, and follow-up for identified depression.[50] Depression is the second part of the mentation component of the 4Ms framework, along with cognition, and screening for depression is a required element of the AWV with a validated tool such as the Patient Health Questionnaire-2 ("Over the past two weeks, have you felt down, depressed, or hopeless?" and "Over the past two weeks, have you felt little interest or pleasure in doing things?"). Other common screening tools include the Geriatric Depression Scale or the one-question screen ("Do you often feel sad or depressed?"). It is important that any positive responses are followed up with a more comprehensive assessment specifically looking for the severity and duration of symptoms, as suicide rates are higher in older adults.

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Visual Impairment

Impaired visual acuity is reported by nearly 10% of older adults over 60 and up to 15–20% in those over 75. Most commonly this is due to presbyopia and cataracts, though causes including glaucoma, diabetic retinopathy, and age-related macular degeneration are also common in older adults. The USPSTF concludes that there is insufficient evidence for or against screening for impaired visual acuity in older adults over 65,[51] though screening is part of the Initial Preventive Physical Examination (IPPE) covered under Medicare and an annual glaucoma screening is covered for those at high risk.

Hearing Impairment

Hearing loss is common with aging, with up to 80% of individuals over age 80 experiencing hearing impairment. This is commonly due to presbycusis and exposures to loud noises or ototoxic agents. Other causes include genetic factors, prior ear infections, and systemic causes such as diabetes, and it is important to do a physical examination of the external auditory canal to exclude cerumen impaction as a cause of hearing loss. The USPSTF has stated there is insufficient evidence to recommend for or against routine screening for hearing impairment, though screening is an essential component of the initial AWV and evidence is growing about the association between hearing impairment and cognitive impairment.[52, 53] While audiometry remains the gold-standard assessment for hearing impairment, the Whisper Test at 2 feet has a positive predictive value of near 75% and is a reasonable option for patients with barriers to obtaining audiometric testing. A simple assistive listening device can be purchased for use in primary care clinics, and visits should be held in a quiet space with good lighting. The clinician should face the patient and speak clearly, as lip-reading is a common adaptation for hearing impairment.

Mistreatment of Older Adults

Mistreatment can include physical, sexual or mental abuse, neglect (which can also include self-neglect), abandonment, and financial exploitation. It is estimated that as many as 14% of older adults experience elder mistreatment in some form, with rates estimated as high as 40–50% in older adults with dementia.[54,55] There are no screening tools that have been validated in primary care, and the USPSTF does not find sufficient evidence to recommend for or against the routine screening for mistreatment of older adults. If concerns are raised, a portion of the history and a thorough physical examination should be obtained without caregivers present. A clinician should examine for signs of mistreatment or neglect including bruises on uncommon areas (abdomen, back, buttocks, thighs), burns or injuries not consistent with history, pressure injuries that are worsening, disheveled appearance or poor hygiene, dehydration, or unintentional weight loss. It is important to engage social work and Adult Protective Services if needed to evaluate further based upon clinical assessment.

Driving Safety

It is estimated that as many as 75% of older adults over age 75 still drive, and age alone is not a reason to recommend that a patient stop driving. Driving risk increases in patients who have had falls in the past one to two years, have visual or cognitive deficits, have had prior collisions, especially in the prior 6 months, or are on medications that have central nervous system effects, in particular benzodiazepines, which increase traffic accident risk up to 50% in the first week after initiating therapy.[56] The AWV is an important time to ask questions regarding how the patient arrived at the clinic, recent traffic violations or near-misses in the past one to two years, episodes of getting lost while driving, and comfort level with driving.[57,58]

Unfortunately, there exist no validated screening tools to assess driving fitness in the primary care setting. The Clinical Assessment of Driving-Related Skills (CADReS) is a tool that assesses motor and sensory function as well as evaluation for visual and cognitive impairment and is included in the American Geriatrics Society's Clinician's Guide to Assessing and Counseling Older Drivers. An on-the-road driving evaluation is available in most communities and often operated by a certified occupational therapist. Cessation of driving can lead to depression, social isolation, and helplessness. It is important that a transportation plan is in place to support an older adult who has to stop driving. Ride-sharing and other transportation services available through cellphone apps improve options, but can be costly. Mobility and senior ride options may be available through the Department of Aging.

Immunizations

Influenza

The Centers for Disease Control and Prevention (CDC) recommends annual influenza vaccination for adults aged

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65 and older.[59] About 60% of influenza-related hospitalizations occur in patients over the age of 65,[60] yet less than 70% of older adults receive the influenza vaccine each year.[61] In a large randomized controlled trial, the high-dose trivalent vaccine was shown to reduce the rate of laboratory-confirmed cases of influenza by 24.2% in older adults as compared to the standard-dose trivalent vaccine.[62] The Advisory Committee on Immunization Practices (ACIP) states that the high-dose vaccine may provide better protection than the standard-dose vaccine in older adults, though the ACIP does not provide specific recommendations regarding the high-dose or standard-dose vaccine in their guidelines.[59] The live-attenuated intranasal influenza vaccine has not been approved for adults aged 50 and older.

Pneumococcus

Pneumococcus (streptococcus pneumoniae) is associated with significant complications for older adults annually, including meningitis, encephalitis, pneumonia, and bacteremia.[63] The CDC recommends vaccination against pneumococcus for all older adults over the age of 65. Currently there are two pneumococcal vaccines approved for use in older adults – the pneumococcal polysaccharide vaccine (PPSV23) and the pneumococcal conjugate vaccine (PCV13). The ACIP recommends one-time administration of PPSV23 for all older adults aged 65 and older, and one-time administration of PCV13 for all adults who are immunocompromised or have a cochlear implant or cerebrospinal fluid (CSF) leak.[64]

In 2014, based on evidence showing the reduction in pneumococcal rates in older adults following pediatric vaccination with PCV13, the ACIP began recommending that all older adults over the age of 65 receive both PCV13 and PPSV23.[65] On review of the evidence in 2018, it was determined that the incidence of PCV13 had been reduced to very low levels in the older adult population as a result of pediatric immunization with PCV13, and there was not an additional population benefit conferred by administering PCV13 to older adults. Consequently, the ACIP updated their recommendations for PCV13 administration to no longer recommend PCV13 administration for all adults over the age of 65. The ACIP now recommends PCV13 administration for all adults with immunocompromised conditions, cochlear implant, or CSF leak, and shared decision-making discussions with other patients over the age of 65, in particular for older adults who have never received a dose of PCV13.[64] For older adults who elect to receive PCV13 after shared decision-making, it is recommended that they receive PCV13 one year prior to PPSV23.

Herpes Zoster

The CDC recommends herpes zoster vaccination for all adults over the age of 50. The herpes zoster vaccine protects against shingles as well as complications such as postherpetic neuralgia, which is seen more commonly in older adults. The preferred shingles vaccine is the more effective recombinant, adjuvanted, subunit vaccine, which is given as two doses administered 2–6 months apart. This vaccine was shown to be 97% effective in preventing herpes zoster in all adults over the age of 50, and 89.8% effective in older adults over the age of 70.[66] A second option, the live-attenuated shingles vaccine, is less effective than the recombinant, adjuvanted vaccine and is no longer the preferred vaccine. Of note, the recombinant, adjuvanted vaccine has not been studied in immunocompromised patients and the ACIP does not have recommendations specific to that population. The ACIP recommends vaccination for older adults with a history of herpes zoster infection, as it can recur, and does not recommend testing for history of varicella prior to vaccine administration.[67]

Tetanus

The CDC recommends a tetanus-diphtheria (Td) booster every 10 years for all adults, and a Tdap booster once over the age of 65. Immunity to pertussis has been shown to wane in adult years, which is why the booster is recommended. Previously this recommendation was only for older adults who have close contact with an infant under the age of 1 year, but the ACIP recommendation now includes all older adults over the age of 65. In addition, newer updates now recommend either Tdap or Td every 10 years for booster doses, after several studies showed potential benefit and no adverse risks of giving Tdap every 10 years.[68]

Hepatitis A and B

Hepatitis A and B vaccination recommendations are the same for older adults as they are for younger adults, and are based on risk factors. Important risk factors to ask about when considering hepatitis vaccination for older adults include: travel outside the USA, exposure to blood products at work, sexual history including number of partners and men who have sex with men, and intravenous drug use, as well as health problems including

chronic liver disease and end-stage renal disease on dialysis. Hepatitis A vaccine is two doses, and hepatitis B is three doses given over a 6-month span.[69,70]

Maintaining a Healthy Lifestyle

Physical Activity

For older adults, physical activity can reduce fall risk, enhance independence, and improve mental health. There is also evidence to suggest that exercise may improve cognition, though it has not been shown to prevent dementia.[71] Remarkably, even a sedentary individual who begins to exercise in later life reaps benefits when compared to those who do not. Despite these known benefits of activity, many older adults do not meet the recommended amount of weekly physical activity. The World Health Organization (WHO), the CDC, and the US Department of Health and Human Services (DHSS) all recommend at least 150 minutes of moderate-intensity or 75 minutes of vigorous-intensity aerobic physical activity throughout the week. Moderate activities such as walking do not require screening by a primary care physician unless a patient has symptoms; however, it is recommended that a patient speak with their clinician before engaging in vigorous physical activity and consider work-up if they are high risk for cardiovascular complications. For patients at high risk for falls, tai-chi has been shown to improve balance and reduce fall risk.[72] The National Institute on Aging has released a guide for exercise and physical activity for older adults: https://order.nia.nih.gov/sites/default/files/2018–04/nia-exercise-guide.pdf.

Nutrition

Monitoring the weight of older adult patients is an important component of primary care. Body mass index (BMI) is often the marker used to monitor and counsel around obesity in adults, and in patients over 70, the lowest mortality rates are in patients with a BMI of 25–29.[73] Loss of muscle mass can lead to a decrease in weight and therefore focusing on weight loss alone is typically not the goal in older patients. That said, weight loss when combined with increased physical activity could be beneficial in obese older adults because of an association with improved functionality and muscle mass.

Perhaps more important to monitor for in older adults is malnutrition, which occurs in as many as 15% of older adults. It is important to screen for food insecurity (lack of access to resources to get food), cognitive impairment, dental pain, alcohol use disorder, and decreased functional status leading to an inability to prepare one's own meals. There are several tools designed to screen for nutritional status. A common, quick, four-item screener used in primary care is the Simplified Nutrition Assessment Questionnaire (SNAQ), which was specifically tested in the community-dwelling older adult and has reasonable sensitivity and specificity for identifying older adults at risk for weight loss.[74]

Alcohol Use

It is estimated that as many as 50% of adults aged 65 and older consume alcohol, with nearly 15% consuming more than recommended weekly amounts.[75] With increasing age, the risks of alcohol to health and function increase, particularly when in combination with certain medications. It is recommended by the American Geriatrics Society that clinicians screen annually for alcohol use in older patients, though this is not a required screening question in the Medicare AWV. Tools such as the Alcohol Use Disorders Identification Test (AUDIT, developed by WHO), also available in a three-question abbreviated version for primary care called the AUDIT-C, and the Short Brief Michigan Alcoholism Screening Test-Geriatric Version (SMAST-G) can be helpful in identifying alcohol use disorder in older adults. This is in comparison to the CAGE questionnaire, which has lower sensitivity in older adults.

Smoking Cessation

It is an important part of regular screening to ask about tobacco use and to counsel older adult patients around cessation. While the rates of tobacco use are lower in older adults when compared to younger cohorts, there are significant smoking-related health outcomes within the older adult population including lung cancer, chronic obstructive pulmonary disease, and heart disease. Cessation at any age will decrease rates of smoking-related poor health outcomes.[76] There is a dearth of data surrounding the benefits of nicotine replacement therapy in older adults; however, this can be tried in addition to counseling as a means of improving rates of cessation. Other agents such as bupropion and varenicline may be used, though with caution and observation for side effects given the risks of adding any medications in older adults.

Sexually Transmitted Infections (STIs)

The USPSTF recommends routine screening for STIs of all adults up to age 65 and screening of adults over

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65 years old who are at increased risk for acquiring an STI.[77–79] Despite common misconceptions, many older adults (upwards of 25%) are still sexually active into their 80s. It is important that older adults are asked about their sexual health and practices yearly to help with identification of risk factors for STIs and to identify challenges with sexual dysfunction that interventions can help. The USPSTF recommends behavioral counseling for all sexually active adults.

Summary

Screening and preventive health are focused on balancing comorbidities and life expectancy and focusing recommendations based on "What Matters Most" to the older adult. These discussions are best had in the context of continuity and trust, which makes primary care clinicians best suited to guide these discussions. This chapter is meant to provide the most up-to-date preventive health recommendations pertinent to common conditions encountered as patients age.

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Chapter 5

Appropriate Use of Medications

Emily R. Hajjar and Lauren Hersh

Introduction

In 2016, 49.2 million people (15% of the population) in the United States were over the age of 65 years and approximately 36% of these individuals consumed five or more prescription medications.[1,2] Because of the high number of prescription medications used and the risk of adverse drug events, increased awareness is needed for prescribing in older adults. Reasons for the increased attention include an increased sensitivity to drug effects secondary to pharmacokinetic and pharmacodynamic changes that occur with aging, the complexity of medication regimens and issues with medication adherence, lack of guidance from treatment guidelines for older adults, and a high incidence of inappropriate prescribing and polypharmacy. These patients are often medically and socially complex, with multiple disease states and varying levels of functional ability and psychosocial and financial support. Addressing these issues, using an interprofessional team-based approach, will help optimize drug therapy in this population.

Pharmacokinetic and Pharmacodynamic Changes in the Elderly

There are a number of age-related changes in drug pharmacokinetics (Table 5.1) and pharmacodynamics that occur in the elderly population. Pharmacokinetics describes drug absorption, distribution, metabolism, and elimination. Pharmacodynamics refers to the effects that a drug has on the body. Although all of the changes described below may occur in the elderly, it is important to remember that these age-related physiologic changes do not occur uniformly in every patient. While many of these changes may be attributed to the aging process alone, many may be due to the combined effects of age with other factors such as concomitant disease states, polypharmacy, genetics, and environment.

Table 5.1 Potential age-related physiologic changes that can affect drug pharmacokinetics in the elderly[3]

Absorption	Metabolism
↑ gastric pH ↓ gastric motility ↓ surface area of small intestine ↓ gastrointestinal blood flow ↓ hepatic mass/hepatic blood flow	↓ hepatic mass/hepatic blood flow ↓ activity of CYP1A2, CYP2C9, CYP2C19, and CYP3A4 enzymes
Distribution	**Elimination**
↑ total body fat ↓ lean body mass and total body water ↓ albumin concentrations ↑ α-1 acid glycoprotein concentrations	↓ kidney size ↓ renal blood flow ↓ glomerular filtration ↓ tubular secretion

Pharmacokinetic Changes

Absorption and Bioavailability

Absorption appears to be the least affected of the pharmacokinetic processes in the elderly. Most medications are absorbed through passive diffusion, so age-related changes in physiology only minimally alter.[3] However, several physiologic changes can affect the absorption of drugs such as an increase in gastric pH and reductions in gastric motility, mucosal absorptive surface area in the small intestine, and gastrointestinal blood flow.[4–6] These potential alterations are more likely to cause a slight delay in the rate of drug absorption, and usually do not significantly impact the overall extent of absorption.

Bioavailability of drugs may be affected by age-related physiologic changes. Clearance of drugs that would normally be subject to extensive first-pass metabolism (e.g., propranolol, morphine) may be impaired by a reduction in liver mass and hepatic blood flow.[7] Consequently, there may be an increase in the bioavailability of these

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drugs. The function of P-glycoprotein, which normally acts as an efflux pump and serves as a barrier for drug absorption, does not appear to be significantly altered by the aging process.[8] Additionally, the bioavailability of drugs administered via the transdermal route does not appear to be affected by age.[9]

Distribution

Age-related changes in body composition and protein binding may affect drug distribution. The elderly population generally exhibits an increase in total body fat and reduction in lean body mass and total body water. It is estimated that lean body mass may be reduced by as much as 15%, while total body fat may be increased by as much as 40%.[10] Consequently, drugs that are more water soluble (hydrophilic) or that distribute primarily to muscle (e.g., digoxin, aminoglycosides, theophylline) may have a reduced volume of distribution, resulting in increased plasma concentrations.[11] In contrast, drugs that are more fat soluble (lipophilic) may have an increased volume of distribution (e.g., diazepam, oxazepam), leading to an increase in tissue concentration and duration of action.[3]

In the elderly, plasma albumin concentration is reduced by 15–20%.[12] While this reduction in albumin may be attributed in part to age-related physiologic changes, it may also be a result of malnutrition and/or comorbid disease states (e.g., heart failure, chronic kidney disease, rheumatoid arthritis, cancer).[12] Acidic drugs (e.g., phenytoin, sulfonylureas, warfarin, levothyroxine) primarily bind to albumin. A reduction in plasma albumin concentration causes an increase in the free (unbound) concentration of these drugs, which may increase risk of toxicity.[13] Basic drugs, such as propranolol, primarily bind to α-1 acid glycoprotein, which is an acute phase reactant protein. Concentrations of this protein are believed to increase with aging, in chronic inflammatory disease states, malignancies, or in reaction to stress, such as post-myocardial infarction.[13] Consequently, plasma binding of these basic drugs may be increased, leading to a reduction in free plasma concentrations. Alterations in drug distribution that occur secondary to changes in protein concentration are less clinically significant for basic than acidic drugs.

Metabolism

Drug metabolism primarily takes place in the liver. Age-related declines in liver mass and hepatic blood flow may account for a decline in drug clearance, which could lead to an increased risk of drug toxicity. In fact, it is estimated that hepatic blood flow decreases by up to 50% in this population.[3] Consequently, hepatic clearance of drugs that have a high hepatic extraction ratio (e.g., morphine, propranolol, verapamil) may be decreased, resulting in increased plasma concentrations.[3]

Age-related hepatic changes affect liver enzymes responsible for metabolism. In general, drug metabolism can be categorized into phase I and phase II reactions. Only phase I reactions appear to be significantly affected by age.[14,15] Phase I reactions include the processes of oxidation, reduction, and hydrolysis. Most oxidative reactions involve the cytochrome P-450 (CYP) enzyme system. Drugs that induce or inhibit certain isoenzymes act to decrease or increase, respectively, the plasma concentration of certain drugs (substrates) metabolized by a particular isoenzyme. Because of the complexity of potential drug interactions that can occur via the CYP enzyme system, it is essential for practitioners to become familiar with common drugs that serve as substrates, inhibitors, or inducers of this system. Phase II reactions, which involve the processes of glucuronidation, acetylation, and sulfation, are not significantly affected by age.[16] Certain benzodiazepines such as oxazepam, lorazepam, and temazepam are subject only to phase II metabolism; therefore, the metabolism of these drugs is unchanged. Other benzodiazepines, such as chlordiazepoxide and diazepam, undergo both phase I and II metabolism, and may have impaired clearance in the elderly.[17] Consequently, selecting a benzodiazepine that exclusively undergoes phase II metabolism may be more prudent in the older patient.

Renal Elimination

Age-related reductions in kidney size, renal blood flow, glomerular filtration, and tubular secretion may contribute to a decline in drug clearance.[15] Renal blood flow decreases by approximately 10% each decade after the age of 40.[18] These changes may be compounded by disease states such as hypertension and diabetes that are frequently present in this population and which may further impair renal function. As a result, clearance of drugs that are primarily excreted unchanged by the kidneys may be significantly reduced.

Serum creatinine (SCr) alone should not be used to estimate the patient's renal function, as the amount of lean body mass decreases with age.[19] Instead, to provide a more accurate approximation for medication dosing, calculation of the creatinine clearance (CrCl) is used. Creatinine is a byproduct of muscle breakdown; age-related reduction in muscle mass leads to a decrease in

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the production of creatinine. Therefore, even in the presence of renal dysfunction, an elderly patient's SCr may appear to be "normal" (i.e., less than 1 mg/dl). The Cockcroft-Gault formula (shown below), which incorporates SCr as well as age and weight to estimate CrCl, can be used in elderly patients:

$$\text{CrCl (mL/min)} = \frac{(140 - \text{age}) * \text{weight (kg)}}{72 * \text{serum creatinine (mg/dL)}} * 0.85 \text{ for females}$$

Although the Cockcroft-Gault formula is frequently used to both assess renal function and make dosage adjustments of renally excreted drugs, it is important for clinicians to be aware that this equation tends to underestimate the glomerular filtration rate (GFR) of older adults.[20] More recently, the Modification of Diet in Renal Disease (MDRD) equation is being used as an alternative method for assessing renal function. Although the MDRD equation was derived from patients with chronic kidney disease, it may overestimate renal function in older adults.[21] Because of a lack of dosing information with the MDRD equation for most medications, health-care providers should continue to use the Cockcroft-Gault formula for this purpose.

Pharmacodynamic Changes

A number of age-related physiologic changes may occur that increase or decrease sensitivity to a drug. Mechanisms for altered age-related pharmacodynamics include changes in receptor number and affinity, changes in drug concentrations at the receptor, alterations in post-receptor signaling, and alterations in homeostatic mechanisms.[22] In the cardiovascular system, because of the decline in β-receptor activity that occurs with age, a decline in β-adrenergic responsiveness may occur, which could minimize heart-rate response to both β-agonists and β-blockers.[23] Older adults also have a lessened reduction in blood pressure from β-blockers.[22] Additionally, blunting of the baroreceptor reflex can occur with aging, resulting in the development of exaggerated postural hypotensive effects during therapy with drugs such as nitrates, diuretics, calcium channel blockers, and α1 blockers.[24] Changes in the pharmacodynamics of central nervous system agents may also occur, including alterations in the permeability of the blood–brain barrier and changes in brain size and alterations of neurotransmitters.[22] The overall result of these changes is an increased sensitivity to central nervous system agents. Although no significant differences in the pharmacokinetics of warfarin have been demonstrated between younger and older patients, the pharmacodynamics of this drug may be altered in the elderly population, potentially resulting in an enhanced anticoagulant effect and an increased risk of bleeding.[22,24] Consequently, it may be prudent for health-care providers to use lower initial and maintenance doses of warfarin in this population.

Beers Criteria

Older adults are particularly susceptible to inappropriate prescribing because of age-related pharmacokinetic and pharmacodynamic changes, increased comorbidities, increased risk of drug interactions, polypharmacy, and adverse drug reactions. The use of medication is considered potentially inappropriate when the possible risk outweighs the expected clinical benefit. The Beers Criteria, initially developed by an expert panel in 1991 to target nursing home residents, are the most widely cited criteria used to identify high-risk, or "potentially inappropriate," medication in older adults.[25] The Beers were most recently updated in 2019, and are intended for use by clinicians in inpatient, community, and institutional settings.[26] The current criteria include the following categorization of medications: potentially inappropriate medications in the older adult (e.g., barbiturates, megestrol, and glyburide), potentially inappropriate medications due to drug–disease interactions (avoidance of medications with anticholinergic properties in patients with cognitive impairment), medications to be used with caution (aspirin for primary prevention of cardiovascular disease in patients >70 years old), drug–drug interactions to be avoided (warfarin and ciprofloxacin), and medications that should be avoided or used in lower doses in renal impairment (apixaban with a CrCl <30 mL/min).[26] Numerous studies have evaluated health-care outcomes associated with the use of the potentially inappropriate drugs included in the Beers Criteria. There is clear evidence that inappropriate medication use is associated with adverse drug reactions and increased costs across all health-care settings (ambulatory, acute, and long-term care).[27]

Although the Beers Criteria have been increasingly used as a quality-of-care measure (as evidenced by the Beers-like list of inappropriate drugs adopted by the National Committee for Quality Assurance [NCQA] and the Pharmacy Quality Alliance [PQA]), the criteria

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Table 5.2 Adverse drug reactions in the elderly[26]

	Increased fall risk	Delirium	Cognitive impairment
Prescription agents (by class)	Antidepressants	Anticholinergics	Anticholinergics
	Anticonvulsants	Antipsychotics	Antipsychotics
	Antipsychotics	Benzodiazepines	Benzodiazepines
	Benzodiazepines	Corticosteroids	Sedative hypnotics
	Sedative/hypnotics	Histamine-2 receptor antagonists	
	Opioid analgesics	Meperidine	
		Sedative hypnotics	

have been criticized for using an explicit method that may not take clinical application into account for assessing drug therapy appropriateness.[28] As explained in the criteria, clinicians are encouraged to make prescribing decisions in the context of a complete clinical picture that includes the entire medication regimen, history of medication use, comorbidities, functional status, and prognosis.[26,29]

STOPP/START Criteria

STOPP (Screening Tool of Older People's potentially inappropriate Prescriptions) and START (Screening Tool to Alert doctors to the Right Treatment) are another set of explicit criteria developed in Europe to reduce potentially inappropriate medication use.[30] STOPP comprises clinically significant criteria for potentially inappropriate prescribing. Each criterion is supported by a concise explanation as to why the prescribing practice is potentially inappropriate and includes consideration of drug–drug interactions and duplication of therapy. START consists of evidence-based prescribing indicators for commonly encountered diseases to prevent the undertreatment of common medical conditions that impact older adults.[30]

Adverse Drug Reactions in the Elderly: An Overview

Adverse drug reactions (ADRs) are noxious responses to medications used in usual doses that require treatment of the effect, modification of the drug regimen, or cessation of treatment.[31] Older adults are particularly vulnerable to experiencing ADRs and have experienced escalating rates of emergency room assessments and hospital admissions as a consequence. An estimated 34.5% of emergency rooms visits for ADRs occurred in older adults (>65 years), with slightly less than half (43.6%) of such assessments leading to hospitalization.[32] In-hospital ADRs cause a 9% increase in length of stay and correspond to a 20% increase in care costs associated with bed utilization, laboratory testing, and treatment costs.[33] Many such admissions are avoidable and play a significant role in increasing patient morbidity or mortality and health system financial strain.[34,35]

ADRs in older adults are frequently exaggerated responses to expected pharmokinetic and pharmacodynamic shifts. Polypharmacy, a common occurrence in this population, increases the risk of ADRs because of drug interactions, synergistic toxic effects, and nonadherence to complicated or expensive medication regimens. Decreased mobility, multiple disease states, low body weight, renal and/or hepatic dysfunction, female sex, and a prior history of ADRs further increase risk for the development of ADRs.[36] Common ADRs in the elderly include an increased risk of falls, changes in mental status, and effects on urinary continence. Table 5.2 identifies some of the common pharmacologic agents that may cause each of these ADRs.

Adverse Drug Reactions: Falls

Falls are of significant concern because of their associated morbidity and mortality. Nearly one third of adults over the age of 65 fall at least once per year, with 10% of such falls resulting in serious injury requiring medical intervention.[37,38] Fall-related injuries are associated with a decline in functional status and an increased likelihood of nursing home placement.[39] In one study, 20% of elderly patients who experienced a hip fracture died within a year of the fracture.[39] Established risk profiles for "high-risk fallers" include increased age, female sex, lower educational attainment or socioeconomic status, depression or dementia, gait dysfunction, poor health, and previous significant falls. While research has tied certain medications to increased

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fall risk, there is no comprehensive, evidence-based list of specific problematic medications in general distribution.[38,40]

Medication use is one of the most modifiable risk factors for falls.[41] Polypharmacy may be a marker of underlying comorbidity and frailty, rendering a patient more susceptible to falling. Regardless of whether agents are considered to be high risk, falls are more common in elderly patients who take more than four drugs per day.[42] Risk of medication-related falls can be mitigated through patient education, slow dose titration, and avoidance of polypharmacy and high-risk medication when possible.

Certain medication classes, when used alone or in combination with other medications, can increase fall risk.[42] Psychotropic and antidepressant medications are frequently prescribed in the elderly to treat depression, psychosis, and insomnia and may increase fall risk when taken with other medications that carry a high risk profile. Antipsychotic medications can cause many adverse effects, including extrapyramidal symptoms, orthostasis, and cognitive impairment.[43] These effects are more commonly associated with the typical antipsychotics, (e.g., haloperidol, thiothixene, droperidol), but have also been documented with the newer atypical agents (quetiapine, risperidone, olanzapine).[43,44] The anticholinergic properties of sedation, orthostatic hypotension, and confusion associated with tricyclic antidepressants (TCAs) were one of the original perceived barriers to prescribing in older adults. While selective serotonin reuptake inhibitors (SSRIs) cause markedly less sedation, studies suggest that SSRIs are associated with fall rates similar to those with TCAs.[45]

Benzodiazepines have been implicated in increasing fall risk, particularly upon initiation of a new prescription.[38] It is frequently stated that short-acting benzodiazepines are safer than long-acting benzodiazepines for the elderly. However, this distinction may be blurred, as the pharmacokinetic half-life of benzodiazepines in the blood may be misleadingly short compared with the duration of pharmacodynamic effect on the nervous system.[46] It is likely that risk increases with increasing dose; therefore, if benzodiazepines must be used, the lowest dose should be used for the shortest duration possible.[43,45]

The use of opioid analgesics, with proper monitoring, is increasingly recognized as a reasonable option for treating moderate to severe pain in older patients, despite concerns about the addictive potential and fall risk.[47] Pain management for older adults presents a particularly challenging dilemma in the context of declining organ function, multimorbidity, and polypharmacy, and inadequate responsiveness by care teams can adversely impact independence and quality of life in this vulnerable demographic. Agent and dose, titration, and use of adjunct agents should be carefully selected to minimize risk.[47]

Any agent that causes hypotension or dizziness may increase fall risk. Agents such as α1 blockers, which are used to treat benign prostatic hypertrophy, can cause hypotension and possible syncope.[26] Evidence linking fall risk with antihypertensive agents, however, is mixed. Some studies show that patients have an increased risk with these agents, while others do not.[48] It has been demonstrated in observational studies that digoxin and diuretics are associated with increased fall rates.[48] Since these agents are commonly used in this population, it is important to educate patients about the risk of falls due to hypotension or dizziness as well as preventive measures that can be taken.

Adverse Drug Reactions: Cognitive Impairment

Patients with an acute illness, such as a urinary tract infection, or a worsening chronic condition, such as heart failure, can present with cognitive dysfunction in the form of mental status changes.[49,50] Many commonly used medications can also precipitate or contribute to the development of cognitive disturbances. It can be challenging for the practitioner to distinguish between cognitive impairment secondary to a disease process versus a medication. It is therefore important to consider the possibility that changes in cognitive function may be partially or wholly due to medications.[51]

Any medication with central nervous system (CNS) effects has the potential to cause cognitive dysfunction. Opioid analgesics can cause sedation, confusion, and even hallucinations.[47] Antipsychotics, used to treat behavioral problems in dementia as well as psychotic disorders, can cause anticholinergic-associated cognitive impairment.[52] Anticholinergic agents are frequently responsible for causing CNS disturbances such as confusion, excitement, disorientation, and delirium.[53] Many commonly used medications, including over-the-counter antihistamines (diphenhydramine, chlorpheniramine), as well as prescription-only antidepressants (e.g., amitriptyline, doxepin, imipramine), skeletal muscle relaxants (cyclobenzaprine, orphenadrine), and bronchodilators (atropine and ipratropium), have anticholinergic actions.[26] The risk of cognitive impairment increases with the number of anticholinergic medications used, as

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does limitation in physical functioning and risk of hospitalization.[54] Physicians must be aware of an agent's potential to cause cognitive dysfunction and must consider the "anticholinergic burden" of a patient's entire medication regimen when prescribing. While a single medication may not result in anticholinergic-associated cognitive changes, the use of multiple drugs with such effects may increase that risk.[54]

Drug-Prescribing Patterns: Underutilization of Drug Therapy

Clinicians historically view polypharmacy negatively because of the associated medication risks, such as drug–drug interactions and adverse drug events. In recent years, the problem of underprescribing potentially beneficial drugs (in accordance with current clinical guidelines) has gained increasing attention in the literature and in clinical care.[35] Underprescribing can reflect "appropriate" or "inappropriate" prescribing patterns, with rational underprescribing representing a well-deliberated and considered decision and inappropriate underprescribing stemming from a number of scenarios (underappreciation of the benefits of therapy, insufficient evidence of clinical benefit in older adults, or clinicians' generalized fear of polypharmacy and perceived potential harm).[55] The interconnectedness of polypharmacy and undertreatment has been repeatedly illustrated, and studies have demonstrated the probability of underprescribing increases as the size of a patient's drug regimen increases.[56,57] Undertreatment of medical conditions has been observed to occur in 64–83% of elderly patients receiving more than five medications.[56,57]

A high incidence of undertreatment has been observed with laxatives used to prevent constipation in patients receiving chronic opioids, and angiotensin-converting enzyme (ACE) inhibitor and β-blocker use in cardiovascular disease.[56,58] Other agents underused in the geriatric population include antihypertensives, aspirin, antihyperlipidemics, oral hypoglycemic agents, and calcium supplements.[57,58] In a study of patients with coronary artery disease in an academic nursing home, only 62% received aspirin, 58% received an ACE inhibitor or an angiotensin II receptor blocker, 57% received a β-blocker, 27% received a calcium channel blocker, and 21% received a statin.[58] None of these patients had a contraindication to the agents with which they were not treated.

In evaluating ways to minimize both polypharmacy and the underutilization of well-studied and supported therapeutic initiatives, a collaborative approach has been observed.[59] When the addition of a clinical pharmacist to the geriatric team (Geriatric Evaluation and Management [GEM] care) was compared to the use of the GEM alone, intervention patients (stratified by age and number of medications) were more likely to benefit from a clinically appropriate medication regimen.[59]

Drug Adherence

The true rate of medication adherence is estimated to be 50%, with a range of 26–59% in patients aged 60 and over.[60] As many as 10% of hospital admissions and 23% of nursing home admissions may be attributed to medication nonadherence.[60] Seventy-five percent of hospital admissions related to nonadherence involved cardiovascular and CNS medications. Falls, postural hypotension, heart failure, and delirium were the most common manifestations of nonadherence.[60]

Factors Affecting Drug Adherence

Once an optimal therapeutic regimen is determined for a patient, adherence becomes a key component to therapeutic success. Adherence is a preferred term to compliance as it implies a collaborative relationship between the patient and/or caregiver and the health-care provider. Compliance, in comparison, implies a one-way relationship wherein the health-care provider makes all decisions and provides "directions" independent of the wants and needs of the patient.[61]

Factors that affect medication adherence include: demographics (e.g., occupation, level of education, and health literacy); medical parameters (type, severity, and duration of disease); medication profile (complexity of regimen and side-effect profile); behavioral factors (patient–provider communication and patient health beliefs); and economic constraints (type of insurance, cost of medication, and patient income). The elderly are at high risk of nonadherence.[62]

Improving Drug Adherence

Patients who self-administer medications typically take less than half of what is prescribed, therefore efforts to improve adherence assume high import.[63,64] Increasing patient communication and discussing the importance of following the drug regimen is one of the few simple interventions that can improve adherence. Involvement of caregivers, supplementation with written material, simplified dosing schedules, and dosing aids to organize medications (e.g., pillboxes) have demonstrated

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additional benefits. Patients who miss medical appointments are more likely to be nonadherent; assistance with scheduling and more frequent visits may provide additional benefit. Minimizing polypharmacy and mindfulness of financial burden to the patient also improve adherence rates.[65–67]

Fixed-dose medication combinations also have a positive impact on adherence. These products typically contain two active agents, each at a specific dose. Fixed-dose agents currently exist for the disease states of hypertension, human immunodeficiency virus, tuberculosis, and diabetes. A meta-analysis showed that the use of these products resulted in a significant 26% decrease in the risk of noncompliance compared to single-drug component regimens. A 24% reduction in the risk of noncompliance was noted for hypertension alone.[68]

Strategies for Improving Medication Use

A thorough medication history that includes prescription, nonprescription, and alternative agents is the first step to improving medication use. Having patients bring in all medications at each health-care visit can help ensure that providers are making clinical decisions with complete medication information. Asking patients about nonprescription or alternative therapy use is necessary, as these agents are frequently omitted from medication lists.

Once a complete list of medications has been obtained, each medication should be evaluated to determine its necessity for the patient at that point in time. Each medication should have an indication, and if no indication exists, the medication should be evaluated for taper or discontinuation. Each medication should also be evaluated for efficacy, especially if a specific symptom is being treated. Medications prescribed for the purpose of treating the adverse effects of other medications should be carefully assessed. Although prescribing cascades may be unavoidable, medication complexity must be taken into account. Undertreated medical conditions should be assessed to determine if the addition of medication is warranted.

If a medication is deemed necessary, its use, therapeutic goals, and response should be monitored regularly, as medical conditions and patient pharmacokinetic and pharmacodynamic responses may change with time. Medication reconciliation should be performed for all transitions in care to ensure no medication omissions have occurred. Risk–benefit assessments should be performed for each medication to see if benefits outweigh potential risks. Duplicate medications should be tapered and discontinued. Drug–drug and drug–disease interactions, medication regimen complexity, prescription formulary information, and financial burden must also be considered to alleviate medication burden to the patient. Consideration should be given to those medications that can be given on a regular basis as few times per day as possible to increase patient adherence. Medication counseling should be provided to both patient and caregiver orally and in print.

Medication necessity should be considered at end of life. Goals of care, time to benefit, and remaining life expectancy are important factors to assess and may render certain medications irrelevant.[69]

Deprescribing

As people age, goals of care and risks of adverse effects often change for individuals. When a medication's known risk surpasses the anticipated benefit or the value of the medication is no longer consistent with the goals of care, patient preference, or remaining life expectancy, clinicians should consider deprescribing.[70] Deprescribing is the planned reduction in dose or discontinuation of a medication to reduce the risk of drug exposure to a patient. While reducing drug exposure may be helpful, a risk of deprescribing is subjecting a patient to an adverse drug withdrawal event (ADWE). An ADWE is a clinically relevant sign or symptom that is caused by the removal of a drug, which may include a physiological withdrawal reaction, an exacerbation of the underlying condition the medication was being used to treat, or new symptoms caused by abrupt withdrawal.[71] To reduce the risk of an ADWE, it is important to know whether a medication can be abruptly discontinued or should be tapered to prevent any adverse effects. Common classes of medications such as cardiovascular and CNS agents are associated with ADWEs.[71] As deprescribing becomes an important part of prescribing in older adults, evidence-based guidelines and algorithms are being developed to provide more information on best practices for medication discontinuation.[72]

Conclusion

The safe, effective, and optimal use of medications in the elderly requires heightened awareness in comparison to other patient populations. The issues of increased sensitivity to drug effects (both desired and adverse), the underuse of proven drug therapies, and a high incidence of medication nonadherence increase the need for due diligence in

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prescribing and monitoring drug therapy in this population. The employment of a multidisciplinary approach to patient care as well as the use of known methods for improving medication adherence are key to long-term success in the medical management of the elderly patient.

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Chapter 6

Nutrition and Aging

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By the year 2050, the global population of older adults over age 60 is projected to be 2 billion, with the number of individuals aged 80 or over projected close to 400 million. With this significant shift in demographics, the practice of important health behaviors becomes paramount, amongst them a focus on optimizing nutrition. The goal is to facilitate healthy aging; not only to increase years of life, but more specifically to maximize healthy active years. Changes in taste, smell, dental troubles, gastrointestinal absorption, limited access to fresh food, cognitive decline, and less social support are some of the factors leading to nutritional frailty in older adults.[1]

Sufficient intake of energy and nutrients is key for the human body's ability to optimize physiology and organ function and contribute to overall health. Adequate nutrition helps strengthen an older adult's immune system to decrease infection, preserve musculoskeletal function, and optimize overall functional and cognitive status.[2] There are variations in the definition of malnutrition, but one all-encompassing definition, "a state resulting from lack of intake or uptake of nutrition that leads to altered body composition (decreased fat-free mass) and body cell mass, leading to diminished physical and mental function and impaired clinical outcome from disease," emphasizes its far-reaching impact on an older adult.[3] The World Health Organization (WHO) describes malnutrition in a broader sense, including undernutrition, overweight conditions, and obesity.[4] The Academy of Nutrition and Dietetics and the American Society for Parenteral and Enteral Nutrition (ASPEN) consensus statement states that the diagnosis of malnutrition can be made if two or more of the following six criteria are met: insufficient energy intake, weight loss, loss of muscle mass, loss of subcutaneous fat, localized or generalized fluid accumulation that may mask weight loss, and diminished functional status as measured by handgrip strength.[5] In 2018, the Global Leadership Initiative on Malnutrition (GLIM) issued new diagnostic criteria for malnutrition highlighting the role of inflammation on nutritional status. The diagnosis requires a combination of at least one phenotypic (nonvolitional weight loss, low body mass index [BMI], or reduced muscle mass) and one etiologic criterion (reduced food intake or absorption, or underlying inflammation due to acute disease/injury or chronic disease).[6]

The consequences of undernutrition include an increase in morbidity and mortality; more specifically, decreased bone mass, impaired muscle function, immune system dysfunction, poor wound healing, and prolonged postoperative recovery.[7] Different segments of the geriatric population have been found to have a varied prevalence of malnutrition: 5–10% of community-living elderly, 30–60% of hospitalized elderly, 51% in geriatric rehabilitation, and between 12% and 85% of nursing home residents are suboptimally nourished.[8] Given the complexity and multifactorial presentation of malnutrition, up to 60% of cases elude recognition at the time of hospital admission.[8]

Malnutrition has a multifactorial etiology, with the main causes identified by the "Nine Ds of weight loss in the elderly"[9] (Table 6.1).

Age-Related Physiological Etiologies of Eating Disturbances in the Elderly

Gastrointestinal

There is a decrease in gustatory and olfactory input, part of the cephalic phase of digestion, resulting in a disturbance of how sight and smell normally regulate appetite and food ingestion.[10] Dry mouth can lead to poor dentition, which adversely impacts chewing. Reduced peristalsis and impaired lower esophageal sphincter contraction result in an increase in the incidence of gastroesophageal reflux disease. Impaired cholinergic function of the myenteric plexus can lead to decreased gastric emptying and early satiety. There is a higher prevalence of hypochlorhydria/achlorhydria,

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Table 6.1 Nine Ds of weight loss in the elderly

Dementia
Dysgeusia
Dysphagia
Diarrhea
Depression
Disease (acute, chronic)
Dentition
Disability
Drugs

which can alter oral medication bioavailability and absorption of food, vitamins (B12, D), and minerals (calcium, iron).[11] The decline in cytochrome P450 (CYP) activity influences the pharmacokinetics of medication and the risk of adverse events and drug–drug interactions.[12]

Neuroendocrine

The central and peripheral nervous system are involved in appetite regulation. The process of aging affects the level of neurotransmitters in the hunger center in the lateral hypothalamic nucleus and the satiety center in the ventromedial hypothalamic nucleus. At the level of the peripheral nervous system, glucagon-like peptide (GLP-1) is an anorexic neurotransmitter that is found at higher levels in the elderly, resulting in delayed gastric emptying. Leptin, produced in adipose cells, regulates energy intake relative to fat stores. Given the change in body composition with aging resulting in an increase in fat mass, leptin levels are high and increase satiety. Cholecystokinin (CCK), released from the intestine after food ingestion, tends to be higher in older adults and increases satiety.

Cytokines

Cytokines have been implicated in many disease processes, malnutrition included. Typically, proinflammatory cytokines are released as a response to a body stressor such as infection, but it seems that aging can be considered a stressor based on the cytokine levels found in older adults. The elderly have higher levels of cortisol, which induces the release of cytokines such as interleukin (IL)-6 and tumor necrosis factor alpha (TNF-α). Increased TNF-α levels correlate with higher levels of leptin and also reduce the sensitivity of muscle to anabolic stimuli. Through complex mechanisms involving the acute phase response in the liver and via the hypothalamic pituitary axis, IL-6 effects lead to bone and muscle breakdown. Overall, the proinflammatory cytokines promote anorexia and are catabolic.

Other Causes of Malnutrition in the Elderly

Older adults may have a decreased desire to eat and drink as a result of a multitude of risk factors that interact with an older adult's changing physiology, environment, and burden of comorbid illness. There is reduced olfactory and gustatory function, along with a decrease in the density of tastebuds. The threshold for recognizing a particular taste is increased for all of the basic tastes: salty, sweet, bitter, and sour.[13] Diminished taste alters the pleasure of eating, which can culminate in protein-energy undernutrition. Other changes with aging include diminished central orexigenic (appetite-stimulating) signals and a relative increase in gastrointestinal satiety. These physiological changes can be looked upon as a balancing act – an adaptation to decreasing energy requirements with advancing age, which may have potential for malnutrition if the reduction in food intake is excessive.

Often, the integrity of the oral cavity, which is key to ingestion and the initiation of digestion, is overlooked. For optimal chewing function, one must maintain good oral health and hygiene.[14] Important oral considerations include the status of the person's teeth, specifically whether the individual has untreated caries or is edentulous/wearing dentures/has missing teeth, and the existence of periodontal disease. If an individual has experienced recent weight loss, the dentures may be ill-fitting and result in gingival and buccal irritation, further compounding potential weight loss by adversely impacting the desire to eat. In addition to tooth considerations, mouth hydration impacts oral health. Saliva, composed of over 99% water, also contains electrolytes, minerals, mucus, antibacterials (IgA and lysozyme), and enzymes. The enzymes are critical to the initiation of digestion and serve a role in minimizing bacterial decay and caries formation. Saliva serves a lubricative function, which facilitates swallowing and prevents drying of the oral cavity. Xerostomia, dry mouth, can hinder chewing, taste perception, and swallowing. This condition is not a consequence of normal aging, but rather can be secondary to medications, comorbidities, and decreased oral fluid intake. For all the above reasons, attention to oral health is paramount.

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Malnutrition in the elderly also has diverse social causes: social isolation, loneliness, food availability and affordability, a person's own values, and even the impact of the governing laws of a person's country can affect the pattern of eating. Other factors include caregiver neglect, education level, and financial exploitation of vulnerable older adults. An older adult may have superimposed acute and chronic illnesses, which when combined with the associated medications required for treatment can adversely affect appetite and simultaneously create increased caloric requirements in the setting of cachexia or malabsorption.[15]

Malnutrition Syndromes

Anorexia

With aging, food choice starts to change secondary to the influences of biological factors, palatability, financial status, social determinants, and psychological factors. Longitudinal studies, such as the National Health and Nutrition Examination Surveys (NHANES) and the Baltimore Longitudinal Study, have shown that the decline of weight and energy intake with advancing age represents the most common eating disturbance in the elderly.

Anorexia of aging can be subdivided into: physiologic, pathologic, environmental, and psychological.[16] Physiologic changes include: impaired chewing, decreased action of salivary glands, impaired esophageal motility, reduced gastric secretions, and decreased sensitivity of taste and smell. Superimposed on physiological changes, pathological anorexia is the consequence of acute or chronic illnesses such as malignancy, stroke, chronic obstructive pulmonary disease (COPD), dementia, and mood disorder. The negative effect of many classes of medications on appetite should not be overlooked.

Cachexia

This is defined as a "complex metabolic syndrome associated with underlying illness and characterized by loss of muscle with or without loss of fat mass. The prominent feature of cachexia is weight loss in adults."[17] Cachexia is not a result of starvation or age-related muscle loss. The illnesses associated with cachexia include cancer, COPD, chronic kidney disease (CKD), chronic liver failure (CLF), chronic infections, and congestive heart failure (CHF).[18] These medical conditions manifest with systemic inflammation and a hypercatabolic state, leading to cachexia. Cachexia is therefore the result of complex interactions between chronic disease, host metabolism (insulin resistance, increased lipolysis and lipid oxidation, increased protein breakdown), and an imbalance between proinflammatory and anti-inflammatory cytokines. The proinflammatory cytokines inhibit transcription of muscle myosin heavy chains and induce proteolysis, leading to cachexia. Progressive muscle loss is accompanied by a decrease in function and decline in quality of life, and is predictive of increased morbidity and mortality.[19,20] As stated by Muscaritoli et al., "though not all malnourished patients are cachectic, all cachectic patients are invariably malnourished."[21] The Society of Sarcopenia, Cachexia and Wasting Disorders (SCWD) developed a set of diagnostic criteria for cachexia, helping differentiate late-stage cachexia from pre-cachexia, in an effort to identify conditions that cause cachexia as well as focus on earlier initiation of interventions.[22]

Sarcopenia

Sarcopenia is characterized by progressive and generalized loss of skeletal muscle mass and strength with a risk of adverse outcomes such as physical disability, poor quality of life, and death.[23,24] This loss of muscle mass compromises strength, immunocompetence, and the body's metabolic homeostasis.[25] With the associated functional impairment resulting from a decrease in muscle mass, there is increased likelihood of falls and associated loss of autonomy.[26] Consensus papers with a goal of defining sarcopenia have been published. Walking speed, handgrip, and muscle mass are the common parameters used for assessment.

Importantly, it is recognized that in some conditions, such as malignancy, rheumatoid arthritis, and aging, lean body mass can be lost while fat mass remains unchanged or increases. This imbalance between a loss of muscle mass and an increase in body fat results in marked weakness. In the elderly, skeletal muscle protein synthesis is resistant to the anabolic action of insulin. This resistance may lead to age-related muscle loss. As adipose tissue is recognized to be metabolically active, it is thought that an increase in visceral fat may lead to an increase in secretion of proinflammatory cytokines (IL-6, TNF-α), which, in turn, have a catabolic effect on muscles, further contributing to sarcopenia.[26]

Sarcopenic Obesity

Older adults with obesity are at increased risk of poor health and disability because of an obesity/muscle impairment syndrome known as sarcopenic obesity. This is a chronic condition and a growing public health concern.

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Its prevalence (4–12%) is related to adverse lifestyle changes over the past decades leading to higher rates of obesity. When obesity (BMI ≥ 30 kg/m^2) and muscle impairment coexist, there is a synergistic increase in the development of adverse health outcomes.[27] Sarcopenic obesity was initially defined by Baumgartner as a muscle mass index (appendicular skeletal muscle mass divided by body height squared in meters) less than 2 SD below the sex-specific reference for a young, healthy population measured with dual X-ray absorptiometry (DXA). There is no widely accepted definition, as the methods used to define obesity are quite variable between studies. The coexistence of sarcopenia and obesity is sufficient to suggest the presence of the condition. Sarcopenic obesity has a greater association with physical functional decline, metabolic syndrome, and atherosclerosis than obesity alone.

In sarcopenic obesity, the decline in muscle function is not proportional to the decline in muscle mass; that is, there is a greater decline in strength than would be expected on the basis of the decline in muscle mass alone. It is muscle quality that makes up this difference. Included are a decrease in fiber size and number, reduction in contractility in intact fibers, fat infiltration into muscle, increase in collagen, and impaired neuroregulation of muscle contractility. The mechanisms leading to the obesity/muscle impairment syndrome are diverse, including an age-related decrease in physical activity, inflammation, insulin resistance, decrease in growth hormone and testosterone, and insufficient protein in the diet.[28] In fact, insulin resistance secondary to obesity may underlie the development of sarcopenia. Interventions such as exercise, resistance, and aerobic forms to promote weight loss, as well as nutritional modifications including increasing protein intake and a high dietary calcium intake, may have favorable effects on the management of sarcopenic obesity. The entity of sarcopenic obesity and its management is an ongoing area of research.

Nutrition Screening

Because of the serious impact of nutritional impairment on health outcomes in older adults, all geriatric patients, both inpatient and outpatient, should be screened for malnutrition. Screening for nutritional status includes checking weight, calculating weight loss, and using a screening tool to identify an individual at risk of poor nutrition.

A handy mnemonic to screen elderly patients for treatable causes of weight loss is MEALS ON WHEELS[29] (Table 6.2):

Table 6.2 MEALS ON WHEELS mnemonic

Medications
Emotional (depression)
Alcoholism, anorexia tardive, abuse (elder), late-life paranoia
Late-life paranoia or bereavement
Swallowing problems
Oral problems
Nosocomial infections, no money (poverty)
Wandering/dementia
Hyperthyroidism, hypercalcemia, hypoadrenalism
Enteric problems (malabsorption)
Eating problems (e.g., tremor)
Low-salt, low-cholesterol diet
Shopping and meal preparation problems, stones (cholecystitis)

Measuring serial weights is the best way to identify a trend. In frail elderly patients, obtaining a weight can pose a challenge because of physical limitations. A chair or bed scale can help those who can't stand on an upright scale. Involuntary weight loss is predictive of mortality. Clinically significant weight loss is defined as >2% decrease of baseline body weight in 1 month, >5% decrease in 3 months, or >10% decrease in 6 months.[30]

There is no gold-standard screening test, nor is there a well-validated general laboratory screening test for diagnosis of malnutrition in the elderly. An older adult with involuntary weight loss of 5–10% or more in less than a year should undergo further evaluation for undernutrition. One must also consider the setting of the patient, as not all tools are validated in a general geriatric population. Consideration must be given to whether the patient is community-dwelling, in residential care, or in acute hospital care.

Commonly used nutrition screening tools include the Mini-Nutritional Assessment (MNA), the Malnutrition Universal Screening Tool (MUST), and the Subjective Global Assessment. A more detailed assessment follows the use of a screening tool and can include: anthropometric measurements (BMI/mid-arm circumference), quantifying weight loss, food intake, and medication history, as well as laboratory investigations comprising serum albumin, lymphocyte count, and total cholesterol.

The two screening tools that exhibit greater than 80% sensitivity and specificity are the Mini-Nutritional Assessment Short Form (MNA-SF) and Malnutrition Screening Tool (MST). The MUST is quick to administer

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and is used for estimating malnutrition risk in both community dwellers and hospitalized patients.

MNA-SF

The MNA-SF uses six questions from the MNA covering the following areas: survey of food intake in the past 3 months, weight loss in the past 3 months, mobility, psychological stress or acute medical illness in the past 3 months, neuropsychological problems, and BMI (calf circumference can be substituted if BMI not available). A score of 12–14 reflects normal nutritional status, while a score of 8–11 suggests a person is at risk of malnutrition and a score of less than 7 is concerning for malnutrition. This tool can be used in the acute-care setting as well as in residential care and in the community. The MNA-SF demonstrates good sensitivity when compared to the full 12-item MNA, and offers the advantage of being a rapid screening tool for nutritional assessment.[31]

MST

The MST has been validated for use in acute care, ambulatory care settings, and residential care.[32] It consists of two questions related to recent unintentional weight loss ("Have you lost weight recently without trying?"), quantifying the weight loss, and screening for poor eating because of a decreased appetite ("Have you been eating poorly because of a decreased appetite?").

MUST

The MUST was developed in the UK in 2003 and is used worldwide.[33] It comprises five steps including gathering nutritional assessment (weight, height, BMI), checking for involuntary weight loss in the past 3–6 months, establishing recent acute illness of greater than five days, during which there has been no nutritional intake, determining the overall risk score of malnutrition (based on adding the scores from the first three steps) giving a score of 0 (low risk) to 2 (high risk), and finally implementing management guidelines for patients. It takes less than 5 minutes to complete.

Weight Loss and BMI

Body weight generally increases through adulthood until the sixth/seventh decade of life, at which point it begins to decline. Cross-sectional studies have shown that the finding of a decline in body weight is at least partially due to the premature death of obese individuals. Both intentional and unintentional weight loss is associated with adverse outcomes. Between 20 and 80 years of age, muscle mass decreases by about 30%. After age 50, approximately 1–2% of muscle mass is lost each year thereafter. Muscle remodeling ensues, consisting of: intramuscular fat accumulation, muscle atrophy (type IIA fibers), decreased satellite cell proliferation, and a decrease in the number of motor units. Any illness that results in bed rest or immobilization for a period of 10 days results in loss of 1 kg of muscle mass.[34]

There is uncertainty as to the optimal BMI for an elderly person. Based on WHO guidelines, a suggested BMI to maintain good health is 18.5–24.9 kg/m^2. Interestingly, a meta-analysis by Flegal showed that being overweight (BMI 25–<30) was, in fact, significantly associated with lower all-cause mortality.[35] Mortality risk in the low-BMI group is increased and, as a result, mortality rates in the higher-BMI groups may be falsely low.[36] This was not exclusive to the elderly. The "obesity paradox," which describes the improved survival associated with obesity, is seen in patient populations (advanced malignancies, CHF, end-stage renal disease) that have protein-calorie malnutrition, whereby those who started off with a higher BMI may have some protection conferred to them in the form of nutritional reserve. Alternatively, it has been argued that the low mortality rates in the higher-BMI groups are artificially low given that the mortality risk in the low-BMI group is increased because of the prevalence of chronic disease. Another consideration amongst the elderly is that BMI may not be as valid a measure as in the younger population because of the disproportionate loss of muscle mass associated with aging. BMI is now sometimes used as a prognosticator for various disease states. In a study assessing optimal treatment strategies for ST-elevation myocardial infarction, BMI (along with serum troponin level) was found to be predictive of failure to discharge home.[37]

Nutritional Requirements of Older Adults

A revised report was published in 2021 by the US Department of Agriculture (USDA) and the US Departments of Health and Human Services (DHHS), on a mandated five-year cycle.[38] The report contains information about nutritional and dietary information and guidelines for the general public, focusing on eating patterns and their food and nutrient characteristics.

These are the main points from the 2015–20 *Dietary Guidelines for Americans*[38] (Table 6.3):

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Table 6.3 Main points from the 2015–20 *Dietary Guidelines for Americans*

1. Follow a healthy eating pattern across the lifespan. All food and beverage choices matter. Choose a healthy eating pattern at an appropriate calorie level to help achieve and maintain a healthy body weight, support nutrient adequacy, and reduce the risk of chronic disease.
2. Focus on variety, nutrient density, and amount. To meet nutrient needs within calorie limits, choose a variety of nutrient-dense foods across and within all food groups in recommended amounts.
3. Limit calories from added sugars and saturated fats and reduce sodium intake. Consume an eating pattern low in added sugars, saturated fats, and sodium.
4. Shift to healthier food and beverage choices.
5. Support healthy eating patterns for all. Everyone has a role in helping to create and support healthy patterns in multiple settings nationwide from home to school to work to communities.

Table 6.4 Quantitative recommendations for dietary components

Consume <10% of calories per day from added sugars
Consume <10% of calories per day from saturated fats
Consume <2,300 milligrams per day of sodium
If alcohol is consumed, it should be consumed in moderation – **up to one drink per day for women** and **up to two drinks per day for men**

The key recommendations in the document are to consume a healthy eating pattern that accounts for all foods and beverages within an appropriate calorie level.

A healthy eating pattern includes: a variety of vegetables from all of the subgroups (dark green, red and orange, legumes, starch), fruits (especially whole fruits), grains (at least half of which are whole grains), fat-free or low-fat dairy, a variety of protein (seafood, lean meats and poultry, eggs, legumes, nuts, seeds, and soy products), oils.

A healthy eating pattern limits: saturated fats and trans fats, added sugar, and sodium.

There are specific quantitative recommendations for several dietary components that need to be limited to maintain healthy eating patterns and avoid adverse health outcomes (Table 6.4).

One of the key recommendations accompanying the *Dietary Guidelines* is to meet the *Physical Activity Guidelines for Americans* with a goal of promoting health and decreasing chronic disease. Physical activity is the counterweight to calorie intake. DHSS published a set of physical activity recommendations entitled *Physical Activity Guidelines for Americans*, 2nd edition.[39] Those 65 years and older should still follow the adult guidelines; however, it is recognized that not all older adults can meet those recommendations. In that case, they should be as physically active as their abilities and conditions allow. It is important to focus on balance exercises to decrease the risk of falls. We must help older adults to understand how their chronic medical conditions may affect their ability to do regular physical activity safely.

In 2019 Canada introduced a revised *Canada's Food Guide*, the first revision since 2007.[40] Previously, the emphasis of *Canada's Food Guide* had been on incorporating foods from different food groups to achieve a balanced diet, a recommendation to reduce consumption of trans fats and replace saturated fats with unsaturated fats, and attention to portion control. With the most recent version of *Canada's Food Guide*, there is a shift from specific portion sizes to a recommendation based on proportions. Specifically, a person's plate should include 50% of calories coming from vegetables and fruits, 25% of calories from whole-grain foods, and 25% of calories from protein. It also recommends that protein foods should come from plants more often and that water be the beverage of choice.

These are based on the premise that the goal is to meet one's nutritional needs through food consumption rather than relying too heavily on supplements or fortified foods. A balanced diet that incorporates the Institute of Medicine of the US National Academy of Sciences Dietary Reference Intakes (DRIs) considers an individual's age, gender, and activity level. The DRIs are nutrient reference values, encompassing more than 40 nutrient substances, which provide a scientific basis for the development of food guidelines in the United States and Canada.[41,42] Multiple reference values are provided by the DRIs, including:

1. **Recommended Dietary Allowances** (RDAs): This represents the daily dietary intake level of a nutrient considered sufficient to meet the requirements of 97.5% of healthy individuals in each life stage and gender group.
2. **Adequate Intake** (AI): For these nutrients, there is insufficient scientific evidence to establish an EAR (see 3 below) or set an RDA. It is a recommended average daily nutrient intake level based on observed or experimentally determined approximations or estimates of nutrient intake by a group of apparently healthy people who are assumed to be maintaining an adequate nutritional state.
3. **Estimated Average Requirement** (EAR): The median value that is estimated to meet the requirement of half

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the healthy individuals in a life stage and gender group. The EAR is used to calculate the RDA: (EAR + 2 standard deviations = RDA).

4. **Tolerable Upper Intake Level** (UL): The maximum level of daily nutrient intake that is likely to pose no risk of adverse health effects. In general, there is no established benefit to healthy individuals of ingesting nutrients in amounts exceeding the RDA or AI. Folate use in pregnant women for the prevention of neural tube defects represents an exception.

Micronutrients

Older adults may have micronutrient deficiency in addition to macronutrient deficiency. Micronutrients include vitamins, minerals, and trace elements. The requirement for micronutrients does not diminish with age; thus, with the encroachment of malnutrition, the elderly can become deficient, particularly in vitamin D, calcium, folic acid, and vitamin B12. After menopause, a woman's need for iron diminishes to the level that is required by men. Antioxidants like β-carotene, α-tocopherol, and ascorbic acid are important parts of the body's response to oxidative stress against free radicals. Trace elements, such as zinc, selenium, and iron as well as vitamins A, C, and E, exhibit antioxidant properties in the process of cell replication and protect the immune cells from oxygen free radicals. The latter is important to support an immune system that is recognized to weaken with advancing age because of declining production of antibodies by B-cells and lower cytokine production by T-cells.[43]

Vitamin B12

There is a 10–20% prevalence of vitamin B12 deficiency in older adults, as shown by low or low normal serum cobalamin levels and elevations in serum methylmalonic acid and homocysteine.[44] It is important to recognize that although someone's cobalamin level may be in the low normal range, they may be deficient and manifest neurologic, psychological, and/or hematologic abnormalities. Traditionally, a deficiency of intrinsic factor was thought to account for vitamin B12 deficiency; however, more is now understood about the pathophysiology. About 15% of older adults absorb protein-bound vitamin B12 poorly, as a result of gastric achlorhydria or atrophic gastritis. This process of chronic inflammation of the stomach mucosa results in decreased secretion of intrinsic factor and a concomitant lowered absorption of the food-protein-B12 complex.[45] There may be some association with *H. pylori* infection. Given the high prevalence of vitamin B12 deficiency and the safety of its treatment, there are some proponents of routine vitamin B12 screening in those over age 65. This is not yet incorporated into any screening guideline. Rather, older adults should take supplements containing vitamin B12 or eat fortified food products. Those with food cobalamin malabsorption-induced vitamin B12 deficiency need oral vitamin B12 (1,000 mcg daily) to optimize the serum vitamin B12 level and correct any hematological abnormality.[30]

Vitamin D and Calcium

There is an age-related decrease of vitamin D and calcium receptor expression in the duodenum in women. This, combined with impaired skin synthesis of previtamin D and decreased hydroxylation in the kidneys, can result in vitamin D deficiency in many older adults.[46] Nutritional intake of vitamin D is also often low in older adults. The consequences of low vitamin D status in the older adult are significant: muscle weakness, low mood, increased risk of falls with resultant fractures, and an overall decrease in functional status.[47] Low vitamin D status has been linked to other chronic comorbidities such as hypertension, diabetes, dyslipidemia, and peripheral vascular disease.[48] Interestingly, in individuals who had severe vitamin D deficiency but did not have cardiovascular risk factors, the risk of developing diabetes, hypertension, and dyslipidemia was increased. Currently, universal screening for vitamin D deficiency is not recommended. Rather, it is important to recognize those who may be at greater risk for being 25-hydroxy vitamin D deficient (less than 20 nmol/L or 8 ng/ml) or insufficient (less than 75 nmol/L or 30 ng/ml): institutionalized older adults and people who are housebound or have limited sun exposure, dark skin, osteoporosis, or malabsorption.

The RDA of vitamin D for adults up to age 70 is 600 IU daily with an increase to 800 IU for those over age 70.[30] Supplementation is recommended, as older adults may not spend enough time outdoors for the skin to produce sufficient vitamin D. At baseline, increased dietary consumption of vitamin D from foods such as salmon, mackerel, snapper, egg yolk, milk, and fortified soy milk should be encouraged.

With advancing age, calcium absorption diminishes, such that individuals who are 80 years old only absorb about two thirds of the calcium compared to a younger adult. Calcium supplementation has become controversial because of its possible risk of increasing cardiovascular

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disease. Two meta-analyses assessing calcium or calcium with or without vitamin D raised concern about an increased risk of myocardial infarction in those patients assigned to calcium versus placebo.[49,50] The criticism of the trials in these meta-analyses was that they were not designed to assess cardiovascular outcomes a priori. Other meta-analyses have not shown an increase in cardiovascular events with calcium. Prospective studies have subsequently been done and, interestingly, when comparing dietary calcium to calcium supplements, dietary calcium intake was associated with no relationship to or even reduction in heart disease, myocardial infarction risk, and death.

Nutritional Issues in Long-Term Care

Naturally, every resident of a long-term care (LTC) facility should be provided with sufficient food and fluids to maintain adequate nutrition and hydration; however, the medical complexity of residents in LTC often results in the development of nutritional challenges in these individuals. In addition to the common comorbidities affecting the older adult, the specific nutritional challenges that are more prevalent in residents of LTC facilities include dehydration; dysphagia due to dementia, stroke or other neurodegenerative disease; emphasis on therapeutic diets (targeted to control diabetes, hypertension); noisy institutional environments; under-recognition of pain; constipation; and mood disorder.[51]

Once an individual moves into LTC, the offered meal plan is often perceived to be restrictive and residents are expected to adapt to existing meal structures. Not only is the meal menu set, but meal times, dining settings, and dining room companions are also preselected. These imposed limitations can lead to lower food consumption, satisfaction, and quality of life, and ultimately to malnutrition. LTC institutions should seek input from their residents on issues related to variety and timing of meal choice, such that, for example, residents are not forced to choose a meal many days in advance.[52] Options for residents with texture-modified diets should be as diverse as for those selecting from a non-texture-modified menu. It was found that residents' food service satisfaction increased by 30% when they were given some autonomy over their food choices.[53]

Nutritional Supplements and Other Interventions

Nutritional supplements are often invoked when an elderly person screens positive for malnutrition; however, there is limited evidence supporting the effectiveness of nutritional supplements. It is important to emphasize that an oral nutritional supplement is not a meal replacement, and thus, if offered, should be given between meals, not instead of a meal, as it is likely to increase satiety. A Cochrane Database systematic review in 2009 comprising 62 trials and 10,187 participants examined whether oral protein and energy supplementation showed a benefit in terms of weight gain, mortality, risk of complications, and any change in functional status in older people.[54] The results showed that supplementation produced a 2.2% weight change and a signal toward a mortality benefit in undernourished individuals – overall, limited gains.

Practical Tips

To address the often missing social aspect of eating for an elderly individual, time- and resource-intense interventions are necessary. Caregiver support includes mealtime oversight, encouragement, and emotional support. In nursing homes, aesthetics to simulate a more homelike environment focusing on lighting, relaxing music, and easy-to-use silverware can help decrease the risk of low caloric intake. Specific disease-related dietary recommendations form a part of the management of several chronic diseases, such as diabetes and CHF. This can be at odds with an older adult who is at risk of malnutrition. For example, an individual with CHF trying to follow a salt-restricted diet may in effect end up limiting caloric intake as the food palatability is unacceptable to the individual without the addition of salt. Educating the patient about salt alternatives would make a meaningful contribution to optimize their food intake. This underscores the multifaceted approach to improve an older adult's nutritional status.

The concept of tailoring food offered based on the cause of anorexia is emerging. For the individual with early satiety, one could offer a more nutrient-dense meal. Alternatively, for someone with diminished taste and/or smell, flavor-enriched foods would be preferred, and in those with an increased inflammatory state, a diet higher in polyunsaturated fats may prove beneficial. These specific, tailored interventions are more likely to be effective once routine screening for malnutrition is in place and there is a better understanding of the particular cause of a specific individual's malnutrition.

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Chapter 7

Physical Activity and Exercise for Older Adults

Jorge Camilo Mora and Willy M. Valencia*

Physical Activity and Exercise among Older Adults

Physical activity and regular exercise are fundamental for healthy aging.[1–6] Among older adults, regular activities reduce risk of all-cause mortality and chronic disease, preserve functional capacity, help in the management of chronic conditions, and reduce health-care costs.[1–16] Given the prevalence of physical inactivity and sedentary behavior among older adults, exercise needs to be more thoroughly integrated into care plans and counseling in primary care settings.

Physical Inactivity and Sedentary Behavior among Older Adults

Although exercise is essential for older adults to achieve healthy aging, nearly one in four adults aged 50 and over in the USA (27.5%; approximately 31 million persons) self-reported as inactive.[5–7,17–20] The 2018 *Physical Activity Guidelines for Americans* by the US Department of Health and Human Services (USDHHS) defines inactivity as "not getting any moderate- or vigorous-intensity physical activity beyond basic movement from daily life activities."[6] According to a systematic literature review of studies published between 1981 and 2014, older adults spend an average of 9.4 hours a day, equating to 65–80% of their waking day, in sedentary behavior, defined as "sitting (non-exercising), reclining, and lying down (posture), or by low energy expenditure."[18] Self-reported data shows that the prevalence of inactivity grows with increased age: adults aged 50–64 years (25.4%), 65–76 years (26.9%), and 75 years and older (35.3%).[7]

Chronic Disease and Physical Inactivity

Nearly half of all American adults – approximately 117 million people – have one or more preventable chronic diseases, such as heart disease, cancer, or diabetes.[4,6,8] While regular physical activity can preventively decrease the risk of chronic disease, it also favorably influences seven of the ten most common chronic diseases for those who already have them.[1–6] Nevertheless, analysis of trend data regarding the percentage of adults meeting the *2008 Physical Activity Guidelines for Americans* over the last decade (2008–18) indicates that nearly 80% of adults are "not meeting the key guidelines for both aerobic and muscle-strengthening activity."[5–6,19–20] Conversely, the prevalence of inactivity was higher in adults reporting at least one of seven diseases than their counterparts (31.9% vs. 19.2%), and the pervasiveness of inactivity depends on chronic disease, from highest to lowest: chronic obstructive pulmonary disease (COPD) (44.4%), stroke (43%), diabetes (38%), depressive disorder (38%), coronary heart disease (37.2%), arthritis (33%), and cancer (31.6%).[7,17]

Physical Inactivity and Health-Care Costs

Physical inactivity among older adults has a significant impact on health-care utilization and expenditures. It is estimated that approximately $117 billion in health-care spending each year is linked to a lack of physical activity.[6]

Benefits of Physical Activity

Extensive studies demonstrate the benefits of physical activity for older adults (see Table 7.1). Even if inactive older adults are not able to meet target ranges of physical activity, *any increase* in physical activity can lead to substantial health gains.[2,5] Since the publication of the *2008 Physical Activity Guidelines Advisory Committee Report*,[2] there has been substantial scholarly literature that demonstrates the positive health benefits of reducing inactivity even if the weekly targets are not achieved.[5] Moreover, the *2018 Physical Activity Guidelines Committee Report* documents that whereas previous evidence focused on bouts of physical activity of 10 minutes in duration or more, more recent studies have

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Table 7.1 Health benefits of exercise for older adults[a]

Overall benefits for older adults	
• **Improves quality of life and well-being**: Physical activity has positive effects for physical, social, and emotional quality of life.[5,21,22] • **Improves physical function**: Exercise improves physical function in older adults with and without frailty.[5] • **Reduces all-cause mortality risk**: A sedentary person has a risk of dying almost 50 times higher than one who exercises about 5 times a week. Adherence to physical activity that burns more than 1,000 kcal/week is associated with up to 30% reduction in all-cause mortality.[23] • **Reduces aging effects**: Exercise attenuates the aging effects in major organ systems and reduces cellular hallmarks of aging.[24] • **Reduces cancer risk**: Exercise reduces overall cancer risk, including risks for breast and colon cancers by 30% and 10%, respectively.[25] It is also related to a lower incidence of bladder, endometrium, esophagus, kidney, stomach, and lung cancers.[5] • **Reduces risk of falls**: Multicomponent exercise programs that include strength, balance, and endurance and last more than 6 months are more likely to reduce the risk of falls.[26] Increased exercise is associated with reduced incidence of falls[2] and reduced incidence of fall-related injuries.[5] • **Reduced feelings of anxiety and depression**: Exercise is associated with reduced feelings of anxiety and depression in healthy people and in people with existing clinical syndromes.[5] • **Reduced risk of excessive weight gain**: Exercise leads to weight loss, and when a sufficient dose of moderate-to-vigorous physical activity is attained, it prevents weight regain.[5] It also functions as an additive effect on weight loss when combined with moderate dietary restriction.[5]	
***Specific physiological health benefits*[27,28]**	***Primary prevention***
Cardiovascular and pulmonary systems	
Increased heart rate variability; better endothelial reactivity; lower inflammatory markers; reduced arterial stiffness, improved cardiac output; less atherosclerotic disease; enhanced microvasculature; improved gas exchange; stronger respiratory muscle; improves muscle function and adaptation to oxidative stress;[29] may improve function of mitochondria in the skeletal muscle;[30] improves vascularization.[31]	• Reduces cardiovascular disease risk.[32] • Reduces the risk of heart failure.[33,34] • Reduces the risk of heart attack.[35] • Reduces the risk of peripheral vascular disease.[36] • Reduces high blood pressure.[37] • Lowers LDL and total cholesterol.[38] • Reduces the risk of stroke[39] and recurrent stroke.[40]
Neurological and neuro-psychological systems	
Faster nerve conduction; improved balance; improved memory, attention, and reaction time by increasing the size of the hippocampus and cortical volumes;[41] improved visual-spatial orientation and proprioception; improved sleep;[42] improved cognitive function following bouts of aerobic activity.[5]	• Reduces the risk of dementia.[5,43,44] • Prevents mild cognitive decline.[45] • Improves memory function.[41] • Physical inactivity is among the top reversible risk factors for Alzheimer's disease (which also includes hypertension, obesity, and diabetes).[46]
Immune and endocrine systems	
Reduced markers of systemic inflammation; increased basal metabolic rate; improved lipid profiles; lower body fat percentage;[47] improved insulin sensitivity and glucose homeostasis.[48–51]	• Reduces risk of obesity-related conditions.[47] • Reduces risk of diabetes mellitus.[48–51]
Musculoskeletal	
Increases muscle mass, strength, and power; preserves and increases bone mass; improves and maintains joint range of movement and flexibility;[52] increases synthesis of collagen in ligaments and tendons.	• Reduces risk of osteoporosis and risk of osteoporotic fracture, especially among postmenopausal women.[53] • Reduces risk of sarcopenia and frailty syndrome.[54,55] • Reduces age-related loss of muscle mass; improves muscle mass and strength, and physical function.[54,55]

[a] Reprinted with modifications from Mora JC, Valencia WM. Exercise and older adults. *Clin Geriatr Med*. 2018 (Feb.); 34(1):145–162, Copyright 2018, with permission from Elsevier. This table has been updated to include key health benefits of exercise documented in the *2018 Physical Activity Guidelines Advisory Committee Scientific Report*[5] and the *2018 Physical Activity Guidelines for Americans*,[6] as well as more recent scientific studies.

demonstrated that any amount of moderate-to-vigorous physical activity counts toward meeting the target range.[5] Physical activity of any duration can cumulatively contribute to health benefits.

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Role of Primary Care Providers

Given the immense benefits that physical activity offers older adults and the global burden of inactivity,[56,57] primary care settings are ideal points of intervention for providing physical activity counseling. Recent studies confirm that older adults are responsive to efforts aimed at promoting physical activity at an individual level.[5,58] Some experts argue that providers have particularly unique opportunities for making meaningful interventions on behavior modification because of the high regard that patients frequently have for clinicians' advice.[59] Nevertheless, primary care providers are not sufficiently capitalizing on their potential to impact the physical activities of older adults.

Earlier national studies helped raise awareness of the lack of integration of physical activity promotion in primary care settings and helped to identify barriers to exercise counseling.[60–63] In a national cross-sectional study of 298 primary care physicians, two major barriers identified were insufficient time and inadequate knowledge and/or experience.[60] A questionnaire survey in the UK found similar barriers, citing lack of time and resources as major barriers.[61]

Since then, there have been widespread calls for greater integration of physical activity promotion in primary care.[62–64] While there has been notable progress in this area, both in actual practice[65] and in the number of medical schools incorporating physical activity promotion into their curricula,[66] studies have found that current efforts have a high degree of variation in the quality and quantity of this intervention[67] and that there remains a significant need for greater integration of physical activity counseling and referral practice among primary care providers.[68]

The Patient Protection and Affordable Care Act of 2010 incentivizes physical activity screening and counseling by primary care providers as part of the national priorities related to health promotion, disability reduction, and chronic conditions management.[69] Physical activity screening is also part of the core measures of the Healthcare Effectiveness Data and Information Set (HEDIS) – a tool used by more than 90% of American health plans to measure performance of care and service. Despite these provisions, current reimbursement structures do not sufficiently incentivize physical activity counseling and referrals. Notably, the Centers for Medicare and Medicaid Services only provide reimbursements for physical activity counseling in cases where patients suffer from obesity, but not for counseling nonobese patients. While providing physical activity promotion is associated with increased provider satisfaction,[62] reimbursement structures need to be adjusted to incentivize such preventive measures. Moreover, primary care providers can and should develop community partnerships that can help promote physical activity in their patients.[70]

Barriers to Exercise

Older adults frequently cite poor health as their main reason for not engaging in more physical activity or exercise. Other prevalently self-reported reasons include: fear of injury, environmental challenges, lack of counseling by primary care providers, lack of knowledge regarding exercise benefits, and self-perception of harm when doing exercise.[71] Moreover, the social and environmental conditions in which people are born, live, learn, work, and age affect the health outcomes of patients. Studies have demonstrated that physical activity resources such as parks, sports facilities, fitness clubs, designated trails, and gyms are less prevalent in lower-income areas than in more affluent settings. A national study on the availability of physical activity-related facilities found that the likelihood of finding a fitness facility in a neighborhood with a median income of $75,000/year is 20% greater than in neighborhoods with a median income of $25,000/year.[72] These sorts of findings have important policy implications and have prompted national calls to integrate physical activity into city development planning.[4] Physicians need to evaluate access-related barriers and preferences when providing physical activity recommendations.

Practical Approach to Exercise Counseling for the Generalist

Careful evaluation of physical activity and physical function is a vital component of the comprehensive geriatric assessment of any older patient with any health condition.[73] These evaluations are prerequisites for properly engaging and educating patients regarding exercise interventions.

Properly understanding a patient's baseline physical activity, physical function, personal goals, and motivations will help physicians provide exercise counseling that is appropriate and safe and that can progress slowly. Pedometers are a useful, low-cost, and unobtrusive tool to assess physical activity in older adults by measuring numbers of steps.[74] Data from two or three days of regular activity levels is sufficient to serve as a reliable indicator of an estimated regular amount of physical

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activity in older adults. Physical activity in adults can be classified into three broad groups, depending on the number of daily steps: highly active older adults (≥10,000 steps daily), moderately active (5,000–10,000 steps daily), and inactive older adults (≤5,000 steps daily).[75]

The National Institute of Aging provides comprehensive educational materials and resources that are useful for patients and providers regarding types of physical activities, exercise and chronic disease, how to start exercising, exercise planning, and motivation.[76] Much of this material was previously housed through its *Go4Life* Campaign. A practical, three-step approach to exercise counseling is recommended (see Table 7.2).

Physical Activity and Exercise Guidelines for Older Adults

According to the USDHSS' *2018 Physical Activity Guidelines for Americans*,[5] the following is the target range of physical activity beyond routine activities: 150 minutes (2 hrs and 30 min) to 300 minutes (5 hrs) a week of moderate-intensity aerobic physical activity, or 75 minutes (1 hr and 15 min) to 150 minutes (2 hrs and 30 min) a week of vigorous-intensity aerobic physical activity, or an equivalent combination of moderate- and vigorous-intensity aerobic activity. See also guidelines from the American Heart Association [89] and the National Institute on Aging [104].

There are additional health benefits if the amount of moderate and vigorous intensity increases beyond 300 minutes per week.[5] In addition to aerobic exercise, older adults should engage in exercise activities promoting muscle-strengthening, balance, and flexibility.[5,78] Physical activity and exercise guidelines are provided across these four types of exercise (see Table 7.3). Older adults should be encouraged to engage in multicomponent recreational activities that include aerobic, balance, flexibility, and muscle-strengthening components, such as tai chi, yoga, or dancing.[5,79] Target levels of physical activity need to be adapted based on level of fitness and presence of chronic conditions.[80]

Exercise and Geriatric Care

Exercise and Chronic Disease

Epidemiological data shows that the prevalence of multiple chronic diseases is highest among older adults.[11] Adults with multiple chronic diseases experience poorer health outcomes, utilize more health resources, and account for a disproportionately greater amount of health-care spending.[8–16,84] Older adults should be as physically active as their condition allows. It is important for providers to tailor exercise prescriptions to patients' chronic conditions, fitness practices, and preferences (see Table 7.4).

Exercise and Medications

The number of prescribed medications increases in older adults. US survey data shows that whereas 90% of adults aged 65 and over take a prescribed medication, 39% take five or more regularly.[105] Since older adults are more likely to be taking multiple medications, including over-the-counter nutraceuticals, it is imperative for health-care professionals to review patients' medications when providing exercise counseling, prescription, and referrals. Commonly prescribed medications for older adults can have various possible impacts on exercise performance, including negative impairment (see Table 7.5).

Common nutraceuticals can also impact exercise performance. For example, turmeric, which is widely used for osteoarthritis, has the potential to increase risk of arrhythmias. Saw palmetto, sometimes used for benign prostate hyperplasia, increases the risk of tachycardia, arrhythmias, and fatigue. Chondroitin sulfate, commonly used for osteoarthritis, can increase risk of musculoskeletal and connective tissue disorders.[106] Red yeast rice, a very popular nutraceutical used for effects on lipids, increases risk of myalgia, arthralgias, and myopathy.[107] Melatonin, used for sleep disorders, can increase reaction time and thereby increase risk for falls. St John's Wort, commonly used for depression, has been associated with fatigue, lethargy, muscle or joint stiffness, and muscle spasms.

Exercise and Injuries

Fear of injury is frequently expressed as a factor that inhibits older adults from starting an exercise program.[121] Injuries in older adults are commonly related to repetitive stress and overuse (see Table 7.6).[122] Muscle strains are the most common injuries because of the decreased capacity of aging muscles to absorb energy. Additionally, aging is associated with decrease in muscle mass and muscle fibers. Tendinosis is extremely common because of changes in the microarchitecture, collagen disorganization, and decrease in the collagen content.[123] Microtrauma and lack of vascularity make tendons stiffer and more prone to inflammation. Since healing takes longer in older adults, adults 65 and over are especially vulnerable to injuries in endurance sports. Arthritis is commonly due to trauma and overuse.

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Table 7.2 Approach to counseling older adults on physical activity and exercise

Step 1. Evaluate

- Evaluate current physical activity and exercise practices.
- Screen for contraindications.

Contraindications [a–b][77]

Absolute	• Recent acute ischemic event • Unstable angina • Uncontrolled heart failure • Symptomatic/severe aortic stenosis • Uncontrolled cardiac arrhythmia	• Acute pulmonary embolism • Acute pericarditis • Suspected or known dissecting aneurysm • Acute systemic infection
Relative (clinical)	• Left main coronary artery stenosis • Moderate stenotic valvular heart disease • Severe arterial hypertension • Hypertrophic cardiomyopathy • High-degree atrioventricular bloc	• Ventricular aneurysm • Uncontrolled electrolyte imbalance • Uncontrolled metabolic disease • Mental or physical impairment that limits ability to exercise safely
Relative (situational)[c]	• Ongoing workup for cardiovascular or pulmonary disease • Ongoing workup for malignancy	• Ongoing evaluation and treatment of pain

Step 2. Educate

- Provide motivation and discuss goals.
- Explain the benefits and importance of physical activity (see Table 7.1).
- Explain the recommended physical activity guidelines (see Table 7.3).
- Discuss how chronic conditions (see Table 7.4), medications (see Table 7.5), injuries (see Table 7.6), or hip and knee arthroplasty may affect physical activity recommendations.
- Discuss access and preferences related to exercise equipment and/or programs at home or in the community.
- Discuss feasibility of different options to accomplish activity goals, accounting for any relevant limitations, according to established preferences.

Step 3. Establish

- Establish an initial intervention plan compatible with current physical activity and exercise levels, motivation, goals, limitations, access, and preferences. It is recommended that patients fill in an exercise planning sheet.
- Establish a plan for monitoring according to patient's preferences (e.g., facility-based functional testing, telephone/technology for monitoring).

[a] Fletcher GF, Ades PA, Kligfield P, et al. Exercise standards for testing and training: A scientific statement from the American Heart Association. *Circulation.* 2013; 128:873–934.

[b] These contraindications are based on known concerns related to cardiologic stress testing. The assumption is that the patient may endure risks for acute exacerbation, especially if exercise is not done without supervision. Nevertheless, this does not preclude the options for cardiac rehabilitation and other specialized interventions to prevent disability and preserve function. Referral to specialists in the field (cardiologist, exercise physiologist) may be warranted.

[c] Ongoing workup can lead to discovering an unknown absolute contraindication. It is recommended to complete any scheduled workup first. It can be a worrisome and stressful scenario. While exercise interventions can make a difference to the functional performance and outcomes from oncologic treatment and prognosis, the patient may not be ready or focused to engage in exercise. It is recommended to swiftly complete the oncologic workup and plan of care, and offer the exercise interventions right after, if consistent with the oncologic plan of care. With regard to pain, it can be best to hold exercise in order to avoid confusion in etiology and exacerbating factors. Once the pain is better controlled, exercise interventions can become part of the therapy.

Table 7.3 Regular exercise guidelines for older adults

Exercise type	Recommendation	Examples[b]
1. *Aerobic and endurance activities*	• Any modality of aerobic or endurance activity that does not impose excessive orthopedic stress. Adults who do *any* amount of moderate-to-vigorous physical activity gain some health benefits. Some physical activity is better than none. • Older adults should strive to do at least 150 minutes a week of moderate-intensity (5 or 6)[a] or 75 minutes a week of vigorous-intensity (7 or 8)[a] aerobic physical activity, or an equivalent combination, for substantial health benefits. • Older adults may incrementally build up stamina and should preferably spread activities throughout the week. Additional and more extensive health benefits are gained by extending this regimen to 300 minutes a week of moderate-intensity (5 or 6)[a] or 150 minutes a week of vigorous-intensity (7 or 8)[a] aerobic physical activity, or an equivalent combination.	Walking, dancing, swimming, water aerobics, jogging, aerobic exercise classes, bicycle riding (stationary or on a path), some activities of gardening, such as raking and pushing a lawn mower, tennis, golf (without a cart).
2. *Muscle-strengthening activities*	• Progressive weight-training program, weight-bearing, or weight-bearing calisthenics. • No specific amount of time is recommended for muscle-strengthening, but muscle-strengthening exercises should be performed to the point at which it would be difficult to do another repetition without help. • Muscle-strengthening exercises that involve all major muscle groups on 2 or more days a week, with moderate to vigorous intensity (≥6).[a] • Grip strength and muscle strength related to major muscles, including biceps, triceps, and quadriceps, are correlated with physical independence and reduction in risk of frailty, falls, and injury.[81] • Development of muscle strength and endurance is progressive over time. This means that gradual increases in the amount of weight or the days per week of exercise will result in stronger muscles.	Exercises using exercise bands, weight machines, hand-held weights, calisthenic exercises (body weight provides resistance to movement), digging, lifting, and carrying as part of gardening, carrying groceries, some yoga exercises, some tai chi exercises.
3. *Flexibility*	• Any activities that maintain or increase flexibility using sustained stretches for each major muscle group. • Minimum of 2 times per week. • This type of exercise is recommended to maintain the normal range of motion for daily activities and to reduce functional decline in the long term.[82,83] • It is typically joined with warm-up or cool-down surrounding aerobic or muscle-strengthening activities.	Neck stretch, shoulder stretch, shoulder and upper arm raise, upper body stretch, chest stretch, back stretch, ankle stretch, back of leg stretch, thigh stretch, hip stretch, lower back, calf stretch.[b]
4. *Balance activities for older adults at risk of falls and/or with mobility problems*	• Reduction in falls is seen for participants in programs that include balance and moderate-intensity[a] muscle-strengthening activities for 90 minutes a week plus moderate-intensity[a] walking for about 1 hour a week. • Older adults at risk of falls should do balance training 3 or more days a week and do standardized exercises from a program demonstrated to reduce falls.	Backward and sideways, heel and toe walking, and standing from a sitting position. Exercises can increase in difficulty by progressing from holding on to a stable support to doing them without support. Tai chi may also help prevent falls.

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Table 7.3 (cont.)

Exercise type	Recommendation	Examples[b]
	• Progressively difficult postures that gradually reduce the base of support, dynamic movements that perturb the center of gravity, and stressing postural muscle groups. • In older adults at increased risk of falls, strong evidence shows that regular physical activity is safe and reduces the risk of falls.	

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[a] On a scale of 0 to 10, where 0 is the level of effort of sitting and 10 is maximal effort, the level of *self-perceived effort* distinguishes between moderate-intensity activity (5 or 6) and vigorous-intensity activity (7 or 8). Level of effort for physical activity is relative to level of fitness.

[b] Helpful lists of exercises with illustrations can be found at US Department of Health and Human Services, National Institute on Aging. Four types of exercise can improve your health and physical ability. Last rev. Apr. 2, 2020. www.nia.nih.gov/health/four-types-exercise-can-improve-your-health-and-physical-ability Accessed on 7/8/2020.

X-ray findings may show joint space narrowing and osteophyte formation. Calcific tendinitis due to accumulation of hydroxyapatite crystals affects older adults. This causes pain and reduction in mobility, symptoms of synovial inflammation, and eventually joint destruction. Adhesive capsulitis presents in the older athlete as stiffness in joints at the extremes of movement.[124,125]

Exercise After Arthroplasty

Arthroplasty has become the gold-standard treatment for severe osteoarthritis of the knee or hip. After arthroplasty, a majority of patients experience improved quality of life and decrease in pain and limitations. In addition, patients predominately show improvements in coordination, muscle strength, balance, and proprioception, contributing to higher mobility and better endurance.[126] Unfortunately, this is not always the case. For example, patients with total knee arthroplasty (TKA) exhibit greater functional limitations, such as lower walking speeds and compromised ability to climb stairs, compared to individuals with no knee pathology.[127]

After undergoing arthroplasty, patients are expected to return to some kind of exercise between 3 and 6 months.[128] Although higher levels of activity are associated with postoperative satisfaction, those activity levels can also affect prosthesis failure. Pathology reports have confirmed the negative adverse effects of physical activity and body weight on the wear and tear of the implant. Postoperative activity is mainly determined by preoperative fitness, body mass index (BMI), and age.[129]

Scientific studies confirm the following recommendations after total hip and knee arthroplasty: high-impact activities should be discouraged since they lead to a higher risk of prosthesis failure. Providers should encourage patients to engage in lower-impact activities as appropriate to activity level. Examples of high-impact activities to be avoided are: rock climbing; snowboarding; singles tennis; volleyball; soccer; squash; racquetball; jogging; gymnastics; hockey; waterskiing; martial arts; softball; football; basketball; and high-impact aerobics. Examples of lower-impact activities to be recommended are: canoeing; doubles tennis; golf; swimming; hiking; speed walking; normal walking; low-impact aerobics; ballroom dancing; road cycling; stationary cycling; bowling; and using elliptical machines, treadmill, weight machines, and stair climber machines.[130–136]

Exercise, Social Isolation, and Loneliness

According to the *2017 Profile of Older Americans* report by the Administration for Community Living's Administration on Aging of the USDHHS, approximately 28% of older adults, or 13.8 million people, live alone.[137] Burgeoning scientific studies have provided consistent evidence that the quality and quantity of social relationships are major factors in broad-based morbidity and mortality.[138–141] The scientific literature has documented a link between social isolation/loneliness and several physiological and mental conditions, including: heart disease, obesity, weakened immune system, anxiety, depression, Alzheimer's disease, cognitive decline, high blood pressure, poorer physical and mental health, cognitive impairment, and greater risk of

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Table 7.4a Exercise prescription and common chronic conditions[a]

Chronic conditions and exercise benefits	Exercise prescription guidelines
Arthritis/ musculoskeletal pain (includes back pain) • Pain reduction and physical function improvement.[85]	• Land- or aquatic-based physical activity • Aerobic training 3–5 times per week and resistance training 2–3 times per week • Aerobic exercises (e.g., walking or cycling), lower-extremity strengthening exercises, tai chi, yoga,[86] and aquatic exercises (e.g., aerobics, strength training performed in a therapeutic/heated indoor pool) can all alleviate pain and improve function in patients with osteoarthritis[87]
Cardiovascular disease (includes peripheral vascular disease and heart failure) • Improves vascularization, possibly decreasing artery stiffness and improving blood flow.[31]	• For peripheral vascular occlusive disease, patients may need to exercise to the limits of pain tolerance each session to extend time to claudication[87] • Stress testing and cardiology evaluation is recommended before vigorous exercise training in persons with known cardiovascular disease[87,88]
Diabetes mellitus • Improves insulin resistance and glycemic control.[48.49,90] • Reduces physical impairment.[87] • Enhances weight control.[87]	• Aerobic training at least 3 days per week with no more than 2 consecutive days between bouts of activity (i.e., 150 minutes per week of moderate to vigorous activities), and • Resistance training at moderate to vigorous intensity at least twice per week on nonconsecutive days.[91] • If the individual is using insulin or insulin secretagogues, decrease the medication doses before, during, and after exercise, and/or ingest carbohydrates if pre-exercise blood glucose levels are less than 100 mg per dL (5.6 mmol per L).[87]
Mental health, includes Insomnia • Improves self-sleep quality, increasing sleep duration up to 1.25 hours.[42] • Improves depression and decreases the severity of symptoms.[92]	• Standard exercise guidelines for older adults (see Table 7.1).
Osteoporosis • Preserves and improves bone loss.[93,94] • Reduces the risk of falls.[88,93,94]	• Most evidence supports resistance exercises; some evidence supports high-intensity and high-impact aerobic exercises.[87] • Add balance training to prevent falls for those with severe osteoporosis, who should avoid forward flexion exercises, using heavy weights, or side-bending exercises, because pushing, pulling, lifting, and bending exert compressive forces on the spine that may lead to fracture.[95] • Individuals with previous vertebral fractures are at risk of further vertebral fractures. It may be beneficial to consult with physical medicine and rehabilitation physicians.[87]
Falls • With balance and strength training, reduces falls and fall-related injuries.[96,97] • Reduces fear of falling.[96]	• For patients at risk of falls, balance exercises are recommended for fall prevention.[88] • Additional effective fall prevention programs are multifaceted interventions that include gait training and strength training, tai chi, Otago Exercise Program, and Stepping On.[87,98] • Patients that have problems with balance or demonstrate frailty may need to be enrolled in an observed physical therapy program and/or encouraged to be as physically active as their abilities or conditions allow.[27]
Obesity • Improves physical function.[47] • Modest weight loss.[47] • Improves quality of life.[21,29,47]	• Aerobic exercises contribute to energy expenditure to induce caloric deficit.[87] • Resistance exercises are recommended during weight-loss period to help maintain lean muscle and bone mineral density.[87]

Table 7.4b Exercise prescription and common chronic conditions continued[a]

Chronic conditions and exercise benefits	Exercise prescription guidelines
Cognitive issues • Improves cognitive function.[99] • Reduces progression to dementia.[41]	• Standard exercise guidelines for older adults (see Table 7.1), and/or tai chi.[100]
Sarcopenia and frailty syndrome • Improves muscle mass and strength.[55,101] • Improves physical function.[55,101]	• *Aerobic exercise:* Moderate to vigorous activity enough to raise the pulse rate to 70–80% of the maximum heart rate. Activity performed for a minimum of 20–30 minutes at least 3 days per week.[102] • *Resistance exercise:* The progressive resistance program should involve all major muscle groups of the upper and lower extremities and trunk. One set of 8 to 10 different exercises, with 10 to 15 repetitions per set, performed 2–3 nonconsecutive days per week. Moderate-/high-intensity training is recommended, in which moderate intensity is 5 or 6 on a 0 to 10 scale.[102] • *Flexibility and balance exercise:* Stretching to the point of tightness and holding the position for a few seconds. Flexibility activities are performed on all days that aerobic or muscle-strengthening activity is performed. Balance training exercise 2–3 times per week.[102]
Pulmonary diseases • Improves cardiorespiratory fitness.[31] • Decreased dyspnea and improvement in respiratory muscle function.[103]	• Exercise training is part of the pulmonary rehabilitation program (usually 6– 12 weeks) for patients with chronic obstructive pulmonary disease.[87] • Exercise sessions should be timed to coincide with bronchodilator medication peak; use oxygen during exercise as needed.[87]

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Table 7.5 Commonly prescribed medications and possible impact on exercise performance[a]

Medication	Exercise performance	Explanations
Nonselective β-blockers	Reduce tolerance to exercise, increase predisposition to hyperthermia, exacerbation of exercise-induced bronchospasm or asthma, impairment of left ventricular function during exercise, reduction in β-2-stimulated glycogenesis, and reduction of VO2 max.[27]	Nonselective β-blockers such as propranolol can reduce tolerance to exercise by causing reduction in β-2-stimulated glycogenesis, producing earlier fatigue and lactate threshold. They can also increase predisposition to hyperthermia during exercise and can potentially exacerbate exercise-induced bronchospasm or asthma.[108]
Thiazides	Muscle cramping, arrhythmias, rhabdomyolysis, hypokalemia.[27]	Thiazides can produce urinary loss of potassium and magnesium, increasing the risk of muscle cramping, arrhythmias, and rhabdomyolysis, especially during warm weather. Converting enzyme inhibitors, calcium channel blockers, α-blockers, and central α-agonists are medications with the least potential for negative effects on exercise performance.[109]
Statins	Fatigue, joint pain, decrease muscle strength, myalgia.[27]	Statins may induce muscle weakness, increase fatigue, and alter energy metabolism during exercise.[110] Statins may also contribute to myopathy in several ways, including reducing cholesterol for sarcolemma, endoplasmic reticular, and membrane stability, decreasing levels of COQ10, which are required for mitochondrial respiration and energy production in the muscle, and by reducing the activity of certain genes in charge of muscle repair.[111]

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Table 7.5 (cont.)

Medication	Exercise performance	Explanations
Metformin	Tachycardia during exercise, increase levels of lactate.[27]	Metformin can increase heart rate and lactate concentrations during exercise. However, there is new evidence in animal models that metformin was able to enhance mitochondrial respiration in skeletal muscle after just 2 weeks of treatment, and thus improve exercise performance.[112]
Quinolones	Tendinitis, tendon ruptures.[27]	Quinolones and steroids increase the risk of tendinitis and tendon ruptures.[113]
Steroids	Tendon ruptures.[27]	
PPIs	Muscle cramps, muscle weakness, arrhythmias and lethargy.	Proton pump inhibitors (PPIs), when used over long periods, have been recognized to cause muscle cramps, muscle weakness, arrhythmias and lethargy, mainly due to hypomagnesemia.[114]
Antihistamines	Reduce reaction time and visual discrimination.	Antihistamines and selective serotonin reuptake inhibitors (SSRIs) can increase reaction time and decrease visual discrimination, thereby increasing the risk for falls in older adults.[116,117] (Further studies are needed to determine the directionality of causation with greater evidentiary probity regarding the documented association between SSRIs and falls.[118])
SSRIs	Increased risk for falls, rhabdomyolysis.[115]	
NSAIDs	Reduce inflammation associated with exercise and improved strength recovery (only short term: less than 1 month).[27]	Nonsteroidal anti-inflammatory drugs (NSAIDs) have been shown to reduce the inflammation associated with exercise and improve strength recovery. Nevertheless, the side effects associated with NSAIDs in older adults (GI bleeding and renal insufficiency) restrict their use in the long term (>1 month).[119,120]

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Table 7.6 Common injuries among older adults

Diagnosis	Cause	Clinical presentation
Arthritis	Trauma, overuse	Pain and limitation
Calcific tendinitis	Accumulation of hydroxyapatite crystals	Joint destruction Bouts of synovial inflammation (pseudogout)
Adhesive capsulitis	Unknown	Stiffness of joints at the extremes of movement
Chronic tendinitis	Decreased amounts of lubricin, decreased collagen turnover, increased collagen cross-linking, reduction of water solubility and water content, micro tears, decreased ability for tensile loads, fiber separation	Insidious onset of pain
Muscle injuries	Decreased muscle fibers, denervation, decreased mitochondria volume, changes in type of muscle fibers, increased collagen content	Decreased ability to adapt to high levels of loading Muscle tears, strains Weakness

falls.[142–148] Engaging in exercise in places where there is a potential for social interaction and promotion of stronger social networks, such as sports clubs, gyms, and senior centers, provides the health benefits associated with increased social connections and friendships beyond the benefits of the exercise itself. Participation in a fitness

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program can reduce isolation and loneliness and thereby provide indirect health benefits.[149]

Exercise in the Age of COVID-19

According to the Centers for Disease Control (CDC), older adults are at higher risk of getting severely ill or dying from coronavirus disease 2019 (COVID-19).[150] Eighty percent of COVID-19 deaths reported in the USA have been in adults aged 65 and older.[150] As a result, older adults may be concerned about engaging in physical activities that might heighten their risk of potential exposure to COVID-19. Older adults should check the CDC website regularly for updates on best preventive practices. Nevertheless, they should also be encouraged to not forgo regular exercise but to modify their exercise practices in order to implement best preventive practices while retaining the substantial benefits of regular exercise. Some basic recommendations include:

1. *Do exercise at home or outdoors*. Based on current evidence, the risk of transmission outdoors is very low relative to indoor settings. One study showed that of 1,245 cases in China, only two were confirmed to have been transmitted in an outdoor setting.[151]
2. *Avoid group activities, such as team sports*. Limiting close contact with others is key to reducing the spread of COVID-19.[152]
3. Keep safe social distancing, even in outdoor settings (stay 6 feet apart).[152]
4. Cover your mouth and nose with a cloth face covering when around others, even in outdoor settings.[153]
5. Wash hands with soap and water, or with hand sanitizer that contains at least 60% alcohol, upon reentering home. After you have been in a public place and touched a public item or surface, do not touch your eyes, nose, or mouth before washing your hands.[154]

Summary

Current levels of physical inactivity and sedentary behavior constitute a global public health burden. The prevalence of chronic disease among older adults is not merely a burden for these patients, but for the health-care system too.[11] Physicians should see this as an opportunity and a challenge to more widely and more consistently integrate physical activity counseling, prescription, and referrals into patient care plans in primary care settings.[27,28]

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Chapter 8

Frailty

Amy S. Klein and Mindy J. Fain

Introduction

Frailty is considered fundamental to geriatrics. A common impression is that the thin, stooped, and slow-moving older adult represents normal aging. However, decades of research have illuminated the differences between normal aging and frailty. Frailty is a geriatric syndrome that is both common and commonly missed by health professionals, and it increases the risk of a wide range of adverse health outcomes and other geriatric conditions. Frailty is characterized by diminished physiologic reserves and function, leading to decreased capacity to withstand stressors.[1,2] More than a third of people older than 85 are estimated to be frail,[1,3] and frail adults are at higher risk of dependency, institutionalization, and death.[4] The recognition of frailty, especially in its early stages, offers the possibility of preventing or mitigating adverse clinical outcomes. However, several issues have limited the advancement of frailty research and translation into practice, including the lack of consensus regarding the definition of frailty, the proliferation of assessment tools, and the gaps in validated best practice guidance for frail patients. This chapter will provide an overview of the frailty syndrome.

Definition

Frailty is a clinical state of increased age-related vulnerability for developing adverse health outcomes when exposed to stressors, and is not normal aging.[2] In this state an older person experiences increased and accelerated declines in reserves that can trigger disproportionate changes in health status from even relatively minor stressors. These cumulative declines in reserves can lead to increased risk of disability, dependence, and accelerated trajectory to death.[5] Frailty is characterized by diminished strength and endurance as well as impaired function within interrelated physiologic, nutritional, cognitive, and psychosocial dimensions. Although advanced age, multimorbidity, and disability are associated with frailty, there is strong evidence that frailty is a distinct entity.[1] It is a dynamic condition along a clinical spectrum that can improve or worsen over time. Although there is some conceptual overlap with physical resilience, frailty is not considered to be the opposite of physical resilience.[6]

Two main constructs have developed to better characterize frailty: (1) a phenotype model and (2) an accumulation of deficits model.[7] However, there is no consensus for a single operational definition of frailty,[8] and the numerous scales and tests developed include combinations of nutritional status, physical activity, mobility, energy, strength, cognition, mood, and social relations and support. Both models are associated with adverse health outcomes, and most operational definitions fall into two approaches. In addition, a biopsychosocial model has recently been described.

The Phenotype Model

The phenotype model, described by Fried, Walston, and colleagues, conceptualizes frailty as a clinical syndrome that is biologically driven.[1] Age-related molecular and biologic changes are the underpinnings of changes that lead to the syndrome of frailty, which leads to associated adverse health outcomes. Based upon a secondary analysis of data from the Cardiovascular Health Study (CHS), the frail phenotype consists of at least three of the following five criteria: weight loss, exhaustion, weakness, slowness, and reduced physical activity. This definition was validated in a community-dwelling population from the Women's Health and Aging Studies.[9] It is the most commonly used model in research, and it correlates with increased risk of adverse outcomes in a variety of settings. It can be challenging for clinicians to use this model because its components are not routinely evaluated in geriatric assessments,[10] including the measurement of gait speed, which requires mobility.

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The Deficit Model

The deficit model, described by Rockwood, Mitnitski, and colleagues, is drawn from the Canadian Study of Health and Aging, and is based on risk accumulation.[7] In this model, frailty is based on accumulation of unrelated disease states, physical impairments, cognitive decline, and psychosocial risk factors. A frailty index score is calculated based on the number of deficits found in relation to the total number of measures used, where the greater the number of deficits, the higher the frailty index score. Disability and underlying diseases are the underpinning of frailty rather than caused by the frailty syndrome. The frailty index is a more sensitive predictor of adverse health outcomes than the phenotype model and is strongly associated with vulnerability to adverse health outcomes. However, it is not easily used in clinical settings because of its complexity and the difficulty in accessing all necessary data. A more accessible scale based on clinical judgment has increased the use of this model in clinical practice.

The Biopsychosocial Model

A third frailty construct, the biopsychosocial model, has emerged in the last decade.[11] This model considers a person's socioeconomic context in understanding vulnerability. It expands the definition of frailty beyond the physical and clinical domains, and brings in wider determinants of health, including health inequalities in older age, that shape the conditions of everyday life.

Screening Tools

Frailty screening tools have been used to assess risk, to test model hypotheses, and to evaluate interventions. There are dozens of frailty scales in use based upon the proposed constructs, and they differ widely in their content validity, feasibility in clinical settings, and predictive ability. Because many of these tools were developed for specific populations and/or settings, results vary across instruments. It is important, therefore, to understand the specific intention of the tools in order to select tools that are appropriate within particular clinical context. For example, there is an inherent tradeoff between achieving the most accurate risk prediction versus selecting a tool that allows for the best timing and targeting of an intervention.[2,12] The Fried Frailty Index, the Clinical Frailty Scale (CFS), and the FRAIL scale are short and simple, although each varies in its ease of integration in routine clinical practice. The Fried Frailty Index (as shown in Table 8.1[13]) is based on the phenotype model of frailty and includes an interview and objective measurements such as grip strength and walking speed. The CFS, derived from the deficit model, is a pictorial measure of frailty based on clinical judgment. The FRAIL scale (using questions on fatigue, resistance, aerobics, illness, and loss of weight) utilizes findings from the African-American Health cohort and incorporates functional, deficit, and biological models in an interview-based tool.[14,15] Other scales such as the Palliative Performance Scale or the Karnofsky Performance scale allow patients to be classified based on their functional impairment, and can correlate with prognosis, but are not designed to screen for or correlate with frailty.[16]

Table 8.1 Cardiovascular Health Study (CHS) index – Fried criteria[1]

Frail = 3 of the following findings present *Pre-frail* = 1 or 2 of the following findings present
Weight loss (≥5% of body weight in the last year)
Exhaustion (positive response to questions regarding effort required for activity)
Weakness (decreased grip strength)
Slow walking speed (>6–7 sec to walk 15 ft)
Decreased physical activity. Males expending <383 kcal/week and females <270 kcal/week in physical activity. (For reference: walking 4 mi in 1 hr = 300 kcal)

Epidemiology

Frailty is a recognized risk factor for older adults. The overall prevalence of frailty in the United States in community-dwelling adults aged 65 or older ranges from 7% to 12%. Similar findings are reported for older adults in Europe and Latin America. In 2013, a systematic literature review examined the association of frailty with survival in community-dwelling older adults.[17] For studies using the phenotype model, the pooled prevalence in adults aged 65 and older was 14%, and studies using the deficit model demonstrated a pooled prevalence of 24%. Although the prevalence of frailty in long-term care settings has not been examined in depth, it is higher than in community-dwellers, carrying much higher mortality rates.

Prevalence by Age, Race/Ethnicity, and Gender

The prevalence of frailty increases with age and stratifies within subgroups. Using the phenotype definition, the prevalence of frailty for 65- to 70-year-olds ranges from

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3% to 6% and increases to 5% to 15% using the deficit definition. For those over age 80, the prevalence of frailty is more than 16% for either definition used. For the phenotype definition, prevalence increased from 16% for those aged 80–85 to 26% in those aged 85 and over. Using the deficit accumulation model, more than half of people over the age of 85 are frail.[17] The different operational definitions of frailty may identify different groups of older adults.

The prevalence of frailty differs among ethnic and racial groups, and it is important to employ ethnically sensitive measures. African Americans have a high prevalence of frailty. According to two studies, and using both accumulation and phenotype definitions, more than half of older African Americans were frail.[18,19] The prevalence of frailty in Hispanic older adults ranged from 8% to 20%, depending upon the scale used.[20] Among Caucasians, the prevalence of frailty ranged from 6% to 12% using the phenotype definition, and from 15% to 40% using the deficit accumulation criteria.[17]

Frailty appears to be higher among women than among men. In older women, the prevalence of frailty defined by phenotype is 13%, and defined by deficit accumulation it is 26%. For older men, the prevalence by phenotype is 7%, rising to 24% using the deficit definition. The prevalence of frailty is the highest amongst African-American women over the age of 85, at 60%.[1,17]

Association with Survival

Frailty is associated with increased mortality in both men and women.[17] When comparing frail with non-frail older adults, the relative increase in mortality risk averages 50% when using the phenotype definition and 15% when using the deficit definition. In a study of the course of disability in the last year of life, frailty was the most common condition leading to death.[21]

Association with Comorbidities and Disability

Comorbidity is defined by having two or more chronic conditions. Disability refers to chronic limitations or dependence in activities of daily living, instrumental activities of daily living, and/or mobility. The phenotype model considers disability to be a frailty outcome, where the deficit model includes measures of disability and comorbidity as contributors to frailty.

Frailty is associated with multiple comorbidities, including an increased rate of cognitive decline.[22] Frail older adults are found to be at higher risk of cognitive impairment and cognitive disorders, especially vascular dementia.[23] A systematic review and meta-analysis found that seven out of ten frail adults present with multimorbidity, and that multimorbidity increases the likelihood of being frail almost twofold.[24] Frailty is related to heart disease (44.5% prevalence),[25] malnourished status (68% prevalence),[26] depression (40% prevalence in older adults),[27] and cancer (42% prevalence in older adults with cancer).[28] The recognition of sarcopenic obesity is important because of the increasing prevalence of obesity in older adults; approximately 20% of US adults over the age of 65 are obese, and obesity is associated with impaired physical functioning and frailty.[29] Frailty is associated with diabetes, and individuals with diabetes have a 40% increased risk of developing frailty.[30]

Pathogenesis

The phenotype model postulates that the clinical manifestations of frailty are due to an interrelated and self-perpetuating cycle of negative energy balance, generalized weakness, diminished strength, reduced exertional tolerance, and sarcopenia. The underlying molecular, cellular, physiological, and functional changes of frailty are likely impacted by genetic and environmental factors, in combination with epigenetic mechanisms (see Figure 8.1). The deficit accumulation model assumes that aging involves the progressive accumulation of subcellular deficits and conditions that become clinically apparent as frailty when they reach a "tipping point." Further investigation into the biological basis of frailty is a key step toward developing interventions to prevent or mitigate frailty.

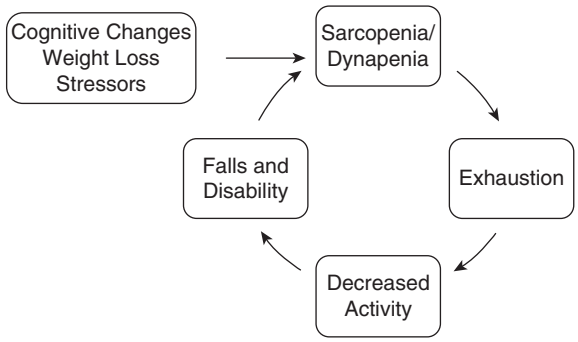

Figure 8.1 The frailty cycle.

Source: Adapted from elder care provider sheet, "Frailty – Elders at Risk," http://aging.arizona.edu/sites/default/files/frailty.pdf

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Natural History of Frailty

The concept of the frailty cycle (Figure 8.1) raises the question as to whether an individual can enter at any point, or if there is an ordered pattern of development of signs and symptoms. Longitudinal studies indicate some hierarchical order in how adults develop frailty, despite heterogeneity in initial symptoms.[31] Differing patterns may represent pathways stemming from varying physiologic dysregulation. Clarifying the common patterns of clinical onset could lead to earlier identification and intervention.

Transitions across frailty stages (e.g., non-frail, pre-frail, frail) are of clinical and research interest. In a longitudinal study of community-living older adults,[32] more than half had at least one transition, and nearly a quarter had two transitions, although improvement from frail to non-frail was extremely unlikely.

Immune System

Abnormal inflammation appears to have a major role in the development of frailty, and an accumulation of proinflammatory responses is one of the fundamental findings in frailty syndrome.[33] These responses are unlike typical acute inflammatory responses; rather, they are characterized by high levels of proinflammatory cytokines.[34] An expanding number of proinflammatory cytokines has been independently associated with the frailty syndrome, including interleukin 6,[35] C-reactive protein (CRP), and tumor necrosis factor-alpha (TNF-α). Since inflammation is associated with catabolism of skeletal muscle and is involved in several core features of frailty including malnutrition, anorexia, sarcopenia, weight loss, and cognitive changes, assigning inflammation a major causative role in frailty seems attractive. However, the elevation of proinflammatory cytokines as a root cause of frailty is not well understood. There is an association between frailty and several clotting markers (factor VIII, fibrinogen, and D-dimer) as well as evidence that immune system activation may generate the clotting cascade. Lastly, white blood cell and monocyte counts are elevated in frail older adults living in the community, which provides further evidence for immune activation.[36]

Endocrine Pathways

Aging is associated with changes in the hypothalamopituitary axis, which have an impact on metabolism and energy through several hormones. There is a decrease in growth hormone synthesis, which results in a reduction in insulin-like growth factor-1 (IGF-1). IGF-1 is involved in anabolic activity, specifically skeletal muscle strength. There are reductions in estradiol and testosterone secretion, reduction in the activity of adrenocortical cells that produce the sex steroid precursors such as adrenal dehydroepiandrosterone sulfate (DHEA-S), and a slow rise in cortisol levels. The impact of these changes in IGF-1, sex hormones, steroid precursors, and cortisol secretion, and their association with frailty, is not well understood. Although levels of IGF-1 are significantly lower in frail than non-frail older women,[37] trials of IGF-1 supplementation in older adults have not shown benefit. There is mixed evidence that lower levels of reproductive hormones are linked to frailty.[38,39] Similarly, the link between frailty and elevated cortisol concentrations that was demonstrated in one cross-sectional study is under investigation.[40,41]

Musculoskeletal System

With aging there is a delicate balance between muscle formation and loss coordinated by hormonal and immune systems, the brain, nutritional factors, and physical activity.[37] Usual age-related musculoskeletal changes include decline in skeletal muscle fibers, satellite cells, and neuromuscular junctions, infiltration by fibrous and adipose tissue, and an increase in apoptosis. There is evidence for alterations in mitochondrial function and the renin–angiotensin axis affecting muscle quality. The physiologic changes in frailty, including inflammatory cytokines, lower levels of growth hormone and sex steroids, and higher levels of cortisol, accelerate the decline in muscle mass and strength.

Sarcopenia is the progressive and generalized disease of muscle that is defined by the accelerated loss of muscle mass and strength, and impaired physical performance. Sarcopenia can be acute or chronic, and it is an important physiologic contributor to frailty.[42,43] It begins between the ages of 20 and 30, accelerates after age 50, and is a major cause of disability, poor quality of life, and death. Skeletal muscle mass measurements alone do not capture functionality, and thus do not define sarcopenia. The societal and economic costs of sarcopenia are significant.[44]

Clinical Applications

Clinicians encounter frail individuals in a wide variety of stages and settings. Some may be able to recover after a stressful event, and others may never regain full

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function. An older patient may appear vigorous yet be unable to tolerate the stress of elective surgery. An individual may experience a seemingly minor insult, such as a mild infection or a new medication, and decline rapidly, transitioning from independent to dependent or from ambulatory to immobile.

Failing to recognize frailty in a patient has potential to expose them to invasive or unrealistic interventions that are unlikely to offer much benefit. Likewise, incorrectly identifying a patient as frail could limit appropriate and beneficial interventions, resulting in denial of elective surgery purely for age. Especially in early stages, frailty is frequently overlooked. A routine and systematic approach to frailty assessment is recommended to help identify patients earlier who are at higher risk or more likely to benefit from treatments or procedures. Routinely evaluating for frailty could help limit potential iatrogenic complications. Despite these potential benefits, it has been challenging to incorporate frailty assessment into clinical practice.

Common Related Signs and Symptoms

Muscle weakness, or dynapenia, is a frequent complaint among older adults and a common symptom of frailty. It is strongly associated with impaired function, disability, and increased risk for future mobility limitations. A recent national consensus project found a relationship between low muscle mass and slower gait speed as well as higher likelihood of serious falls.[45] However, studies into the relationships among weakness, muscle mass, muscle quality, and function have demonstrated that, although low muscle mass is associated with weakness, low muscle mass alone does not cause disability. Sarcopenia often contributes to frailty, but not all patients with sarcopenia are frail.[46]

Cachexia is associated with prominent muscle wasting and may be confused with sarcopenia. Cachexia is a severe wasting condition accompanying underlying illness such as cancer, characterized by loss of muscle with or without loss of fat mass. It is frequently associated with inflammation, anorexia, and breakdown of muscles, and is distinct from starvation, malabsorption, and age-related loss of muscle mass.

Malnutrition occurs in approximately 5% of older community-dwellers, 35% of hospitalized older adults, and 50% of patients in rehabilitation settings. It implies a mechanism of protein-energy status imbalance and should be applied to conditions that clearly respond to feeding. The nutritional needs of older adults are determined by multiple factors including underlying diseases and their activity level. Older individuals are at greater risk for undernutrition and involuntary weight loss. Older adults are less able to adapt to periods of low food intake that can occur during hospitalization, secondary to illness, or from poor socioeconomic conditions. Older adults may also have limitations in their ability to access, prepare, and easily digest meals. The six-question Mini-Nutritional Assessment – Short Form includes domains related to mobility, psychological stress, acute disease, and ability to eat and is easy to administer and well validated.

Clinical Decision-Making

Identification of frailty has potential to be used as a core component to guide clinical care and aid with decision-making. Frail patients may fail to launch an adequate immune response to the influenza and pneumococcal vaccines.[47,48] Frailty risk assessment is important in caring for older patients with cardiovascular conditions; it predicts increased morbidity and mortality.[49] Screening is recommended by the American College of Surgeons, as frailty independently predicts postoperative complications, length of stay, and need for transition to a skilled or assisted living facility in older adults undergoing surgery.[50] A comprehensive frailty score has been shown to be more predictive for adverse postoperative outcomes in older surgical patients than traditional assessment tools.[51] For geriatric trauma patients, the Trauma Specific Frailty Index has been shown to be an independent predictor of in-hospital complications and adverse discharge disposition, and superior to age for clinical risk stratification.[52] In a study of ICU patients, frailty was associated with increased risk of extubation failure, discharge to long-term care, tracheostomy, and hospital death.[53] Post-ICU frailty is associated with incident disability, high 6-month mortality, and high burden of emotional and physical distress in older ICU survivors.[54]

Palliative Care

Frailty can be a key identifier of older adults who could benefit from more targeted conversations about their medical care preferences. Frail older adults are more susceptible than their non-frail counterparts to developing dependence and disability, and experience increased side-effect burden and worsening health outcomes for the same disease processes or stressors.[55] More than half of older cancer patients have pre-frailty or frailty,

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and are at increased risk of chemotherapy intolerance, postoperative complications, and mortality.[56] A study of pre-frail and frail older cancer patients receiving chemotherapy found them more likely to develop grade 3+ chemotherapy toxicity, discontinue the drug, and be hospitalized.[57]

Evidence exists that older adults with frailty experience pain and emotional distress at frequencies similar to people with cancer.[58] Frail patients also experience a higher occurrence of shortness of breath, drowsiness, fatigue, and weakness than their non-frail counterparts.

Compared to others, people with frailty are more likely to prefer less invasive or intense treatments.[58] One study found that, after initiating frailty screening prior to surgery, palliative care consultations increased and were associated with a 180-day mortality reduction of 33%. It also found an increase in patients who did not undergo surgery, suggesting that implementation of frailty screening may have prevented unnecessary operations in patients who may not have benefited from or did not want surgery.[59]

Frailty is a dynamic syndrome, and it is important to continue discussing goals and supportive care over time. Goals that were realistic before an illness or other stressor may become unlikely to achieve, as the frail older adult may be unable to rebound.

Interventions

Goals for interventions are targeted at reducing prevalence, slowing progression, and decreasing the severity of frailty. However, there remain no available curative treatments for frailty. As frailty likely has a complex etiology, multidomain trials that include combinations of interventions, such as exercise and nutrition, have been explored.

Physical Activity and Exercise

Exercise is currently the most effective intervention to improve function and quality of life among frail elders. Demonstrable benefits of exercise in older adults include increased muscle strength, decreased falls, enhanced gait and mobility, reduced inflammation, enhanced cognition, and improved well-being. Several studies have demonstrated that even the most frail adults benefit from physical activity that includes resistance training, aerobic activity, and focus on flexibility and balance.[60–62] A randomized clinical trial in obese older adults comparing the independent and combined effects of weight loss and exercise demonstrated that a combination program resulted in the greatest improvement in physical function.[29]

The Health, Aging, and Body Composition observational study demonstrated that participation in self-selected exercise could both prevent the onset of frailty and delay its progression.[63] A randomized clinical trial of a home-based physical activity program for frail elders helped reduce progression of functional decline among those with moderate frailty, but not with severe frailty.[64] A systematic review demonstrated that long-term multicomponent exercise interventions performed several times per week for 30–45 minutes may be helpful for the management of frail elders.[61] There is growing evidence that frail adults in a wide variety of settings can improve their functional performance, activity of living performance, and quality of life by regular exercise training, but more high-quality trials are still needed.[65]

Nutritional Intervention

Despite the possibility that several core components of frailty, including weight loss and weakness, may respond to nutritional intervention, evidence supporting this approach is scarce. According to a Cochrane review, nutritional supplementation with extra protein and energy produced a small but consistent weight gain in older people and a small reduction in mortality for undernourished elders, yet yielded no improvement in function.[66] While there is some indication that high-protein supplements may counteract the catabolic effects of disease and improve recovery from illness, strong evidence for their use in frail elders has not been demonstrated.[67] For frail elders, facilitating access to food, optimizing food preparation, and encouraging socialization at meals can be helpful in improving nutritional status. As food is frequently symbolic in many cultures as a source of life, patients and their families frequently struggle as an older adult loses their appetite, causing conflict.[68]

Appetite stimulants and micronutrient supplements are not recommended. While appetite stimulants may be beneficial for malnutrition, the adverse side effects associated with them can be potentially more harmful and include risk of blood clots, cirrhosis, hypotension, and delirium.[69]

There is growing evidence that a healthy diet may mitigate the onset of frailty.[70,71] A systematic review highlighted an association between diet quality and incidence of frailty,[72] but more studies are needed to further define the potential role of nutrition in the prevention and treatment of frailty.

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Hormonal Intervention

Various hormonal therapies have been proposed, but little evidence exists to support their use. Testosterone improves muscle mass by increasing protein synthesis and muscle strength. Although there may be a role for low-dose testosterone in combination with nutritional supplementation in frail older men who are hypogonadal, testosterone therapy brings significant systemic side effects and is not recommended in patients who are not hypogonadal.[73] Vitamin D prescription for elderly people who are deficient might reduce the number of falls. Vitamin D supplements along with calcium can improve muscle strength and balance.[74] Despite the evidence that low vitamin D levels are strongly associated with frailty,[75] the benefits of vitamin D to treat frailty in nondeficient elders have not been demonstrated.[2] Growth hormone therapy results in an increase in muscle mass in normal elderly, but there is no increase in strength unless exercise is added to the intervention. Since there is no demonstrated clinical benefit, and the short- and long-term safety is not known, supplementation with growth hormone is not recommended.[76]

Other Pharmacological Approaches

Polypharmacy contributes to frailty, and avoidance of inappropriate medications is highly recommended. Several guidelines are helpful in reducing unnecessary or potentially harmful medications in this vulnerable group.[77] Other areas of investigation include angiotensin-converting enzyme inhibitors because of their impact on the structure and function of skeletal muscle.[78] Although inflammation seems to be a key component of frailty, the use of systemic anti-inflammatory agents carries significant adverse effects.[4]

Comprehensive Geriatric Evaluation and Specialized Clinical Programs

Comprehensive geriatric evaluation by a skilled interprofessional team is commonly considered to be the gold standard for developing and implementing a plan of care that is consistent with the patient's goals, values, and preferences. A targeted approach to prevent the range of biological, socioeconomic, and environmental stressors that could potentiate frailty and thus improve clinical outcomes is appealing. Evidence for improved outcomes especially among frail elders, however, is still emerging.

Clinical programs targeted to the frail elderly have been shown to improve clinical outcomes in a variety of settings.[40] Frail patients cared for in specialized inpatient units are more likely to return to their homes, less likely to suffer functional or cognitive decline, and have lower mortality rates than usual ward care.[79] A study examined the impact of frailty on rehabilitation outcomes in a geriatric evaluation and management unit. All patients experienced functional improvement, with the frailest patients experiencing greater improvement than those who were less frail.[80]

Resilience

Aging successfully frequently depends on one's ability to respond to life's inevitable stressors. Resilience is defined as one's ability to withstand functional decline or recover from stressors. Study into the development of resilience is likely important for older adults, as enhancing physical resilience could potentially help to limit susceptibility to stressors. However, resilience and resistance to stressors is likely important across the lifespan, and is not the opposite of frailty.[6] There is little evidence of how resilience relates to frailty or if it can prevent the development of frailty, and further research is needed.

Conclusion

Frailty is an important geriatric syndrome associated with high morbidity and mortality. Prevention, early diagnosis, and management across the spectrum of frailty represent crucial areas in the clinical care of older adults, and our understanding of frailty is still evolving. Early risk assessment, implementation of exercise and nutritional support programs, and access to interprofessional geriatric assessment and management represent the most effective ways to improve health outcomes. However, more research is needed, including further understanding of the underlying mechanisms of frailty that may open doors to prevention and treatment strategies.[34] Objective, validated, and easily administered assessment tools including biomarkers are sorely needed, although there is a general consensus that there is likely no single biomarker or measurement tool adequate to fully capture frailty risk. Frailty is positioned to become a major public health issue because of its large burden, major impact on individuals and society, and potential for public health intervention in the future. High-value team-based models of care linked with community resources are required to help improve quality of life for this rapidly growing and vulnerable population.

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Chapter 9

Fall Prevention in Community-Dwelling Older Adults

Levan Atanelov

Key Messages

- Falls epidemiology and impact
  - Falls are common and deadly for older adults
  - Fall risk factors can be classified as intrinsic and extrinsic
  - Use acute trigger versus predisposing condition for determining cause of falls
  - Functional deficits comprise the common denominator for many fall risk factors
  - Biomechanical deficits in gait and balance are the primary culprit
- Assessment for fall prevention
  - A fall risk assessment should be performed annually and with change in health status
  - Individuals at risk of falls should undergo comprehensive medical and biomechanical assessment
  - Know the common patterns of gait and causes of falls
- Interventions to enhance gait and prevent falls
  - An individualized and multidisciplinary approach is necessary for fall prevention
  - Know the current literature on fall prevention

Epidemiology and Impact

Incidence

Current estimates of annual rate of falls in older adults (65 years old and older) in the United States are alarming: one in three to one in four older adults experience a fall.[1,2] Fall rates increase with age: 25% of older adults <75 years old report at least one fall a year vs. 30% of those in the 85+ age group.[2,3] These numbers underestimate the actual incidence of falls, as roughly half of older adults do not report falls to their physicians.[4]

Mortality

Falls are the number one cause of unintentional death in older adults.[3] The fall-related death rate in those 75 and older tripled from 2000 to 2016.[5] Fall-related deaths increase with age, with those aged ≥85 years at particularly high risk.[6]

Morbidity

Falls are the number one cause of nonfatal injury in older adults, accounting for almost 3 million emergency visits and under a million hospitalizations annually.[3] Approximately 10% of falls in those in the 65–75 age group cause injuries of any type.[2] One in five falls causes major trauma, e.g., bone fractures or brain trauma.[7,8] Fall-related injuries increase with age: those in the 85+ age group have almost double the rate of fall-related injuries compared to those in the 65–69 age group.[3] Over 95% of hip fractures are attributed to falls[9] and falls are the most common cause of traumatic brain injuries.[10] Over half of rhabdomyolysis cases present after a fall, commonly after a patient is found down and unable to get up for a prolonged time period.[11] Following a fall, fear of falls develops in up to a third of older adults, resulting in reduced overall activity and quality of life.[12] Falls are three times more common than coronary artery events.[3,13] Falls resulting in hip fracture are responsible for an all-cause annual mortality rate of 21.2%, comparable to that of myocardial infarction.[14,15]

Fall Risk Factors

Demographic and Socioeconomic Risk Factors

Fall risk is increased in women compared to men, as well as in select minorities, those who are widowed/divorced, or those who come from a lower income or educational level.[2,3]

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Functional Risk Factors

Falls are attributed to both intrinsic and extrinsic risk factors. Extrinsic factors include environmental hazards like a curb or a "throw-rug." Though 20%[16] to 59%[17] of falls were attributed to trips and slips, modifying environmental hazards as a single intervention would not necessarily decrease the rate of falls.[16,18] Intrinsic factors include impaired vision, somatosensory abnormalities, foot problems, and mobility deficits. The majority of falls occurred at night; 55% took place in the bedroom or adjoining bathroom, and 67% occurred during ambulation.[18] Commonly reported causes of falls are slips, trips, and loss of balance.[19] Factors associated with falls include impaired health, poor balance, impaired activities of daily living (ADLs), the need for community support, use of psychoactive medications, and polypharmacy (i.e., use of four or more medications).[19]

Medical Comorbidities

Several age-related medical comorbidities are associated with falls. Falls are eight times more common in patients with dementia.[20] Other medical conditions associated with falls and/or gait/balance deficits include frailty,[21] sarcopenia,[22] chronic obstructive pulmonary disease (COPD),[23] knee osteoarthritis,[24] cancer,[25] diabetes,[26] spinal stenosis,[27] Parkinson's disease,[28] and stroke.[29]

Syncope versus "Mechanical Falls"

Although syncope and presyncope explain a significant number of falls and loss-of-balance episodes, 62% of falls are deemed to be "mechanical" in nature. "Mechanical" falls are commonly implied to be less likely due to syncope and more likely due to associated environmental factors.[30] We recommend using the term "nonsyncopal falls" instead.

Acute Trigger versus Predisposing Condition for Falls

We recommend differentiating between the acute trigger of the fall (often extrinsic) and the predisposing health condition(s) that put the patient at risk of falls. For instance, a patient with diabetic peripheral neuropathy causing decreased somatosensory input and diabetic retinopathy causing impaired vision may trip over a stair and fall. The stair is the acute trigger; neuropathy and retinopathy are the predisposing conditions. A patient with Parkinson's disease and benign prostate hypertrophy starts an alpha-blocker and falls. The alpha-blocker is the trigger; autonomic dysfunction, common in Parkinsonian patients, is the predisposing condition. Similarly, a patient with premorbid cognitive and somatosensory deficits develops a urinary tract infection (UTI), becomes confused, and falls. The urinary tract infection is the acute trigger; the cognitive and somatosensory impairments are the predisposing conditions.

Patients often attribute a fall to an external impediment. It is intuitive to see that the impediment or trigger did not "cause" the falls. For example, if you ask the patient who believes that they fell three times in a year because of the stairs: "Did you fall three times a year by tripping on stairs when you were a teenager?" the answer is usually "No." What has changed is that now the patient suffers from impaired somatosensory and visual input, predisposing them to trip and fall on the stairs.

Understanding and specifying the role of acute triggers and chronic predisposing factors in falls is more useful than labeling a fall as "mechanical." The term "mechanical falls" is too often misleading since it implies that environmental barriers – that is, the triggers – are the primary cause of falls. This approach detracts from identifying the underlying chronic predisposing factors contributing to the fall, and is of limited clinical utility for guiding patient management or predicting patient outcomes.[30]

Functional Risk Factors as Final Common Pathway

The list of different geriatric conditions associated with falls is long (see partial list above). We recommend focusing on a few key functional deficits that form the final common pathway for many of these fall risk factors.[31] Functional deficits making up the final common pathway for different medical conditions often comprise most of the chronic predisposing fall risk factors. Predisposing factors include gait and balance deficits, impaired vision, orthostatic hypotension, and cognitive, ADL, and instrumental ADL impairments. Gait and balance problems predominate among fall risk factors.[31]

Clinical Assessment for Fall Prevention

Screening

Annual screening for falls is recommended by the American Geriatrics Society,[32] since older adults rarely volunteer that they have suffered a fall.[33]

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The Centers for Disease Control and Prevention (CDC) has developed an algorithmic approach for identifying, risk-stratifying, and managing older adults at risk of falls: STopping Elderly Accidents, Deaths, and Injuries (STEADI).[34] The STEADI algorithm (a) screens patients for risk of falls: "Do you feel unsteady when standing or walking? Are you worried about falling? Have you fallen in the past year?"; (b) stratifies patients by their risk of falls, e.g., those with one traumatic fall or two nontraumatic falls a year with gait and balance deficits (e.g., Timed Get Up and Go test score of at least 12 seconds screens positive for future falls); and (c) recommends a multifactorial assessing for balance and gait deficits, vision deficits, polypharmacy, somatosensory deficits, foot and ankle problems, and vitamin D deficiency. Although the STEADI strategy implemented in the primary care setting reduced fall-related hospitalizations in a large study, its conclusion should be interpreted with caution because of methodological limitations.[35]

Since falls are common and cause high mortality and morbidity, but can be prevented, we recommend including a fall risk assessment and treatment plan in every progress note: e.g., "Fall risk low, never had falls, no new falls, on last exam scored well on functional balance and gait tests," or "Fall risk high, three falls a month despite physical therapy, increased rate from one fall a year last year, no fractures/brain trauma, neuro ruled out movement disorder, appear to be not-syncopal, scheduled to see fall prevention specialist."

History

To better understand the key etiology of falls, we recommend understanding the biomechanical and situational context associated with falls and loss-of-balance episodes. For instance, a patient with diabetic peripheral neuropathy may present with either balance or gait deficits, both functional chronic predisposing fall risk factors.[36] A history of consistently losing balance during standing rather than ambulation is more consistent with balance deficits as the cause of falls, even though the patient may have decreased gait speed and stride length. In this situation, an acute fall risk may be present when stretching to reach for the phone while standing at the sink washing dishes. Below we present a sample history-taking template by adapting the mnemonic "OLDCARTS," remembering that many patients do not report falls as a "Chief Complaint."[33]

Onset: Insidious or was there an inciting event?

Location: Where do you primarily fall/lose balance: indoors or outdoors? In which rooms?

Duration: When did you start falling/losing balance?

Characteristics:

Frequency: How many times did you fall/lose balance in the past 3 months? In the past year?

Direction of falls: forward (usually tripping over a barrier; may cause upper-limb fracture), sideways (often while standing or losing balance during ambulation; may cause hip fracture), or backwards (usually the most dangerous as may cause head trauma). Vestibular-related falls/loss-of-balance episodes may occur with transfers and cause backwards or sideways momentum. Falls due to Parkinsonian disease spectrum may present as backwards falls. For instance, pre-frail older adults appear to fall more often while in transit, while frail older adults fall more commonly while standing and in indoor public places.[37] Fall direction is a better predictor of fractures than bone strength.[38] Tripping over an object and/or stepping down usually result in forward falls, while slower ambulatory gait speed is a risk factor for slips causing sideways or backwards falls with impact near the hip.[39]

Aggravating factors:

Acute triggers: Ask about head turns causing dizziness, tripping over curbs/stairs, losing balance, slipping, new medications, change in health condition, standing up from a seated/lying-down position, dizziness, "passing out," environmental hazards such as throw rugs, loose electric cords, slippery areas, poor lighting, dysuria, cough, or acute pain.

Chronic predisposing conditions: Screen for depression and cognitive impairment; motor, balance, vision, and sensory deficits; trouble walking without an assistive device; trouble with activities of daily living and instrumental activities of daily living; medications; and chronic functional impairments due to medical conditions.

Associated mobility and/or balance condition: during ambulation, during transfers, during quiet standing.

Relieving factors: getting rest, standing up slowly from the bed, not rushing to the bathroom, well-lit room, cane, walker, wheelchair, ankle foot orthosis (AFO), assistance with mobility and ADLs, etc.

Therapeutics tried: medication adjustment or discontinued, durable medical equipment, physical therapy, and cardiac and neurological interventions.

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Severity: trauma, fractures, bruising, head trauma? Facial trauma with no evidence of upper-limb trauma is often a sign of a syncopal fall, as often patients with non-syncopal falls tend to at least attempt to catch themselves and protect the face.

Screening: screen for long bone fracture, head trauma, mood disorders, dementia, osteoporosis, blood clots, arrhythmias, spinal surgeries, spinal stenosis, and chronic pain. Screen substance use disorders and sexually transmitted diseases (e.g., neurosyphilis). Screen for polypharmacy and medications associated with orthostatic and/or psychoactive profile, anticoagulants and drugs associated with osteoporosis (e.g., steroids, phenytoin).

Physical Exam Components

Usually a gait and balance evaluation comprises the key element of a physical exam in this patient population, but comprehensive physical evaluation is often indicated. There are several well-validated tools used to assess for risk of falls based primarily on balance and gait exam findings.[40,41] Upon clinical evaluation we recommend starting with observing transfers from chair, static and dynamic balance, and gait evaluation.

Balance Examination

Background

Postural control (being able to maintain desired upright position) is not accomplished by one static "balance system." Balance is maintained by the interaction of multiple systems, including (a) cognitive processing (especially attention), (b) control of balance during gait (e.g., increased lateral sway during ambulation), (c) movement strategies to regain equilibrium (with stepping and hip strategy to recover equilibrium more common in older adults vs. ankle strategy in younger individuals), (d) biomechanical constraints (ankle range of motion, foot pain), (e) sensory strategies (proprioception, visual and vestibular system integration with the central nervous system), and (f) orientation in space (e.g., "pusher syndrome" in some strokes causes patient to internally perceive an upright stance to be at diagonal instead of vertical position).[42] Implications of the multisystem control of balance are twofold: (1) Simple global measures of balance (e.g., 40, 41) are insufficient for diagnosing the particular faulty component of the balance system; (2) Impairments in one component of the balance system can be compensated by other components and only become apparent in certain conditions (e.g., an individual with neuropathy can compensate well with visual information unless they are placed in a dark environment). Comprehensive evaluation by a clinician trained in systematically assessing balance disorders is optimal.[42]

Exam Findings

A simple clinically useful test is to ask a patient to stand on each foot with hands at the waist without leaning on a chair or table for up to 5–6 seconds. Balance deficits can be due to (1) leg weakness, e.g., due to stroke hemiparesis or knee osteoarthritis; (2) proprioceptive deficits due to diabetic peripheral neuropathy or posterior column damage in spinal stenosis; (3) vestibular deficits; or (4) musculoskeletal abnormalities, e.g., hamstring contractures or leg-length discrepancy. Leg muscle proprioceptive signals provide the primary source of information for postural control, and that there is an age-related decrease in sensitivity, acuity, and integration of the proprioception signals.[43] Clinically, this means that physical exams should also include tests of proprioception.

Patients with vestibular deficits may present with loss of balance or vertigo upon lateral or horizontal head turns while seated or ambulatory. They may also have findings of nystagmus on static or dynamic gaze examination. A common vestibular problem, benign paroxysmal positional vertigo, can be diagnosed with the Dix-Hallpike Test and is often well treated with the Epley maneuver.

Gait Examination

Background

The Einstein Aging Study identified abnormal gait patterns in 35% of the patients aged 70 years or older, 50% of which were neurological: ataxic/unsteady (swaying and losing balance during ambulation, turning, or tandem walking), hemiparetic (e.g., circumduction gait), frontal (short strides, wide base, and poor foot lift), and Parkinsonian (en bloc turns, festination, stooped posture, absent arm swing, shuffling). The other 50% were non-neurological, with 85% of these due to orthopedic impairments, 10% cardiac, and 5% pulmonary.[44]

Physical Exam Elements

Vital signs: check for fever and orthostatic vital signs.

Functional exam: balance and gait evaluation as above.

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Psych: screening for depression is important, as decreased mental processing speed and activity avoidance in the context of depression have been associated with falls.[31]

Neuro: cognitive evaluation should focus on dual tasking and executive function, not memory.[45] Any deficits in light-touch or proprioception sensation should be noted. Reflexes and joint range of motion should be assessed for hyperreflexia and spasticity (upper motor neuro signs). Brief cerebellar examination for rapid alternating and repeating movements, as well as finger-to-nose test, is appropriate. Assessment for movement disorder-associated findings including asymmetrical tremors, en bloc turning, shuffling gait, bradykinesia, and masked facies may be indicated.

Eyes: visual assessment for reduced contrast sensitivity and depth perception is more important than visual acuity.[46] Screening for cataracts is indicated, as first cataract surgery is shown to reduce falls.[47] Examining a patient's eyeglasses is helpful; those with multifocal lenses can benefit from switching to long-distance single-lens glasses for outdoor mobility.[48] Finally, a visual acuity exam can still be helpful, since those with corrected visual acuity of 20/80 in the better eye (significantly impaired) benefit from occupational therapy evaluation of home safety.[49]

Cardiac: assessment for orthostatic hypotension is commonly recommended, though data from adequately powered randomized controlled studies showing benefits of medication adjustment is lacking.[31] Check for arrhythmias or signs of valve stenosis that may contribute to risk of syncope.

Pulmonary: patient may present with hypoxia causing pre-syncopal episodes at times; check for signs of chronic obstructive lung disease, assess pulse-ox with ambulation if clinically indicated (e.g., if patient reports syncopal/pre-syncopal episodes with fatigue after ambulation).

Musculoskeletal: check for joint range of motion, muscle bulk (e.g., for sarcopenia or signs of denervation, e.g., thenar muscles), manual-muscle testing including weakness for ankle dorsiflexion, which may cause foot drop. Seventy-two percent of participants in a study of footwear and falls were wearing footwear that did not fit correctly on both feet, 90% had shoes with smooth, partly worn, or fully worn sole treads, and 67% reported wearing slippers at home (a fall risk).[50] Foot and footwear examination should assess for foot pain, decreased sensation, foot deformities, and decreased range of motion, as well as for footwear and brace as indicated.[51] Check for joint pain, which may affect weight-bearing during balancing or gaiting tasks.

Skin: check for skin lesions on feet that may be impairing ability to bear weight.

Durable Medical Equipment evaluation: evaluate AFO functionality if applicable. Cane or walker should be roughly at the height of the greater trochanteric bursa for most people.

Fall Prevention Interventions

Multifactorial management for preventing falls in older adults has repeatedly shown to be effective in systematic reviews.[31,47,55,56] The best evidence favors balance, gait, and strength-training for reducing falls in older adults.

Exercise as a single intervention can prevent falls in community-dwelling older people.[57] Overall, exercise reduced the rate of falls in community-dwelling older people by 21%, with greater effects seen from exercise programs that challenged balance and involved more than 3 hours/week of exercise.[58] Moderate- to high-challenge balance training with cumulative duration of over 50 hours that did not include walking programs[58] and those with strengthening programs were shown to be effective.[59] Exercise alone was shown to be effective for patients with Parkinson's disease or those with cognitive impairment, but not those with stroke or those who resided at residential care settings or were recently discharged from the hospital.[60] Notably, combined exercise and cognitive training improved balance in patients with mild cognitive impairment.[61]

Visual interventions may include first-eye cataract surgery[47] and single-lens glasses for outdoor ambulators using bifocals.[48]

Foot-related interventions include recommendations for using anti-slip shoes,[47] wearing low heels with firm, slip-resistant soles indoors and outdoors,[62] avoiding walking barefoot or only with socks,[63] and wearing high-collared shoes with openings at the ankle to possibly improve the sense of position and stability.[64]

Patients with a history of dementia and/or depression should ideally be treated non-pharmacologically when possible.[31] Studies showed that those with dementia taking cholinesterase inhibitors had an increased risk of syncope with no decrease in risk of falls.[65] Unfortunately, cognitive behavioral therapy did not show a decrease in falls, but resulted in fewer people experiencing multiple falls.[66]

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Environmental modification interventions led by an occupational therapist are best suited for the highest-risk groups, i.e., those with at least one fall in the past year, using a mobility aid, needing assistance with ADLs, taking psychoactive medications, or concerned about falling.[67]

Medication review and elimination of those with high risk of falls and low margin of benefit is recommended. However, medication management alone may have limited impact on reducing falls.[52,53] In one study resulting in a reduction in falls after psychotropic medication withdrawal, almost half of those in the medication-withdrawal arm opted to resume the medication 1 month after conclusion of the trial, limiting the long-term impact.[54]

Vitamin D supplementation in the absence of an indication for supplementation has not been shown to result in a reduction in falls.[68] Patients with a history of osteoporotic fractures or at risk of osteoporosis should be given treatment for osteoporosis to help reduce risk of fractures.[69] Hip protectors did not show reduction in hip fractures in community-dwelling older adults.[70]

Patients taking anticoagulants were at low risk of traumatic intracranial hemorrhage from ground-level low-impact falls.[71] Antiplatelet agents appear to have a higher risk of brain bleeds compared to anticoagulants.[72]

Novel Approaches for Fall Prevention

Whole-body vibration exercise reduces the risk of falls.[73] Community-based (e.g., at senior centers) fall prevention programs usually focus on education and exercise. These programs are part of the current CDC recommendations to help prevent falls.[34] Roughly half of senior centers nationally offer evidence-supported, community-based fall prevention programs (e.g., Stepping On, an educational and exercise program), while roughly a third don't offer any.[74] STEADI recommends utilizing these community-based programs to help reduce falls in older adults.[41]

There has been an emergence of electronic means to help monitor and prevent falls. These technologies may be utilized in the future to help track patients at risk of falls and detect their increased risk before they start falling. Though there are promising studies about wearable sensors and "smart" homes electronically detecting patient movement patterns, the accuracy of the technology is still lacking for reliable clinical consumption.[75]

Fall Prevention Across the Continuum of Care

Hospital-based fall prevention programs. Typical hospital-based fall prevention interventions published included a multicomponent approach integrating fall risk assessments, visual risk alerts, patient education, care rounds, bed-exit alarms, and post-fall evaluations. Unfortunately, a systematic review on hospital-based fall prevention programs returned equivocal results, and no individual single intervention showed significant benefit. Later studies showed that providing patient-centered interventions in addition to tailored patient education may be effective in reducing falls in acute-care hospitals.[76–78]

Long-term and subacute facility-based fall prevention programs. The efficacy of fall prevention interventions is more promising in subacute and long-term care facilities. In care facilities, vitamin D supplementation was shown to reduce the rate of falls, which should of course only be provided for patients with low vitamin D. Multifactorial interventions in subacute facilities reduced falls in recurrent fallers but not in first-time fallers. Exercise interventions that included balance training and were combined with tailored other interventions improved the risk of falls in subacute facilities.[79–81]

Fall prevention with home health care. Geriatric-based care together with the Otago Exercise program comprising strength- and balance-training components and delivered at patients' homes by a physical therapist prevents falls compared to usual geriatric care alone.[82]

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Chapter 10

Evaluation and Management of Dizziness

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Introduction

Dizziness is a common symptom of the elderly. Patients may use a variety of terms such as "woozy," "lightheaded," "off-balance," or "spinning" to describe the phenomenon of dizziness, which is sometimes used interchangeably with vertigo. However, dizziness, which is characterized by a distorted sense of spatial orientation, lacks the illusion of self-motion that is a feature of vertigo.[1,2] Either dizziness or vertigo may present as a primary complaint. Alternatively, they may accompany another primary symptom such as chest pain, dyspnea, or nausea.

Spatial orientation is governed by multiple sensory inputs from and affecting a wide array of physiological systems, including vestibular, visual, cerebellar, motor, and sensory systems. Dizziness (spatial disorientation without a false sense of self-motion) tends to occur when distorted input to the vestibular system is relatively symmetrical, while vertigo often occurs when the input is relatively asymmetrical. As a result, whether an individual experiences dizziness or vertigo is less a function of the cause for the spatial disorientation than the balance of inputs to the vestibular system. Furthermore, because the sensation of dizziness is necessarily subjective, individuals may describe the experience of dizziness resulting from similar mechanisms in different terms, particularly if they are from different cultures, different native/preferred languages, or have cognitive impairment.[3] For the purpose of this chapter, "dizziness" will denote both dizziness and vertigo unless otherwise specified.

Presyncope and gait unsteadiness may evoke descriptions by patients that overlap with those used to describe dizziness or vertigo, or the symptoms may co-occur.[3] Syncope, which is a transient loss of consciousness due to transient global cerebral hypoperfusion, usually leads to a fall and full recovery of consciousness within 30 seconds. Presyncope is the sensation of impending faint without actual loss of consciousness. While many causes of presyncope overlap with causes of dizziness or vertigo, the presence of true syncope begets a differential diagnosis that usually differs from that seen with typical dizziness and vertigo. Gait unsteadiness or directional pulsion (a sense of falling or leaning in a particular direction) frequently accompany dizziness or vertigo sensations "in the head." However, these may present as more isolated vestibular symptoms (i.e., without dizziness or vertigo) in sensorimotor disorders (e.g., myelopathy causing sensory loss and weakness) or parkinsonism (e.g., retropulsion in progressive supranuclear palsy) that often do not produce sensations of dizziness or vertigo. Similarly, difficulties of standing or walking balance may occur as isolated features of musculoskeletal conditions.

Dizziness leads to more than 5 million primary care visits each year in the USA, disproportionately among the elderly,[4,5] and is one of the top three reported primary care symptoms.[6] Prevalence is age-dependent, ranging from about 30% at age 65 to 50% in those over 85.[5,7] In addition, dizziness is a major risk for falls and hip fractures.[8,9] Because dysfunction of multiple physiological systems (e.g., visual, cardiovascular, neurological) with input to and from the vestibular system is common in the elderly, the description of the experience of dizziness often varies by individual irrespective of the precipitating cause (e.g., postural hypotension, medication side effect, benign positional vertigo). Furthermore, many older adults have more than one condition that produces or predisposes to dizziness. For example, an older adult may have dizziness caused by impaired balance due to Parkinson's disease and have dizziness related to side effects of the drugs used to treat that condition. For this reason, some propose thinking of dizziness as a geriatric syndrome.[10] Although it may be useful to consider dizziness as a geriatric syndrome, still, the elderly, like younger patients, often have discrete causes for dizziness. When possible, it is important to accurately identify the underlying cause(s) to differentiate emergencies from less urgent causes, thus optimizing treatment and assigning prognosis.

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Classification

To diagnose the cause of dizziness, physicians have traditionally focused on asking the patient, "What do you mean by dizziness?"[11] However, research over the last decade has shown that characterizing the type of dizziness qualitatively is not sufficiently specific to identify the cause.[13] For one thing, patients often use qualitative descriptions inconsistently when describing an episode of dizziness.[3] Furthermore, cardiac arrhythmia, orthostatic hypotension, stroke, and panic attacks may cause frank vertigo, and dizziness without vertigo may occur with a full range of vestibular diseases.[3,14] Instead of determining the type (quality) of dizziness, the clinician should use the Triage – TiTrATE – Test method.[15] The first step, Triage, refers to identifying the most obvious, especially life-threatening, causes of dizziness through overt evidence such as abnormal vital signs, altered mental state, or clear exposures. Acute conditions would include such things as cardiovascular disease (e.g., acute hypertensive urgency, cardiac arrhythmia), infections (e.g., pneumonia, encephalitis), and metabolic disturbances (e.g., hypoglycemia). Somewhat less emergent, but no less important, are medication toxicity (e.g., antiseizure, benzodiazepines, tricyclic antidepressants, amiodarone), use of illicit drugs and alcohol, and psychiatric (e.g., panic attack) etiologies.

For those with not-so-obvious causes of dizziness, the second step is TiTrATE, which refers to Timing, Triggers And Targeted Exam. Timing characterizes the onset, continuity, and duration of symptoms. For example, dizziness may be discretely *episodic* without symptoms between episodes or *continuous* with or without fluctuations in intensity. Once timing is determined, the patient's vestibular syndrome can be categorized as an episodic vestibular syndrome (EVS – recurrent episodes typically lasting seconds, minutes, or hours), an acute vestibular syndrome (AVS – >24 hours in duration, usually monophasic), or chronic vestibular syndrome (CVS – weeks, months, or years). Distinguishing AVS from EVS from CVS allows the clinician to narrow the differential diagnoses substantially (Tables 10.1–10.3). Patients with EVS can be further classified by whether the episode was "triggered" (e.g., reproduction of dizziness when rolling over in bed due to otoconia floating within a semicircular canal) or "spontaneous" (e.g., a patient with abrupt onset unprovoked dizziness due to a transient ischemic attack [TIA]).

Triggers are maneuvers or actions (e.g., specific head movements, standing posture, or exercise) that initiate episodic dizziness. It is critically important to distinguish *triggers* from *provocative factors* – head movement that brings on dizziness from a baseline of no dizziness is a "trigger," while head movement that exacerbates dizziness from a baseline of spontaneous dizziness is a "provocative factor." The lack of a trigger (i.e., spontaneous onset) is also meaningful, and should raise concern for a cerebrovascular etiology when vascular risk factors are present. Associated symptoms, such as chest pain, weakness, headache, or hearing loss, can provide more specific clues to the underlying cause. Following a methodical history, the physician then performs a Targeted Exam. For example, in a patient with episodic dizziness consisting of recurrent, seconds-long vertigo attacks (Timing) brought on by rolling over in bed (Trigger), the Dix-Hallpike maneuver should be performed (Targeted Exam). If the Dix-Hallpike reproduces vertigo and brings on the expected pattern of upbeat-torsional nystagmus (top poles beating toward the lowermost or affected ear), benign paroxysmal positional vertigo (BPPV) is confirmed. For this patient, the Epley maneuver may be performed and no further diagnostic investigations are needed. When diagnostic uncertainty remains after the history and physical exam, and depending on clinical relevance and concern for dangerous causes such as stroke or TIA, further testing with brain and vascular imaging is indicated (Test).

> "What do you mean by dizziness?" should not be your first question. Focus on timing and triggers, rather than type.

However, when using the Triage – TiTrATE – Test method, it is important to remember that the elderly often experience more than one cause of dizziness, either concurrently or sequentially over time. Concurrent causes (e.g., simultaneous presence of vestibular disease, medication side effect, and balance disorder) may cause overlapping dizziness syndromes. For example, a patient with CVS (e.g., due to cerebellar degeneration) associated with unsteadiness that is always present when standing or walking may develop new "triggered" EVS due to BPPV. Or, an older adult who has experienced one cause of EVS in the past (e.g., BPPV) may present with a new cause for episodic symptoms that is qualitatively similar (producing vertigo) but is due to a new underlying mechanism such as postural hypotension, cardiac arrhythmia, or TIAs. While the diagnosis may be straightforward when associated symptoms are present (i.e., temporal relation to starting a new medication, obvious anemia, new neurological symptoms or signs), the diagnosis

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might be missed when associated symptoms are absent and attention is paid only to the qualitative similarity to the previous episode. Therefore, accurate assessment (and reassessment) of Timing and Triggers, heightened vigilance for associated symptoms (e.g., neurologic or cardiopulmonary), relationship to medication change, or illness is necessary. In the elderly population, identifying so-called benign causes (e.g., benign paroxysmal positional vertigo, self-limited drug reactions, mild volume depletion) remains important, since these disorders impair balance and can lead to dangerous falls or other harms. Likewise, mild chronic dizziness can be debilitating and lead to deconditioning if it prevents the patient from staying physically active. Therefore, any complaint of dizziness must be addressed comprehensively.

Intermittent, Brief Episodes of Dizziness Lasting Seconds to Hours

The EVS is the most common vestibular syndrome, and is characterized by intermittent, brief episodes of dizziness or vertigo lasting seconds to hours. Because symptoms typically resolve quickly, most patients seek evaluation in the ambulatory care setting.

Differential Diagnosis

The differential diagnosis of EVS is broad, and "timing" and "triggers" are most helpful for uncovering the etiology. BPPV and orthostatic hypotension are the most common etiologies, though this category includes a number of cardiac and central nervous system causes that are less common but must be considered (Table 10.1). Arising from a recumbent position will trigger both BPPV and orthostatic hypotension, though BPPV will also be triggered by lying back down and by turning while supine, while orthostatic hypotension should not occur when reclining or supine. Dangerous mimics of BPPV include central nervous system disease (e.g., posterior fossa tumors or stroke – both rare) and orthostatic hypotension due to bleeding, sepsis, or other acute disorders.[16]

If the history suggests that the symptoms are triggered, then the dizziness should be reproduced by provocation at the bedside. For positional symptoms that are brief and postural, this is done by measuring the orthostatic blood pressure and by performing provocative positional testing for BPPV. The Dix-Hallpike maneuver (Figure 10.1) tests for posterior canal (PC) otoconia, which is the most common cause of BPPV. A positive test produces mixed upbeat and torsional nystagmus with the top poles of the eyes beating toward the affected (tested or lowermost) ear. In horizontal canal (HC) BPPV, the Dix-Hallpike maneuver will usually trigger horizontal nystagmus that beats toward the ground (geotropic with right *or* left ear down) or, less frequently, toward the ceiling (apogeotropic with right *or* left ear down). However, the supine roll test (patient is supine, head is flexed 20–30 degrees and then turned – or the body and head are rolled together when cervical range of motion is poor – 90 degrees to the right and left) triggers more robust nystagmus and symptoms in HC BPPV. Patients who have geotropic nystagmus during Dix-Hallpike and supine roll almost always have HC BPPV. These patients can be referred for repositioning maneuvers or treated in the office (e.g., BBQ roll or Gufoni). Patients with apogeotropic nystagmus usually have HC BPPV, although some cases are due to a posterior fossa tumor, stroke, or cerebellar dysfunction. Repositioning maneuvers exist for the apogeotropic variant as well (e.g., Gufoni), but if the clinician is uncomfortable diagnosing or treating the patient, contrast-enhanced brain MRI and simultaneous referral to a vestibular physical therapist, otolaryngologist, or neurologist is reasonable. When downbeat nystagmus is seen during Dix-Hallpike, contrast-enhanced brain MRI and neurology referral are indicated (see

Table 10.1 Core differential diagnosis of episodic (duration of seconds to hours) dizziness or vertigo

Nonspontaneous (triggered)	Spontaneous
Position/ Posture: BPPV, orthostatic hypotension *Valsalva*: situational presyncope, inner ear fistula *Exertion*: valvular heart disease, myocardial ischemia or cardiac insufficiency	*Less urgent*: vestibular migraine, Ménière's disease, panic disorder, vasovagal presyncope *More urgent*: TIA, cardiac arrhythmia, hypoglycemia, pheochromocytoma, occult carbon monoxide exposure

Abbreviations: BPPV – benign paroxysmal positional vertigo; TIA – transient ischemic attack. Modified from Newman-Toker.[29]

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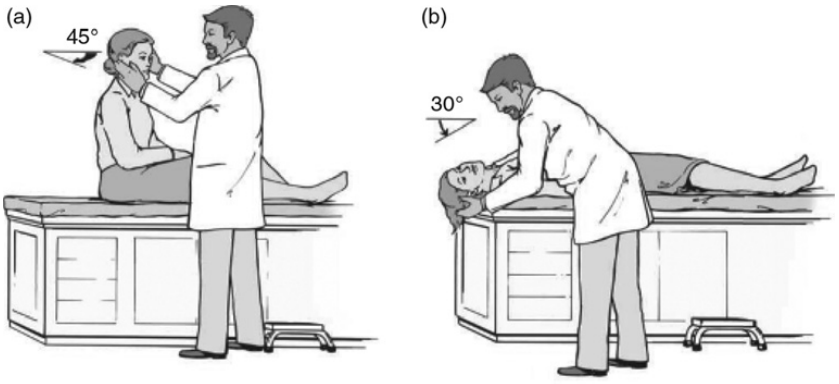

Figure 10.1 Dix-Hallpike maneuver (right ear). The patient is seated and positioned so that the head will extend over the top edge of the table when supine. The head is turned 45° toward the ear being tested (position [a]). The patient is quickly lowered into the supine position with the head extending about 30° below the horizontal (position [b]). The patient's head is held in this position, and the examiner observes the patient's eyes for nystagmus. In this case with the right side being tested, the physician should expect to see a fast-phase counter-clockwise nystagmus. To complete the maneuver, the patient is returned to the seated position (position [a]) and the eyes are observed for reversal nystagmus, in this case a fast-phase clockwise nystagmus.

Useful Websites at the end of this chapter for diagnosis and treatment of PC and HC BPPV).

In those with episodic symptoms, worry when spells are spontaneous or triggered by exertion. When symptoms are positional, do test maneuvers for BPPV.

Spontaneous episodes of dizziness (lacking a clear, reproducible trigger) may be vestibular in origin (i.e., EVS) or may be due to non-vestibular disorders including reflex presyncope or panic disorder. The diagnosis may be straightforward when the underlying condition is accompanied by typical features or accompanying symptoms, such as headache with vestibular migraine or fear and palpitations with panic disorder. In the absence of discriminating features, one must be careful to consider TIAs, cardiac arrhythmias without palpitations, and hypoglycemia. If chest pain, dyspnea, or syncope is associated with a spontaneous episode, then cardiovascular cause or pulmonary embolus is more likely than a neurological cause. Furthermore, it is important to note (and not widely recognized) that cardiac causes may produce spinning-type vertigo.[17] Similarly, a TIA may present with isolated dizziness or vertigo in the absence of other neurological symptoms or signs in over 50% of individuals with vertebrobasilar events before proceeding to major stroke.[16] Therefore, a high index of suspicion is necessary when encountering recurring, spontaneous EVS, particularly if the symptoms are new in the prior 6 months and the frequency of attacks is increasing.

As noted earlier, it is important to differentiate truly episodic symptoms (brief episodes separated by asymptomatic intervals) from dizziness that is persistent but exacerbated by intolerance for head movement or dizziness that is always present with standing or walking in a patient with unsteadiness.

Treatment

Patients with BPPV usually benefit from physical maneuvers to reposition canal otoconia (i.e., "canalith repositioning maneuvers") (Figure 10.2).[18] Dizziness typically lasts only seconds and becomes progressively less intense with each episode. Antihistamines are not helpful in BPPV and may produce side effects including drowsiness that increase the risk of falls. Vestibular migraine is probably more common than previously imagined;[19] treatment is generally by a combination of lifestyle modification and prophylactic medication.[20] However, new headaches and vertigo in an elderly patient are a red flag for vascular disease unless there is a strong and long history of migraine.

Acute, Continuous Episodes of Dizziness Lasting Days to Weeks

The acute vestibular syndrome is characterized by acute onset, continuous dizziness, or vertigo lasting days to weeks associated with spontaneous nystagmus, nausea or vomiting, intolerance to head movement, and gait unsteadiness. During an attack, most patients seek help in an emergency department or urgent care setting. Rarely, bouts of AVS may occur as part of a relapsing and remitting illness (usually multiple sclerosis), or result from sequential vestibular neuritis, either of which can cause chronic residual dizziness. Accurate assessment of the "timing" characteristic depends on recognition of *continuous* symptoms over days to weeks, unlike EVS, which consists of brief episodes lasting seconds to hours even if they *recur* over days or weeks. When symptoms are present at the time of

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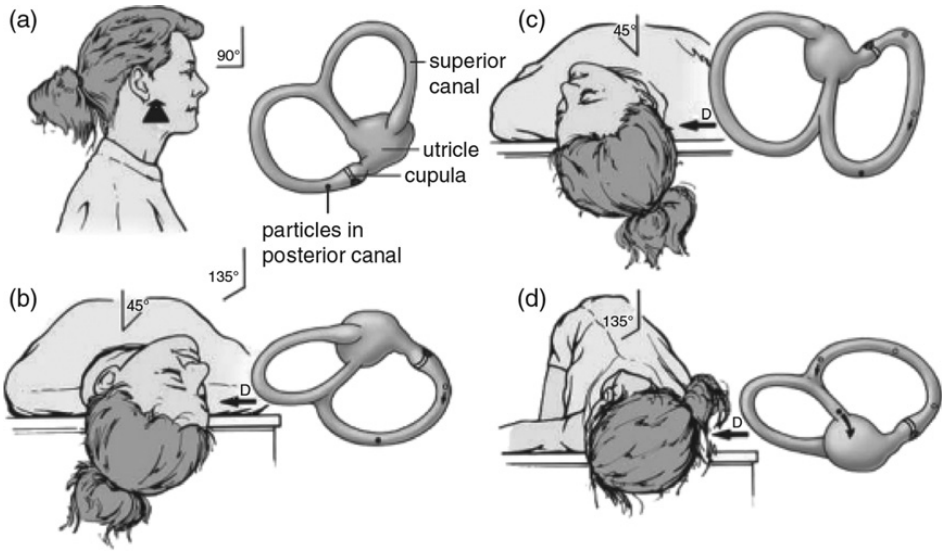

Figure 10.2 Particle repositioning maneuver (right ear): schema of patient and concurrent movement of posterior/superior semicircular canals and utricle.[30] The patient is seated on a table as viewed from the right side (a); (b)–(d) show the sequential head and body positions of a patient lying down as viewed from the top. Before moving the patient into position (b), turn the head 45° to the side being treated (in this case it would be the right side): patient is in normal Dix-Hallpike head-hanging position (b). Particles gravitate in an ampullofugal direction and induce utriculofugal cupular displacement and subsequent counter-clockwise rotatory nystagmus. This position is maintained for 1–2 minutes. The patient's head is then rotated toward the opposite side with the neck in full extension through position (c) and into position (d) in a steady motion by rolling the patient onto the opposite lateral side. The change from position (b) to (d) should take no longer than 3–5 seconds. Particles continue gravitating in an ampullofugal direction through the common crus into the utricle. The patient's eyes are immediately observed for nystagmus. Position (d) is maintained for another 1–2 minutes, and then the patient sits back up to position (a). D = direction of view of labyrinth, dark circle = position of particle conglomerate, open circle = previous position.
Source: Adapted from LS Parnes and J Robichaud. Further observations during the practice repositioning maneuver for benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg.* 1997; 116:238–243.

assessment, physical exam can be useful for localizing the cause (e.g., focal neurological deficits, otitis media, or herpes zoster rash of Ramsay Hunt syndrome). Likewise, the history may reveal a toxic or drug exposure (i.e., carbon monoxide or phenytoin) or head trauma. One must be careful not to prematurely ascribe AVS to a peripheral labyrinth disorder as a result of exposure to a recent viral upper-respiratory infection before appropriate evaluation has excluded dangerous causes such as stroke.

In acute, continuous dizziness, patients will feel worse when their head is moved, but this exacerbating feature has no diagnostic value. All AVS patients, whether due to stroke or vestibular neuritis, will feel worse when moved. Do not perform the Dix-Hallpike test in these patients.

Differential Diagnosis

When a patient presents with AVS it is critical to differentiate between urgent and less urgent causes. An abridged differential diagnosis is provided in Table 10.2. Head trauma and medication are the most common exposures causing AVS. When the onset of AVS is spontaneous (i.e., lacking an obvious antecedent exposure), then acute unilateral peripheral vestibular neuritis (dizziness or vertigo only) or labyrinthitis (dizziness or vertigo with hearing loss) is most common. Caution should be advised when diagnosing labyrinthitis in a patient with vascular risk factors and normal otoscopy – some of these patients have labyrinthine ischemia[21] that can go on to more extensive brainstem infarction.

Strokes (particularly of the posterior fossa) can mimic either presentation very closely. Note that focal neurologic signs and symptoms may be absent in ~80% of patients with stroke presenting with AVS.[22] Computed tomography (CT) has extremely low sensitivity (16%)[23] for acute ischemic stroke, so unless the patient has severe headache, lethargy, or hemiparesis (signs of cerebellar hemorrhage), CT should not be performed when imaging is required.[24] If imaging is

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Table 10.2 Core differential diagnosis of acute onset continuous (duration of days to weeks) dizziness or vertigo

Nonspontaneous (postexposure)	Spontaneous
Treatments: anticonvulsants, intra-tympanic gentamicin *Trauma*: acute traumatic brain injury, surgery (e.g., cochlear implantation) *Toxins*: chemicals (e.g., toluene), biotoxins	*Less urgent*: vestibular neuritis, labyrinthitis *More urgent*: stroke, cerebellar hemorrhage, bacterial mastoiditis, herpes zoster oticus (Ramsay Hunt syndrome), brainstem encephalitis, Wernicke's syndrome (thiamine deficiency)

Modified from Newman-Toker.[29]

Table 10.3 Tests to evaluate for stroke in acute vestibular syndrome

Test	Sensitivity for stroke
Focal findings on general neurologic exam	~19%[22]
Brain CT scan	~16%[23]
Brain MRI MRI-DWI (first 48 hours)	~80–85%[25]
HINTS to INFARCT	~99%*[25]

* The point estimate for sensitivity is 99% with 97% specificity in acute vestibular syndrome without hearing symptoms. Sensitivity is slightly lower (96%) when loss of hearing accompanies the AVS.[25]

Abbreviations: CT – computed tomography; HINTS to INFARCT – head impulse, nystagmus, test of skew; impulse normal, fast phase alternating, refixation on cover test; MRI-DWI – magnetic resonance imaging with diffusion-weighted images

needed, magnetic resonance imaging (MRI) with diffusion-weighted imaging (DWI) is the test of choice. MRI-DWI is the current gold-standard test when obtained from 72 hours to 7 days, but misses ~15–20% in the first 24–48 hours after onset of continuous symptoms.[25] When encountering a patient with isolated AVS, strong evidence indicates that a careful eye exam ("HINTS to INFARCT") outperforms MRI in ruling out stroke (Table 10.3).[25,26] The three-step ocular motor exam "HINTS" exam consists of the following: Head Impulse, Nystagmus, Test of Skew. The addition of a fourth step – a bedside assessment of auditory function – constitutes the "HINTS Plus" exam. The AVS due to posterior fossa stroke (brainstem and/or cerebellum) is very likely when the patient has *any* (i.e., one or more) of the following three dangerous eye movement signs: (1) bilaterally normal head impulse test (suggesting that the peripheral vestibulo-ocular reflexes are spared), (2) direction-changing nystagmus (e.g., right-beating nystagmus when looking right and left-beating nystagmus when looking left), or (3) abnormal test of skew – visible vertical ocular misalignment and refixations seen during alternate cover testing suggests a skew deviation in the AVS. Note that, paradoxically, a normal head impulse test is highly suggestive of a central etiology (normal, in the context of AVS, supports stroke). Additionally, acute hearing loss with normal otoscopy should be considered to be labyrinthine ischemia until proven otherwise, especially when vascular risk factors are present.

In contrast, (right) vestibular neuritis is likely when *all* of the following are present: (1) abnormal head impulse test to the right (suggesting that the ipsilateral peripheral vestibulo-ocular reflex is involved), (2) unidirectional (left-beating) nystagmus (stays left-beating regardless of the gaze position; increases in intensity when looking leftward; and decreases in intensity when looking rightward), (3) normal test of skew (no vertical movement with alternate cover testing), and (4) no acute unilateral or bilateral hearing loss (subjectively and with bedside testing using finger rub). The three-step HINTS exam has a sensitivity of 96.8% and specificity of 98.5% to detect a central etiology, while the four-step HINTS Plus exam has a sensitivity of 99.2% and specificity of 97% to detect a central etiology[21,27] (Table 10.5). Although this approach is now well validated in expert hands, it has not been tested when applied by non-specialists.

In acute, continuous dizziness, eye movement exams (HINTS) outperform our current gold standard, MRI, to look for ischemic stroke. New devices may eventually make it easier for primary care physicians to test these eye movements. Do not bother with CT unless a rare case of cerebellar hemorrhage is suspected.

Treatment

If the HINTS Plus exam is consistent with a benign or peripheral etiology of the AVS (i.e., vestibular neuritis), these patients can usually be sent home from the

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emergency department with medication to suppress nausea and dizziness (although using these medications for longer than three days or so can impede normal vestibular compensation). Frail older adults, by contrast, may require hospitalization to ensure that symptoms are controlled and hydration is maintained.

Chronic Dizziness Lasting from Months to Years

Many older adults have more chronic symptoms. This category of dizziness is usually related to a chronic, progressive neurological or vestibular condition with pathological findings that are present at the time of examination that often suggest the diagnosis. Symptoms may be stable or progressive over months to years, even though they may fluctuate in severity over time. Dizziness is often context-dependent, such as when the patient reports feeling dizzy only or always when walking. This should not be interpreted as "episodic" or as "triggered" by an upright posture. Because dizziness typically develops gradually with the progression of the underlying neurological or vestibular condition, patients most often seek evaluation in an outpatient setting.

In the elderly, causes of imbalance or unsteadiness include sensory peripheral neuropathy (e.g., diabetes, B12 and thyroid deficiency) or a gait disorder resulting from Parkinson's disease, other neurodegenerative disease, vascular parkinsonism, normal pressure hydrocephalus, or degenerative joint disease of the lower extremities with instability. A patient who experiences visual jumping or bouncing while walking following intravenous gentamicin therapy (or another vestibulo-toxic medication) almost certainly has bilateral vestibular loss, which is a peripheral cause of the chronic vestibular syndrome. A patient with a cerebellar neurodegenerative condition (e.g., spinocerebellar ataxia) may experience constant dizziness and imbalance, which is a central cause of the CVS. Other causes are listed in Table 10.4. A thorough assessment of the patient includes bedside examination of musculoskeletal function, and testing for upper motor neuron and extrapyramidal motor abnormalities, autonomic system dysfunction, peripheral sensory abnormalities, and vestibular, ocular motor, visual, and cerebellar impairment. Neuroimaging may be indicated to rule out treatable conditions such as normal pressure hydrocephalus, depending on the patient's general health and preferences.

Treatment

Chronic vestibular syndrome associated with gait unsteadiness may improve, depending on the cause, with physical conditioning and gait training, and with the use of an appropriate assistive device (e.g., cane, walker, or wheelchair). Correctable causes of dizziness may be uncovered by careful history and physical exam, and targeted laboratory tests. Medications, whether recently started or longstanding, are common causes of dizziness. Careful medication assessment and reduction should be considered whenever possible. Correcting electrolyte and metabolic abnormalities such as hyponatremia, hyperglycemia, hypothyroidism, and B12 deficiency may also lead to resolution or improvement.

Dizziness in the elderly is often multifactorial. Fix everything that is "fixable," emphasizing treatable neurologic and iatrogenic conditions (e.g., medication toxicity).

Useful Websites

American Academy of Neurology (Treatment Guidelines and Videos for BPPV Maneuvers)
www.aan.com/guidelines
www.neurology.org/content/70/22/2067/suppl/DC2

Dizziness and Balance (Timothy Hain)
www.dizziness-and-balance.com/index.html

Journal of Vestibular Research – Barany Society International Classification of Vestibular Disorders Page (consensus criteria and definitions for vestibular disorders)
www.jvr-web.org/Barany.html

Table 10.4 Core differential diagnosis of chronic (duration of months to years) dizziness or vertigo

Nonspontaneous (context-dependent)	Spontaneous
While walking: Parkinson's disease, multisensory dizziness *While head moving:* chronic uni- or bilateral vestibulopathy *While eyes open:* visual dizziness (e.g., diplopia, new glasses)	*Less urgent:* cerebellar degeneration, post-concussive dizziness, presbylibrium, PPPD *More urgent:* cerebellar tumor, hydrocephalus, metabolic deficiency (e.g., Wilson's, B12, E), autoimmune or paraneoplastic cerebellopathy

PPPD – persistent perceptual postural dizziness

Modified from Newman-Toker.[29]

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Table 10.5 The most common vestibular conditions categorized by timing and triggers, with specific ocular motor and vestibular features that should be sought for each

	Vestibular conditions to consider	Targeted ocular motor and vestibular exam
Acute vestibular syndrome (>24 hours)	1) Vestibular neuritis 2) Stroke (demyelination and other central etiologies less common) 3) Wernicke (Korsakoff) syndrome	1) HINTS+: HIT abnormal (see example – https://collections.lib.utah.edu/ark:/87278/s6x398q2) AND unidirectional nystagmus that obeys Alexander's law [see example – https://collections.lib.utah.edu/ark:/87278/s64205qx] AND skew deviation absent (see alternate cover testing at 1 and 3 minutes – https://collections.lib.utah.edu/ark:/87278/s6tn1htv) AND no acute hearing loss; peripheral pattern of HSN 2) HINTS+: HIT normal (see demonstration in a normal patient – https://collections.lib.utah.edu/ark:/87278/s63b97tz) OR gaze-evoked nystagmus (see example – https://collections.lib.utah.edu/ark:/87278/s6kh4n5k) OR skew deviation present (see example – https://collections.lib.utah.edu/ark:/87278/s6c0045t) OR acute hearing loss; look for central patterns of HSN (see example – https://collections.lib.utah.edu/ark:/87278/s61c5vkg) 3) Bilaterally abnormal HIT, spontaneous vertical (see example – https://collections.lib.utah.edu/ark:/87278/s6h74j6d) and gaze-evoked nystagmus are common, also 6th NP, ataxia
Episodic *spontaneous* vestibular syndrome*	1) TIA 2) Vestibular migraine 3) Ménière's (endolymphatic hydrops) 4) Vestibular paroxysmia	1) Usually symptoms have resolved and eye movement exam is normal; otherwise, may use HINTS** 2) Can see peripheral or central patterns of nystagmus (spontaneous, gaze-evoked, head-shaking-induced, positional) during the attack, often normal interictally; often spontaneous, but typical migraine triggers are common too 3) Nystagmus can be in excitatory or inhibitory patterns during the attack, often normal interictally 4) Hyperventilation-induced nystagmus (see demonstration in a normal patient – https://collections.lib.utah.edu/ark:/87278/s6pz98ht)
Episodic *triggered* vestibular syndrome	1) Benign paroxysmal positional vertigo (BPPV) 2) Central positional vertigo or nystagmus 3) Superior canal dehiscence syndrome (SCDS)	
Chronic vestibular syndrome	1) Bilateral vestibular loss (BVL) 2) Persistent postural positional dizziness (PPPD) 3) Cerebellar disease 4) Acoustic neuroma 5) Oculopalatal tremor	1) Bilaterally abnormal HIT (see example – https://collections.lib.utah.edu/ark:/87278/s62z4z8g), additional cerebellar signs can narrow differential and loss of 4 or more lines with dynamic visual acuity (see example of how to perform this maneuver – https://collections.lib.utah.edu/ark:/87278/s6tn19w8) 2) No characteristic ocular motor/vestibular findings 3) Flocculus/paraflocculus: Gaze-evoked nystagmus, spontaneous downbeat nystagmus, saccadic pursuit and VORS (when VOR is present), saccadic dysmetria (see example – https://collections.lib.utah.edu/ark:/87278/s6dj8q9h), alternating skew deviation (see example – https://collections.lib.utah.edu/ark:/87278/s6d83n91) while additional vestibular loss can narrow differential (e.g., CANVAS, see example – https://collections.lib.utah.edu/ark:/87278/s6s50fth) Nodulus/uvula: periodic alternating nystagmus (see example – https://collections.lib.utah.edu/ark:/87278/s62k013r) 4) Hyperventilation-induced nystagmus (see example – https://collections.lib.utah.edu/ark:/87278/s63f8cgs), and Bruns nystagmus (see example – https://collections.lib.utah.edu/ark:/87278/s60p4p3j) 5) Vertical or vertical-torsional pendular nystagmus with palatal tremor (see example – https://collections.lib.utah.edu/ark:/87278/s6mh1mnm)

HINTS+ = head impulse, nystagmus, test of skew, "plus" bedside assessment of auditory function; HIT = head impulse test; NP = nerve palsy; BPPV = benign paroxysmal positional vertigo; SCDS = superior canal dehiscence syndrome; BVL = bilateral vestibular loss; PPPD = persistent postural perceptual dizziness; CANVAS = cerebellar ataxia, neuropathy, vestibular areflexia syndrome

* If first attack of TIA, vestibular migraine or Ménière's, may be better described as the acute transient vestibular syndrome (<24 hours)

** HINTS has been studied in the acute vestibular syndrome, and should not be relied upon in the episodic or acute transient vestibular syndrome unless the patient remains symptomatic at the time of bedside assessment

Source: Neuro-Ophthalmology Virtual Education Library: NOVEL. https://collections.lib.utah.edu/ark:/87278/s6tr0d0h [Accessed May 2020].

Neuro-Ophthalmology Virtual Education Library (NOVEL) (Newman-Toker Collection)

http://novel.utah.edu/Newman-Toker

Neuro-Ophthalmology Virtual Education Library (NOVEL) (Gold Collection)

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Chapter 11

Evaluation and Management of Dementia

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Dementia is a clinical syndrome characterized by cognitive and functional symptoms that can be caused by a range of diseases or injuries to the brain. The *Diagnostic and Statistical Manual of Mental Disorders*, 5th edition (DSM-5) separates the relevant diagnostic criteria into major (dementia) (Table 11.1) and minor (mild) neurocognitive disorder.[3] Major neurocognitive disorder is characterized by significant decline in at least one cognitive domain (such as memory, learning, processing speed, attention, or executive functioning) leading to a significant worsening of function that interferes with activities of daily living (ADLs). Minor neurocognitive disorder, on the other hand, describes modest cognitive decline from a previous level of performance in one or more cognitive domains, not interfering with ADLs.

Additional terms have been used to refer to cognitive impairment not meeting criteria of dementia (Table 11.2). Cognitive impairment not dementia (CIND) is a clinical syndrome characterized by a measurable decline in memory or other areas of cognition, beyond what might be expected by age alone, with preserved day-to-day functioning and not meeting criteria for dementia. Mild cognitive impairment (MCI) is a clinical subset of CIND that includes cognitive symptoms and objectively impaired cognitive function relative to age and education.

Epidemiology

There are over 47 million persons with dementia worldwide, with the number expected to increase to 66 million by 2030 and 115 million by 2050.[39] At age 45, the estimated lifetime risk for dementia is 20% for women and 10% for men.[10] The costs of health care and long-term care for individuals with dementias in the United States in 2015 are estimated at $818 billion.[39] In the setting of an aging population, the financial impact of dementia is estimated to increase to more than $1.1 trillion in 2050.[2]

Risk Factors

A review of potentially modifiable risk factors for dementia found that 35% of dementia is attributable to a combination of the following risk factors: low education, midlife hypertension, midlife obesity, hearing loss, late-life depression, diabetes, physical inactivity, smoking, and social isolation.[39] Education is a widely accepted risk factor for dementia, with higher education associated with reduced dementia risk in a linear dose-response manner.[64] Increased engagement in social and intellectual pursuits has also been associated with decreased risk of cognitive decline.[41]

While modifiable risk factors are important in identifying prevention strategies, age remains the greatest risk factor for dementia. Overall, about 80% of dementias occur in persons aged 75 years or older.[48] A number of genetic variants play a role in the pathogenesis of dementia. As these pertain to specific types of dementia, they will be discussed in greater detail later in the chapter.

Evaluation Approach

While various diagnostic criteria exist for its etiologic subtypes (which will be discussed later in this chapter), dementia is a clinical syndrome defined solely on clinical grounds.[3] Thus, a thorough approach including a comprehensive history and clinical examination is crucial in the assessment of dementia.

The DSM-5 diagnostic criteria (Table 11.1) for major neurocognitive disorder, or dementia, specify four critical elements for the diagnosis. First, cognitive decline, defined as decline in mental processes used to obtain knowledge or to interact with the environment, must be present in one or multiple domains. To differentiate dementia from other causes of cognitive deficits such as intellectual disability, cognitive symptoms must represent a cognitive decline for the individual. The decline should be substantial enough to be of concern to the individual or observed by a knowledgeable informant or

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Table 11.1 DSM-5 criteria for major neurocognitive disorder

A. Evidence of significant cognitive decline from a previous level of performance in one or more cognitive domains (complex attention, executive function, learning and memory, language, perceptual-motor, or social cognition) based on:
   1. Concern of the individual, a knowledgeable informant, or the clinician that there has been a significant decline in cognitive function; and
   2. A substantial impairment in cognitive performance, preferably documented by standardized neuropsychological testing or, in its absence, another quantified clinical assessment.
B. The cognitive deficits interfere with independence in everyday activities (i.e., at a minimum, requiring assistance with complex instrumental activities of daily living such as paying bills or managing medications).
C. The cognitive deficits do not occur exclusively in the context of a delirium.
D. The cognitive deficits are not better explained by another mental disorder (e.g., major depressive disorder, schizophrenia).

Specify whether due to:

Alzheimer's disease
Frontotemporal lobar degeneration
Lewy body disease
Vascular disease
Traumatic brain injury
Substance/medication use
HIV infection
Prion disease
Parkinson's disease
Huntington's disease
Another medical condition
Multiple etiologies
Unspecified

Specify:

Without behavioral disturbance
With behavioral disturbance

Specify current severity:

Mild
Moderate
Severe

Source: DSM-5 criteria for major neurocognitive disorder reprinted from *Diagnostic and Statistical Manual of Mental Disorders*, 5th edition.

Table 11.2 Definitions related to dementia

Dementia	A clinical syndrome of cognitive decline with significant effect on day-to-day functioning.
Cognitive impairment not dementia (CIND)	A clinical syndrome describing subjective or measurable deficits in memory or other areas of cognition that are not sufficiently severe to meet criteria for dementia.
Mild cognitive impairment	A clinical subsyndrome of CIND. It can involve memory deficits (amnestic) or other cognitive domains (nonamnestic). Amnestic MCI is like a prodrome of Alzheimer's dementia.

clinician; be documented by objective cognitive assessment; *and* affect the individual's independence in everyday activities.

Functional impairments may not only include ADLs but also extend into social and interpersonal functioning. Individuals with mild dementia may have difficulties with instrumental activities of daily living (IADLs) such as social activities, managing finances, meal preparation, shopping, and driving. As dementia progresses, individuals may develop impairments in basic ADLs such as grooming, dressing, toileting, eating, and transferring. Neuropsychiatric impairments, also known as neuropsychiatric symptoms (NPSs), affect almost all persons with dementia over the course of illness and can affect social and interpersonal functioning.

History-Taking

Because of the nature of cognitive impairment, a patient with suspected dementia may not be able to provide an accurate history because of barriers such as lack of

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insight, memory loss, and/or language problems. It is therefore critical to involve a reliable and knowledgeable informant during history-taking. Because informants themselves can be influenced by their own mental state, it is often useful to speak with more than one informant.

It is critical to elucidate a timeline of cognitive symptoms and establish the progression of symptoms over time, starting from when the patient was last well. These characteristics can influence the differential diagnosis since the type of symptoms, pattern of deficits, and pattern of progression can be specific to different dementia etiologies. History-taking should assess for cortical and subcortical cognitive symptoms, functional losses, and neuropsychiatric and neurological deficits. Examples of cognitive symptoms across a range of cognitive domains can be found in Table 11.3.

It is important to inquire about a family history of dementia or other neuropsychiatric illnesses, which may help identify syndromic patterns. A review of neurological symptoms (e.g., stroke, seizures, traumatic brain injury) and psychiatric symptoms (e.g., depression, anxiety, psychosis, substance use disorder) should be performed to help identify potential etiologies and mimics of dementia. Clinicians should ask about problems with vision and hearing, behavioral or personality changes (which may suggest frontotemporal dementia, thyroid disease, or major depression), visual hallucinations (Lewy body dementia, psychosis), symptoms of neuropathy, changes in speech (stroke), and changes in gait (Parkinson's disease [PD], stroke, normal pressure hydrocephalus). These differentials will be described in greater detail later in the chapter.

Medication Review

Certain classes and combinations of medications may contribute to cognitive impairment.[56] Classes of medications most likely to be associated with cognitive impairment, largely due to anticholinergic effects, include opiates, anticholinergics, benzodiazepines, antihistamines, tricyclic antidepressants, muscle relaxants, and antiepileptic medications (Table 11.4).

Cognitive Assessment

A cognitive assessment provides critical information on the severity and characteristics of cognitive deficits in the evaluation of dementia. A lot can be learned about an individual's cognitive status through history-taking. For example, difficulties with tasks that require planning may suggest deficits in executive functioning, word-finding

Table 11.3 Examples of symptoms of deficits in cognitive domains affected by dementia

Cognitive domain	Examples of symptoms
Executive function	Difficulty completing tasks that require multiple steps or planning Difficulty with previously familiar tasks Difficulty problem-solving Becoming uncharacteristically disorganized
Complex attention	Taking longer to finish tasks Requiring more overview or rechecking of work Increased distractibility
Learning and memory	Repetition in conversations Difficulty in recalling recent events Increasing reliance on lists Forgetting to pay bills Forgetting to buy items or buying the same items multiple times
Language	Difficulty in finding the correct words Substituting names for general descriptions or pronouns Mispronunciation of common words Difficulty understanding verbal or written directions
Social cognition	Losing interest in social or work situations Increase in socially inappropriate behaviors Loss of empathy Impaired judgment
Perceptual-motor function	Difficulty using previously familiar tools, appliances, and technology Becoming lost in familiar environments

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Table 11.4 Medications associated with cognitive side effects

Medication class	Examples of medications
Anticholinergics	Benztropine Medications for overactive bladder or urge incontinence (oxybutynin, darifenacin, flavoxate)
Benzodiazepines	Clonazepam Lorazepam Diazepam Alprazolam
Antihistamines	Diphenhydramine Cetirizine Hydroxyzine Chlorpheniramine
Opiates	Oxycodone Morphine
Antidepressants	Tricyclic antidepressants (amitriptyline, imipramine, doxepin) Paroxetine
Antipsychotics	Quetiapine Olanzapine Haloperidol
Muscle relaxants	Methocarbamol Cyclobenzaprine Baclofen
Anticonvulsants	Carbamazepine Phenobarbital Phenytoin

difficulties may suggest language deficits, and difficulties recalling information may suggest deficits in short-term memory.

Many assessments have been developed to characterize deficits in specific domains of memory. While a full battery of cognitive testing requiring assessment by a neuropsychologist may be used, a number of measures can inform the differential diagnosis.

Composite measures of global cognitive function can provide a simple way to screen for and monitor progression of cognitive decline. These measures combine elements to examine multiple domains of cognition, some of which have previously been discussed. The Mini-Mental State Examination (MMSE)[24] is frequently used as a primary tool for its brevity and ease of use. While a cutoff point of <24 on the MMSE has been recommended as suggesting the presence of dementia,[37] it is by no means definitive. MMSE score thresholds may vary between different education levels.

Compared to the MMSE, the Montreal Cognitive Assessment (MoCA)[47] can provide a more comprehensive assessment of cognition, assessing abstract thinking, delayed recall, verbal fluency, and executive functioning.

Neuropsychological testing is not required in every case. It may be useful, however, in differentiating dementia from milder cognitive syndromes or normal aging or clarifying the etiology of the cognitive disorder. If neuropsychological testing is needed, clinicians should direct specific questions to address, such as how to clarify the differential diagnosis.

Physical Exam

A complete neurological exam should be completed in evaluating patients with suspected dementia. The neurological exam should include an assessment of the cranial nerves, speech, motor and sensory skills, balance and coordination, posture, and gait. While the physical exam does not necessarily assess for the presence or absence of dementia, it may reveal important clues to its etiology.

Diagnostic Testing

The term "reversible dementia" has previously been used to describe dementias associated with potentially reversible etiologies. These potentially reversible causes are estimated to comprise 9% of dementia cases.[12] However, more recent literature has emphasized that dementia is usually not reversible, even when what was previously thought to be a potentially reversible cause has been found.[46] Still, routine laboratory tests are recommended to identify the very rare instances of severe abnormalities for which (1) a correction may stop progression or (2) a modest metabolic abnormality may be contributing to cognitive impairment on top of a presumed degenerative process.

Laboratory testing of complete blood count, electrolytes, glucose, vitamin B12, calcium, thyroid function tests, and folates are recommended to identify infection, hyper- or hypoglycemia, hyper- or hypothyroidism, electrolyte imbalance, vitamin B12 deficiency, and folate deficiency. Syphilis, Lyme titers, and HIV tests rarely reveal infectious causes for cognitive impairment. Urinalysis and urine culture in the presence of signs or symptoms of infection may be useful if delirium is part of the differential diagnosis.

Neuroimaging

Structural neuroimaging in the routine initial evaluation of patients with suspected dementia is recommended.[23,55]

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There is no clear consensus on the use of computed tomography (CT) versus magnetic resonance imaging (MRI) for structural imaging in dementia evaluation, though MRI is generally regarded as the superior tool for brain imaging.[62] While structural imaging in the context of dementia evaluation has historically been used to exclude abnormalities potentially amenable to surgical treatment, such as tumor, hematoma, or hydrocephalus, imaging can also provide valuable etiological clues including patterns of atrophy and visualization of vascular lesions.[23] Functional brain imaging using positron emission tomography (PET) or single-photon emission computed tomography (SPECT) may reveal regional low metabolism/hypoperfusion. For example, 18-fluordeoxyglucose positron emission tomography (FDG-PET) is used to provide quantitative and qualitative measures of brain glucose metabolism. Identifying patterns of glucose metabolism may narrow the diagnostic differential.

Genetic and Biomarker Testing

The majority of family history of dementia is due to genetically complex disease, comprising many genetic variations of small effects. Rarely, families may have an autosomal dominant family history of early-onset dementia due to Mendelian disease. Testing for specific genetic mutations associated with dementia may be appropriate in rare instances where there is a clear familial linkage.

Routine lumbar puncture is not recommended for clinical evaluation unless Creutzfeldt-Jakob disease (CJD), or other forms of rapidly progressive dementias, is suspected. Similarly, electroencephalography (EEG) is not routinely used unless CJD or delirium is suspected.

Alzheimer's Disease-Specific Diagnostic Testing

The National Institute on Aging-Alzheimer's Association (NIA-AA) diagnostic guidelines do not recommend the use of Alzheimer's disease (AD) biomarker tests for routine diagnostic purposes for individuals who meet core clinical criteria for probable AD dementia.[44] AD dementia remains a clinical diagnosis, though biomarker support may help increase certainty that the etiology of a dementia syndrome is secondary to an AD pathological process.

Three core cerebrospinal fluid (CSF) biomarkers have been identified and extensively studied: T-tau, P-tau, and Aβ-42. Changes in levels of these CSF biomarkers may be observed even in the preclinical phase of AD.[66] The combination of high CSF T-tau and high P-tau levels with low concentration of Aβ-42 is a pattern commonly referred to as the "AD signature," which can potentially be used to add to the diagnostic accuracy of AD.[67]

In the USA, amyloid imaging is not covered by Medicare except for use in clinical trials that assess how amyloid imaging improves patient outcomes or advances treatment options. PET tracers for tau imaging are currently under investigation but do not yet have enough evidence to support their use in the clinical setting.[50]

Dementia Subtypes

Alzheimer's Dementia

Alzheimer's disease is a progressive neurodegenerative disorder characterized pathologically by intracellular neurofibrillary tangles of hyperphosphorylated tau protein and extracellular β-amyloid (Aβ) deposits. It is the most common cause of dementia, accounting for 60–80% of cases.[2]

Although old age is the primary risk factor for AD, many other factors are associated with the progression or development of AD. Risk factors for cardiovascular disease are also associated with higher risk of AD dementia.[45] Obesity, type 2 diabetes mellitus, low exercise, traumatic brain injury, and low cognitive reserve (e.g., fewer years of formal education, lower cognitive engagement) have been associated with AD onset.[2] While a family history of AD is not necessary for one to develop AD, genetics are a major risk factor. Genetically, AD is divided into two categories: (1) familial cases, with Mendelian inheritance and (2) sporadic cases, with less apparent or no familial aggregation. Sporadic cases usually have later age of onset (≥60 years), with an estimated heritability of 60–80%.[25] Individuals who are carriers of APOE-ε4 have a dose-dependent increased risk of developing late-onset AD.[38] Ultimately, AD is a heterogeneous condition representing a range of etiologies involving interactions between different sets of genetic and environmental risk factors.

The diagnosis of AD is clinical, though additional investigations may be utilized for diagnostic clarification, as described in the Diagnostic Testing section. Probable AD is diagnosed when the patient meets criteria for dementia, in addition to insidious onset and a clear history of worsening of cognition. The most common syndromic presentation of AD is amnestic, with initial presentation of deficits in learning and recall of recently

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learned information. However, initial and prominent nonamnestic presentations involving deficits predominantly in language, visuospatial, or executive function domains are also possible.

The median time from diagnosis to death is approximately 10 years, with considerable variability. Despite variability in the rate of decline, patients who progress typically first experience loss of memory, followed by the development of agnosia, apraxia, and aphasia. Functional impairments progress as cognitive impairments increase. In later stages, patients universally develop problems with mobility and continence. Neuropsychiatric symptoms such as depression, apathy, and sleep disturbance are prevalent across the spectrum of AD and increase as disease severity progresses.[65]

Vascular Dementia

After AD, vascular dementia is believed to be the second most common cause of dementia, accounting for about 15% of cases.[49] The term "vascular dementia" remains a controversial nosological entity, partially because of the lack of consensus for pathological criteria and difficulty differentiating clinically from dementia due to AD.[31] The clinical presentation of vascular dementia is variable and highly dependent on the specific neural substrates (brain circuits and regions) affected by the vascular pathology. Typically, it presents with acute or subacute onset after a cerebrovascular event.[49] Other early features of vascular dementia may include apathy, depression, motor symptoms (such as gait disorders and parkinsonism), and incontinence. The diagnosis requires brain imaging that shows completed infarcts or lacunes in brain areas associated with the observed cognitive changes. Radiological findings of nonspecific white-matter change without evidence of completed strokes or associated examination findings *are not supportive* of a diagnosis of vascular dementia.

Frontotemporal Dementia

Frontotemporal dementia (FTD) is the third most common form of dementia across all age groups.[60] It is the most common type of early-onset dementia in individuals <65 years old.[5] Criteria for clinical features differ based on clinical variant.

The most common presentation of FTD is the behavioral variant (bvFTD). bvFTD is characterized by early behavioral and executive deficits. The most pronounced early symptoms include personality changes, disinhibition, apathy, socially inappropriate behavior, and increased impulsivity. These changes may manifest as reduced interest in hobbies or social interactions, new criminal behaviors, careless actions, or embarrassing personal remarks. Because of the prevalence of apathy in this variant, bvFTD may be misdiagnosed as clinical depression early in the disease course.[8] Left-lobe degeneration may also be accompanied by compulsive behaviors such as selective eating (often focused around one particular type of food), collecting, and repetitive activities, while the right-temporal-lobe variant may be associated with verbal compulsions involving words, symbols, numbers, and making puns.[5]

The second most common presentation of FTD is primary progressive aphasia (PPA). PPA is characterized by prominent progressive impairment in speech and language of insidious onset. The three main variants of PPA are logopenic PPA, non-fluent variant (NFV-PPA), and semantic variant (SV-PPA). Logopenic PPA is characterized by a paucity and slowed rate of speech, impairment in word retrieval, and repetition.

Non-fluent variant primary progressive aphasia is characterized by progressive deficits in speech, grammar, and word output. Patients may experience deficits in language production, object-naming, syntax, and word comprehension. Semantic-variant primary progressive aphasia is characterized by semantic aphasia and associative agnosia. While correct grammar and fluent speech is usually retained at initial stages, there is impaired comprehension of individual words, and especially for words that are not routinely used.[29]

Dementia with Lewy Bodies

Dementia with Lewy bodies (DLB) accounts for 4–8% of cases.[59] Visuospatial or constructional impairments are common, even in the early stages of dementia; they are associated with the presence of visual hallucinations[32] and highly specific for pathological diagnosis of DLB.[57]

Clinical diagnostic criteria have been laid out by the DLB Consortium for the diagnosis of "probable" and "possible" DLB.[43] The core clinical features of DLB include: fluctuating cognition with pronounced variations in attention and alertness, recurrent visual hallucinations (that are typically well formed and detailed), rapid eye movement (REM) sleep behavior disorder (which may precede cognitive decline), and one or more spontaneous cardinal features of parkinsonism (e.g., bradykinesia, rest tremor, or rigidity).

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Parkinson's Disease Dementia

About 25% of patients with Parkinson's disease have dementia.[1] The risk of dementia increases with disease duration, and prevalence reaches 50% at 10 years after diagnosis.[63] PD dementia is defined clinically as dementia starting 1 or more years after well-established PD.[20] Patients with PD typically experience impairments in executive functioning, deficiencies in attention, and poor fluency. Risk factors for the development of dementia in individuals with PD include old age, PD onset after age 60 years, higher duration of PD, severity of motor symptoms, REM sleep behavior disorder, and visual hallucinations.[61]

Prion Disease

Although relatively rare, prion diseases represent a large portion of neurodegenerative causes of rapidly progressive dementias. The most common cause of human prion dementia is Creutzfeldt-Jakob disease (CJD). While most cases of CJD are sporadic (75–85%), 10–22% are genetic, and <1–3% are acquired.[4] Patients often experience rapidly progressive multidomain cognitive impairment, occasionally accompanied by cortical visual disturbances, ataxia, myoclonus, and heightened reactivity to external stimuli. There are often preceding mild psychiatric symptoms such as malaise, anxiety, mood changes, and poor concentration.

Rapidly Progressive Dementias and Dementia with Specific Treatments

While rapidly progressive dementia describes dementia that occurs within less than 1–2 years from illness onset, it commonly develops over weeks to months.[27] Prion disease is the most common cause, followed by neurodegenerative, autoimmune, and infectious causes.[51] While there are no disease-modifying therapies available, many causes of rapidly progressive dementia are potentially treatable.[51] The differential diagnosis of rapidly progressive dementia is summarized in Table 11.5.

Table 11.5 Differential diagnosis of rapidly progressive dementias

Etiology	Differential diagnosis
Neurodegenerative	Creutzfeldt-Jakob disease Alzheimer's disease Frontotemporal degeneration Dementia with Lewy bodies Cortico-basal degeneration Progressive supranuclear palsy
Infectious	Viral encephalitis Fungal infections Parasitic infections Syphilis Whipple disease Acquired immune deficiency syndrome (AIDS) HIV dementia Progressive multifocal leukoencephalopathy
Metabolic	Vitamin deficiencies (i.e., vitamin B12, folate, niacin) Uremia Wernicke syndrome Extrapontine myelinolysis Acute intermittent porphyria
Toxicity	Heavy metals (i.e., bismuth, mercury, arsenic) Lithium Alcohol Carbon monoxide Iatrogenic, medication side effect
Autoimmune	Hashimoto's encephalopathy Paraneoplastic syndrome Limbic encephalitis Sjogren's syndrome Multiple sclerosis Lupus cerebritis Sarcoid CNS vasculitis Anti-NMDA-receptor encephalopathy Giant cell arteritis
Endocrine	Hypo- or hyperthyroidism Hypo- or hyperparathyroidism Adrenal insufficiency
Vascular	Infarct Subcortical arteriosclerotic encephalopathy Hyper-viscosity syndromes
Oncologic	Primary central nervous system tumor Metastatic tumor

Depression and Delirium

Major depression and delirium are clinical syndromes that do not infrequently co-occur with dementia. Severe major depression can cause dementia ("dementia of depression," in the past referred to as "pseudodementia"). Both depression and delirium symptoms overlap with symptoms of dementia and should be ruled out in a dementia evaluation. See Chapter 12 on recognition, management, and prevention of delirium and Chapter 20 on clinical geropsychiatry for further details.

Management and Care

Management and care options for dementia have four basic elements, or pillars. The first pillar pertains to the

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management of the primary pathology underlying dementia, with the goal of reversing its effects or delaying its progression. The second pillar relates to the management of symptoms, which includes cognitive, neuropsychiatric, and functional symptoms. While symptomatic therapies do not target underlying pathology, they can be effective in managing cognitive and neuropsychiatric symptoms. Though pharmacotherapy has been the focus of many clinical trials, specific, evidence-based nonpharmacological approaches play a central role in management. The third and fourth pillars focus on providing supportive care to patients and their caregivers.

Disease-Oriented Therapies

Alzheimer's Disease

Though there have been many clinical trials, for the most part they have not been successful at producing safe disease-modifying therapies for AD.[13] There is currently no Food and Drug Administration (FDA)-approved disease-modifying treatment for AD.

Off-label uses for medications targeting risk factors associated with the pathogenesis of AD, such as increased inflammation and reduced estrogen, have produced negative or mixed results for the treatment of dementia. The American Association for Geriatric Psychiatry (AAGP) does not recommend the use of estrogen, anti-inflammatory agents (e.g., prednisone, NSAIDs), or Ginkgo biloba as treatments for AD because of evidence that they do not work and can be harmful.[40] Evidence for the use of vitamin E is limited, and trials of vitamin E in AD have typically used doses much higher than the daily recommended allowance.[35] Given potential adverse effects, the AAGP recommends avoiding doses above 400 IU a day.[40] Omega-3 polyunsaturated fatty acids, which are present in fish, vegetable oil, and nuts, were not associated with benefit in cognition for mild to moderate AD in a Cochrane meta-analysis of three randomized, placebo-controlled trials.[9] Medical foods, such as AC-1202 (Axona; containing ketogenic medium chain triglycerides) and Souvenaid with Fortasyn Connect (a multinutrient drink), have mixed evidence for improvements in AD.[35]

Nonspecific Disease-Oriented Therapies

Managing Medical Comorbidities

Patients with dementia are more likely to be admitted to the hospital and require more outpatient visits than individuals without dementia.[11] Having a higher number of medical comorbidities in AD is associated with worse cognitive performance and functional decline.[16] Common medical comorbidities in AD include cardiovascular disease, thyroid dysfunction, osteoporosis, glaucoma, sleep apnea, and incontinence.[18]

One of the most effective therapies for AD is the aggressive management of associated vascular risk factors such as managing hypertension, hypercholesterolemia, diabetes, obesity, smoking cessation, and sedentary lifestyle.[45] In addition to the management of cardiovascular risk factors, the Alzheimer's Association recommends maintaining a healthy diet and cognitive stimulation to reduce the pace of cognitive decline.[6]

Preventing Delirium

Precautions should be taken to reduce the risk and duration of delirium in patients with dementia, as delirium itself is a risk factor for accelerated cognitive decline.[34] Polypharmacy, pain, urinary retention, acute illness (i.e., upper-respiratory tract infections, urinary tract infections), constipation, and dehydration are some common causes of such acute declines in this population.[30,54]

Optimization of perioperative care is important for the prevention of delirium for patients undergoing either emergent or elective surgeries or other procedures such as colonoscopies.

Symptomatic Therapies

Acetylcholinesterase Inhibitors

One of the earliest pathologic findings associated with AD was the loss of neurons in the nucleus basalis, the main origin of cholinergic neurotransmission to the cortex. Cholinesterase inhibitors (ChEIs) inhibit the enzymes that degrade acetylcholine, increasing the concentration of acetylcholine at synaptic clefts of the brain. Donepezil, galantamine, and rivastigmine (Table 11.6) are FDA-approved ChEIs for the symptomatic treatment of cognitive symptoms in mild to moderate AD. Donepezil and the patch formulation of rivastigmine are approved for use in severe AD. There is currently no evidence to support the use of one anticholinesterase over another.[53]

ChEIs provide modest, largely time-limited stabilization of cognitive and functional decline associated with AD. They do not, however, reverse or stop the degenerative process. The optimal duration of ChEI treatment has not been established, though the medications are generally well tolerated and patients in clinical

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Table 11.6 FDA-approved treatments for AD: cholinesterase inhibitors donepezil, galantamine, rivastigmine

Medication	Disease stage	Formulations	Initial dosage	Maximum dosage	Common side effects
Donepezil	All stages of AD	Oral tablet Oral disintegrating tablet	5 mg/day	10–23 mg/day	Nausea, diarrhea, insomnia, vomiting, muscle cramps, fatigue, anorexia, vagotonic effects leading to bradycardia and heart block. May require reduced dosage in hepatic impairment.
Rivastigmine	Mild to moderate AD for oral formulation Transdermal patch also approved for severe AD and mild to moderate dementia associated with Parkinson's disease	Oral capsule Oral solution Transdermal patch	1.5 mg twice daily 4.6 mg (transdermal patch)	6 mg twice daily (oral) 13.3 mg (transdermal patch)	Nausea, vomiting and diarrhea, anorexia, dyspepsia, asthenia, extrapyramidal symptoms, vagotonic effects leading to bradycardia and heart block. May require dose reduction in renal and hepatic impairment.
Galantamine	Mild to moderate AD	Oral tablet Extended-release capsule Oral solution	4 mg twice daily 8 mg/day (extended release)	12 mg twice daily 24 mg/day (extended release)	Nausea, vomiting, diarrhea, anorexia, weight loss, muscle cramps, vagotonic effects leading to bradycardia and heart block. Use in severe hepatic or renal impairment not recommended.
Memantine	Moderate to severe AD (monotherapy and in combination with acetylcholinesterase inhibitor)	Oral tablet Extended-release capsule Oral solution	5 mg/day 7 mg/day (extended release)	10 mg twice daily 28 mg daily (extended release)	Dizziness, headache, confusion, constipation, fatigue. May require reduced dosage in severe renal impairment.

trials have shown benefit for up to 5 years.[14] Therefore, continuation of therapy with family support, as long as patients tolerate the medication, is reasonable.

Memantine

The addition of memantine to ongoing ChEI therapy is frequently used for severe dementia. Evidence for its use in mild AD is limited. Namzaric, a combination pill of memantine hydrochloride extended release (Namenda) and donepezil hydrochloride (Aricept), is approved by the FDA for moderate to severe AD. Systematic reviews and meta-analyses show that combination therapy in moderate to severe AD has greater effects on cognition and global impression on short-term follow-up compared to ChEI therapy alone.[22,28,42] However, the effects of long-term combination therapy or effects on nursing home placement are still unclear.

Evaluation and Management of Neuropsychiatric Symptoms

Although cognitive deficits are the hallmark of dementia, more than 90% of patients with AD experience neuropsychiatric symptoms such as depression, agitation, anxiety, psychosis, hallucinations, apathy, eating disorders, disinhibition, and sleep disturbances.[26] After ruling out delirium and potential medical causes, nonpharmacological approaches should first be used to manage symptoms.[36] The "DICE" (Describe, Investigate, Create, and Evaluate) approach (Table 11.7) provides a useful mnemonic for a methodic approach to managing neuropsychiatric symptoms.

Some patients who do not respond to nonpharmacological approaches may require targeted medication therapy. It should be noted that while psychotropics are frequently prescribed for NPSs in dementia, there are currently no pharmacotherapies with FDA approval for this purpose. The evidence for efficacy for most psychotropics used in neuropsychiatric symptoms is mixed and current research is limited.[52]

Because of the inherent risks of using medications to treat NPSs in dementia, nonpharmacological approaches should be used as first-line therapy (Table 11.8). Psychotropics should be used only after significant efforts have been made to mitigate NPSs, with three exceptions: (1) clear-cut major depression; (2) psychosis causing harm or with significant potential of harm to self or others; and (3) aggression causing harm or risk of harm to self or others.[36]

Table 11.7 "DICE" (Describe, Investigate, Create, and Evaluate)

Describe	Caregiver describes problematic behavior Context Social and physical environment Patient perspective Degree of distress to patient and caregiver
Investigate	Provider investigates possible causes of problem behavior Undiagnosed medical conditions Underlying psychiatric comorbidity Limitations in functional ability Poor sleep hygiene Boredom, fear, sense of loss of control Medication side effects Sensory impairment Environmental factors Unmet needs
Create	Provider, caregiver, and team collaborate to create and implement treatment plan Respond to medical problems Strategize behavioral interventions Provide caregiver education and support Create meaningful activities for the patient Simplifying tasks Ensuring the environment is safe Enhancing communication with the patient Increasing or decreasing stimulation in the environment
Evaluate	Provider evaluates whether the interventions have been implemented by caregiver and whether they are effective

Source: Kales et al., 2014[36]

Supportive Care for the Patient

Beyond the management of symptoms of dementia, there are many additional practicalities such as advanced directives, safety, and transition of household responsibilities that also need to be considered. The goals of supportive care are to maintain the patient's quality of life and prevent morbidity. Concerns and interventions related to communication, maximizing function, nutrition and hydration, sleep hygiene, establishing a daily routine, and travel are summarized in Table 11.9.

Advance Directives

Advance care planning comprises discussions between a patient and their health-care provider that usually take place in anticipation of a future deterioration of a person's condition.[33] The use of written advance directives in dementia is associated with decreased distress at end of life and quality of dying.[58] However, given the unpredictable nature of anticipating every

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Table 11.8 Nonpharmacological strategies for common neuropsychiatric symptoms

Behavior	Key strategies
Memory-related problems	Provide reminders, cues, or prompts Provision of familiar objects Use of pictures to provide cues Changes in activity demand Simplify language Day-to-day living of patients should be structured to maximize their abilities and function
Hearing voices or noises	Adjustment of environmental noise level Reduce clutter or visual distractions
Repetitive questioning	Respond in a calm, reassuring voice Use a light touch to reassure, calm, or redirect Inform patient of events as they occur Use distraction, engage the person in an activity Use memory aids
Agitation	Focus on patient's wishes, interests, and concerns Create a calm environment Respond in a calm, reassuring voice Use a light touch to reassure, calm, or redirect Use distraction Consider psychotherapy in early dementia with patients who are anxious, depressed, or demoralized Offer simple choices Simplify tasks and routines
Nighttime wakefulness and sundowning	Encourage good sleep hygiene Evaluate environment for disturbances that may affect level of comfort Avoid caffeinated beverages starting in the afternoon Limit daytime napping, plan active days Create a quiet routine for bedtime
Psychosis	Avoid arguing or trying to reason with the person about their symptoms as this can make the situation worse Ignore symptoms that are not distressing to the person
Anxiety	Allow extra time for tasks and activities Filter information shared with the person, particularly events occurring in the future
Depression	Provide reassurance Ask family or friends to spend one-on-one time with the person Rule out hypoactive delirium
Apathy	Give the person a job at home, such as folding laundry Focus on the process of doing things rather than the results
Disinhibition	Distract from an inappropriate topic by calmly and firmly changing the subject Avoid television shows with violence or sexual content
Irritability	Create a calm environment Acknowledge and validate the patient's frustration

possible situation that will require a health-care decision, it is preferable that the patient designate a durable power of attorney for health care early on.

Safety

For individuals living alone, a professional evaluation should be sought to assess the many skills needed to live independently. A summary of common safety concerns and interventions for them can be found in Table 11.10. If there is concern for significant risk of harm, steps should be made to intervene in independent living.

Driving

Driving is a complex skill that relies on many physical and cognitive skills. Individuals with dementia are at a 2 to 8 times higher risk of being involved in a motor vehicle accident than age-matched controls.[17] While a diagnosis of dementia does not automatically preclude one from driving, the progressive nature of the disease means that

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Table 11.9 Supportive care for the patient

	Concerns	Key strategies
Establish a relationship with the patient	Impairments in communication can impede the ability to establish and maintain a relationship Patients with cortical dementias develop expressive and receptive aphasia	Respect the dignity of patients Most moderately to severely impaired patients have limited insight. Avoid arguing with individuals who have dementia when they make incorrect statements Provide directions one step at a time Ask one question at a time and give the patient time to respond
Maximize function and identify abilities	Maximizing functions can help preserve patient's dignity, improve caregiver burden, and likely enable remaining abilities to be maximally used for as long as possible	Work with caregivers to find settings and environments in which limitations are minimized and remaining abilities maximized Maintain a structured schedule, taking into consideration the patients' prior interests, wishes, and usual activity level Identify activities that the patient can participate in, where they require little cueing and seem to enjoy themselves
Nutrition and hydration	Dementia can impair the ability to plan and prepare meals, to differentiate fresh from spoiled foods, to eat a balanced diet, or to remember to eat at all	Meals should be organized and monitored by the caregiver Inedible garnish should not be used Decorative wax or plastic fruits should be removed from the home Avoid tough meats Small amounts of food can be offered gradually to avoid gorging Weigh-ins can be used to monitor intake Fluid intake should be monitored carefully Offer fluids every 2 hours
Sleep hygiene	Adequate rest is important for ensuring optimal functioning Day/night sleep cycles can be disturbed or even reversed in dementia	Establish a routine of activities leading up to bedtime Daily mild exercise can help in maintaining normal sleep cycles Keep the bedroom environment comfortable Avoid medications that may cause frightening dreams or nocturia
Establish a daily routine	Dementia can reduce cognitive flexibility and adaptability Patients do better in a stable, predictable environment, even early in the illness Predictable patterns of activity can maximize the ability to learn	Develop a routine patterned after those that the patient and family have followed for years
Emotional support	Emotional support is needed particularly when the disease causes frustration and difficulty with daily living	Patients with milder dementia may benefit from counseling-based professional intervention Acknowledge and validate the source of distress Encourage patient to refocus their thinking on things that they can accomplish
Travel	Travel can be desired or necessary, but can also impose significant difficulties for a person with dementia and for their care providers	Consider if travel will be beneficial to the person with dementia and whether he or she wants to go It is best to travel using the fastest method, to as familiar an environment as possible Provide distraction and personal time for the patient during the trip. Plan activities for distraction, such as playing a card game or listening to music Minimize what is expected of the patient during travel (i.e., care of luggage) Ensure patient is adequately fed and hydrated during the trip

Table 11.10 Common safety concerns and key strategies

	Safety concerns	Key strategies
Early stage	Medication management	Use a pill box organizer Develop a routine for giving medications Place medications in locked drawer or cabinet to ensure that they are taken safely
	Cooking	Install a gas valve or circuit breaker on the stove so that it cannot be turned on Use appliances that have an auto shut-off feature
	Falls	Remove unnecessary objects and clutter Use a fall alert system if the patient can activate it Consider referral to occupational therapy for home safety evaluation Consider referral to physical therapy for balance exercise Minimize alcohol intake Avoid polypharmacy
	Driving	Driving privileges should be revoked once there is clear evidence of impairment in the skills required for driving Individuals with dementia should be evaluated by occupational therapist with expertise in driving evaluations
Middle stage	Wandering	Educate caregiver about the need to supervise patient Have a routine for daily activities Identify the most likely times of the day that wandering may occur Avoid places that may be confusing and cause disorientation Remove environmental cues such as car keys Hang bells on doors, cabinets, or drawers to alert caregiver when opened GPS-tracking devices or apps can be used to find a person who has become lost Use a medical ID bracelet that can provide information about individuals with dementia if they become lost
	Safety at home	Reverse or remove bathroom locks so that patients do not lock themselves in Windows should be securely locked or capable of being opened only 6 inches to prevent patients from crawling out Guns and weapons should be removed from the home Remove any potentially dangerous tools, utensils, and machines from the patient's environment
Late stage	Ingestion	Clinicians should assume that patients may ingest anything Everyday items that can cause poisoning if ingested (such as plants, flowers, paint, cleaning solutions) should be removed from the patient's environment Remove decorative fruits

there should be continuous reassessment for impairment in their ability to drive. Laws regarding notification of the motor vehicle bureau vary greatly throughout the United States. In some states, physicians are required to report all persons with a diagnosis of dementia, while in other states, notification is not permissible because of confidentiality laws. Driving evaluations that place demands on both visuospatial abilities and motor responses are the best predictors of driving safety.[15] Single tests, such as the MMSE, should not be used as an indicator of fitness to drive, as they do not sufficiently assess the many cognitive and physical abilities required to operate a vehicle safely.[7]

Transition Out of the Home

Placement usually refers to the transition of a patient from the home environment to an organized, supported living situation such as an assisted living facility or a nursing home. The need for placement usually arises because of cognitive decline leading to an inability to recognize or interact with the environment, functional decline causing the patient to become completely dependent on others, the development of chronic behavioral disturbances that require constant supervision, and comorbid medical disorders requiring specialized nursing care. When possible, transitions should be planned ahead of time.

Support for the Caregiver

High caregiver burden is reported in 46% of AD caregivers[2] and is associated with poor quality-of-life outcomes for both the caregiver and patient as well as early nursing home placement.[21] Elements of caregiver support should include providing education, patient symptom reduction, addressing the emotional needs of

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Table 11.11 Supportive care for the caregiver

Elements of caregiver support	Key strategies
Education	Educate the caregiver about dementia Written material is helpful for many people, and several organizations, including the Alzheimer's Association, provide excellent pamphlets on specific topics
Patient symptom reduction	Instruct caregiver on the skills of caregiving Support the caregiver with problem-solving techniques Help the person with dementia maintain active
Address the emotional needs of caregivers	Provide comfort and emotional support Encourage respite from caregiving Encourage the caregiver to maintain a social network
Address the personal needs of caregivers	Remind caregivers to attend to their own needs Attend to the caregiver's general and mental health, including scheduling preventive health-care visits

caregivers, and helping caregivers address personal needs (Table 11.11). Providing education about the disease and skills training in communication for dementia care can significantly improve the quality of life for patients and increase positive interactions.[19] Case management can be a resource for providing assessment, information, planning, referral, and care coordination.

Key Points

- Dementia is a clinical syndrome characterized by global cognitive decline, involving deficits in memory and at least one other area of cognition.
- The evaluation and differential diagnosis of dementia involves history-taking with outside informants and examination focused on phenomenology and associated features, followed by a workup for potential causes.
- Neuropsychiatric symptoms in later life may represent a noncognitive symptom of a dementia prodrome. Cognitive symptoms in the presence of psychiatric symptoms, such as depression, do not rule out dementia.
- The four pillars of dementia care are: (1) disease-oriented treatment, (2) symptomatic treatments, (3) supportive care for the patient, and (4) supportive care for the caregiver.
- Neuropsychiatric symptoms are common throughout the spectrum of dementia severity. Management usually involves psychological, social, and environmental adjustments. No pharmacological measures have been approved by the FDA and should be reserved for more severe symptoms. The Describe, Investigate, Create, and Evaluate (DICE) approach offers a structured method in evaluating and managing neuropsychiatric symptoms.
- Disease-modifying therapies in AD have largely been unsuccessful. Symptomatic treatments such as cholinesterase inhibitors and memantine provide a modest and temporary stabilization of cognitive changes associated with the disease, but do not reverse or stop the degenerative process.
- There are no disease-modifying treatments for dementia with Lewy bodies, Parkinson's disease dementia, frontotemporal lobar degeneration, or Creutzfeldt-Jakob disease.

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Chapter 12

Recognition, Management, and Prevention of Delirium

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Introduction

Delirium is a serious neuropsychiatric condition characterized by an acute change in cognition and attention that affects a significant proportion of hospitalized older adults. The prevalence of delirium (Table 12.1) varies depending on the care setting and patient population; in one large tertiary care teaching hospital, 34.8% of patients over the age of 80 met the diagnostic criteria for delirium.[1]

Delirium portends a poor prognosis; it is associated with an increased risk of death, functional decline, and institutionalization.[2] The 1-year mortality rate for delirium is high, found to be 39% in one study.[3] Institutionalization is more common among those who have been delirious, with one study determining an odds ratio of 4.53.[4] The association is not necessarily causal; delirium may initiate a decline in health, or patients who develop delirium may be more ill.

The economic costs of delirium are significant. In one study, delirium more than doubled a patient's average health-care costs per day for the entire year.[5] Total costs per patient for the delirium alone ranged from $16,303 to $64,421, which translated to an overall burden to the US health-care system of $38 billion to $152 billion per year.[5] The study did not account for indirect costs, such as caregiver burden or decreased quality of life.

Table 12.1 Prevalence and incidence of delirium in different care settings

Care setting	Prevalence (%)	Incidence (%)
General medical ward	18–35	11–14
Medical ward, patients with dementia	18	56
Emergency department	8–17	
Nursing home	14	20–22
Intensive care	7–50	19–82

Adapted from Inouye 2013.[6]

Despite its association with substantial adverse events and health-care costs, delirium remains underdiagnosed.[1] Challenges to diagnosing delirium are often present but vary depending on the provider and the patient. One common challenge results from the difficulty in differentiating between delirium and dementia, as providers might attribute the signs and symptoms of delirium to "dementia," assuming that the cognitive change is not new to the patient. Determining the acuity of onset of the cognitive change often requires some detective work such as calling a nursing facility or querying a caregiver. The physician must then trust this assessment of the patient's baseline mental status. Providers might also have difficulty determining if a somnolent or lethargic patient has delirium, as this takes time and effort. The provider must attempt to arouse the patient to perform a cognitive assessment or initiate a conversation. Another challenge arises from the natural history of delirium. With delirium, cognition waxes and wanes, making brief assessments at discrete points in time sometimes inadequate. Providers again have to obtain collateral information from a caregiver to better determine whether the patient has displayed signs of delirium at other points in time. Finally, delirium can be called many names; examples include altered mental status, sun-downing, agitation, and encephalopathy. Health-care providers might not know if their patients have delirium because of the lack of consistent terminology used by caregivers and colleagues. Despite these challenges, delirium prevention, recognition, and management are key to the care of the older patient.

Definition

The American Psychiatric Association's *Diagnostic and Statistical Manual*, 5th edition (DSM-5) provides the following diagnostic criteria for delirium:

(1) Inattention and reduced awareness
(2) Change in cognition
(3) Acute onset and fluctuating course
(4) Presence of underlying medical cause based on evidence from the history and physical exam

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Pathophysiology

The pathophysiology of delirium is complex, remains incompletely understood, and the causes are likely diverse depending on the individual and the factors contributing to the delirium. It involves a combination of neurotransmitter imbalances, cerebral hypoperfusion, microglial dysregulation, diurnal dysregulation, and a cytokine surge in the central nervous system (CNS), among other things. The microglial and cytokine alterations lead to a neuroinflammatory state and accelerated neuronal aging.[8] A combination of these alterations leads to the clinical syndrome that we observe as delirium.

Delirium is most commonly associated with a deficiency in acetylcholine, excess dopamine, and excess glutamate. Of note, imbalances of other common neurotransmitters such as serotonin and GABA-aminobutyric acid vary depending on the driving insult that led to the delirious event (i.e., trauma, sepsis, alcohol withdrawal, etc.).[8] It is important to consider these interpatient differences, as CNS active medications can have varying effects in delirious patients depending on the nature of the CNS action and driving mechanism of the patient's delirium. For example, GABA potentiating medications may be useful when treating alcohol withdrawal but can be detrimental for patients who are experiencing delirium due to other causes.[8]

A thorough and detailed review of the pathophysiology is beyond the scope of this chapter. If one wishes more detailed information, we would suggest one of Dr. Maldonado's prior reviews.[8]

Etiology

Like many geriatric syndromes, the cause of delirium is usually multifactorial, and the multifactorial model of delirium is well described and widely accepted.[6] This model focuses on the interplay between predisposing factors and precipitating factors.[6] Predisposing factors, also termed intrinsic factors, are characteristics that patients have at baseline that increase their vulnerability to delirium.[6] Precipitating factors, or extrinsic factors, are the additional insults that patients encounter that result in delirium when combined with the predisposing factors.[6] Patients with significant predisposing factors, such as cognitive impairment, high medical comorbidity, and polypharmacy, may only need a small precipitating factor, such as constipation or bedrest, to tip them into delirium.[6] Patients with either few or minor predisposing factors will need the addition of several significant precipitating factors, such as heart failure, pneumonia, and electrolyte abnormalities, to develop delirium.[6] The most common predisposing factor is cognitive impairment, but other examples of predisposing factors and precipitating factors are listed in Table 12.2.

Table 12.2 Predisposing and precipitating factors for delirium

Predisposing	Precipitating
Cognitive impairment	Infection
Comorbidity	New medication
Polypharmacy	Surgery
Frailty	Restraint use
Malnutrition	Immobility
Dementia	
Depression	Sleep deprivation
History of falls	Illness
Alcohol abuse	Dehydration
Vision impairment	Fracture
Hearing impairment	Neurologic disease
Age >65	Constipation
Male sex	Urinary retention
Neurologic disease	New environment or schedule
Low level of activity	Polypharmacy
Functional dependence	Pain

Adapted from NEJM 2006.[10]

Certain medications, such as sedatives, opioids, and anticholinergics, are associated with the development of delirium.[6] These medications should be used cautiously in older adults at risk for delirium, and stopping or tapering these medications may be part of the management plan (refer to Table 12.3 and Management section). An important caveat is that uncontrolled pain can also precipitate delirium, so opioid pain medications should be used if necessary to alleviate discomfort.[9]

The cause of delirium is usually multifactorial, resulting from a combination of predisposing and precipitating factors.

Prediction of Delirium

The multifactorial model forms the basis for tools used to predict delirium in patient populations. For example, a prediction tool designed specifically for patients undergoing cardiac surgery gives points for prior stroke or transient ischemic attack, Mini-Mental State Examination score, abnormal albumin, and Geriatric Depression Scale

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Table 12.3 Recommended strategies for using medications in patients at risk for delirium

Opioids	Utilize opioid-sparing techniques and medications such as routine administration of acetaminophen, heating pads, gentle massage, patches, or gels. Start with a low dose of opioids and frequently reassess, increasing dose as needed to achieve pain control. Use a bowel regimen to prevent constipation.
Benzodiazepines	Use the lowest dose possible for the shortest duration possible. Address anxiety and insomnia with nonpharmacological measures such as psychotherapy and sleep hygiene. Taper when discontinuing to avoid withdrawal, as withdrawal can also precipitate delirium.
Diphenhydramine	If possible, avoid and use second-generation antihistamines. Use the lowest dose possible and for the shortest duration possible.

score. Higher scores are associated with higher rates of delirium.[11] Other tools have been developed for other specific patient populations, such as patients in the emergency department[12], or at hospital discharge.[13] While helpful for population use, these tools cannot accurately predict which individual will develop delirium.

Prevention

Inouye et al. reported important results of a multicomponent intervention to prevent delirium in hospitalized older patients in the *New England Journal of Medicine* in 1999.[14] Hospitalized patients at risk for delirium were studied using a prospective matching strategy rather than a randomized controlled trial design, as this was the most cost-effective approach. Patients in the experimental arm received a multicomponent, protocoled intervention addressing six risk factors for delirium: cognitive impairment, sleep deprivation, immobility, visual impairment, hearing impairment, and dehydration. The study found that 15% of patients in the control arm developed delirium compared with 9% in the experimental arm.[14]

This pivotal study resulted in the development of the Hospital Elder Life Program, which has been implemented at more than 60 sites in five countries.[15] More information on the components of the Hospital Elder Life Program is provided in Table 12.4.

Geriatric consultation may have a role in the prevention of delirium in hip fracture patients, as demonstrated by a small randomized controlled trial.[16] Medications, including haloperidol, atypical antipsychotics, gabapentin, acetylcholinesterase inhibitors, and melatonin, have been studied, usually in the postoperative setting, but they were not efficacious in delirium prevention.[17]

Table 12.4 Components of the Hospital Elder Life Program

Daily Visitor Program: Orientation, socialization
Therapeutic Activities Program: Cognitive and social stimulation
Early Mobilization Program: Daily exercise
Nonpharmacological Sleep Protocol: Back rub, warm drink, relaxation tapes
Hearing and Vision Protocol: Adaptations provided
Oral Volume Repletion and Feeding Assistance Program: Assistance with meals

Adapted from the Hospital Elder Life Program.[14]

Up to one third of delirium cases can be prevented with a multifactorial intervention (the Hospital Elder Life Program).

Diagnosis

The diagnosis of delirium is best made using the Confusion Assessment Method, or CAM.[18] This algorithm, which was developed by an expert panel, utilizes four cardinal diagnostic criteria for delirium: acute onset and fluctuating course, inattention, disorganized thinking, and altered level of consciousness. The patient must have the first two criteria and either the third or fourth to have a positive test. The test was validated against psychiatrists' diagnoses and was determined to have a sensitivity of 94–100% and specificity of 90–95%.[17]

An acute onset of cognitive change (criterion 1 of the CAM) is key to the diagnosis of delirium and helps differentiate delirium from dementia. The interviewer must question the caregiver regarding the patient's baseline mental status and rely on the history provided to determine the acuity of onset and fluctuating course.

Patients with inattention (criterion 2 of the CAM) have difficulty engaging in conversation, following commands, and performing simple repetitive tasks, such as subtracting serial sevens, tapping on the letter *A* in SAVEAHEART, or spelling "world" backwards. They may attend to other stimuli, either internal such as their own thoughts, or external, such as their bedsheets,

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clothing, or IVs. A classic presentation of an inattentive delirious patient is the patient who is too busy picking at the bedsheets to focus on conversation.

Disorganized thinking (criterion 3 of the CAM) is not required for the diagnosis of delirium unless the patient has a normal level of consciousness (see criterion 4 of the CAM).[18] Disorganized thinking is noted when a patient's speech is unpredictable, incoherent, paranoid, or illogical. A patient might, for example, refuse medications, believing that they are poison.

The last diagnostic criterion is an altered level of consciousness. A patient with an altered level of consciousness is either more or less active than a person with normal alertness. A patient with delirium who is agitated, hypervigilant, easily startled, and restless has hyperactive delirium. Alternatively, a patient who is difficult to arouse, lethargic, and sleepy has hypoactive delirium. Both of these variations can be alarming and difficult to manage.

The Confusion Assessment Method is very sensitive and specific for the detection of delirium.

Evaluation

Once a patient has been diagnosed with delirium using the CAM, an evaluation to determine the possible underlying causes of the delirium starts with a full history, including a complete medication review, and a thorough physical exam.[9] If the history is limited by the patient's mental state or ability to cooperate, an attempt should be made to question available caregivers about the patient's health and behavior in the days prior to the presentation.[9]

Possible underlying etiologies of delirium may be remembered using the mnemonic DELIRIUM (see Table 12.5), which can guide the history and physical.

Delirium may present as the only manifestation of a severe illness, but it usually results from more than one factor.[9] Clinical suspicion coupled with findings on the history and physical exam should guide the diagnostic workup.[9] Additionally, the evaluation and treatment plan should take into account the patient's goals of care. Delirium is often present at the end of life when a palliative or comfort-oriented approach should be considered with the patient and his/her primary decision-maker. Patients with delirium often lack capacity to make significant medical decisions, so this role may be delegated to the designated decision-maker: the person with health-care power of attorney or next of kin.

While many additional studies may be part of the delirium evaluation, some tests to consider are listed in Table 12.6.

Management

The management of delirium requires multiple interventions targeting the precipitating and predisposing factors that contributed to its development.[9] Management should focus on treatments that enhance recovery, maximize function, and improve outcomes while minimizing the negative consequences of delirium.[9] These vary from patient to patient; therefore, certain aspects of the management approach are unique to each patient. For example, a palliative or comfort-oriented approach may be preferred by a patient at the end of life. Research on the effectiveness of these individualized management plans is difficult to perform and not available at this time; nonetheless, this approach is recommended until further research results are available.

Precipitating factors, such as infections, dehydration, and constipation, are often easier to address in the management plan than predisposing factors, such as cognitive impairment or hearing impairment, which may be long-standing issues. Nonetheless, even persistent conditions may be improved; for example, providers can address hearing impairment by removing cerumen impaction and providing noise amplifiers or hearing aids.

Pain can be difficult to assess in a patient with delirium since patients may have difficulty self-reporting, and agitation and somnolence from the delirium can mimic

- Does the patient have inattention?
- If yes, is it an acute onset and/or fluctuating?
- If yes, does the patient have either disorganized thinking OR altered level of consciousness?
- If yes to all three questions, the patient is CAM-positive.

Figure 12.1 Confusion Assessment Method (CAM).[18]

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Table 12.5 DELIRIUM mnemonic to guide evaluation

Letter		Examples	Questions for caregiver	Examples of diagnostic studies
D	Drugs/lack of drugs	New or recent medications, e.g., benzodiazepines; alcohol intoxication/withdrawal	Any new or recent medication changes? Any OTC medications? Illicit drugs? Alcohol use? When was his/her last drink of alcohol?	Urine drug screen, blood alcohol level
E	Electrolyte abnormalities	High or low glucose, sodium, calcium, urea nitrogen	Has the patient been eating and drinking well? How does s/he access food? Has s/he lost/gained weight?	Chemistry panel, albumin, prealbumin
L	Lack of sleep/consistent routine	New home, new caregiver	Describe the patient's routine. Any changes recently?	
I	Infections	Pneumonia, UTI, acute cholecystitis, cellulitis, abscess, diverticulitis, appendicitis, gastroenteritis	Has the patient had a fever? Cough? Abdominal pain? Nausea or vomiting?	Lung, skin, and abdominal exams; CBC, urinalysis, urine culture, CT scan, *C. difficile* assay
R	Reduced sensory input	Vision or hearing loss	Does the patient wear eyeglasses or hearing aids?	Ear exam, eye exam, examination of hearing aids
I	Intracranial	Intracranial hemorrhage, stroke, seizure	Has the patient hit his/her head? Is the patient on an anticoagulant?	Neuro exam, head CT, EEG
U	Urinary retention, constipation	Fecal impaction, acute urinary retention	Has the patient had difficulty with urination, bowel movements? When was the last bowel movement?	Abdominal X-ray, bladder scan, rectal exam
M	Major organ dysfunction	Myocardial infarction, kidney failure, respiratory failure with CO_2 retention, mesenteric ischemia	Has the patient complained of chest pain, shortness of breath, abdominal pain, blood in the stools?	Cardiac exam, EKG, CBC, cardiac enzymes, chemistry panel, ABG, hemoccult

Table 12.6 Examples of diagnostic studies in the evaluation of patients with delirium

Test	Diagnoses considered	Threshold for ordering	Comments
Chest X-ray	Pneumonia, pulmonary edema from congestive heart failure	low	Chest X-ray can rarely be negative early in the course of pneumonia
EKG	Arrhythmia, ischemia	low	Compare to baseline EKG, if available
Basic metabolic panel	Renal failure, electrolyte abnormalities	low	Compare renal function to baseline renal function, if available
CBC	Leukocytosis, anemia	low	Compare to baseline counts, if available
Cardiac enzymes	Ischemia	low	
Urinalysis	Infection	low	Older women may have abnormal findings that do not indicate infection unless signs and symptoms of infection are present
Urine culture	Infection	low	Older women may have positive cultures (bacteriuria) that do not indicate infection unless signs and symptoms of infection are present
Liver function tests	Cholestasis, hepatitis	intermediate	If fever is present, consider obtaining a right-upper-quadrant ultrasound to evaluate for acute cholecystitis, which can present atypically in older adults
Abdominal X-ray	Constipation, bowel obstruction	intermediate	Consider CT if significant concern for intra-abdominal pathology (such as diverticulitis, appendicitis)
Head CT or MRI	Hemorrhage, stroke	intermediate	Lower threshold in patients who have fallen or are on anticoagulants, such as warfarin
EEG	Seizure	very high	Can help differentiate delirium from other conditions, assess for occult seizures
Urine drug screen	Drug exposure	low	
Blood alcohol level	Intoxication	low	Only helpful in acute intoxication
Vitamin B12 level	Low B12	intermediate	Rare as the cause of acute delirium
Thyroid function tests	Hypothyroidism, hyperthyroidism	low/intermediate	
Arterial blood gas	Hypercarbia, hypoxia	intermediate	
Lumbar puncture	Meningitis	high	Consider in patients with fever and/or headache and/or nuchal rigidity

under- and overtreatment with opioids. Undertreatment of pain can lead to delirium; therefore, adequate pain control is essential in the management of delirium.[9] A combination of opioid-sparing agents, such as acetaminophen, lidocaine patches, and topical NSAID creams, and approaches, such as heating pads and gentle massage, in addition to opioids may ensure pain relief (see Table 12.3).

All patients with delirium would likely benefit from certain interventions, and this recommendation is extrapolated from the delirium prevention trials[14] and studies on the "delirium room," also called the "restraint-free room."[19] This room operates on the principle of "Tolerate, Anticipate, and Don't Agitate," and has special features (outlined in Table 12.7) that may lessen the negative outcomes associated with delirium.[19] For example, in the delirium room, physicians try to limit continuous intravenous infusions that tether the patient to an IV pole for prolonged periods, instead using boluses through IVs that are covered by soft gauze.

Physicians should try to untether patients as much as possible to allow for maximum mobility.[19] Restraints,

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ranging from soft wrist restraints to telemetry leads and intravenous infusions, should be minimized.[19] Bladder catheters should be removed as soon as possible, and patients should be encouraged and, if needed, assisted to use the toilet.[19]

Another recommendation in the management of delirium is to encourage a normal sleep–wake cycle.[14] Allowing natural daylight and artificial light to keep a room well lit during the day and avoiding night-time disruptions, such as lab draws and vital sign checks, help patients attend to their natural biological rhythm.

Family members or familiar caregivers should be encouraged to stay with a patient with delirium to provide reassurance and orientation.[14] Education on how to interact with the patient, particularly if the patient is agitated, may be needed, but families and caregivers are usually able to quickly learn this skillset (see Figure 12.2).

If these measures are not able to adequately address severe behavioral and emotional symptoms, most experts in the field concede that antipsychotics may be added to the nonpharmacological approach. Antipsychotics and their role in treating delirium are discussed further in the next section.

Table 12.7 Features and strategies of the delirium room (DR)

All patients can be seen by the nursing staff
The DR is near the nurse's station
Physical restraints are banned
Patients are screened for delirium every shift
Nurse and physician are involved in evaluation of delirium
Pharmacological interventions are a last resort
Physicians should evaluate an agitated patient at the bedside
If reorientation is not successful, do not continue to attempt reorientation
Hide necessary and remove unnecessary "attachments"
Encourage patient to be out of bed and assist or observe depending on patient safety

Adapted from Flaherty 2011.[19]

The management of delirium first involves addressing predisposing and precipitating factors that contributed to its development.

Pharmacological Treatment

Delirium can resolve and improve over time without pharmacological intervention, and studies do not consistently show that any medication lessens the severity or reduces the duration of delirium.[20] There are two medications worth mentioning that have been considered recently for treatment: antipsychotics and melatonin.

Hyperactive

- Remain calm
- Approach the patient slowly
- Use a reassuring and calming tone
- Use gentle touch if patient appears receptive; if patient appears guarded, respect his/her personal space as much as possible
- Ask easy-to-follow questions or speak using easy-to-follow statements
- Avoid restricting the patient's movements with tethers, such as oxygen tubing, telemetry, catheters, and IVs
- Distract the patient from any distressing stimuli by providing alternative stimuli such as a vest or apron that has strings, buttons, and other textures to provide tactile stimulation (often called a busy-vest/apron)

Hypoactive

- Get close to the patient's face so that s/he can hear and see you as easily as possible
- Speak using easy-to-follow statements
- Touch the patient's arm or shoulder with increasing firmness to attempt to arouse
- Put glasses and hearing aids on the patient after ensuring that these assistive devices are clean and functional
- Help the patient sit in an upright position, preferably in a chair

Figure 12.2 Recommendations for how to interact with patients with delirium.

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Both typical and atypical antipsychotics have been used clinically for the treatment of delirium. The MIND-USA study, published in 2019, was a multicenter, randomized, controlled trial comparing placebo to haloperidol (typical antipsychotic) to ziprasidone (atypical antipsychotic).[21] This study randomized 566 patients who had developed delirium in the setting of critical illness to receive intervention from one of these three arms. The primary outcome was a delirium and coma-free days, a commonly used outcome that allows for inclusion of comatose patients in delirium studies. There was no difference in delirium and coma-free days in the three arms. Based on these results, one can conclude that antipsychotics do not have a role in the management of delirium.[21] However, clinical experience has demonstrated that some patients with hyperactive delirium do respond to these medications. It is the opinion of the authors that antipsychotics do have a role in agitated delirious patients who are a danger to themselves or others. When utilizing these medications, one should follow standard geriatric practices. as these medications are associated with harms such as extrapyramidal symptoms, prolonged QT interval, and sedation.[20] They should be used for as short a duration and at as low a dose as necessary. We recommend that these medications in the inpatient setting should be reviewed for necessity on at least a daily basis. Given that these medications have a black box warning for use in older adults, providers should attempt to communicate with families and caregivers about the risks and benefits. If time allows, a baseline EKG should be obtained to ensure that the patient does not have a prolonged QT interval, as almost all antipsychotics prolong the QT interval.

Interest in using melatonin for the management of delirium has recently grown. There are multiple studies that have suggested that delirium induces a melatonin-deficient state, which likely leads to circadian disruption given the role that melatonin has in regulating sleep–wake cycles.[8] There have been multiple studies that examine the use of melatonin for delirium treatment in older adults. To date, there is not strong evidence that melatonin results in less severe or shorter duration of delirium once it has developed.[22] Given that it is well tolerated with a favorable side-effect profile, however, it can be considered in certain patients who are experiencing night-time hyperactivity. Further large studies are required to fully delineate the role of melatonin in the management of delirium.

Conclusions

Delirium, a neurocognitive disorder characterized by an acute change in attention, usually results from a combination of precipitating and predisposing factors and can be the only manifestation of a severe underlying illness. The prevention, recognition, and management of delirium are essential to reducing the morbidity and mortality associated with this disorder. The components of the Hospital Elder Life Program should be part of the daily routine of our hospitalized older patients to reduce the incidence of delirium. Providers should regularly screen hospitalized older adults for delirium using the CAM to improve detection. Once identified, a thorough history and physical should guide further evaluation and management, which involves addressing the precipitating and predisposing factors and providing support to the patient to maximize independence and functionality. Addressing delirium in these ways is a fundamental and critical part of geriatric patient care.

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Chapter 13

Diagnosis and Management of Heart Disease

Joshua M. Stolker and Michael W. Rich

Global Burden of Heart Disease in Older Adults

Cardiovascular disease is the leading cause of death and major disability worldwide, and the global burden of heart disease is increasing, in large part because of the aging of the population. In the United States, nearly two thirds of all cardiovascular hospitalizations occur in patients aged 65 and older, over 80% of cardiovascular deaths occur in geriatric individuals, and an estimated 70% of Americans over age 70 have clinically recognized cardiovascular disease.[1,2] For these reasons, practitioners should become familiar with strategies for the prevention and management of cardiovascular disease in older adults.

Effects of Aging on the Cardiovascular System

Aging is associated with diverse alterations in cardiovascular structure and function (Table 13.1).[3] Some of the most clinically relevant changes include increasing vascular stiffness, impaired endothelial function, impaired left ventricular (LV) relaxation and compliance, diminished responsiveness to neurohormonal signals such as β-adrenergic stimulation, and degeneration of the sinus node and electrical conduction system. These factors contribute to the development of systolic hypertension, heart failure (HF), coronary artery disease (CAD), aortic and mitral valve disease, and electrical disturbances including bradyarrhythmias and atrial fibrillation (AF). In addition, the effects of aging modulate the clinical presentation and response to cardiovascular therapies in the geriatric population. Similarly, aging affects other organ systems (Table 13.2) that frequently interact with cardiovascular diseases and therapeutics. Medical comorbidities, altered pharmacokinetics and pharmacodynamics, behavioral and social factors, and financial concerns also impact prognosis and goals of care in older individuals with cardiovascular disease.[4]

Ischemic Heart Disease

Epidemiology and Risk Factors

Based on data from the Atherosclerosis Risk in Communities study, approximately 720,000 Americans will have a new ischemic heart disease (IHD) event each year, defined as first hospitalization for myocardial infarction (MI) or coronary-related death.[2] These numbers have declined modestly since the 1990s and early 2000s, but the average age for first MI remains around 66 years for men and 72 years for women. In other studies, the median age at death from IHD was about 70 years for men and 80 years for women (i.e., half of all IHD deaths in women occurred in the small fraction of the population 80 years of age or older).[2]

Increasing age is the strongest predictor of CAD, as exemplified in risk assessment tools such as the Framingham Risk Score[5] and demonstrated by incidence and prevalence data from epidemiologic studies (Figure 13.1).[2]

Age-related changes in the arterial wall and prolonged exposure to traditional atherosclerotic risk factors contribute to the paramount importance of advancing age as a potent predictor of CAD. Apart from age, major risk factors for CAD include hypertension, dyslipidemia, tobacco use, family history (via incompletely defined genetic factors), and behavioral and environmental factors such as atherogenic diet and sedentary lifestyle. Diabetes mellitus is considered a CAD risk-equivalent, since people with diabetes but without known CAD experience 7-year MI rates that are similar to patients without diabetes who already have experienced an MI.[6] Importantly, the prevalence of hypertension, dyslipidemia, diabetes, and sedentary lifestyle all increase with age,[1] further predisposing older individuals to the development of CAD. In addition, chronic inflammatory conditions and subclinical cardiovascular disease are highly prevalent in the geriatric population, which magnifies the risk of ultimately developing symptomatic CAD.[1,7]

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Table 13.1 Effects of aging on the cardiovascular system

- Gross anatomy
  - Increased left ventricular wall thickness and decreased cavity size
  - Endocardial thickening and sclerosis
  - Increased left atrial size
  - Valvular fibrosis and sclerosis
  - Increased epicardial fat
- Histology
  - Increased lipid and amyloid deposition
  - Increased collagen degeneration and fibrosis
  - Calcification of fibrous skeleton, valve rings, and coronary arteries
  - Shrinkage of myocardial fibers with focal hypertrophy
  - Decreased mitochondria, altered mitochondrial membranes
  - Decreased nucleus: myofibril size ratio
- Biochemical changes
  - Decreased protein elasticity
  - Numerous changes in enzyme content and activity affecting most metabolic pathways, but no change in myosin ATPase activity
  - Decreased catecholamine synthesis, esp. norepinephrine
  - Decreased acetylcholine synthesis
  - Decreased activity of nitric oxide synthase
- Conduction system
  - Degeneration of sinus node pacemaker and transition cells
  - Decreased number of conducting cells in the atrioventricular node and His-Purkinje system
  - Increased connective tissue, fat, and amyloid
  - Increased fibrosis in and around the atrial conduction system
  - Increased calcification around conduction system
- Vasculature
  - Decreased distensibility of large- and medium-sized arteries
  - Impaired endothelial function
  - Aorta and muscular arteries become dilated, elongated, and tortuous
  - Increased wall thickness
  - Increased connective tissue and calcification
- Autonomic nervous system
  - Decreased responsiveness to β-adrenergic stimulation
  - Increased circulating catecholamines, decreased tissue catecholamines
  - Decreased α-adrenergic receptors in left ventricle
  - Decreased cholinergic responsiveness
  - Diminished response to Valsalva and baroreceptor stimulation
  - Decreased heart-rate variability

Clinical Presentation

Whereas substernal chest pain or pressure is considered the hallmark of obstructive CAD, geriatric individuals frequently present without classic angina. Exertional angina may not occur because of sedentary lifestyle or limited functional capacity in older individuals, so the diagnosis of CAD may be delayed. In addition, older patients may experience dyspnea, nausea or gastrointestinal distress, presyncope or syncope, generalized malaise or fatigue, diaphoresis, altered mental status, or even no symptoms at all when experiencing acute coronary syndromes (ACS). Thus, the proportion of subjects with acute MI who present with chest pain decreases with age, although dyspnea and exertional fatigue remain prominent presenting symptoms.

Physical findings are variable, but subacute or late presentations in the geriatric population with MI may lead to more profound cardiovascular decompensation and associated signs of HF, hypotension, or shock. Other findings may include pallor, confusion, tachycardia, low-grade fever, leukocytosis, or elevated C-reactive protein. Individuals presenting with hemodynamic compromise, prolonged ischemia, or life-threatening arrhythmias have

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a markedly increased risk of death, and the mortality rate following acute MI increases exponentially with age.[8]

Diagnosis of Myocardial Infarction

In patients with myocardial ischemia, the electrocardiogram (ECG) often demonstrates ST-segment elevations and/or depressions, as well as T-wave abnormalities. However, older patients are less likely to exhibit diagnostic ECG changes because of preexisting conduction system disease (e.g., left bundle branch block), ventricular paced rhythm, LV hypertrophy, prior MI, metabolic and electrolyte abnormalities, or medications (e.g., digoxin or antiarrhythmic drugs).

The diagnosis of MI is established when acute or subacute clinical symptoms are associated with elevated serum biomarkers of myocardial necrosis (troponin or creatine kinase-MB fraction).[9] Troponin-I and troponin-T have excellent sensitivity and specificity for diagnosing acute MI, and the development of high-sensitivity troponin assays has led to improved early detection of myocardial injury. However, these newer tests also result in higher "false-positive" rates,[10] leading to overdiagnosis of acute MI. This is particularly problematic in older adults who tend to have higher ambulatory troponin levels[11] or other medical conditions associated with non-MI troponin elevations (e.g., pneumonia, pulmonary embolus, renal dysfunction).[9]

The high prevalence of atypical presentations and nondiagnostic ECG findings in the geriatric population therefore requires a higher index of suspicion for acute MI in older subjects. Delayed diagnosis is common, reducing the window of opportunity for implementing appropriate treatment and limiting the extent of ischemic damage. Treatment delays also increase the risk for complications, including HF, arrhythmias, hypotension, myocardial rupture, and shock.

Table 13.2 Effects of aging on other organ systems

- Kidneys
  - Gradual decline in glomerular filtration rate, ~ 8 mL/min/decade
  - Impaired fluid and electrolyte homeostasis
- Lungs
  - Reduced ventilatory capacity
  - Increased ventilation/perfusion mismatching
- Neurohumoral system
  - Reduced cerebral perfusion autoregulatory capacity
  - Diminished reflex responsiveness
  - Impaired thirst mechanism
- Hemostatic system
  - Increased levels of coagulation factors
  - Increased platelet activity and aggregability
  - Increased inflammatory cytokines and C-reactive protein
  - Increased inhibitors of fibrinolysis and angiogenesis
- Musculoskeletal system
  - Decreased muscle mass (sarcopenia)
  - Decreased bone mass (osteopenia), esp. in women

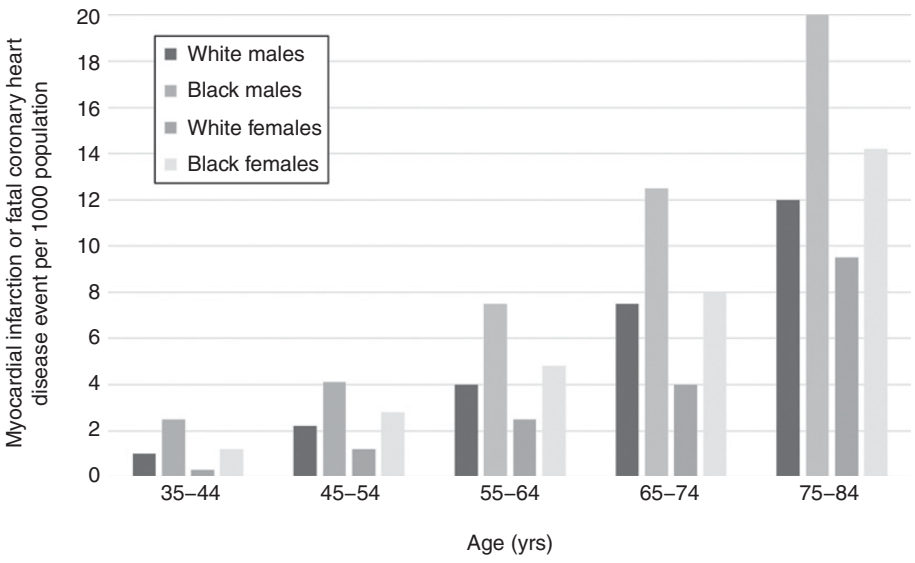

Figure 13.1 Incidence of myocardial infarction or fatal coronary heart disease in the Atherosclerosis Risk in Communities study, 2005 to 2014. (A black-and-white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

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Other cardiovascular and medical conditions with similar symptom complexes should be included in the differential diagnosis for MI, especially because many of these diseases also occur more frequently in older individuals. Chest pain without ECG changes could signify unstable angina, but other life-threatening conditions such as pulmonary embolism, aortic dissection, acute pericardial disease, pneumonia, severe peptic ulcer disease, cholecystitis, pancreatitis, or esophageal rupture must also be considered.

Pharmacologic Management of Myocardial Infarction

Acute MI with ST-segment elevation usually involves atherosclerotic plaque rupture and associated thrombotic occlusion of an epicardial coronary artery. However, MI without ST-segment elevation is more common in the geriatric population, often because of baseline ECG abnormalities, severe stenosis of multiple arterial territories (thus causing "balanced" ECG changes that can mask ST-segment elevation), or plaque rupture and thrombosis in smaller vessels. Alternatively, acute MI may result from a mismatch between oxygen supply and demand in the setting of fixed coronary obstruction. These latter infarcts are designated "Type II" in the universal classification of MI schema.[9] Thus, MI in older adults may occur in association with an infection (e.g., pneumonia or sepsis), significant hypertension or hypotension, tachy- or bradyarrhythmias, anemia, perioperative volume shifts, or other systemic illness such as thyroid disease. For both mechanisms of MI, therapies are directed toward improving coronary blood flow, reducing myocardial oxygen demand, reducing the risk of coronary thrombosis, correcting any precipitating illness, reducing sympathetic tone, and preventing adverse remodeling of hypoperfused myocardium.

The major therapeutic options for acute MI are listed in Table 13.3. Supplemental oxygen should be administered as needed to maintain an arterial oxygen saturation ≥92%, but routine administration of oxygen in patients with normal oxygen saturation is not beneficial. Intravenous access and telemetry monitoring are imperative to identify and treat potential complications of MI. As allowable by an individual's blood pressure, careful administration of nitroglycerin and analgesics should be provided as needed to control pain and dyspnea.

Antiplatelet therapy. Aspirin 160–325 mg should be administered immediately and traditionally has been continued indefinitely at a dose of 75–100 mg daily.[12–14] Aspirin reduces mortality in patients with unstable angina or acute MI, and the benefit of aspirin therapy increases with age, from a 1% absolute mortality reduction below age 60 to a 4.7% absolute mortality reduction at age 70 and older.[15] Clopidogrel, which binds irreversibly to the $P2Y_{12}$ receptor on circulating platelets, is a reasonable alternative (300–600 mg loading dose followed by 75 mg daily) in subjects unable to take

Table 13.3 Management of acute myocardial infarction

- General measures
  - Supplemental oxygen if arterial saturation <92%
  - Telemetry monitoring
  - Serial electrocardiograms and troponin measurements
- Antiplatelet and antithrombotic therapy
  - Aspirin
  - Platelet $P2Y_{12}$ receptor inhibitors (clopidogrel, prasugrel*, ticagrelor)
  - Systemic parenteral anticoagulants during MI (unfractionated heparin, enoxaparin, dalteparin, bivalirudin)
  - Rarely glycoprotein IIb/IIIa inhibitors (abciximab*, eptifibatide, tirofiban)
- Disease-modifying medications
  - Beta-blockers (if heart rate and blood pressure adequate), preferably oral
  - Angiotensin-converting enzyme inhibitors (ACEIs), or angiotensin receptor blockers if ACEIs contraindicated, if blood pressure and renal function adequate
  - Eplerenone (if reduced left ventricular systolic function or diabetes, in the absence of significant renal dysfunction or hyperkalemia)
  - High-potency statins
- Reperfusion therapy
  - Fibrinolysis (ST-elevation or new left bundle branch block only)
  - Percutaneous coronary intervention (PCI)
  - Urgent coronary bypass surgery in selected cases
- Other medications, only for compelling indications
  - Morphine for pain and dyspnea
  - Nitroglycerin for ischemia and heart failure
  - Calcium channel blockers (if persistent hypertension, immediate-release nifedipine contraindicated)
  - Antiarrhythmic agents (if refractory ventricular arrhythmias after acute MI)
  - Oral anticoagulation (select patients with anterior MI, atrial fibrillation, mechanical heart valves)
- Prior to hospital discharge
  - Assessment of left ventricular systolic function (e.g., echocardiogram)
  - Tobacco cessation counseling
  - Nutritional evaluation
  - Screening for depression
  - Exercise counseling and cardiac rehabilitation referral

* Only indicated for patients with acute coronary syndrome undergoing PCI.

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aspirin. Among patients with ACS, 12 months of aspirin plus clopidogrel reduces the risk of death or reinfarction by about 20% compared to aspirin alone.[16] Newer $P2Y_{12}$ receptor antagonists, such as prasugrel and ticagrelor, have been associated with improved outcomes relative to clopidogrel in younger patients with ACS, but prasugrel should be avoided in patients ≥75 years of age because of the increased risk of bleeding.[17] In contrast, ticagrelor improves outcomes relative to clopidogrel but without higher rates of major bleeding complications in older patients with ACS.[18,19]

Regardless of the antiplatelet medication chosen, the recommended duration of dual antiplatelet therapy after ACS remains 12 months in most cases.[14] However, some studies suggest longer treatment (up to 5 years after ACS) reduces ischemic events at a cost of increased bleeding,[20,21] while other data suggests that stopping the $P2Y_{12}$ inhibitor at 6 months after ACS may be reasonable among select patients at very high bleeding risk.[14] Thus, use of prolonged dual antiplatelet therapy after ACS in the geriatric population should be individualized.

Of note, older patients with ACS requiring systemic anticoagulation therapy for chronic indications such as AF or mechanical heart valves should generally be treated with aspirin and clopidogrel, avoiding the more potent agents prasugrel and ticagrelor, to mitigate bleeding risk. Whenever possible, all of the $P2Y_{12}$ agents should be withheld for 5–7 days prior to coronary bypass or other surgery because of the increased risk of perioperative bleeding.

Intravenous antiplatelet therapies include glycoprotein IIb/IIIa inhibitors (eptifibatide, tirofiban, abciximab), which block the final common pathway of platelet aggregation and reduce infarct size and reinfarction in subjects with non-ST-elevation ACS. Many interventional cardiologists use these medications for patients with large thrombus burdens during cardiac catheterization procedures as well. Overall, high-risk patients tend to gain the most benefit, particularly when undergoing percutaneous coronary intervention (PCI), but the risk of bleeding increases with age. Few studies have enrolled individuals over age 75, and one study demonstrated higher event rates in patients over age 80 receiving eptifibatide,[22] so the value of these agents in older patients remains unclear. Because of a high risk of hemorrhage, glycoprotein IIb/IIIa inhibitors are contraindicated in patients over age 75 receiving thrombolytic therapy for ST-elevation MI.[12] Nearly all of the IIb/IIIa clinical trials were performed before oral $P2Y_{12}$ inhibitor therapy became standard practice, so the incremental benefit of upstream intravenous loading in the absence of PCI – for older patients in particular – remains unclear. In contrast, the intravenous $P2Y_{12}$ inhibitor Cangrelor reduces ischemic events at 48 hours among patients undergoing PCI, with similar benefits among patients older or younger than 75 years of age.[23] Bleeding rates were similar between patients receiving or not receiving the drug, but high cost and greater adoption of the more potent oral $P2Y_{12}$ inhibitors have limited widespread acceptance of Cangrelor.

Antithrombotic therapy. In patients with acute MI, unfractionated or low-molecular-weight heparin should be administered immediately in addition to the antiplatelet agents described earlier – particularly in patients with high-risk features such as anterior MI, large infarct size, associated AF, or recurrent ischemia.[12,13] Low-molecular-weight heparins (e.g., enoxaparin, dalteparin) offer more predictable anticoagulation than unfractionated heparin, but declining renal function and increasing age are associated with increased rates of hemorrhagic complications. Nevertheless, the combined endpoint of death, MI, and recurrent angina is reduced,[24] with significant benefits in older subjects.[25] Low-molecular-weight heparin must be used with caution, if at all, in patients with severe renal insufficiency (creatinine clearance <30 mL/min).

Balancing risk of thrombosis versus bleeding. During hospitalization for ACS, many clinical decisions in older patients revolve around the competing risks of thrombosis versus bleeding. These include choice of antiplatelet and anticoagulant drugs, careful attention to medication dosing (especially in the setting of renal dysfunction and lower body weight), deciding whether to perform cardiac catheterization, radial versus femoral approach for catheterization, and stent choice during PCI. However, longer-term decisions must be evaluated as well, particularly when using more potent $P2Y_{12}$ inhibitors after ACS, and with many individuals having indications for chronic systemic anticoagulation therapy (e.g., AF, mechanical prosthetic heart valves, or thromboembolic disease).

An approach to minimizing chronic bleeding risk, studied in several recent clinical trials, involves the removal of aspirin after starting $P2Y_{12}$ inhibitor therapy. One study of patients undergoing PCI who also required chronic systemic anticoagulation demonstrated lower rates of bleeding and similar ischemic outcomes when withholding aspirin and treating patients with clopidogrel and warfarin alone.[26] However, this trial enrolled only a small number of older subjects (maximum age was

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80 years). Other related studies have shown similar findings, with lower bleeding rates but unclear effect on ischemic events when withholding aspirin therapy.[27] When evaluating antiplatelet therapy alone, one study of more than 7,000 patients receiving ticagrelor after PCI (two thirds for ACS indications) demonstrated lower bleeding rates when aspirin was stopped after 3 months, without an increase in ischemic events.[28] Similar findings have been reported with clopidogrel monotherapy when aspirin was stopped 1 month after PCI in patients with stable CAD.[29]

Despite these trends toward withholding aspirin while continuing $P2Y_{12}$ inhibitor therapy (and/or systemic anticoagulation, if indicated), no consensus exists within the cardiology community, and additional study of this clinical conundrum is needed. In geriatric patients with indications for antiplatelet therapy plus systemic anticoagulation, an individualized, patient-centered approach is warranted to balance the potential reductions in ischemic or thromboembolic events against bleeding risks.

Beta-blockers. Beta-blockers reduce mortality, recurrent ischemia, and arrhythmias in patients with acute MI.[12,13] Contraindications include bradyarrhythmias, hypotension, acute HF during MI, or active bronchospasm with wheezing. A history of obstructive lung disease alone should not preclude beta-blocker therapy. In a pooled analysis of several clinical trials, early treatment with beta-blockers reduced mortality by 23% in older patients with ACS, but had no effect in younger patients.[4] In addition, long-term beta-blocker treatment after MI is associated with 6 lives saved per 100 older patients treated, versus only 2.1 lives saved per 100 younger patients. However, older patients with acute MI are also at increased risk for serious adverse events associated with beta-blocker therapy, including HF, hypotension, shock, and death.[30] Current guidelines recommend institution of oral metoprolol or carvedilol within 24 to 48 hours in hemodynamically stable patients.[12] Doses should be lower and titration slower in older adults. Following MI, metoprolol, propranolol, and timolol are approved for long-term use, while carvedilol and metoprolol succinate are recommended after MI in patients with LV ejection fractions less than 40%.

Angiotensin and aldosterone inhibition. Angiotensin-converting enzyme inhibitors (ACEIs) reduce mortality in MI, particularly in geriatric individuals age 65–74,[31] and in the setting of HF, LV dysfunction, or anterior ST-elevation MI. Following MI, ACEIs reduce mortality by 17–34% in older patients, with an absolute benefit that is three times greater than in younger individuals.[32–34] Angiotensin receptor blockers (ARBs) (e.g., candesartan or valsartan) are suitable alternatives for ACEI-intolerant patients, but head-to-head trials have confirmed that ACEIs are the preferred medications in subjects with MI.[35] Serious potential adverse effects with both classes of drugs include hypotension, renal failure, and hyperkalemia; initiation and up-titration of these medications must be performed with caution in older patients with renal dysfunction or low blood pressure in the MI setting.

Eplerenone is a selective aldosterone antagonist that reduces mortality and cardiovascular hospitalizations in acute MI patients with LV systolic dysfunction (ejection fraction ≤40% at time of event) and either HF or diabetes.[36] Like spironolactone, eplerenone is a potassium-sparing diuretic that requires close monitoring of renal function and serum potassium levels during initiation and follow-up, particularly in the geriatric population.

Statins. Statin therapy improves clinical outcomes after acute MI and should be initiated in all patients prior to hospital discharge.[5,12,13] The benefits of statin treatment have been verified in geriatric patients with known CAD or vascular disease,[37–39] with one study demonstrating a 15% reduction in recurrent MI, stroke, or cardiovascular death in subjects age 70–82 years.[40] Nonetheless, the utility of statins after MI in patients over 80 years of age is unproven, as none of the major clinical trials enrolled patients in this age group.[41,42] In addition, older patients are at increased risk for statin-related myalgias during longer-term therapy,[43] and statins may be associated with fatigue and reduced physical activity levels in susceptible older individuals.[44,45] In general, secondary prevention after MI should prompt more aggressive attempts to achieve a low-density lipoprotein level below 70 mg/dL, including the addition of ezetimibe or injectable lipid therapies (alirocumab, evolocumab) if needed.[46] However, the intensity of lipid-lowering therapy in patients of advanced age should be individualized, taking into consideration the cardiovascular risk profile, life expectancy, comorbidities, potential side effects, and patient preferences.

Other agents. Calcium channel blockers have not been shown to improve outcomes in patients with acute MI, but may be useful after maximizing beta-blocker therapy in patients with ongoing ischemia, poorly controlled hypertension, or supraventricular tachyarrhythmias (diltiazem or verapamil). Empiric antiarrhythmic drugs (e.g., lidocaine or amiodarone), magnesium

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therapy, and glucose-insulin-potassium infusions are not recommended in acute MI in the absence of a specific indication.

Reperfusion in Acute ST-Segment Elevation Myocardial Infarction (STEMI)

Acute MI associated with ST-segment elevation or new left bundle branch block is usually caused by thrombotic occlusion of the infarct-related coronary artery, and numerous large trials have confirmed that prompt pharmacological or mechanical reperfusion reduces mortality and morbidity.[12] Importantly, although the potential risks of pharmacological reperfusion with fibrinolytic therapy or mechanical reperfusion with PCI are higher in older patients, the potential benefits are also higher, and the net benefit of reperfusion is at least as great in older as in younger patients. Therefore, older age per se is not a contraindication to reperfusion therapy, but the benefits must be weighed against the inherent risks, which increase progressively above 75 years of age.

Primary PCI is the preferred reperfusion strategy in patients of all ages with STEMI if it can be performed within 90 minutes of presentation (preferably within 60 minutes) because it is associated with higher efficacy and lower risk of intracranial hemorrhage (ICH) relative to fibrinolytic therapy.[12,47,48] In situations where PCI cannot be performed within 90 minutes, fibrinolysis is considered a suitable alternative, provided that the risk of bleeding is acceptable. In this regard, the risk of ICH is approximately twofold higher in patients ≥75 years of age treated with fibrinolysis compared to younger patients (1–2% vs. 0.5–1%). Of note, multiple observational registries and nonblinded clinical trials have suggested that complete revascularization reduces the risk of subsequent cardiovascular events among patients with STEMI and multivessel CAD.[49] Since the majority of older patients with STEMI have diffuse CAD involving multiple coronary territories, careful evaluation of each individual's clinical status and prognosis should be undertaken when planning the therapeutic approach (i.e., bypass surgery vs. multivessel PCI vs. medical therapy).

Reperfusion in Non-ST-Elevation Acute Coronary Syndromes

In older patients with ACS and no ST-segment elevation, thrombolytic therapy is contraindicated and the role of coronary revascularization is less clear. Investigators from the Thrombolysis In Myocardial Infarction (TIMI) group identified seven variables that confer increased risk in individuals presenting with unstable angina or non-ST-elevation MI (Table 13.4).[50] This TIMI Risk Score, and similar risk assessment algorithms from other sources, helps triage patients to an early invasive (i.e., coronary angiography) versus early conservative strategy (i.e., medical therapy, with invasive assessment only for subjects with recurrent ischemia or other clinical indications).[13] Several of the most important discriminators – including age 65 and older, elevated troponin, and ST-segment deviation at presentation – have also been associated with higher rates of in-hospital mortality in large observational databases.[13,51] Since older age is associated with worse outcomes in acute MI, early coronary angiography is recommended in geriatric patients with MI complicated by recurrent ischemia, HF, or hemodynamic instability who are suitable candidates for percutaneous or surgical revascularization.[52] However, individualized decision-making is required to carefully weigh the risks (e.g., contrast nephropathy, bleeding, potential for stroke or other embolic events) versus benefits related to invasive procedures in frail patients, those with complex comorbidities, and those with very advanced age (i.e., ≥85 years).

Patients with acute MI at highest risk for death are those presenting with cardiac arrest, HF, hypotension, or significant tachycardia, and the in-hospital mortality rate approaches 50% for subjects with cardiogenic shock.[53] Although early coronary revascularization is beneficial in patients up to age 75 with acute MI complicated by cardiogenic shock, the value of this approach in patients over age 75 is less certain.[54,55] Nevertheless, urgent revascularization is a reasonable option in selected older patients in the absence of other life-threatening conditions.

Table 13.4 TIMI Risk Score variables for predicting adverse clinical outcomes in non-ST-elevation acute coronary syndromes*

- Age 65 years or older
- 3 or more risk factors for coronary artery disease
- Prior coronary stenosis of 50% or more
- ST-segment deviation on presenting electrocardiogram
- At least 2 anginal events in the prior 24 hours
- Use of aspirin in prior 7 days
- Elevated serum cardiac markers

* The combined endpoint of mortality, myocardial infarction, and urgent revascularization increases in linear fashion, with Risk Scores 0–1 associated with a 4.7% risk of events at 2 weeks versus 8.3% for Risk Score 2, 13.2% for Risk Score 3, 19.9% for Risk Score 4, 26.2% for Risk Score 5, and 40.9% for Risk Scores 6–7.

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Complications of Myocardial Infarction

Heart failure occurs in up to 50% of older patients with acute MI and is the most common cause of in-hospital death. Treatment includes diuretics and vasodilator therapy, especially nitroglycerin and ACEIs. Beta-blockers should be administered if blood pressure and heart rate are adequate, and if volume overload is manageable with diuretics. In more advanced HF, inotropic agents may be required transiently until the patient stabilizes (e.g., dopamine, dobutamine, milrinone – see section on Heart Failure and Cardiomyopathy below).

Clinically significant right ventricular (RV) ischemia or infarction occurs in 10–20% of patients with acute inferior STEMI and portends an ominous prognosis in older adults.[56] Manifestations of RV infarction include hypotension and signs of right-sided HF. Treatment involves intravenous fluid administration to maintain RV filling pressure and inotropic therapy if needed.

In addition, patients with large anterior MI or evidence for LV thrombus have historically been treated with warfarin for 3 months after MI to maintain an international normalized ratio (INR) of 2.0–3.0. However, more rapid coronary revascularization and more aggressive medical therapies during and after ACS may contribute to a lower risk of thromboembolic complications than reported prior to the reperfusion era. As such, many practitioners have adopted strategies designed to mitigate bleeding risks after ACS, particularly among older patients. For example, scheduling a contrast-enhanced echocardiogram several weeks after the MI may permit earlier removal of systemic anticoagulation therapy if the LV wall motion has adequately recovered. Other practitioners may choose a direct oral anticoagulant (DOAC) such as apixaban, rivaroxaban, dabigatran, or edoxaban in order to reduce exposure to warfarin, and thus potentially reduce bleeding risks while prescribing concomitant antiplatelet therapy after ACS. However, one retrospective study suggested higher rates of stroke or systemic embolism with DOAC therapy among patients with established LV thrombus.[57] No studies have evaluated DOACs for the prevention of thrombus in this context, or the use of alternative DOAC dosing strategies (e.g., higher doses for the first several weeks, as currently recommended after diagnosing deep venous thrombosis or pulmonary embolus). As such, the safety and efficacy of DOACs as alternatives to warfarin following acute MI require further investigation.

Life-threatening mechanical complications occur in 1–2% of all patients with MI and have decreased in incidence during the reperfusion era. Mechanical complications include LV free wall rupture with pericardial tamponade, papillary muscle dysfunction or rupture with severe mitral regurgitation, rupture of the interventricular septum, and LV aneurysm or pseudoaneurysm formation. Advanced age is a potent risk factor for each of these catastrophic consequences of acute MI.[58] Care should be individualized, but all forms of rupture require urgent attention and surgical or transcatheter repair, if clinically feasible. Management of LV aneurysm depends on the size and degree of hemodynamic instability or thrombosis risk; in most cases, smaller aneurysms can be managed medically, whereas surgical repair should be considered for large aneurysms associated with HF or thromboembolic complications. LV pseudoaneurysm refers to a situation in which a free wall rupture has been locally contained by adherent pericardium. Pseudoaneurysms are prone to expand, leading to pericardial tamponade, so surgical repair is recommended.

Sustained ventricular tachyarrhythmias are generally treated with direct-current cardioversion or defibrillation, beta-blocker therapy, and correction of electrolyte abnormalities and ischemia. Selected individuals may require antiarrhythmic therapy or placement of a temporary external wearable defibrillator (LifeVest) for several weeks after MI,[59] particularly if new, life-threatening ventricular arrhythmias occur more than 48 hours after MI. However, none of these approaches are indicated for routine management or for prophylactic purposes.[12,60] Supraventricular arrhythmias such as AF should be treated according to standard recommendations (see section on Arrhythmias and Conduction Disturbances below). Bradyarrhythmias are common in patients with inferior wall MI but frequently resolve spontaneously once ischemia has been treated; some patients may require temporary transvenous or transcutaneous pacing. Permanent pacing may be necessary in subjects with persistent high-grade heart block (e.g., Mobitz type II second-degree atrioventricular block or complete heart block) after MI.

Diagnosis and Management of Stable Ischemic Heart Disease

After experiencing MI, or in the setting of stable ischemic heart disease (SIHD), all patients should be counseled in conjunction with their families or caregivers regarding the medication regimen, diet, and long-term recommendations for CAD management.[61] This is particularly important in older patients, for whom sensory or memory deficits combined with polypharmacy may adversely

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affect medication adherence. Modifiable risk factors should be targeted during the convalescent phase after MI or coronary revascularization, with nutritional counseling and tobacco cessation efforts addressed prior to discharge.[12,13] Systolic blood pressure should be controlled in accordance with current guidelines,[62] lipid therapy should be initiated or titrated appropriately using higher-potency statins,[63] diabetes management should be addressed,[64] and medical follow-up should be arranged. Cardiac rehabilitation reduces mortality and improves quality of life after MI, with similar benefits in younger and older patients. However, cardiac rehabilitation is significantly underutilized in the geriatric population relative to younger patients.[65]

With regard to type 2 diabetes mellitus in particular, patients with vascular disease and favorable life expectancy should be evaluated for cardioprotective therapy using metformin plus sodium-glucose cotransporter (SGLT2) inhibitors or glucagon-like peptide (GLP1) agonists shown to reduce cardiovascular events, independent of glycated hemoglobin (A1C) levels.[64,66,67] However, whereas long-term follow-up studies of metformin and all of the major SGLT2 inhibitor cardiovascular outcome trials have shown significant benefit for the subgroups of patients age 65 and older,[68–72] the GLP1 agonist trials have not consistently suggested benefit in older patient subgroups.[73–76] Thus, when renal function and blood pressure allow, and with counseling regarding surveillance for dehydration or urinary tract or yeast infections, select older diabetic patients with SIHD or other vascular disease may be considered for metformin and appropriate cardioprotective diabetes therapy.[64,66,67]

Chronic CAD, with or without prior MI, increases in prevalence with age – likely as a result of prolonged exposure to multiple cardiac risk factors in conjunction with structural and metabolic changes related to vascular aging. Atherosclerotic changes tend to be more diffuse in older adults, with a higher likelihood of left main and multivessel CAD. Compared to younger patients, geriatric patients tend to present with more advanced disease and fewer or no anginal symptoms because of comorbidities (e.g., diabetes), neuropsychiatric changes, and more sedentary lifestyles.

Indications for stress testing in geriatric patients are similar to those for younger individuals, including consideration of coronary calcium scoring or coronary computed tomography (CT) angiography, in select clinical circumstances.[63,77,78] However, clinicians should carefully screen older patients prior to stress testing, taking into consideration whether the patient is a suitable candidate for coronary angiography and revascularization, in the event the CT scan or stress test is abnormal and symptoms persist despite antianginal therapy. These decisions are particularly relevant given the low rate of cardiovascular events in the setting of contemporary medical therapy for chronic atherosclerotic CAD, irrespective of the choice of functional or anatomic testing.[78] In deciding whether to pursue noninvasive testing and possible coronary angiography, it should be recognized that revascularization has been shown to reduce symptoms and improve quality of life in patients with SIHD, but there is no evidence that it decreases the risk of MI or all-cause mortality beyond optimal medical therapy for CAD (see discussion below).[79,80]

Medical management of chronic stable angina includes aspirin, beta-blockers, nitrates, calcium channel blockers, and ranolazine – with beta-blockers being the anti-ischemic agents of first choice unless contraindicated.[61] Antiplatelet therapy after PCI for SIHD is generally less intensive than for patients with ACS. In the absence of bleeding events, patients with SIHD receiving bare metal stents should receive 6 months of $P2Y_{12}$ inhibitor therapy, but this can be held after 1 month if required because of bleeding or the need for elective surgical procedures. Those SIHD patients receiving drug-eluting stents should preferably be treated for 12 months, but several studies have demonstrated safety when holding the $P2Y_{12}$ inhibitor after 6 months (and sometimes after only 3 months, if needed) with the newer generations of drug-eluting stents.[14] Other studies have suggested that stopping aspirin after 1–3 months, and instead continuing the $P2Y_{12}$ inhibitor, may be another option.[28,29] Although some studies have shown modest reductions in cardiovascular events when using ticagrelor or low-dose rivaroxaban for certain subgroups of patients with SIHD, benefits appear to be attenuated among patients age 75 and older in these trials, and bleeding rates are substantially elevated.[81,82] Thus, these therapies should be used with caution among carefully selected older patients with persistently elevated ischemic risk and relatively low risk of bleeding.

When compared with medical management, coronary revascularization with PCI or coronary artery bypass graft (CABG) surgery reduces symptoms and improves quality of life,[79,80] including in the older patient population.[83] CABG also decreases mortality in certain high-risk subgroups (e.g., left main coronary disease, three-vessel disease with LV dysfunction, diabetes mellitus). However, optimal medical therapy in SIHD patients – even in those with significant ischemia on

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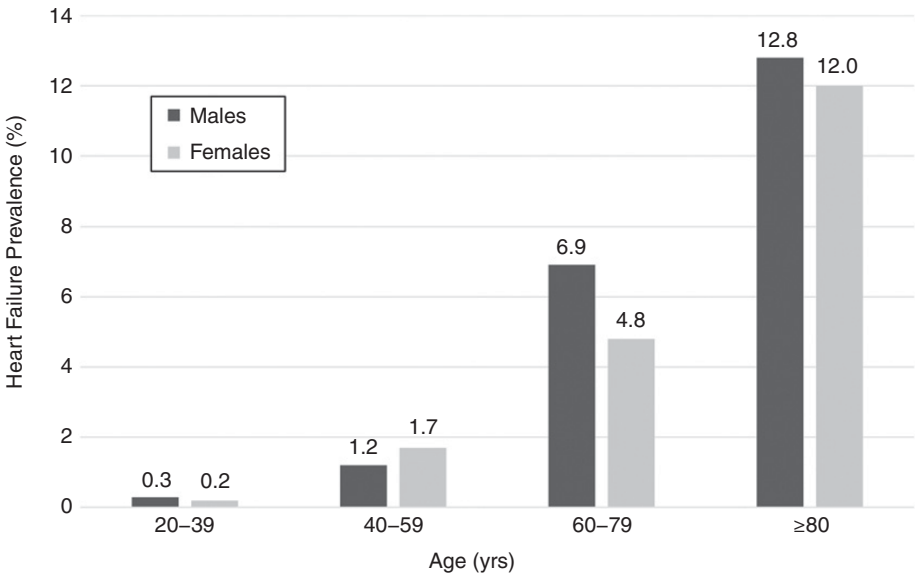

Figure 13.2 Prevalence of heart failure among adults in the National Health and Nutrition Examination Survey (NHANES) between 2013 and 2016. (A black-and-white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

stress testing – helps prevent subsequent cardiovascular events as effectively as routine revascularization.[79,80] Coronary angiography, PCI, and CABG in the geriatric population are associated with higher complication rates than in younger patients, likely as a result of more advanced and diffuse CAD, higher rates of LV dysfunction, and diminished cardiac reserve related to aging itself. Comorbidities play an important role as well, with vascular and renal disease contributing to procedural difficulty, bleeding complications, contrast-induced nephropathy, and stroke and other thromboembolic events. In addition, older patients undergoing CABG experience higher rates of postprocedural HF, arrhythmias (particularly AF), cognitive dysfunction,[84] and pulmonary complications than younger patients, and these adverse events contribute to increased length of stay and mortality. Despite the attendant risks, outcomes following PCI and CABG in older adults are generally favorable, and over 50% of patients undergoing these procedures in the United States are over age 65.[2]

Heart Failure and Cardiomyopathy

Epidemiology and Pathophysiology

Over 5 million Americans have HF, and there are more than 1 million hospitalizations with HF as a primary diagnosis each year.[85] With the aging of our population, HF prevalence continues to increase, from 5.7 million American adults in a cross-sectional survey between 2009 and 2012, up to 6.2 million adults between 2013 and 2016.[2] HF increases in both incidence and prevalence with increasing age, with up to 75% of HF hospitalizations occurring in patients age 65 and older and approximately 50% occurring in patients over age 75 (Figure 13.2).

In addition, HF is the costliest Medicare diagnosis-related group (DRG), and mortality from HF rises exponentially with age. Age-related changes in cardiovascular structure and function – including increased arterial stiffness, impaired LV diastolic relaxation and compliance, diminished responsiveness to beta-adrenergic stimulation, and dysfunction of the sinus node – all contribute to a marked reduction in cardiovascular reserve, predisposing older adults to the development of HF. In addition, increased vascular stiffness leads to a progressive rise in systolic blood pressure, which is a major risk factor for the development of HF in geriatric patients. Indeed, approximately 75% of HF cases have antecedent hypertension, although the increasing prevalence rates of CAD, diabetes, and valvular disease also contribute to the exponential rise in geriatric HF.

Etiology and Prevention

Most HF in the geriatric population is related to hypertension, CAD, valvular disease, or other nonischemic causes of dilated cardiomyopathy. Less common etiologies include tachycardia-induced cardiomyopathy (from chronic arrhythmias like AF, or high-output states such

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as anemia), hypertrophic cardiomyopathy (HCM), myocarditis, constrictive pericarditis, thyroid disease, and infiltrative diseases such as amyloid or hemochromatosis. Individuals with exposure to certain drugs, such as cocaine or chemotherapeutic agents (e.g., anthracyclines and trastuzumab), are also at risk of developing LV dysfunction.

Clinical guidelines emphasize prevention in high-risk populations – especially in subjects with multiple cardiovascular risk factors – and more aggressive titration of therapies in the presence of asymptomatic LV dysfunction, significant valvular disease, or symptomatic HF.[85] Large-scale clinical trials have verified that lowering blood pressure reduces the risk of developing HF,[86] and the greatest benefit is derived from control of systolic hypertension in subjects over age 75, with more intensive systolic blood pressure reduction to a target value below 120 mm Hg being more effective than treatment to a target of less than 140 mm Hg.[87] Heightened surveillance for adverse events is imperative, particularly among older or frail individuals, given higher rates of hypotension, renal dysfunction, or electrolyte abnormalities with more intensive blood pressure reduction.

Clinical guidelines reflect this data, recommending target systolic blood pressures below 130 mm Hg in all patients at risk of HF or with prior HF diagnosis, and irrespective of whether the ejection fraction is reduced or preserved (i.e., HFrEF or HFpEF, respectively).[88] In the geriatric population, deconditioning and pulmonary disease may contribute to exercise intolerance beyond that due to HF alone. Exercise training is recommended for patients with asymptomatic LV dysfunction or chronic HF in the absence of severe symptoms. Pharmacologic regression of LV hypertrophy in hypertensive subjects also reduces the incidence of HF and cardiovascular events.[89] Dietary counseling, including modest sodium restriction and avoidance of excessive fluid intake, may help reduce fluid retention and subsequent HF exacerbations. The importance of taking medications regularly should be discussed early after diagnosing HF, since medication nonadherence is a leading cause of rehospitalization for HF.

Clinical Features and Diagnosis

Classic symptoms of HF include shortness of breath (especially with exertion), exercise intolerance, orthopnea, paroxysmal nocturnal dyspnea, lower-extremity edema, fatigue, and weakness. Older patients with HF also commonly experience anorexia, bloating, psychomotor slowing, lethargy, altered sensorium, and gastrointestinal disturbances. Because older persons are often sedentary, exertional symptoms may be less prominent than in younger patients. Conversely, "atypical" symptoms such as anorexia and altered cognition become increasingly prevalent.

Assessing symptom severity in patients with HF is useful for identifying therapeutic goals, monitoring disease progression, and determining prognosis. Although there are several metrics available, the New York Heart Association (NYHA) functional classification is most widely used (Table 13.5).[90] Nearly 70% of patients with HF are in Class I or II, with mild limitations to routine physical activities. About 25% of patients experience more severe activity limitations (Class III), whereas only 5% of patients are Class IV, with symptoms during minimal exertion (e.g., going to the bathroom) or at rest. Patients with Class IV HF have a 1-year mortality rate of 25–50% – worse than for many forms of metastatic cancer.

Initial assessment should include a detailed history and physical examination. This is essential in the geriatric population, since patients may not present with typical symptoms or signs, and other medical conditions such as pulmonary disease or deconditioning may confound the clinical picture. Common precipitants of HF exacerbations in older adults include medication or dietary nonadherence, ischemia, uncontrolled hypertension, new arrhythmias (especially AF), infection, volume overload

Table 13.5 New York Heart Association functional classification

Class	General characteristics
I	Cardiac disease does not limit physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea, or anginal pain.
II	Cardiac disease results in slight limitation of physical activity. Patients are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea, or anginal pain.
III	Cardiac disease results in marked limitation of physical activity. Patients are comfortable at rest. Less than ordinary physical activity causes fatigue, palpitation, dyspnea, or anginal pain.
IV	Cardiac disease results in inability to carry on any physical activity without discomfort. Symptoms of cardiac insufficiency of anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased.

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(e.g., perioperatively or with blood transfusions), anemia, and drug interactions that adversely affect renal or cardiac function. Vital signs and volume status should be evaluated, and complete examination of the neck, chest, cardiovascular system, abdomen, and extremities should be performed. Routine laboratory studies include an assessment of electrolytes and renal function, and a complete blood count.[88] Select patients may require thyroid hormone assessment. An ECG is indicated, since HF is often precipitated by ischemia or arrhythmia. Chest radiography may be useful for diagnosing volume overload. Echocardiography should be performed at the time of initial diagnosis or when there is unexplained clinical deterioration in order to assess LV systolic and diastolic function, and to identify other structural abnormalities that may be contributing to the HF syndrome.

B-type natriuretic peptide (BNP) and its precursor N-terminal proBNP (NT-proBNP) are released by the myocardium when ventricular filling pressures are elevated, and these biomarkers are useful for both the diagnosis and management of HF.[91,92] In one large trial, BNP was effective in distinguishing cardiac versus noncardiac causes of dyspnea in the emergency department.[91] Natriuretic peptide levels increase with age and tend to be higher in women than in men.[93,94] Other disorders in the geriatric population also contribute to higher natriuretic peptide levels, including AF, renal dysfunction (especially for NT-proBNP), and pulmonary hypertension. As a result, the predictive accuracy of natriuretic peptides declines with increasing age.[93] To address this issue, several studies have demonstrated that the positive predictive value of NT-proBNP is improved by using age-stratified thresholds to diagnose HF: ≥450 pg/ml for age <50 years, ≥900 pg/ml for age 50–75 years, and ≥1800 pg/ml for age >75 years.[95] For all ages, an NT-proBNP <300 pg/ml effectively rules out acute HF. Thus, a normal BNP or NT-proBNP level makes acute HF unlikely, whereas a substantially elevated level of either biomarker greatly increases the likelihood of active HF. Similarly, persistently elevated biomarker levels despite aggressive treatment portend a less favorable prognosis.[88]

Management

The initial goals of therapy for acute HF exacerbations include hemodynamic stabilization and correction of volume overload. Care for older patients must be individualized, with comorbid conditions, functional limitations, and personal preferences being taken into consideration in designing a therapeutic plan. In addition, since few clinical trials have enrolled substantial numbers of subjects over age 75, most HF therapies are of uncertain benefit in older patients.[96] Multidisciplinary programs that provide individualized patient education and close follow-up have been shown to reduce hospitalizations and improve quality of life in older HF subjects.[97,98] Furthermore, exercise training and cardiac rehabilitation have been associated with improved exercise tolerance in all age groups,[99] leading to endorsement of cardiac rehabilitation for stable HF patients with HFrEF (EF ≤35%) by the Centers for Medicare and Medicaid Services.

Diuretics. Loop diuretics are an essential component of the acute management of HF with volume overload. Administered intravenously, these agents promote rapid natriuresis and increased urine output. Older patients are more sensitive to diuretic-induced electrolyte disturbances and volume shifts than younger subjects, so close monitoring is imperative in conjunction with regular assessments of renal function and electrolytes. Although loop diuretics have not been shown to improve survival, and there are conflicting data concerning the short- and long-term benefits of these agents,[100–102] relief of congestion is a primary goal of initial HF therapy.[103] In patients who do not respond adequately to loop diuretics, the addition of metolazone or other thiazide diuretics (usually administered 30–60 minutes before the loop diuretic) may facilitate diuresis, but renal and electrolyte disturbances are common. Dietary sodium restriction helps prevent fluid retention, and patients should be counseled to avoid salty foods and limit sodium intake to no more than 2 grams per day. Conversely, overzealous sodium restriction has not been shown to improve outcomes and may be harmful.[104] Patients should also be instructed to monitor daily weights at home. A baseline "dry weight" should be defined, and subjects should be advised to adjust their diuretic dosage or contact their health-care provider if their weight varies by more than 2–3 pounds from baseline over several days.

ACEIs. In patients with HFrEF, ACEIs reduce HF hospitalizations, improve quality of life, and decrease mortality by 25–30% – with similar effects in older and younger subjects.[105,106] Older patients experience higher rates of hypotension, hyperkalemia, and renal dysfunction during ACEI titration than younger individuals; close monitoring of these parameters is warranted. Up to 20% of patients may experience cough and a small percentage may experience angioedema from ACEIs, but these side effects do not appear to increase in frequency with

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advancing age. In the absence of adverse effects, ACEI dosages should be titrated to those studied in clinical trials (e.g., captopril 50 mg three times daily, enalapril 10 mg twice daily, lisinopril 20–40 mg daily, ramipril 10 mg daily). In addition, ACEI therapy in patients with asymptomatic LV dysfunction reduces progression to clinical HF,[107] so ACEIs are indicated in patients with LV systolic dysfunction (ejection fraction <40–45%) regardless of NYHA functional class.[88]

ARBs. While ACEIs are recommended as first-line therapy for patients with HFrEF, ARBs are suitable alternatives in individuals intolerant to ACEI because of cough or angioedema.[108,109] In a series of three trials involving patients with HF, the ARB candesartan reduced the composite endpoint of death or HF hospitalization by 13.8% among 1,736 subjects ≥75 years of age (P = 0.007), and the benefit was at least as great in this age group as in younger patients.[110] As with ACEIs, older patients receiving ARBs are at increased risk of hypotension, renal dysfunction, and hyperkalemia. In addition, combination therapy with an ACEI and ARB is not recommended because of a lack of proven benefit and increased risk for adverse effects.[111]

Angiotensin plus neprilysin inhibition. In a clinical trial of >8,000 patients with HFrEF (70% of whom reported Class II HF symptoms at baseline), the combination of the ARB valsartan with the neprilysin inhibitor sacubitril reduced the composite endpoint of cardiovascular death or hospitalization for HF (HHF) by more than 20%, when compared with ACEI therapy.[112] Approximately half of this benefit was due to a reduction in mortality and half from a reduction of HHF, at a median follow-up of 27 months. Benefits were similar in older and younger patients. Most notably, however, there was more hypotension in the group receiving combination therapy, which has been confirmed in real-world experiences after introduction of sacubitril-valsartan in the United States. As such, judicious selection of older patients with careful medication titration is imperative when considering combined ARB and neprilysin inhibitor therapy.

Hydralazine-nitrates. The combination of hydralazine and nitrates improves clinical outcomes in HFrEF.[113] Although mortality reduction is less than with ACEI therapy,[114] this combination is useful in subjects with renal dysfunction, hyperkalemia, or angioedema that may preclude the use of ACEIs or ARBs. Moreover, in the African-American Heart Failure Trial (A-HeFT), the combination of hydralazine and isosorbide dinitrate taken three times daily improved clinical outcomes in Class III–IV HF subjects already treated with standard therapies including ACEIs and beta-blockers.[115] All subjects in A-HeFT were African American and the mean age was 57 years, so the role of these agents when added to standard HFrEF therapy in older patients and in other racial/ethnic groups remains to be determined. Another important concern is the need for multiple daily doses of both agents, which may be problematic in older patients with polypharmacy and medication adherence difficulties.

Beta-blockers. Beta-blockers improve LV function, decrease hospitalizations, and reduce mortality in a broad range of patients with HFrEF, and three beta-blockers are indicated for management of HFrEF (metoprolol succinate, carvedilol, and bisoprolol),[116–118] including for older individuals.[119] However, geriatric patients are often sensitive to beta blockade, so initiation at a low dose with slow upward titration is imperative in order to avoid bradycardia, heart block, hypotension, or worsening HF. In particular, patients with Class III–IV HF require meticulous and slow titration because of the potential for transient worsening of HF symptoms. Active bronchospasm is a contraindication to beta-blocker therapy, but chronic pulmonary disease does not preclude use of these drugs. Other contraindications include marked bradycardia, advanced heart block, hypotension, and severe decompensated HF.

Ivabradine. Despite maximally tolerated beta-blocker therapy, many patients with HFrEF have persistently elevated resting heart rates, which is a marker for adverse outcomes. The selective sinus node inhibitor ivabradine was studied in 6,558 patients with HFrEF and sinus rhythm at 70 beats/minute or higher. After a median follow-up of 23 months, ivabradine was associated with an 18% relative risk reduction in the primary outcome of cardiovascular death or HHF.[120] Symptomatic bradycardia and visual side effects were more common in patients randomized to ivabradine. Findings were similar in older and younger patients, so ivabradine is a reasonable therapeutic option in select older individuals with symptomatic HFrEF and persistently elevated heart rates.

Mineralocorticoid antagonists (MRAs). Spironolactone and eplerenone are MRAs with weak diuretic potency but with antifibrotic properties and other beneficial cardiovascular effects. When added to other therapies in patients with HFrEF, LVEF ≤35%, and Class II–IV symptoms, these agents reduce mortality and hospitalizations by approximately 25–30%.[121,122] The benefits are

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similar in older and younger subjects, but older patients are more susceptible to worsening renal function and hyperkalemia.[123] Meticulous electrolyte and renal surveillance is therefore required in geriatric HF patients treated with MRA. Up to 10% of patients may develop gynecomastia and breast tenderness with spironolactone, but these side effects are rare with eplerenone.

SGLT2 inhibition. The SGLT2 inhibitor class was introduced earlier in this chapter with regard to reducing cardiovascular death, MI, and stroke in patients with established vascular disease. Despite some differences in the ischemic outcomes across the major clinical trials, all of the trials consistently demonstrated 30–35% reduction in HHF (as a secondary clinical endpoint) among patients treated with an SGLT2 inhibitor.[68–70] Some of this benefit is believed to occur as a result of the natriuresis induced by these drugs, which does not cause the hypokalemia commonly seen with loop diuretics, but there appear to be antifibrotic effects, reductions in adrenergic tone, and renal preservation over time as well. In a trial of 4,744 patients with HFrEF, only 42% of whom had known diabetes, dapagliflozin was associated with a 26% reduction in cardiovascular death and HHF after a median follow-up of only 18 months.[71] The reduction in events was driven by both components of the composite endpoint, with similar findings among the diabetic and nondiabetic subgroups, as well as among patients above or below age 65 years. A similar trial evaluating empagliflozin in 3,730 patients with HFrEF (50% of whom had diabetes) demonstrated similar results after a median of 16 months of therapy.[72]

Digoxin. Digoxin reduces HF symptoms and hospitalizations but has no effect on mortality.[124] The benefits and adverse effects of digoxin are similar in older and younger patients – including those over age 80.[125] In addition, retrospective analysis of the Digitalis Investigation Group (DIG) trial suggests that digoxin may have a favorable effect on mortality when the serum digoxin level is maintained at <1.0 ng/mL.[126] In geriatric patients with persistent HF symptoms despite other therapeutic measures, digoxin should be initiated at a low dose (0.125 mg daily in the absence of renal dysfunction) with close monitoring for side effects such as bradycardia, heart block, arrhythmias, gastrointestinal symptoms, and mental status changes or visual disturbances. Digoxin toxicity occurs more frequently in the setting of increasing age, worsening renal function, hypokalemia, hypomagnesemia, hypercalcemia, and concurrent use of amiodarone, verapamil, and several other medications.

Anticoagulant and anti-inflammatory drugs. Although the thromboembolic risk associated with HFrEF approaches that seen in AF, two large clinical trials failed to demonstrate a net clinical benefit from therapeutic anticoagulation with warfarin in HF patients.[127,128] Warfarin or other anticoagulant treatment should therefore be reserved for patients with mechanical heart valves, AF, LV thrombus, or other compelling indications.[88] The value of aspirin in HF patients without clear indications for its use is uncertain. Nonsteroidal anti-inflammatory medications (other than aspirin) – commonly used to treat arthritis and chronic pain in older individuals – should be avoided in HF patients whenever possible because these agents promote sodium and water retention, antagonize ACEIs and other HF medications, and may worsen renal function.

Inotropic agents and intravenous vasodilators. Intravenous inotropic agents, such as dobutamine and milrinone, have not been shown to improve clinical outcomes and have been associated with increased mortality rates in patients with advanced HF.[129,130] Nevertheless, many clinicians utilize intravenous inotropes to palliate symptoms in patients with severe intractable HF unresponsive to standard therapies (often through an indwelling catheter and with a defibrillator in place to mitigate the risk of sudden death due to ventricular tachyarrhythmias).[152] Intravenous nitroglycerin and nitroprusside have favorable short-term hemodynamic effects in patients with severe HF, especially in those with hypertensive emergency or significant aortic or mitral regurgitation. Both agents may cause hypotension, and caution is advised in using nitroprusside in older patients, especially those with impaired renal function. Nesiritide, a recombinant form of BNP administered intravenously, reduces LV filling pressures more effectively than intravenous nitrates or standard therapy (including diuretics), but nesiritide has not been shown to reduce morbidity or mortality.[131,132] Routine use of nesiritide in patients with HF is not recommended.[88]

Heart Transplantation and Mechanical Circulatory Support

While age ≥75 years is considered a contraindication to orthotopic heart transplantation (OHT) at most centers in the USA, a growing proportion of transplant recipients are 65 to 74 years of age, and outcomes are similar in this age group compared to younger patients.[133] However, the number of candidates for OHT greatly exceeds the

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number of donor hearts available, so that only a small proportion of highly selected patients with advanced HF undergo the procedure. Technological advances in mechanical circulatory support systems, particularly the development of continuous-flow left ventricular assist devices (LVADs), have led to more widespread use of LVADs as "destination therapy" rather than as a bridge to transplantation. Experience with LVADs in patients 70 to 74 years of age has been generally favorable with improved quality of life and functional status and an acceptable complication rate.[134] Data is limited in patients ≥80 years of age, but use of LVADs in this population is likely to increase as the technology continues to improve and complication rates decline.[135] The most common complications associated with continuous-flow LVADs include bleeding (especially gastrointestinal), infections, and stroke. Outcomes following LVAD implantation are also dependent on comorbidity burden, baseline functional status, and frailty, so that careful patient selection is critical to the long-term success of mechanical circulatory support.

Heart Failure with Preserved Ejection Fraction (HFpEF)

Heart failure with preserved ejection fraction (HFpEF), once viewed as primarily a disorder of abnormal LV diastolic function, is now considered to be a multifactorial systemic illness with pathogenic links to aging, inflammation, multimorbidity, lifestyle, and genetic predisposition.[136] Approximately half of older adults with HF have a preserved LV ejection fraction,[137,138] with women more commonly affected than men. The impact of HFpEF on exercise tolerance, symptoms, and hospitalization rates is similar to that of HFrEF, although mortality rates tend to be somewhat lower.[139–14] In contrast to HFrEF, however, for which multiple pharmacological and device-based interventions have lowered mortality, to date no therapies have been shown to reduce mortality in patients with HFpEF. Some studies have reported favorable effects on hospitalizations and/or exercise tolerance (Table 13.6),[142–152] but in general the magnitude of these effects has been modest. For example, in the PARAGON-HF trial of 4,822 patients with symptomatic HFpEF, those randomized to sacubitril-valsartan (vs. valsartan alone) experienced a nonsignificant 13% reduction (P = 0.06) in the primary composite outcome of CV death and HHF after a median follow-up of 35 months, with more hypotension and angioedema but less hyperkalemia or worsening renal function compared to valsartan.[152] Patients ≥75 years of age comprised 46% of PARAGON-HF participants, and results were similar in older and younger subjects.

Current recommendations for managing HFpEF focus on controlling heart rate, blood pressure, and volume status.[85] Hypertension should be treated in accordance with existing guidelines.[88] Precipitating factors, such as ischemia or arrhythmia, should be managed appropriately, and judicious diuresis should be undertaken with close monitoring of blood pressure and renal function. Important differential diagnostic considerations in patients with HFpEF include valvular heart disease, pericardial constriction, restrictive cardiomyopathy (e.g., amyloidosis, hemochromatosis, sarcoidosis), and noncardiac etiologies such as pulmonary disease or sleep apnea with right HF. HCM may also mimic HFpEF and may be associated with exertional chest pain and syncope. Since the therapeutic armamentarium for HFpEF is more limited than for HFrEF, identification and treatment of potentially reversible causes of the patient's symptoms is essential.

Amyloid cardiomyopathy. Amyloid cardiomyopathy is an increasingly recognized cause of HFpEF in older adults.[153] Amyloidosis results from deposition of misfolded amyloid fibrils in the extracellular matrix. There are two main types of cardiac amyloid: light chain amyloid (AL) secreted by plasma cells, and transthyretin amyloid (ATTR) due to misfolding of the transthyretin (formerly prealbumin) protein secreted by the liver. Wild-type ATTR (ATTRwt) is an age-related disorder with a strong male predominance (>80% of cases), which was formerly called "senile cardiac amyloid" and may contribute to as much as 10–15% of HFpEF cases in older adults. Hereditary ATTR (ATTRh) is caused by specific genetic mutations, and the most common form is present in 3–4% of African Americans.

The cardiac manifestations of cardiac amyloid are similar to other forms of HFpEF, but noncardiac manifestations often include peripheral neuropathy, autonomic neuropathy (with orthostatic hypotension), bilateral carpal tunnel syndrome, and lumbar spinal stenosis. Diagnosis is based on maintaining a high index of suspicion and selected imaging studies. Low QRS voltage on electrocardiography is a classic feature of cardiac amyloid but is present in <50% of cases. Characteristic findings on echocardiography include increased LV wall thickness with normal or small LV cavity, markers of diastolic dysfunction, and abnormal global longitudinal strain with an "apical sparing" pattern. Cardiac magnetic

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Table 13.6 Pharmacotherapy trials for heart failure with preserved ejection fraction (HFpEF)

Trial	Patients	Treatment	LVEF (%)	Age (yrs)	Outcomes compared to placebo*
PEP-CHF	850	Perindopril	65 (56–66)	75 (72–79)	Death/hospitalization by 1 year – HR 0.69 (0.47–1.01, P = 0.055) HHF alone – HR 0.63 (0.41–0.97, P = 0.033)
CHARM-Preserved	3023	Candesartan	54 ± 9	67 ± 11	CV death/HHF – HR 0.89 (0.77–1.03, P = 0.118) HHF admission – HR 0.85 (0.72–1.01, P = 0.072)
I-PRESERVE	4128	Irbesartan	60 ± 9	72 ± 7	Death/hospitalization – HR 0.95 (0.86–1.05, P = 0.35)
SENIORS (EF >35% subgroup)	643	Nebivolol	49 ± 10	76 ± 5	All-cause death/CV hospitalization – HR 0.81 (0.63–1.04)
TOPCAT	3445	Spironolactone	56 (51–62)	69 (61–76)	CV death/HHF/aborted SCD – HR 0.89 (0.77–1.04, P = 0.14) HHF – HR 0.83 (0.69–0.99, P = 0.04)
Aldo-DHF	422	Spironolactone	67 ± 8	67 ± 8	Reduced E/e' avg 1.5 (P < 0.001)
RELAX	216	Sildenafil	60 (56–65)	69 (62–77)	No difference in VO2 peak at 24 wks
ESS-DHF	192	Sitaxsentan	61 ± 12	65 ± 10	Median 43-second relative increase in Naughton treadmill time (P = 0.03)
DIG Ancillary	988	Digoxin	55 ± 8	67 ± 10	HHF – HR 0.79 (0.59–1.04, P = 0.09) Hospitalization for unstable angina – HR 1.37 (0.99–1.91, P = 0.06)
PARAGON-HF	4822	Sacubitril-valsartan	58 ± 8	73 ± 8	Nonsignificant 13% relative risk reduction in CV death or HHF

Abbreviations: CV = cardiovascular. E/e' avg = echocardiographic ratio of mitral inflow velocity/tissue Doppler velocity. HHF = hospitalization for heart failure. LVEF = left ventricular ejection fraction. SCD = sudden cardiac death. VO2 peak = peak oxygen consumption.

Statistics: Age and LVEF presented as mean ± SD or median (IQR). HR = hazard ratio with (95% confidence interval).

Trial acronyms: PEP-CHF (Perindopril in Elderly People with Chronic Heart Failure), CHARM-Preserved (Candesartan in Heart failure: Assessment of Reduction in Mortality and morbidity – Preserved LVEF), I-PRESERVE (Irbesartan in Heart Failure with Preserved Ejection Fraction Study), SENIORS (Study of the Effects of Nebivolol Intervention on Outcomes and Rehospitalisation in Seniors with Heart Failure), TOPCAT (Treatment of Preserved Cardiac Function Heart Failure with an Aldosterone Antagonist), Aldo-DHF (Aldosterone Receptor Blockade in Diastolic Heart Failure), RELAX (Phosphodiesterase-5 Inhibition to Improve Clinical Status and Exercise Capacity in Heart Failure with Preserved Ejection Fraction), ESS-DHF (Effectiveness of Sitaxsentan Sodium in Patients With Diastolic Heart Failure), DIG Ancillary (Digitalis Investigation Group Ancillary Trial), PARAGON-HF (Prospective Comparison of Angiotensin Receptor-Neprilysin Inhibitor with Angiotensin Receptor Blocker Global Outcomes in Heart Failure with Preserved Ejection Fraction trial).

* All-cause mortality was not significantly reduced in *any* trial.

resonance may also support a diagnosis of cardiac amyloid, but recently bone-avid nuclear imaging has emerged as the noninvasive test of choice with high sensitivity and moderate specificity. In patients in whom multiple myeloma or monoclonal gammopathy of unclear significance (MGUS) has been ruled out, a strongly positive bone-avid nuclear scan is diagnostic for ATTR amyloid, and biopsy is not required. In other cases, myocardial biopsy is needed to confirm the diagnosis. Once a diagnosis of ATTR cardiac amyloid has been established, gene sequencing is needed to differentiate ATTRwt from ATTRh.

Until recently there was no effective therapy for cardiac amyloid and the prognosis was poor, with median survival of 2–4 years. However, there are now several promising therapies that attack the pathogenesis of cardiac amyloidosis at multiple levels. In addition, tafamidis, a transthyretin-binding agent, was recently approved for treatment of ATTR amyloid (both forms) based on the results of the ATTR-ACT trial.[154] In this study, 441 patients with ATTR cardiomyopathy (median age 75 years, 90% male, 81% white) were randomized to receive one of two doses of tafamidis or placebo and followed for 30 months. Compared to placebo, tafamidis was associated with a 30% reduction in all-cause mortality (the primary endpoint), 32% reduction in cardiovascular hospitalizations, and better exercise tolerance and HF-related quality of life. Tafamidis was generally well tolerated, and side effects tended to be more common in the placebo group.

Device Therapy in Advanced Heart Failure

Implantable devices. Although pharmacological and behavioral therapies are the cornerstones of HF management, implantable devices are playing an increasingly important role in the treatment of HF patients. Advanced HF frequently is associated with dyssynchronous LV contraction related to abnormalities of electrical conduction, and cardiac resynchronization therapy (CRT) improves symptoms and clinical outcomes in patients with NYHA Class II–IV HF, left ventricular ejection fraction (LVEF) ≤35%, and a QRS duration ≥150 msec on the ECG, especially with left bundle branch block morphology.[155–157] Interestingly, the benefits of CRT appear to be greater in women than in men, and two studies suggest that some women with QRS duration ≥130 msec but <150 msec may also benefit.[158,159] Although none of the CRT trials enrolled patients over 80 years of age, observational studies indicate that selected octogenarians may derive significant quality of life benefit from CRT.[160,161] Thus, CRT should be considered in older patients with persistent advanced HF symptoms who otherwise meet criteria for the device.

Implantable cardioverter-defibrillators (ICDs) reduce the risk for sudden cardiac death (SCD) in selected patients at increased risk for such events.[162,163] Indications for ICD implantation include an LVEF ≤35%, NYHA Class II–III symptoms, and a life expectancy of at least 1 year with good functional status; an ICD is also indicated for patients with Class I symptoms and LVEF ≤30% due to ischemic cardiomyopathy.[60] ICD implantation should be delayed for at least 40 days after MI, and for sufficient time to allow titration of guideline-directed medical therapies such as beta-blockers (usually 3 months in patients with nonischemic cardiomyopathy). However, few patients over 80 years of age were enrolled in the major ICD trials, and there is evidence that the life-saving benefits of ICDs decline with age, most likely due to competing causes of mortality.[164] Thus, although advanced age is not an absolute contraindication to an ICD, device implantation should be undertaken only after careful consideration of the potential benefits and risks through a process of shared decision-making. In contrast to CRT devices, which improve symptoms and morbidity (i.e., functional status and HHF), ICD therapy only reduces the risk of SCD – which is not always the primary goal of care in older individuals who often value quality rather than quantity of life for their remaining lifespan. In addition, all patients considering ICD placement, regardless of age, should be involved in a discussion of circumstances under which the patient would want to disable the ICD to avoid painful shocks (e.g., in the event of terminal illness).[165]

Rehospitalization and Prognosis

Approximately 20–25% of patients discharged from the hospital with a primary diagnosis of HF are readmitted within 30 days, and up to 50% are readmitted within 6 months. These readmissions contribute substantially to the total cost of caring for HF patients, and hospitals with excess risk-adjusted 30-day readmission rates are subject to financial penalties under the Affordable Care Act's Hospital Readmission Reduction Program. In addition, 30-day readmission rates are widely used as quality metrics to assess performance of both hospitals and physicians.

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Over the past two decades, numerous studies have tested various nonpharmacologic interventions designed to reduce readmissions in patients with HF. These interventions generally include individualized patient and family education aimed at enhancing HF self-care in conjunction with close follow-up in the days and weeks immediately following discharge. While randomized trials and meta-analyses have shown that such interventions reduce all-cause readmissions by 20–25%,[166] the effectiveness of "disease management" programs in improving outcomes on a population-wide basis has not been established. Furthermore, since approximately two thirds of early readmissions are for reasons other than HF, additional research is needed to develop and test novel interventions designed to manage HF in the context of multiple chronic conditions.

The overall prognosis for older patients with HF remains poor, with 1-year mortality rates of up to 25% and 5-year mortality in excess of 50%. Factors associated with worse prognosis include older age, more severe symptoms (NYHA functional class III–IV), ischemic etiology, renal insufficiency, hyponatremia, peripheral arterial disease, and dementia.[167] Patients with HFpEF have a somewhat more favorable short-term prognosis than subjects with HFrEF, but long-term prognosis is similar. In light of the high mortality associated with HF, which is equivalent to or worse than many forms of cancer, it is appropriate to begin to address goals of care and end-of-life preferences early in the course of illness.[168] HF patients should be advised to develop a living will or advance directive and to designate durable power of attorney for health care in the event that decision-making capacity is lost, either temporarily or permanently. In patients with advanced HF and NYHA Class IV symptoms despite optimal therapy, life expectancy is less than 6 months. In such cases, transition to palliative care or hospice should be considered.

Valvular Heart Disease

Aortic Stenosis

The prevalence of aortic stenosis (AS) increases with age, from 0.2% in persons 50–59 years of age to 9.8% in octogenarians.[169] AS is the most common valvular abnormality requiring intervention in older adults, and over 70% of aortic valve (AV) procedures are performed in patients over 65 years of age.[170] In the geriatric population, most AS is related to progressive calcific and fibrotic changes of the valve leaflets. LV hypertrophy commonly results from chronic LV pressure overload, and conduction system disease may be present as a result of concurrent calcium deposition. CAD frequently coexists in calcific AS because of the overlap in underlying pathophysiology and the age of the affected population.

Classic symptoms of AS include chest pain, shortness of breath, and lightheadedness or syncope. Physical examination findings include a harsh systolic ejection murmur that tends to peak later in systole as the degree of stenosis worsens, and delayed and low-amplitude carotid arterial pulses. Once symptoms attributable to AS develop, the prognosis is poor, with an average survival of 2–3 years without intervention.[170] As with CAD and HF, the presentation of severe AS may be delayed in the geriatric population because of reduced physical activity levels. Symptoms may not be evident until another medical illness or need for surgery arises, at which time stenosis may be more advanced than in younger patients at the time of diagnosis.

Several echocardiographic criteria define severe AS, including a mean gradient of 40 mm Hg or greater across the AV, peak velocity 4 m/sec or greater, and calculated valve area of 1.0 cm^2 or less. However, the most important criterion determining the need for surgical or transcatheter intervention is the development of symptoms.[170]

Traditionally, the treatment of AS has required surgical aortic valve replacement, as medical therapy does not delay disease progression or improve long-term outcomes. In addition, percutaneous balloon aortic valvuloplasty is not consistently effective for long-term management. The development of transcatheter aortic valve replacement (TAVR), however, has revolutionized the treatment of severe AS in older patients. Initially, TAVR was indicated only for patients at high or prohibitive risk for surgical aortic valve replacement, but progressive improvements in the devices, techniques, and clinical experience of physicians performing TAVR have made this approach an option for a much wider spectrum of patients with severe AS.[170] Most commonly, TAVR is performed via the femoral artery in a manner analogous to PCI, as a bioprosthetic valve mounted on a large stent is inserted in the aortic valve position and expanded. In a series of prospective randomized trials, TAVR was superior to medical therapy with respect to survival and quality of life in patients deemed inoperable by cardiac surgeons, and TAVR was equivalent to surgical AVR in high-risk patients.[171–174] Importantly, the average age

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of patients enrolled in these trials was 83–84 years, many patients were over 90 years of age, and the majority had significant comorbidities. Multiple studies then confirmed the safety and durability of this approach in other clinical scenarios and patient populations, including those at low or moderate surgical risk, with consistent benefit among patients above age 75 years.[175,176]

A limitation of TAVR is that in some older patients the femoral artery is too small to allow introduction of the catheter, thus necessitating the use of an alternate approach (transapical or transaortic). Complication rates through all approaches are generally acceptable, with somewhat higher need for pacemaker placement compared with surgical valve replacement. Conversely, hospital length of stay and postprocedural recovery times are markedly shorter with TAVR. However, despite the fact that TAVR is associated with excellent outcomes in the majority of patients, not all patients derive benefit. Indeed, up to 25% of higher-risk TAVR patients die within 1 year of the procedure or survive without improvement in exercise tolerance or quality of life. Several factors, including frailty and the clinical factors that placed these individuals into a high-risk category, help identify older patients less likely to benefit from TAVR.[177] As with other medical or procedural therapies, individualized patient selection is critical to help determine those situations in which TAVR or other aggressive interventions are likely to be futile.

Prior to valve replacement by either the surgical or transcatheter approach, coronary angiography is warranted in most older individuals to assess for CAD and the potential need for concomitant coronary revascularization. In geriatric patients referred for surgical AV replacement, bioprosthetic valves are preferred over mechanical valves because they offer satisfactory durability while avoiding the need for long-term systemic anticoagulation. Procedural mortality is less than 5% for both isolated surgical AV replacement and TAVR. Postprocedural quality of life is substantially improved among patients with uncomplicated recovery from valve intervention, and long-term survival is excellent – even in octogenarians. Lifelong endocarditis prophylaxis is indicated for dental or surgical procedures in all patients with prosthetic heart valves.[170]

Aortic Insufficiency

Chronic regurgitation at the AV is often well tolerated for many years, as the LV can effectively compensate for chronic volume overload. Common geriatric causes of chronic AV insufficiency include longstanding hypertension, myxomatous degeneration of the AV leaflets, and aneurysmal dilation of the ascending aorta. Other less common causes include rheumatic valve disease, autoimmune conditions, prior endocarditis, and tumors. Many patients are asymptomatic but may exhibit findings of elevated stroke volume such as increased pulse pressure or prominent arterial pulsations ("bounding pulses"). A decrescendo diastolic murmur is usually heard along the left sternal border. LV hypertrophy is often present on the ECG.

Optimal medical therapy for severe chronic AV regurgitation is undefined. Vasodilators such as ACEIs or nifedipine are often prescribed, but the value of such treatment in delaying surgery and improving clinical outcomes remains controversial.[178,179] Progressive exertional dyspnea or HF signifies a failing LV and necessitates AV replacement.[170] Other indications for surgery include increasing LV dilation or a reduction in LV systolic function with exercise. Operative mortality in geriatric patients undergoing AV replacement is acceptable, and long-term outcomes are generally favorable, with some data suggesting TAVR may be a reasonable option as well.[180]

Mitral Stenosis

In younger patients, stenosis of the mitral valve (MV) is almost always rheumatic in origin, although rare inflammatory or infectious processes may result in MV scarring and narrowing. In older individuals, calcification of the MV annulus with impingement into the valve orifice is the most common cause of MV stenosis, although it is rarely severe enough to warrant surgery. Mitral stenosis in older patients usually runs an indolent course, with gradual progression over several decades. Common symptoms include exertional dyspnea and fatigue. In advanced cases, signs of biventricular failure and pulmonary hypertension are evident. The left atrium is often markedly enlarged, increasing the risk for AF and systemic thromboembolism. Pulmonary hypertension is also common, which contributes to the development of right-sided HF. Classic physical findings include an early diastolic opening snap and a mid-diastolic low-pitched rumbling murmur best heard at the cardiac apex, often with presystolic accentuation. An MV area less than 1.0 cm^2 and a mean gradient greater than 10 mm Hg across the valve at rest indicate severe stenosis.

Diuretics and sodium restriction are recommended to maintain euvolemia in patients with MV stenosis. If AF is

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present, anticoagulation with warfarin should be initiated to maintain an INR of 2.0–3.0.[170] Importantly, the DOACs have not been studied in valvular AF and are not approved for use in patients with significant mitral stenosis. Unlike AV stenosis, dilation of the MV via percutaneous valvuloplasty has good intermediate-to-long-term success, provided there is no significant MV regurgitation or other technical factors such as heavy valvular calcification. As these contraindications to valvuloplasty are more common in older than in younger patients, most older patients with severe symptomatic MV stenosis will require MV replacement. Bioprosthetic valves are recommended in geriatric patients, although many older patients may still require anticoagulation for AF or other indications. Operative mortality is generally higher with MV replacement than with AV replacement, ranging from 5–15% depending on age, functional status, prevalent comorbidities, and the experience of the surgical team. The risk of developing AF after surgery increases with age, and is particularly high after MV surgery.[181] Preoperative beta-blocker or amiodarone therapy reduces postoperative morbidity from AF.[182,183] Recovery following MV surgery may be slow, often requiring prolonged rehabilitation.

Mitral Regurgitation

MV regurgitation can occur as a result of CAD, myxomatous degeneration, mitral valve prolapse, rheumatic MV disease, or prior endocarditis. With improved HF therapies helping patients to live longer with chronic cardiomyopathy, one of the more common causes of mitral regurgitation in older patients is stretching of the MV annulus from progressive LV dilation. Severe chronic MV regurgitation may be asymptomatic until the LV begins to fail, particularly in older patients with reduced activity levels, at which point symptoms typical of HF may ensue. An apical holosystolic murmur with radiation to the axilla is usually present, occasionally accompanied by an S3 gallop. Echocardiography – especially transesophageal echocardiography – is useful for defining the severity of MV regurgitation and identifying other structural abnormalities.

Medical therapy has not been shown to reduce progression of MV regurgitation, although many physicians recommend empiric afterload reduction with ACEIs or other vasodilators. Surgical or percutaneous MV intervention is indicated for symptomatic patients with severe MV regurgitation and an LVEF of 30% or higher, as well as for asymptomatic individuals with LV dilation and/or mild to moderate LV dysfunction, since early intervention improves long-term outcomes.[184] MV repair, when feasible, is preferable to MV replacement. The majority of MV surgeries are performed in the Medicare population, and operative mortality and long-term outcomes following MV repair for nonischemic regurgitation are generally good in patients with preserved LV systolic function. However, outcomes are substantially worse in subjects with an ischemic etiology or with impaired LV systolic function.[170]

More recently, transcatheter MV repair has been shown to be an effective alternative to surgery in selected patients, including older adults.[185,186] Although the clinical trial findings have not been as consistent as the TAVR trials for AS, the largest randomized trial comparing transcatheter MV repair to medical therapy showed relative risk reductions of 47% and 38% for HHF and all-cause mortality at 24 months, respectively.[187] Findings were similar for patients above and below age 75 years, suggesting that this therapy may be a suitable option for older adults with significant mitral regurgitation when performed by experienced operators.[170] Since older patients often have other comorbid conditions that affect postprocedural outcomes and long-term prognosis, the decision to proceed with MV intervention must be individualized as well.

Tricuspid and Pulmonic Valve Disease

In geriatric patients, the right-sided heart valves generally become dysfunctional as a consequence of left heart problems or cor pulmonale from chronic lung disease. Tricuspid regurgitation often results from RV dilation in the setting of pulmonary hypertension or chronic HF. Symptoms and signs reflect right-sided HF and may include dyspnea, elevated jugular venous pressure, a murmur of tricuspid regurgitation, hepatic congestion, abdominal bloating and anorexia, and lower-extremity edema. Tricuspid valve repair (i.e., annuloplasty) is occasionally performed at the time of cardiac surgery for other reasons, but tricuspid regurgitation is rarely the primary indication for heart surgery. Studies to evaluate percutaneous approaches to managing severe tricuspid regurgitation are in progress. Similarly, pulmonic insufficiency may result from left-sided HF or pulmonary hypertension but usually does not require surgical intervention.

Endocarditis

Age-associated valvular degeneration and stenosis increase the risk for infective endocarditis in older adults.

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The risk is further compounded by higher rates of bloodborne infection in older patients, as conditions such as cancer, hemodialysis, dental disease, pneumonia, indwelling catheters, and noncardiac surgery are common sources of bacteremia. Management of endocarditis includes intravenous antibiotic therapy and consideration of valve repair or replacement in patients with high-risk features such as embolic phenomena, large vegetations (>1 cm), perivalvular abscess, HF, hemodynamic instability, or failure to respond to antibiotics.[170]

Arrhythmias and Conduction Disturbances

Most forms of arrhythmia and conduction system disease increase with age, and over 75% of pacemakers and more than half of ICDs are placed in patients 65 years of age or older.[2] Aging is associated with fibrosis and calcification throughout the cardiac skeleton, leading to slower impulse generation and conduction and an increased frequency of atrial and ventricular premature depolarizations. In the absence of symptoms, most age-associated bradyarrhythmias, conduction abnormalities, and nonsustained tachyarrhythmias do not require treatment.

Bradycardia

Sinoatrial dysfunction, or "sick sinus syndrome," refers to various disorders related to impaired function of the sinus node and supraventricular conduction system. Sinus node pacemaker cells degenerate with age, and only around 10% of these cells continue to function normally by age 75. Similarly, conduction of the sinus impulse to atrial tissue or to the ventricles may become impaired, causing sinus node exit block or atrioventricular nodal block, respectively. Age-related stiffening of the carotid arteries also predisposes older individuals to carotid sinus hypersensitivity and associated vagally mediated bradyarrhythmias. Symptoms attributable to bradycardia may include dizziness and lightheadedness, angina, dyspnea, exercise intolerance, impaired mental function, fatigue, falls, and syncope. Symptoms may occur at rest, upon standing, with positional changes of the head or neck (e.g., in patients with carotid sinus hypersensitivity), or during exertion (representing chronotropic incompetence, the inability to increase heart rate with increased metabolic demands). Often, ambulatory cardiac monitoring is required to correctly diagnose symptomatic bradycardia. A treadmill exercise test may facilitate the diagnosis of chronotropic incompetence.

Acute bradycardia is treated with atropine but the effect is short-lived. Reversible causes such as CAD (especially inferior ischemia), autonomic dysfunction, hypothyroidism, and electrolyte abnormalities should be treated if possible. Beta-blockers, certain calcium channel blockers (diltiazem, verapamil), digoxin, clonidine, and many antiarrhythmic drugs may also precipitate symptomatic bradycardia, and a reduction in dosage or discontinuation of these agents may be required. Cholinesterase inhibitors, such as donepezil, can also cause bradycardia. Pacemaker implantation is indicated for nonreversible symptomatic bradycardia.[157] A prophylactic pacemaker is occasionally recommended in asymptomatic patients with severe bradycardia (heart rate less than 35–40 beats per minute) or high-degree atrioventricular block. In sick sinus syndrome, dual-chamber pacing is associated with lower risks of developing AF or requiring hospitalization compared to single-chamber pacing, but dual-chamber pacing does not reduce mortality.[188]

Supraventricular Tachycardias

Supraventricular tachycardias (SVTs) increase in prevalence with advancing age. In many cases, brief episodes of SVT respond to beta-blockers or calcium channel blockers. Some individuals may require the addition of digoxin or treatment with an antiarrhythmic drug. Radiofrequency ablation is also effective in selected cases. Of note, termination of SVT may be associated with prolonged sinus pauses and syncope in older patients ("tachy-brady syndrome"), occasionally requiring pacemaker placement to enable suppression of the tachyarrhythmias with medications. Multifocal atrial tachycardia (MAT) is an irregular SVT characterized by three or more P-wave morphologies on the ECG. MAT is most commonly seen in subjects with severe chronic lung disease, and often responds to treatment of the underlying lung disorder in conjunction with diltiazem, verapamil, or a beta-blocker (if tolerated).

The most common sustained tachyarrhythmia in the geriatric population is AF. Nearly 20–25% of individuals will develop AF or atrial flutter during their lifetime,[2] but the prevalence increases from <1% below age 40 to approximately 10% after age 80 (Figure 13.3).[189] In older adults, most AF arises in the context of age-related atrial fibrosis leading to adverse remodeling and intra-atrial electrical abnormalities. AF may be precipitated by uncontrolled hypertension, ischemia or acute MI, hyperthyroidism, alcohol excess or illicit drug use, HF, progressive valvular disease, acute or chronic lung conditions

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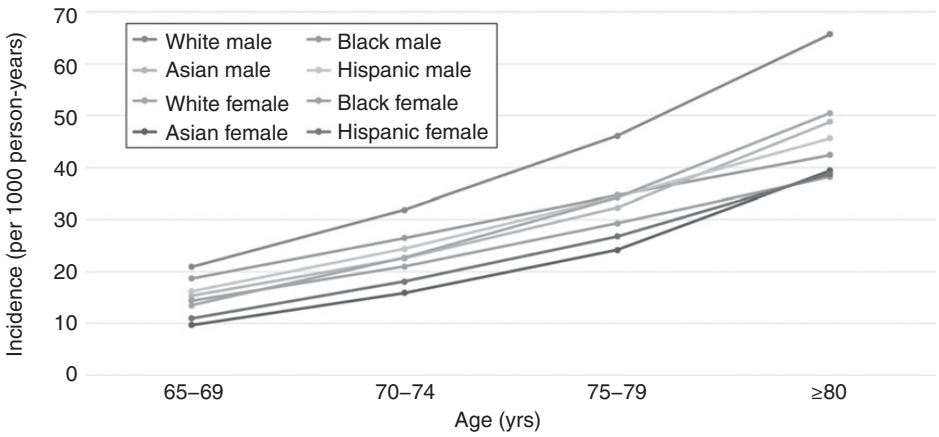

Figure 13.3 Incidence of atrial fibrillation by age, sex, and race in California between 2005 and 2009. (A black-and-white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

(e.g., pulmonary embolus or pneumonia), hypokalemia, cardiac or noncardiac surgery, stimulant medications (caffeine, pseudoephedrine), and chemotherapy for cancer. AF may also occur spontaneously without apparent triggers.

Echocardiography is indicated in patients with new AF to evaluate for structural heart disease or pulmonary hypertension.[190] Electrolyte and thyroid hormone levels should be assessed as well. Patients with acute or chronic AF may be asymptomatic or experience palpitations, chest discomfort, effort intolerance, lightheadedness, syncope, or symptoms of HF. Occasionally older patients with acute-onset AF present with pulmonary edema due to the sudden loss of the atrial contribution to LV filling (the "atrial kick") and associated fall in cardiac output. Some older subjects present with bradycardia in the setting of AF due to underlying conduction system disease or therapy with atrioventricular nodal blocking agents. Unfortunately, in some cases AF is first diagnosed after a thromboembolic event such as a stroke.

Postoperative AF occurs in up to 50% of older patients undergoing major cardiac or thoracic surgery. Beta-blockers, sotalol, and amiodarone reduce the risk of AF after cardiac surgery, including among geriatric patients.[182,183,191,192] Although no mortality benefit has been demonstrated with these perioperative interventions, length of stay is reduced relative to subjects experiencing postoperative AF.

Management of acute AF focuses on symptom control and prevention of thromboembolism. The majority of patients presenting with AF have a rapid heart rate that is irregularly irregular, so therapy is directed at slowing the ventricular rate with a beta-blocker or calcium channel blocker (diltiazem or verapamil). Digoxin is less effective than other agents but may be used as adjunctive therapy or when beta-blockers and calcium channel blockers are contraindicated (e.g., because of hypotension). Concurrent HF should be treated with diuretics, and hemodynamically unstable patients should undergo immediate electrical cardioversion. In hemodynamically stable patients, the need for and timing of pharmacologic or electrical cardioversion is unclear, since clinical trials indicate that cardioversion of asymptomatic or mildly symptomatic patients with AF does not improve quality of life or reduce mortality or stroke risk compared with a strategy of rate control and long-term anticoagulation.[193,194] Nonetheless, these studies suggest that individuals maintaining sinus rhythm have better quality of life than those with persistent AF (irrespective of treatment strategy), so many clinicians recommend at least one attempt to restore sinus rhythm for recent-onset AF.

In cases where AF has clearly been present for less than 48 hours (e.g., in the postoperative setting), the risk of thromboembolism is low, so pharmacologic or electrical cardioversion (if necessary) can be undertaken with relative safety. In patients with AF of longer or unknown duration, elective cardioversion should be preceded by a period of 3 weeks of systemic anticoagulation.[190] Alternatively, transesophageal echocardiography may be performed, and if no atrial thrombus is demonstrated, electrical or pharmacologic cardioversion can then be undertaken. In either case, systemic anticoagulation is essential for a minimum of 4 weeks following cardioversion because of transient atrial stunning and significantly higher thromboembolic risk during the first several weeks

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after cardioversion. Furthermore, long-term anticoagulation is warranted for most older patients with AF (see below).

In subjects who remain highly symptomatic despite efforts to control rate and/or maintain sinus rhythm with antiarrhythmic drug therapy, additional therapeutic options include catheter-based pulmonary vein isolation, ablation of the atrioventricular node with pacemaker implantation, and the surgical "Maze" procedure. Of note, catheter ablation may be a reasonable strategy for treating select patients with highly symptomatic AF, but a routine strategy of ablation does not reduce major cardiovascular or hemorrhagic events when compared with contemporary medical and anticoagulation therapy.[195]

Systemic thromboembolism, most commonly an acute stroke, is the most devastating complication of AF, and usually results from thrombus arising in the left atrial appendage. In addition to being a risk factor for the development of AF, increasing age is a potent risk factor for stroke in patients with either paroxysmal or persistent AF. In the Framingham Heart Study, prior to the use of contemporary anticoagulation therapies, the proportion of strokes related to AF increased from 1.5% for patients in their 50s to 23.5% for patients over 80 years old.[196]

In clinical practice, the $CHADS_2$ and CHA_2DS_2-VASc scores are often used to estimate stroke risk in patients with AF and to identify patients who may benefit from long-term anticoagulation.[197,198] $CHADS_2$ assigns 1 point each for congestive HF, hypertension, age ≥75 years, and diabetes, and 2 points for prior stroke or transient ischemic attack (maximum score = 6). CHA_2DS_2-VASc is similar to the original $CHADS_2$ calculation, but there is further stratification by age (1 point for age 65–74 years, 2 points for age ≥75 years) and 1 additional point for vascular disease (CAD, peripheral arterial disease, or abdominal aortic aneurysm) and for female sex (maximum score = 9). Current guidelines recommend using the CHA_2DS_2-VASc score for thromboembolic risk assessment. Patients with AF and CHA_2DS_2-VASc score ≥2 have an annual stroke risk of at least 2–4%, and systemic anticoagulation is recommended in the absence of major contraindications (Table 13.7).[190,199,200] In patients with a score of 1, anticoagulation is the preferred treatment, but antiplatelet therapy with aspirin and/or clopidogrel may be considered based on individual circumstances (e.g., moderate or high risk of bleeding). Patients with a score of 0 are at relatively low risk and may be managed with antiplatelet therapy alone or no antithrombotic treatment. Note that all men ≥75 years of age and all women ≥65 years of age have a CHA_2DS_2-VASc score of at least 2 and thus anticoagulation is recommended, whenever feasible.

Table 13.7 CHA_2DS_2-VASc score for predicting stroke risk in 7,329 patients with nonvalvular atrial fibrillation

Variable	Points	Total score	Adjusted stroke rate (% per year)
CHF or LVEF <40%	1	0	0
Hypertension	1	1	1.3
Age ≥75 years	2	2	2.2
Diabetes	1	3	3.2
Prior stroke/ thromboembolic event	2	4	4.0
Vascular disease	1	5	6.7
Age 65–74 years	1	≥6	9.6
Female sex	1		

Historically, vitamin K antagonists such as warfarin were the only effective orally administered agents for long-term anticoagulation, at a dose adjusted to maintain the INR in the range of 2.0–3.0. Multiple studies demonstrated that stroke risk was reduced by 60–70% in patients with AF treated with warfarin, including among patients over age 75.[190] However, the development of DOAC therapy has shifted the approach to systemic anticoagulation, as each of the newer agents is at least as effective as warfarin in reducing the risk of stroke with equivalent or lower risk of major bleeding.[201–203] In addition, the benefits of DOACs, relative to warfarin, extend to patients over 75 years of age.[204] Furthermore, the newer agents have fixed dosing, lack of need for monitoring INR or other blood tests, and markedly fewer drug–drug and drug–food interactions relative to warfarin. Despite concerns about patients with severe renal impairment, in whom warfarin and apixaban remain the preferred agents,[190] effective antidotes to the DOACs are now available, if needed, to treat life-threatening bleeding. The major disadvantage of DOAC therapy is higher cost, which may be prohibitive in some older adults. Nonetheless, guidelines recommend DOAC therapy as the preferred option over warfarin, for nearly all patients with AF in the absence of mechanical heart valves, severe mitral stenosis, or end-stage renal disease on dialysis.[190]

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The most important and serious adverse event associated with the use of anticoagulants is bleeding, and older patients are at increased risk for this complication. Although several bleeding risk scores have been developed, the HAS-BLED score is most widely used. HAS-BLED assigns 1 point for hypertension (systolic blood pressure ≥160 mm Hg), abnormal renal function, abnormal hepatic function, prior stroke, prior major bleeding or bleeding disorder, labile INR (<60% of time in therapeutic range), age >65 years, drugs predisposing to bleeding (antiplatelet agents, nonsteroidal anti-inflammatory drugs), and alcohol use (maximum score = 9).[205] Patients with a HAS-BLED score ≥3 are at increased risk for bleeding and warrant close follow-up. Utilizing the HAS-BLED score in combination with the CHA_2DS_2-VASc score may be useful for evaluating the risks and benefits of anticoagulation in individual patients.[206] For example, a patient with a CHA_2DS_2VASc score of 1 and HAS-BLED score of 4 may not be a good candidate for anticoagulation, whereas a similar patient with a HAS-BLED score of 1 may benefit from treatment.

A common conundrum in older patients with AF is an increased risk for falls, and concern about fall-related bleeding is the most common reason for withholding anticoagulants in older patients. However, several studies have shown that in the majority of patients, the risk of serious bleeding related to a fall is greatly outweighed by the beneficial effects of anticoagulation in reducing the risk of stroke and other embolic events in AF.[207,208] Therefore, in most cases, a perceived high risk of falls should not be considered a contraindication to anticoagulation. Alternatively, recent studies have demonstrated efficacy and safety of percutaneous placement of a left atrial appendage occlusion device ("Watchman") to reduce the risk of stroke in patients with AF.[209,210] Real-world registry data suggests acceptable procedural complication rates when implanting Watchman devices, even among patients older than those studied in the clinical trials of the device.[211] Thus, this device should be considered a reasonable option for higher-risk older patients with AF, to mitigate the risk of stroke in the setting of high bleeding risk.

Of note, atrial flutter is managed using the same approach as for AF, since AF and atrial flutter frequently coexist in the same patient. In some patients with clear evidence of isolated atrial flutter and no other AF on outpatient monitoring, radiofrequency ablation of the re-entrant pathway may be curative, and success rates in this setting are higher than for ablation of AF.

Ventricular Arrhythmias

Ventricular arrhythmias, both isolated ventricular premature depolarizations (VPDs) and ventricular tachycardia (VT), increase in frequency with age – in part due to age-related changes in the cardiac conduction system, and in part related to the higher prevalence of cardiac disease at older age (especially CAD, cardiomyopathy, and hypertensive heart disease). In general, management of ventricular arrhythmias is similar in older and younger individuals and is dependent on symptoms, hemodynamic impact, and the severity of underlying heart disease. CAD, hypertension, and valvular heart disease should be treated as previously discussed. Electrolyte abnormalities, including hypokalemia, hyperkalemia, and hypomagnesemia, should be corrected. Isolated VPDs require no specific therapy unless the patient experiences disabling symptoms, in which case beta-blockers are first-line treatment, followed by antiarrhythmic drugs or VPD ablation, if needed.

In the absence of HF or LV systolic dysfunction, asymptomatic non-sustained VT also requires no treatment, and symptomatic patients should be managed in the same manner as for symptomatic VPDs. Sustained VT in geriatric patients almost always occurs in the context of advanced structural heart disease and portends an increased risk for sudden death. Acute sustained VT with hypotension or hypoperfusion should be treated with immediate electrical cardioversion, followed by an evaluation for precipitating causes. Recurrent sustained VT, whether symptomatic or asymptomatic, may warrant antiarrhythmic drug therapy and/or implantation of an ICD.[60,210] Finally, as noted previously, patients with NYHA Class II–III HF and an LVEF of 35% or lower should be considered for an ICD on an individual basis in the absence of very advanced age, frailty, or other major life-limiting comorbidities.[60] In this context, subjects with CAD and documented VT (sustained or non-sustained) are at greatest risk of sudden cardiac death – a factor that should be taken into account in the decision-making process. Ventricular fibrillation is immediately life-threatening and requires emergent defibrillation. Since primary ventricular fibrillation is often caused by ischemia, urgent coronary angiography is usually warranted in survivors of this arrhythmia.

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Pericardial Diseases

Pericarditis

Acute pericarditis usually presents as pleuritic chest pain that is often worse in the supine position and improved with sitting. Fever and leukocytosis are common with infectious etiologies, which represent the majority of cases worldwide. Additional etiologies in the geriatric population include recent MI, hypothyroidism, uremia, recent cardiac or thoracic surgery, malignancy, and prior radiation therapy to the chest. The ECG may show diffuse ST-segment elevation with PR-segment depression. Sinus tachycardia is common, but new pathologic Q-waves are absent. The cardinal sign is a pericardial friction rub, although this may be transient or absent. Serum troponin levels may be slightly elevated, indicating myocardial involvement. Echocardiography may demonstrate a pericardial effusion, but the absence of an effusion does not preclude the diagnosis of pericarditis. Most cases of acute pericarditis respond to a nonsteroidal anti-inflammatory drug, alone or in combination with colchicine, which has been shown to reduce recurrences. Corticosteroids should be reserved for refractory cases. Anticoagulation should be avoided because of the risk of hemorrhagic transformation.

Pericardial Effusion and Tamponade

Common etiologies of pericardial effusion are similar to those for pericarditis but also include HF, hypoalbuminemia, rheumatologic disorders, chest wall trauma, hemorrhage, and certain medications (e.g., minoxidil). Although most pericardial effusions do not progress to tamponade, patients with malignant, traumatic, or infectious etiologies are at increased risk. Clinical manifestations of pericardial tamponade include dyspnea, tachycardia, hypotension, jugular venous distension, and pulsus paradoxus (although not always present). In addition to a moderate or large pericardial effusion, echocardiographic features of tamponade include respiratory variability in flow velocities, right atrial or ventricular compression by the effusion, and a dilated inferior vena cava. Treatment of tamponade involves percutaneous pericardiocentesis (preferably with echocardiographic, hemodynamic, and/or fluoroscopic guidance) or surgical drainage with creation of a pericardial "window" – the therapy of choice when the effusion is likely to recur (e.g., with malignancy).

Pericardial Constriction

Pericardial constriction is a late complication of an inflammatory or infectious pericarditis. The pericardium becomes thickened and scarred, inhibiting ventricular filling. In the past, tuberculous pericarditis was the most commonly identified cause of constriction. Currently, most cases occur following one or more episodes of acute pericarditis, radiation therapy for thoracic cancer, or after open-heart surgery; as a result, the diagnosis is becoming more common in the geriatric population. The clinical course is usually characterized by exertional dyspnea and fatigue, lower-extremity edema, ascites, hepatic congestion, and bowel edema leading to bloating and anorexia. Cirrhosis of the liver may occur in longstanding cases. Inspiratory expansion of the jugular veins (Kussmaul's sign) is a hallmark of pericardial constriction but may also occur in other disease states (e.g., pulmonary hypertension). Echocardiographic findings include small ventricular cavities with normal systolic function but restrictive filling, a thickened pericardium, and a characteristic pattern of respiratory variation in hepatic vein blood flow. Chest imaging may reveal pericardial thickening or calcification. Simultaneous right and left heart catheterization demonstrates equalization of diastolic pressures throughout all chambers with an early plateau in the right and left ventricular diastolic wave form ("square root sign") and discordant respiratory variation of right and left ventricular systolic pressures.

The only effective treatment for constrictive pericarditis is surgical pericardiectomy, which is associated with substantial morbidity and mortality and may not be feasible for older patients with advanced comorbidities. Constriction must be differentiated from restrictive cardiomyopathy, which presents a similar clinical and hemodynamic picture because of ventricular stiffness or infiltrative cardiomyopathy, but which cannot be effectively treated by surgical pericardial removal.

Summary

Aging is associated with diffuse changes throughout the cardiovascular system as well as increasing prevalence of most forms of cardiovascular disease. As a result, the majority of men and women over age 65 have clinically manifest cardiovascular disorders, and such disorders are the leading cause of death, as well as a major source of disability and impaired quality of life in older adults. In general, the diagnosis and treatment of cardiovascular diseases are similar in older and younger patients.

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However, most cardiovascular therapies have been less well studied in older subjects, especially women, patients with more advanced age (e.g., over age 80), and individuals with multiple comorbid conditions. As a result, treatment of the older patient with cardiac disease must be individualized, taking into consideration each patient's unique set of circumstances, needs, and personal preferences. As our population continues to age, there is a clear need for additional research focusing specifically on the prevention and management of cardiovascular diseases in the geriatric age group.

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Chapter 14

Hypertension

Zeke Zamora and Jeff Williamson

Introduction

The exponential growth of the older population is associated with the increased opportunity to understand and improve the management of chronic medical conditions such as hypertension. Defined by the 2017 American College of Cardiology/American Heart Association (ACC/AHA) guidelines as blood pressure ≥130/80 mmHg, hypertension, because of its high prevalence, is a major contributor to cardiovascular disease (CVD), cognitive decline, disability, and mortality in older adults. Despite advances in diagnosis and treatment, only 54% of adults >60 years old have their blood pressures controlled.[1] Hypertension has been viewed as the costliest cardiovascular risk factor, accounting for approximately $131 billion per year in annual health-care expenditures.[2] It is an important target for the prevention of CVD, cognitive impairment, and disability. Because of the increased heterogeneity of older adults, the treatment recommendations for this population require special consideration. This chapter will review both the evaluation and management of hypertension in elderly persons.

Epidemiology

Globally, hypertension is a major public health concern because of its high prevalence that increases with age. It is a major risk factor for cardiovascular and cerebrovascular events, renal disease, cognitive decline, disability, and death. The prevalence of hypertension increases with age for men and women, affecting 63.6% of males and 65.8% of females between the ages of 65 and 74 and increasing to 73.4% and 81.2%, respectively, in those ≥75 years old.[1] A 2017 study found that hypertension was a major contributor to global disability, with disability-adjusted life years of 218 million.[3] Data from randomized clinical trials has been critical to the development of national guidelines, including the refinement of the definition of hypertension. Because of clinically significant event rates at lower systolic blood pressure (SBP), the recent 2017 ACC/AHA guidelines defined hypertension as blood pressure ≥130/80 mmHg compared to the decade earlier Joint National Commission 7 (JNC) definition of blood pressure at ≥140/90. This resulted in a significant increase in the prevalence of hypertension in US adults from 31.9% to 45.6%.[4] The prevalence of hypertension varies across racial/ethnic groups with prevalence in Black Americans being among the highest in the world. According to the Centers for Disease Control and Prevention (CDC) 54% of non-Hispanic Black adults have hypertension compared to 46% of non-Hispanic White adults, 39% of non-Hispanic Asian adults, and 36% of Hispanic adults.[5] Although increasing awareness has improved control rates among all age groups, only 49.4% of adults older than 60 years of age have their blood pressures controlled. Several observational studies including a recent large 2019 analysis showed a continuous relationship between increasing SBPs and cardiovascular mortality throughout all ages.

Pathophysiology

Age-related changes in arterial structure and function predispose older individuals to hypertension. Large vessels stiffen because of impaired function of elastin protein, increased collagen, and loss of smooth muscle cells. Also contributing to this vascular dysfunction is atherosclerosis and calcium deposition within the vessel wall. This increased arterial stiffness leads to increased peripheral vascular resistance and decreased vascular compliance. Increased afterload and left ventricular hypertrophy follow.

Progressive renal dysfunction with a decrease in glomerular filtration rate and changes in the kidneys' ability to manage sodium regulation result in heightened sensitivity to dietary sodium intake and elevated blood pressures. Two thirds of older adults have salt-sensitive hypertension.

Age-associated changes in the sympathetic nervous system also contribute to impaired blood pressure regulation. Decreased baroreflex sensitivity leads to increased

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blood pressure variability – most noticeably a delayed response in heart rate to decreased blood pressure.

Additional factors contributing to blood pressure dysfunction in the elderly include increased risk of orthostatic and postprandial hypotension – often complicating management strategies. Also, circadian variability in blood pressure leads to relatively higher nighttime and early morning blood pressures, increasing an older adult's risk of myocardial infarction and stroke. Lifestyle, tobacco, alcohol, caffeine, and certain medications such as nonsteroidal anti-inflammatory drugs (NSAIDs), steroids, antidepressants, decongestants, and migraine medications also negatively impact blood pressure control in the elderly patient.

Diagnosis and Evaluation

New guidelines from the ACC and the AHA, based on data from a number of observational trials and treatment results from the Systolic Blood Pressure Intervention Trial (SPRINT), lowered the classification of hypertension to blood pressure ≥130/80 mmHg (Table 14.1).

The diagnosis and management of hypertension in older adults requires accurate measurements of blood pressures. In the office setting, time constraints, staff training, measurement methods, and the devices used widely vary and tend to deviate from recommended guidelines. All of these factors affect the accuracy of blood pressure measurements.[6] The 2017 ACC/AHA key components of blood pressure measurement in the office setting are listed in Table 14.2.[7] To diagnose hypertension in the elderly, at least three separate blood pressure measurements obtained on two different office visits are needed. Additional measurements such as a recording of orthostatic changes may be helpful because of increased variability in blood pressure in older adults and increased frequency of conditions such as undetected dehydration.

Ambulatory (home) blood pressure monitoring is useful for the evaluation of "white coat" hypertension or patients with a great deal of variability between readings. New analyses of data on the usefulness of home blood pressure measurement are forthcoming. Increased

Table 14.1 Evolution of blood pressure guidelines and treatment threshold

Hypertension guidelines	BP classification (mmHg)		Treatment threshold (mmHg)
JNC 7	SBP <120 or DBP <80	Normal	BP: ≥140/90 or ≥130/80 in patients with diabetes or CKD
	SBP 120–139 or DBP 80–89	*Prehypertension*	
	SBP 140–159 or DBP 90–99	Stage 1 HTN	
	SBP ≥160 or DBP ≥100	Stage 2 HTN	
JNC 8	SBP <120 or DBP <80	Normal	BP: ≥140/90 for general population (includes those with diabetes and CKD) BP: ≥150/90 for adults >60 yrs
	SBP 120–139 or DBP 80–89	*Prehypertension*	
	SBP 140–159 or DBP 90–99	Stage 1 HTN	
	SBP ≥160 or DBP ≥100	Stage 2 HTN	
2017 AHA/ACC	SBP <120 or DBP <80	Normal	BP: ≥130/80 for general population (includes those with diabetes and CKD) SBP ≥130 in older adults
	SBP 130–139 or DBP 80–89	Stage 1 HTN	
	SBP 140–159 or DBP 90–99	Stage 2 HTN	
	SBP ≥160 or DBP ≥100	Stage 3 HTN	

Abbreviations: SBP = systolic blood pressure; DBP = diastolic blood pressure; CKD = chronic kidney disease.

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Table 14.2 ACC/AHA recommendations for blood pressure measurement

	ACC/AHA recommendations
Preparation	• Patient should be sitting in a chair with both feet on the floor for >5 min • Avoid caffeine, exercise, smoking for at least 30 min prior to BP measurement • Ensure patient has emptied bladder • Neither patient nor clinician should talk during the rest period or BP measurement • Remove clothing from upper arm in area of cuff placement
Technique	• Use validated and previously calibrated BP device • Patient's arm should be supported • BP cuff should be at heart level • Use the appropriate cuff size with ideal bladder length 80% of arm circumference
Measurement	• At first visit check BP in both arms and use arm with higher reading for subsequent BP measurements • Allow 1–2 min in between consecutive measurements • In using the auscultatory technique inflate cuff to 30 mmHg above obliteration of radial pulse and deflate at a rate of 2–3 mmHg/sec listening for Korotkoff sounds
BP recording	• In the auscultatory technique the first and last Korotkoff sound represent systolic and diastolic pressures • Note when patient last took BP medication(s)

rigidity of the peripheral arteries due to calcification and atherosclerosis in the rare patient can lead to pseudohypertension. In these patients, because of the incomplete compression of the brachial artery when the blood pressure cuff is inflated, falsely elevated systolic readings are obtained. Pseudohypertension should be suspected in patients with refractory hypertension without expected end-organ damage or when significant hypotension results unexpectedly after starting antihypertensive medications. When measuring blood pressures, the auscultatory gap (an indication of arterial stiffness) can lead to artificially low readings. To avoid such inaccuracies, inflate the blood pressure cuff 40 mmHg higher than the pressure needed to occlude the brachial pulse.

Initial history should include a review of personal cardiac risk factors, family history as it relates to cardiac disease, evaluation of medications (including prescribed and over the counter), herbal remedies, and other supplements. In addition, discussing lifestyle choices (tobacco, alcohol and substance abuse, exercise history, and dietary preferences) is useful. Compliance with sodium restriction and antihypertensive medications may be useful for those patients diagnosed with hypertension who have poor control. High-risk comorbid conditions such as diabetes mellitus, congestive heart failure, stroke, and hyperlipidemia should also be assessed.

Physical exam maneuvers should focus on the cardiovascular system and potential sites of end-organ damage from uncontrolled blood pressure. Funduscopic examination to identify papilledema or retinopathy, carotid examination to evaluate bruits, heart examination with particular focus on murmurs, cardiac rhythm, and heart size as well as peripheral pulses and abdominal bruits are cardinal elements of the physical exam needed to evaluate a patient with hypertension. Laboratory and investigative studies should include an electrocardiogram, basic metabolic panel to examine electrolytes, kidney function and glucose, a fasting lipid panel, and urinalysis.

As in younger patients, secondary causes of hypertension such as renal artery stenosis, renal failure, hyperaldosteronism, Cushing's syndrome, pheochromocytoma, obstructive sleep apnea, and hyperthyroidism should be evaluated in those with refractory hypertension.

Comorbidities and End-Organ Effects

Uncontrolled hypertension affects multiple organ systems and can exacerbate other chronic conditions. Most commonly effects are seen in the cardiovascular, cerebrovascular, and renal systems, and there is a growing body of evidence for adverse impact on the brain and cognition. Coronary artery disease, including myocardial infarction and angina as well as left ventricular dysfunction, is prevalent among people with hypertension. Arrhythmias, such as atrial fibrillation, may also be related to longstanding hypertension. Uncontrolled blood pressures can also increase the risk of aortic or peripheral arterial disease, such as abdominal or thoracic aortic aneurysms and dissection. Hypertension is linked to cerebrovascular disease, in particular ischemic stroke

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and cerebral hemorrhage. Studies have now clearly demonstrated the link between cognitive impairment (vascular dementia and Alzheimer's disease) and uncontrolled blood pressure.[8,9] Although renal function declines with age, hypertension is an independent risk factor for further kidney disease. Retinopathy can also result from uncontrolled blood pressure, with increased risk for retinal artery occlusion and ischemic optic neuropathy.

Diabetes mellitus and hypertension affect many of the same end organs, and an additive effect is seen in the risk of complications. The combined presence of both conditions accelerates the risk of CVD, retinopathy, nephropathy, and cerebral disease.

Benefits of Treatment

There is compelling evidence that treatment of elevated blood pressures in older adults including those over the age of 80 is effective at reducing cardiovascular events, morbidity, and mortality. Despite the benefits of treating hypertension in elderly patients, medical providers remain reluctant to treat moderately elevated blood pressures in older adults. Appropriate treatment of hypertension reduces the risk of cerebrovascular disease, congestive heart failure, CVD, and Mild Cognitive Impairment (MCI), regardless of age. Multiple studies have shown significant reduction in morbidity and mortality when elevated blood pressures are appropriately treated to goal. The Systolic Hypertension in the Elderly Program (SHEP) trial compared cardiovascular and cerebrovascular outcomes and mortality in adults ≥60 years old receiving antihypertensive treatment for an SBP goal <160 mmHg (in participants with baseline SBP >179 mmHg) and a goal of a 21 mmHg decrease from baseline (in participants with baseline SBP between 160 and 179 mmHg) vs. participants not reaching the blood pressure goal. Study participants achieving target SBPs had a 36% reduction in strokes, 27% reduction in myocardial infarctions, 27% reduction in coronary artery disease, 32% reduction in CVD, and 13% reduction in mortality.[10] A further analysis of the data obtained in this study revealed a reduction of all stroke types, including subtypes of both ischemic and hemorrhagic strokes. Similar benefits were seen in the HYpertension in the Very Elderly Trial (HYVET) study, which included individuals aged 80 to 105 years. This study, which had a targeted blood pressure of 150/80 in the intervention arm, compared indapamide with or without perindopril vs. placebo. There was a mean SBP difference of 15 mmHg between study arms. HYVET showed a reduction in strokes by 34%, heart failure by 72%, cardiovascular mortality by 27%, and all-cause mortality by 28%.[11]

The more recent SPRINT study compared cardiovascular outcomes, cognitive function, and mortality in subjects randomly assigned to intensive blood pressure control (targeted blood pressure <120 mmHg) vs. standard blood pressure control (targeted blood pressure <140 mmHg). This study included 9,361 participants having a mean age of 68 years. SPRINT showed a significant decrease in cardiovascular events (HR, 0.77; 95% CI, 0.64 to 0.89; $P < 0.001$) and all-cause mortality (HR, 0.73; 95% CI, 0.60 to 0.90; $P = 0.003$) in the intensive blood pressure control arm.[12] Analysis of a subset of SPRINT participants >80 years (n = 1167) showed similar benefits in this age group, with intensive blood pressure treatment resulting in a significant reduction in cardiovascular events (HR, 0.66; 95% CI, 0.49–0.90; $P < 0.001$) and mortality (HR, 0.67; 95% CI, 0.48–0.93; $P < 0.001$).[13] The study also showed cognitive benefit with intensive blood pressure control, with decreased MCI (HR, 0.70; 95% CI, 0.51–0.96; $P < 0.001$) and a 15% reduction in the combined incidence of MCI or dementia (HR, 0.85; 95% CI, 0.74–0.97). In persons over age 75, those with higher baseline Montreal Cognitive Assessment (MoCA) scores had the greatest impact relative to CVD events and mortality, and importantly, persons with baseline poor performance on the MoCA had no benefit, while those with frailty and lower gait speed had the same benefit as those without frailty and those with high gait speed. Regardless of age, intensive blood pressure control led to higher rates of self-reported syncope, but this was not statistically significant (HR, 1.24; $P = 0.33$). However, there was no difference in the injurious fall rate between both groups, and orthostatic hypotension was less common (not significant) in the intensive control group.

The Systolic Blood Pressure Intervention Trial Memory and cognition IN Decreased HTN (SPRINT MIND) examined the effect of intensive blood pressure control on adjudicated all-cause dementia, MCI, and the composite outcome of MCI or probable dementia.[8] Although SPRINT was terminated early because of overwhelming CVD and mortality benefit, funding continued for follow-up cognitive assessments, leading to a mean follow-up period of 5.5 years. This trial showed a statistically significant 19% reduction in MCI in the intensive blood pressure control group: 14.6 vs. 18.3 per 1,000 person-years (HR, 0.81; 95% CI, 0.67–0.95). There was a 17% reduction in relative risk of probable dementia in the intensive blood pressure group; however, statistical

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significance was not achieved (HR, 0.83; 95% CI, 0.67–1.04; P = 0.1). SPRINT MIND was the first randomized controlled trial to demonstrate prevention of MCI through an intervention.

Treatment

The heterogeneity in older adult phenotypes related to vitality, frailty, multimorbidity, cognitive impairment, and polypharmacy makes treating hypertension both challenging and rewarding. While the highest level of scientific evidence, randomized clinical trials, have made it clear that, independent of age, treating cognitively intact ambulatory adults with blood pressure ≥130/80 is beneficial, clinical trials have excluded persons with cognitive impairment, immobility, and nursing home residence because of the limited life expectancy of these latter populations. Although SPRINT did not include those with chronic diabetes mellitus, congestive heart failure (CHF), or a history of stroke, other trials have included these populations (other than CHF, which requires many blood pressure-lowering medications), and all have shown CVD prevention benefit while also demonstrating no harm. Thus, with the heterogeneity of the older population, age alone should not be the only determinant of attention to blood pressure management in older adults.

Current recommendations developed by national working groups provide recommendations based on randomized trial evidence. However, the guidelines from the AHA and ACC, affirmed by the American Geriatrics Society, also specifically state that individualized treatment plans should be created for each patient, paying particular attention to concurrent medical conditions, current life expectancy, and social situation (such as whom they are living with and where). If appropriate, family members and caregivers should be included in the discussion to determine treatment goals. The high prevalence of these patients in community samples and in general practice is the reason that the AHA/ACC blood pressure control guidelines have a second recommendation to assist those caring for persons who are older. The first recommendation recommends intensive blood pressure management for healthy older adults and the second recommendation recognizes the fact that there are many older patients with "a high burden of comorbidity and limited life expectancy [where] clinical judgment, patient preference, and a team-based approach to assess risk/benefit is reasonable for decisions regarding intensity of BP lowering and choice of antihypertensive drugs."[7]

Earlier guidelines emphasized the importance of controlling DBP. At one time, SBP elevation was attributed to the physiologic changes associated with aging. However, newer recommendations that focus on the management of SBP values known to increase cardiovascular risk and randomized clinical trial data have not shown adverse DBP effects when SBP is the focus of management.[14]

Nonpharmacologic Treatment

For patients with mild hypertension, lifestyle modification may be the only adjustment required to manage elevated blood pressure. For those with moderate to severe hypertension, nonpharmacologic therapies will augment medication management and likely result in lower doses of medications needed to adequately control the disease. A detailed social history including elements of tobacco and alcohol use as well as a formal dietary history, including how meals are prepared (for example, premade meals may be salt rich), help to determine modifiable risk factors.[7] Additionally, stress reduction and maintaining a healthy body weight may help control elevated blood pressure levels. Moderate exercise and a diet rich in fruits and vegetables also help to control mild hypertension. The Dietary Approaches to Stop Hypertension (DASH) diet incorporates foods high in fiber, calcium, potassium, and magnesium and is low in cholesterol, and has been shown to reduce blood pressure. Consultation with a dietitian may be beneficial for some patients. Weight loss can be considered in an elderly patient with a body mass index greater than 26. Patients who are able to ambulate on their own and who are not wheelchair bound should be encouraged to participate in exercise and walk at least 30 minutes per day for the majority of the week.[7]

Considerations in Treatment

Nonpharmacologic therapies should be attempted prior to medication management in stable patients. Individual drugs can be selected based on patient comorbidities and side-effect profiles. The risks and benefits of medication management of hypertension should be discussed with each patient. The side-effect profile of medications, such as electrolyte disturbances, edema, or cough, should be considered in regard to the patient's current medical conditions and/or comorbidities. Antihypertensive medications should be initiated at the lowest possible dose and slowly increased if needed. If, after 1 month of treatment, the blood pressure is not at goal, this medication dose should be increased.[15] A second agent from

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another class can be added if further blood pressure control is needed after reaching the full dose of the first drug – again, starting at the lowest possible dose. The SPRINT study showed that three medications are often necessary for adequate blood pressure control and that even with three medications, the cost-effectiveness is substantial on a population basis. Nonadherence and drug interactions should be considered with each dose escalation and addition of new drugs.

Classes of Antihypertensive Medications

Thiazide-type diuretics, angiotensin-converting enzyme inhibitors (ACEIs), angiotensin receptor blockers, and calcium channel blockers are appropriate first-line antihypertensive medications to consider when managing elevated blood pressure in the geriatric population.[4]

Thiazide-Type Diuretics

Randomized controlled trials have shown significant benefit in the prevention of stroke, cardiovascular events, and mortality when low-dose thiazide diuretics are used for the management of hypertension in the elderly. Side effects to be noted with this class of medication include hypokalemia, hyponatremia, hypomagnesemia, and glucose intolerance. Caution should be exercised when using this medication in a patient known to have gout, as uric acid levels can rise with the drug. Side effects can be minimized when using these drugs at a lower dose. Potassium replacement may be needed in rare cases. Loop diuretics can be used in the management of hypertension but are generally reserved for patients with CHF.

Angiotensin Receptor Blockers

This class of medication can be considered first-line therapy for elevated blood pressures, particularly in patients with diabetic nephropathy and CHF. The side-effect profile is similar to ACEIs, except for much-decreased risk of cough. These medications may be considered for those who are not able to tolerate ACEIs.

Calcium Channel Blockers

Dihydropyridine calcium channel blockers are effective in decreasing stroke risk in older patients with elevated blood pressure. Calcium channel blockers act by causing vasodilation, increasing vascular permeability, or affecting cardiac contractility. Common side effects include peripheral edema, constipation, and gastroesophageal disease. Headaches and postural hypotension may occur. Non-dihydropyridine and short-acting calcium channel blockers should be avoided as first-line agents for the management of hypertension. Non-dihydropyridine drugs can induce heart block and should be avoided in patients with conduction defects.

Angiotensin-Converting Enzyme Inhibitors

This class of medication can also be used as monotherapy for the management of hypertension. Clinicians may prefer this class of medication in the elderly because of their end-organ protective features for patients with either cardiac or renal disease, such as CHF and diabetic nephropathy, respectively.[8] In addition, a protective effect is seen in patients with hypertensive nephrosclerosis. Related side effects include hyperkalemia, elevated creatinine (in those with renal artery stenosis), dry cough, angioedema, neutropenia, and agranulocytosis. These medications should not be used in combination with an angiotensin receptor blocker.

Other Classes

Beta-blockers are not a preferred first-line therapy for the management of uncomplicated hypertension. Beta-blockers, as compared to placebo, provided no reduction in all-cause mortality and myocardial infarction. Risk reduction for stroke was much less in beta-blockers as compared to other medications.[9] Adults with coronary artery disease, CHF, prior myocardial infarction, angina, and hypertrophic cardiomyopathy may benefit from these medications because of effectiveness in secondary prevention of cardiac events. Beta-blockers are often considered first-line therapy for patients with these medical problems. Alpha receptor antagonists should not be used as first-line therapy for the management of elevated blood pressure because of potential orthostatic hypotension and CHF exacerbation.

Summary

The 2017 ACC/AHA guidelines define hypertension as blood pressure ≥130/80 mmHg. There is extensive data from clinical trials and epidemiologic studies indicating that treating hypertension in ambulatory, cognitively intact older adults is safe and effective. The SPRINT results have provided evidence for treatment to an SBP goal <130 mmHg in ambulatory, cognitively intact older adults with a life expectancy of more than 2 years. For the many older adults outside this group, treatment should be individualized, and consideration of functional status and

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life expectancy is recommended. When considering treatment options, both nonpharmacologic and pharmacologic treatments should be reviewed in the context of an individual treatment plan with consideration of quality of life, comorbid conditions, and patient goals. The possible risks and benefits of antihypertensive medications should be discussed with the patient and/or caregivers. Several medications are available for treatment, and the "start low, go slow" adage should dictate management strategies.

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Chapter 15

Peripheral Artery Disease

Rachelle Hamadi and Casey N. Rust

Introduction

Peripheral artery disease (PAD) refers to narrowing of arteries outside of the heart, including the abdominal aorta, iliac arteries, and arteries of the extremities. PAD results from the obstruction of peripheral arteries leading to a reduction in blood flow to the extremities. While PAD can affect the upper- and lower-extremity vessels, it more commonly affects the lower extremities, which will be the focus of this chapter. PAD most commonly results from atherosclerosis. Less common causes of arterial narrowing include inflammation, thromboembolism (such as in the setting of atrial fibrillation), and arterial insufficiency. PAD may be asymptomatic or lead to a variety of symptoms, including claudication. Claudication refers to exercise-induced pain in the lower extremities comparable to angina in coronary artery disease (CAD). Other signs and symptoms of PAD may include atypical pain, ulcers, or rest pain. Often, symptoms are not present until later stages of the disease, leading PAD to be underdiagnosed in the earlier stages.[1] PAD prevalence increases with increasing age, thus elderly patients should be routinely evaluated for symptoms of extremity pain and nonhealing wounds. Risk factors for PAD are similar to those for other cardiovascular diseases, including smoking, hypertension, diabetes, hyperlipidemia, and family history. An ankle-brachial index (ABI) of less than 0.9 is diagnostic for PAD. Treatments for PAD may include the management of medical comorbidities, exercise, smoking cessation, medications, and surgical interventions. Early intervention can prevent limb ischemia and ultimately limb loss. Without proper management, PAD can lead to significant morbidity, mortality, and a reduction in quality of life.

Epidemiology

PAD affects more than 200 million individuals worldwide, including an estimated 8 to 10 million people in the United States.[1,2] PAD is more commonly seen in low- and middle-income countries, including ones in Southeast Asia and the Western Pacific.

PAD prevalence increases progressively after age 40, affecting approximately 15% of adults aged 70 and older. In addition to having higher rates of PAD, older adults may have fewer additional risk factors for PAD compared to younger adults affected by PAD (see Table 15.1).

PAD is more common in certain ethnic groups. African Americans have a higher prevalence of PAD compared to other ethnicities. According to the United States National Health and Nutrition Examination Survey(NHANES) of asymptomatic individuals, Hispanic American women have higher rates of PAD compared to non-Hispanic Caucasian women.

Although historically PAD was thought to be a male-dominant disease, more recent studies show that PAD is at least as common in females, and females may experience a higher disease burden.[3] In men, the prevalence of PAD is higher in high-income countries, while in women prevalence is higher in low- and middle-income countries.[2]

Risk factors for PAD are similar to risk factors for CAD, including cigarette smoking, diabetes, family history, hypertension, and hyperlipidemia. Cigarette smoking and diabetes are associated with earlier onset of PAD. Known atherosclerosis in other vascular beds is also associated with an increased risk of PAD.[4] Likewise, the presence of PAD is a major risk factor for myocardial infarction and stroke. The

Table 15.1 Peripheral artery disease prevalence according to the National Health and Nutrition Examination Survey

Age	Prevalence
40–49	0.9%
50–59	2.5%
60–69	4.7%
70+	14.5%
80+	23.2%

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morbidity and mortality associated with PAD are similar to those for CAD and stroke.[3]

Studies have shown an association between PAD and various inflammatory markers, including high-sensitivity C-reactive protein (CRP), homocysteine, lipoprotein A, and fibrinogen. CRP can be used as a marker for atherosclerosis in CAD, cerebrovascular disease, and PAD.

While individuals with a family history of PAD seem to be at higher risk for developing PAD, there is no single gene responsible for the increase in risk. Twin studies have shown mixed results regarding the role of genetic versus environmental factors contributing to PAD.[5,6]

Pathophysiology

Peripheral artery disease is caused by atherosclerosis of the arteries of the extremities. The arteries of the lower extremities are more commonly involved than the upper extremities. Atherosclerosis is a narrowing of arterial lumen due to the accumulation of lipid and fibrous material. Atherosclerosis can lead to partial or complete occlusion of vessels and a chronic reduction in blood flow.

Smoking is one of the most significant risk factors for the development of PAD. Smoking promotes endothelial damage, smooth muscle proliferation, thrombophilia, inflammation, and increased sympathetic tone, which may result in PAD.

Ischemia often results from occlusion in more than one artery, thereby reducing collateral circulation. However, isolated tibial vessel disease may be seen in patients with diabetes and in the elderly. While smoking is more likely to lead to proximal artery disease, diabetes more commonly leads to distal artery disease.[7] Depending on the location of arterial stenosis, patients may report pain anywhere from the hips down to the feet. Bilateral aortoiliac disease may lead to a condition known as Leriche syndrome, which involves claudication, absent or diminished femoral pulses, and erectile dysfunction (see Table 15.2).

Table 15.2 Presentation of symptoms based on distribution of lesion

Location of lesion	Location of symptoms
Aortoiliac	Hip, buttocks, thigh
Common femoral	Thigh, calf
Superficial femoral	Upper two thirds of calf
Popliteal	Lower third of calf
Tibial, peroneal	Foot

Presentation

PAD is initially asymptomatic until the reduction in blood flow to muscle and tissue is inadequate to meet metabolic demands. Thus, patients with PAD who lead a sedentary lifestyle may have a prolonged asymptomatic phase.

According to the American College of Cardiology (ACC) and American Heart Association (AHA), claudication (derived from the Latin word for limp) is defined as fatigue, discomfort, cramping, or pain of vascular origin in the muscles of the lower extremities that is consistently induced by exercise and consistently relieved by rest. Claudication is intermittent and reproducible. Onset is typically related to walking a certain distance and is relieved within 10 minutes of rest.

Atypical pain may be seen more commonly in patients with PAD than intermittent claudication. Atypical pain may be described as throbbing, burning, or shooting pain. Atypical pain may be the result of comorbid conditions such as arthritis, neuropathy, spinal stenosis, or fibromyalgia.

Pain related to PAD may be in the buttocks, hip, thigh, calf, or foot and may be unilateral or bilateral. Pain may be mild or may become debilitating as the degree of arterial stenosis progresses. Claudication severity can be classified according to various scales such as the Fontaine and Rutherford scales.

When the severity of arterial narrowing leads to inadequate blood flow at rest, patients develop rest pain. Rest pain is often worse with the elevation of the legs and may be reported as pain in the feet or legs during sleep that is improved by hanging the legs over the side of the bed.

PAD can cause chronic skin discoloration, extremity hair loss, and nail changes. Ischemia can lead to nonhealing ulcers, infection, or gangrene. Ulcers may develop from minor trauma that fails to heal because of inadequate blood supply. Ischemic ulcers are typically circular and may be described as punched-out lesions. Ulcers are often found at the tips of the toes, between digits, or may be at points of pressure. Ulcers are susceptible to infection and may lead to osteomyelitis.

Limb ischemia can be recognized with one or more of the six Ps: pain, paresthesias, pallor, pulselessness, poikilothermia, and paralysis. Critical limb ischemia can be acute or chronic. Acute limb ischemia (ALI) is defined as a sudden decrease in limb perfusion presenting within 2 weeks of the inciting event.[8] ALI is more likely to be embolic than chronic ischemia. Critical limb ischemia (CLI) is characterized by more than 2 weeks of ischemic

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rest pain, nonhealing wounds or ulcers, or gangrene in one or both legs.[4]

Screening and Diagnosis

Screening

Screening for PAD aims to detect disease and intervene early in order to prevent disease progression and complications related to PAD. Diagnosing PAD can also help identify individuals at high risk for CAD or other cardiovascular diseases (CVDs) in order to aggressively treat risk factors and prevent events related to CVD. PAD is a common cause of impaired ambulation and lower-extremity wounds and amputations. Various studies suggest that the majority of cases of PAD remain undetected in routine clinical practice.

PAD is now identified as a risk factor for other cardiovascular diseases. PAD screening is one of the proposed tests for the identification of high-CVD-risk patients in addition to other tools such as the Framingham CVD risk score. Other markers for CVD risk include CRP, apoprotein B, low-density lipoprotein (LDL), homocysteine, and computed tomography cardiac screening. Serum high-sensitivity CRP (hsCRP) is a marker for the severity of atherosclerosis that can be used to evaluate the severity of PAD as well as cardiovascular events in patients with PAD.[9]

A systematic review of guidelines for PAD screening found conflicting recommendations regarding the eight screening guidelines.[10] There have been no randomized trials evaluating the outcomes of screening in asymptomatic individuals. The US Preventive Services Task Force (USPSTF) concluded in 2018 that evidence is insufficient to assess the balance of benefits and harms of screening for PAD and CVD with the ABI in asymptomatic adults.[11] A systematic review found that screening for PAD with an ABI had low sensitivity. Treatment for PAD detected by screening with aspirin or exercise lacked an effect in most studies. Nevertheless, these studies had important limitations, with no trials evaluating the effect of ABI screening on morbidity and mortality.[12]

The 2016 AHA/ACC guidelines recommend that patients at increased risk of PAD undergo comprehensive medical history and assessment for exertional leg symptoms, physical examination including palpation of lower-extremity pulses, and noninvasive blood pressure measurement (Level of Evidence B). Special consideration should be given to screening for PAD in frail older adults, in whom the risks of medical and surgical interventions for PAD may outweigh the benefits.

Diagnosis

PAD is often an unrecognized condition by clinicians and can frequently be underdiagnosed even when the patient is symptomatic. This may be due in part to the high prevalence of atypical pain. Classic symptoms of claudication are only present in a minority of patients. In a study by McDermott et al. in 2001, only 32% of patients had classic symptoms of claudication, 20% had no exertional pain, and half had atypical symptoms.[13] In elderly subjects, comorbidities are common, which may impact presentation in a number of ways. The occurrence of respiratory, cardiac, or musculoskeletal problems is common in the elderly and may complicate the diagnosis since patients may have other factors limiting ambulation, making it difficult to appreciate symptoms of PAD. In addition, the interpretation of results may be more complex in the elderly.

There is no specific biomarker for PAD. Routine laboratory studies may include complete blood count with differential metabolic panel, lipid profile, and possibly homocysteine, lipoprotein A, and CRP. Findings from clinical examination (including cold temperature, diminished pulses, and bruits) have poor sensitivity in the detection of PAD.[14]

The ABI is a simple and accurate test that assesses the ratio of systolic pressures in the lower and upper extremities using a Doppler ultrasound. For patients with suspected PAD, a cutoff of <0.9 had a sensitivity ranging from 79 to 95% and a specificity above 95%. (See classification based on ABI in Table 15.3.)[15] When the ABI is greater than 1.40, because of noncompressible arteries, it is considered uninterpretable and the toe-brachial index is used. A toe-brachial index of less than 0.70 is diagnostic for PAD. An ABI between 0.91 and 1.40 is considered within the normal

Table 15.3 Classification of ankle-brachial index according to American Heart Association guidelines

Ankle-brachial index	Classification
>1.4	Noncompressible arteries
1.00–1.40	Normal range
0.91–0.99	Borderline PAD
0.71–0.90	Mild PAD
0.41–0.70	Moderate PAD
<0.41	Severe PAD

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range; however, when there is a high pretest probability of disease, the exercise ABI may be used. A decrease in the ABI with exercise of more than 20% or a decrease of more than 30 mmHg is considered diagnostic of PAD.

ABI showed an excellent diagnostic accuracy compared with angiography with an area under the ROC curve equal to 0.95 in detecting PAD. As previously stated, there are not randomized controlled trials assessing the sensitivity and specificity of ABI for screening asymptomatic patients. There has been a modified approach to calculating ABI in a screening population, but this approach still requires validation. Time and equipment may be limitations to physicians performing ABI in the office, in which case patients can be referred to vascular surgery clinics for ABI testing.[16]

Plain radiographs are not routinely recommended but may demonstrate arterial calcification in locations consistent with PAD, such as at the arterial branch or along the mid- to distal thigh.

There is an increased preference to use anatomical imaging tests, such as lower-limb duplex imaging, computed tomographic angiography (CTA), or magnetic resonance angiography (MRA). The advantages of duplex imaging include its noninvasive nature and absence of established complications; however, it requires a high level of training and experience. Both CTA and MRA provide detailed mapping of the lower-limb arteries with sufficient detail to plan interventional management. CTA involves ionizing radiation and the injection of intravenous contrast with the associated risks. MRA is frequently less available than CTA.

Formal digital subtraction angiography (DSA) involves percutaneous placement of femoral or brachial sheath and selective catheterization of lower-limb arteries to provide a detailed and high-quality assessment of the lumen of the lower-limb arteries. Despite the fact that DSA is the gold standard, it is unusual to undergo DSA for diagnostic purposes alone owing to its interventional nature and the associated risks of serious bleeding, atherothrombotic embolization, renal impairment, and contrast allergy. DSA is usually undertaken as part of directing and assessing the quality of therapeutic revascularization by angioplasty stenting, or atherectomy. Unlike CTA and MRA, DSA does not provide any information about the arterial wall, which can limit its ability to identify and assess pathologies such as aneurysms.

Differential Diagnosis

Patients presenting with intermittent claudication can have an array of diagnoses. Any vascular disease that results in occlusion or stenosis can cause symptoms of extremity pain or tissue loss, such as arterial aneurysm, dissection, embolism, popliteal entrapment syndrome, adventitial cystic disease, and thromboangiitis obliterans.

Nonarterial etiologies such as neurological causes should also be considered. Neurologic pain can be divided into neuro-spinal causes such as disc disease, spinal stenosis, or tumor, and neuropathic causes such as diabetes or alcohol abuse. Neurogenic claudication, also called "pseudo-claudication," is due to neuro-spinal canal disorder. Unlike vascular causes where pain occurs when walking and is relieved by rest, in the neurogenic causes pain happens with erect posture and is relieved by sitting or lying down, leaning forward, and straightening the spine (like pushing a shopping cart or leaning against a wall).

Chronic venous disease can cause venous claudication. Venous claudication is differentiated from arterial claudication in that it increases with limb dependency. Venous disease can also be associated with limb swelling and varicosities.

Other differential diagnoses include musculoskeletal pain derived from the bones, joints, ligaments, tendons, and fascial elements such as osteoarthritis of the hip and knee. In many elderly patients, several pathologies coexist.

Classification

Classification of chronic PAD is based upon the severity of symptoms as well as markers for severe, chronic disease such as ulceration and gangrene.[8,17] The various classifications include Rutherford, Fontaine, and WIfI systems.

The Fontaine classification system focuses exclusively on symptoms and classifies patients into one of four stages ranging from asymptomatic to ischemia and gangrene. The Rutherford system is also a functional classification that evaluates symptoms as well as objective findings (walking distance and pulse measurement). Rutherford has distinct scales for acute and chronic limb ischemia.

The global vascular guidelines using the Society for Vascular Surgery Lower Extremity guidelines known as WIfI recommend staging the limb. The WIfI system uses three criteria to identify threatened limbs: wounds, ischemia, and foot infection. Each criterion is scored 0 to 3. The WIfI classification system takes into account important clinical considerations that impact management and amputation risk.[8,17]

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Using TASC-II, PAD can also be classified by the atherosclerotic patterns of disease, according to the anatomic distribution, the multiplicity of lesions, and the nature of the lesion.[17] The Global Anatomic Staging System (GLASS) is an anatomic classification that grades the level of disease in the femoropopliteal and infrapopliteal segments of the preferred target artery path.[18]

Management

Management of peripheral artery disease includes medications, surgical interventions, and wound care as well as secondary prevention of adverse events through smoking cessation, weight reduction, blood pressure control, glycemic control, and anti-lipid therapy. PAD management should take a multidisciplinary approach and may include primary care physicians, vascular specialists, wound care physicians or nurses, orthopedic surgeons, podiatrists, endocrinologists, physical medicine and rehabilitation clinicians, physical and occupational therapists, and nutritionists. The ACC and AHA released clinical guidelines for the management of peripheral artery disease in 2016.

Diabetes

Patients with PAD who also have diabetes are at an increased risk of death compared to those without diabetes.[19] Glycemic control improves PAD outcomes, even in those with critical limb ischemia.[20] Hemoglobin A1c targets are based on individual patient factors, including age, comorbidities, and history of hypoglycemia. The choice of antihyperglycemic agent is not dependent on a diagnosis of PAD. The sodium-glucose cotransporter 2 inhibitors (SGLT2-I) and glucagon-like peptide-1 receptor agonists (GLP1-RA) have been shown to reduce mortality from cardiovascular events. However, the SGLT2-I Canagliflozin has been associated with an increased risk for lower-extremity amputations, and as a result it may be safer to avoid use of SGLT2-I in patients with PAD. An interdisciplinary approach to diabetes management is recommended in patients with PAD, which may include primary care physicians, endocrinologists, podiatrists, and wound care specialists. Patients with PAD and diabetes should be educated on foot self-exams, and a physician should perform clinical foot exams at least twice per year (Level of Evidence C).[20] Proper footwear is also important for adults with diabetes and PAD.

Hypertension

According to the 2015 SPRINT trial, patients with hypertension and atherosclerotic disease, including PAD, had reduced cardiovascular events and death with intensive hypertension management.[21] Intensive therapy aimed for a systolic blood pressure goal of less than 120 mmHg. In 2017 the ACC released updated guidelines for hypertension management that recommended a blood pressure goal of less than 130/80 mmHg.[20] Unlike the JNC-8 guidelines, which recommended a less strict goal of below 150/90 in older adults, the new ACC guidelines did not recommend a different goal for older adults. The 2017 ACC guidelines reported that there were no differences in blood pressure management between patients with hypertension and PAD compared to those with hypertension without PAD. There was also no recommendation for a specific antihypertensive in patients with PAD.

Hyperlipidemia

Statin therapy is recommended in all patients with PAD, regardless of baseline cholesterol levels. Moderate- to high-intensity statins are preferred for patients with PAD. The AHA recommends high-intensity statin therapy for patients with symptomatic PAD.[4]

Smoking

Smoking cessation decreases morbidity related to PAD.[22] All patients with PAD who are current tobacco users should be strongly advised to stop smoking at every visit and offered assistance with smoking cessation (Class I, Level of Evidence A). Patients should be offered appropriate pharmacotherapy, which may include nicotine replacement therapy, varenicline, or bupropion. Patients may also be referred to smoking cessation programs or counseling.

Exercise

There are several mechanisms by which exercise improves claudication, including increased calf blood flow, improved endothelial function, reduced local inflammation, improved muscle architecture, improved muscular strength, induction of vascular angiogenesis, improved mitochondrial and muscle function, and reduced red cell aggregation and blood viscosity. Many studies and meta-analyses have shown that exercise significantly improved pain-free walking distance but did not improve ABI. Other studies have shown no effect of exercise on mortality when compared with placebo or usual care.[18,23,24]

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A supervised exercise program is recommended as part of the initial treatment regimen for patients who experience exertional lower-limb pain. Exercise programs have been shown to improve walking parameters in multiple randomized trials.[25,26] Patients should be screened for sufficient cardiopulmonary reserve and other comorbidities to determine their ability to tolerate an exercise program.[27]

Home and community exercise programs are also effective for improving walking tolerance but are less effective than supervised exercise and are associated with a high dropout rate.

Current AHA recommendations for exercise and lower-extremity PAD rehabilitation include:

1. In patients with claudication, a supervised exercise program is recommended to improve functional status and quality of life and to reduce leg symptoms.
2. A supervised exercise program should be discussed as a treatment option for claudication before possible revascularization.
3. In patients with PAD, a structured community or home-based exercise program with behavioral change techniques can be beneficial to improve walking ability and functional status.
4. In patients with claudication, alternative strategies of exercise therapy, including upper-body ergometry, cycling, and pain-free or low-intensity walking that avoids moderate to maximum claudication while walking, can be beneficial to improve walking ability and functional status.

Medications

Antithrombotic Therapy

Chronic antiplatelet therapy with aspirin or clopidogrel is recommended for people diagnosed with symptomatic PAD. For individuals with asymptomatic PAD, treatment with aspirin is reasonable. Dual antiplatelet therapy in patients with PAD and no other clear indications is generally not recommended, given the increased risk of bleeding and the lack of proven benefit.[27–29] Aspirin is generally the first-line agent at a dose of 75 to 325 mg daily, depending on the case.[27]

Clopidogrel 75 mg daily is an alternative and has been proven to be safe and effective in treating PAD to reduce the risk of myocardial infarction, stroke, and vascular death. Some studies, including the PEGASUS-TIMI 54 trial, have demonstrated the benefits of ticagrelor when compared to clopidogrel, but with the increased risk of bleeding.

Vorapaxar is a novel antagonist of protease activated receptor (PAR-1) that is located on platelets, vascular endothelium, and smooth muscle and is the primary receptor for thrombin in human platelets. While Vorapaxar has been shown to reduce the rate of initial ALI events, especially among those who had undergone revascularization, it was also associated with an increased risk of moderate to severe bleeding events, including intracranial hemorrhage.[25,26,30]

Vasodilators

For patients with lifestyle-limiting claudication, the addition of the phosphodiesterase inhibitor cilostazol may improve symptoms. Evidence of decreased pain with ambulation or increased walking distance has only been demonstrated with cilostazol. Cilostazol was shown to significantly increase maximal walking distance and pain-free walking distance in one randomized controlled trial.[31,32] Benefit can be noted as early as 4 weeks after the initiation of therapy.[33,34] A trial period of 3–6 months is generally recommended, as these medications are often expensive. Cilostazol 100 mg twice-daily dosing is recommended 1–2 hours before or after meals.

Data for pentoxifylline has only shown marginal improvements in walking distance. The benefits are generally substantially less with pentoxifylline than those achieved with a supervised exercise program.

Surgery

For patients who are significantly disabled by claudication, invasive intervention may be an option provided the patient meets the following criteria:

1. The patient is significantly disabled by claudication, resulting in inability to perform normal work or other activities that are important to the patient. This criterion reflects the substantial variability among patients and between the patient's and the physician's assessments of the quality of life.
2. The patient has not had or is not predicted to have an adequate response to exercise rehabilitation and pharmacologic therapy.
3. The characteristics of the lesion permit appropriate intervention at low risk with a high likelihood of initial and long-term success.
4. The projected natural history and prognosis of the patient have been taken into account.
5. The patient is able to benefit from an improvement in claudication (i.e., exercise is not limited by other factors).

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Vascular imaging is used to define vascular anatomy prior to intervention.

Different options for interventions are available, including percutaneous intervention, surgical revascularization, or a combination of both. Percutaneous intervention typically involves accessing the femoral artery with an arterial sheath and passing various wires or catheters to guide the placement of a balloon, stent, or other devices. Surgical revascularization involves identifying an appropriate vessel above and another below the arterial obstruction onto which to suture a graft to bypass the obstruction.

Patients with lifestyle-limiting intermittent claudication and rest pain can be considered for revascularization therapy, which includes endovascular or surgical therapy. Options for the vascularization depend upon the level of obstruction. Aortoiliac disease is referred to as inflow disease, with femoropopliteal disease referred to as outflow disease.

Choosing between endovascular versus open surgery depends on the patient's age, comorbidities, extent and location of disease, and the patient's risk for the intervention. Surgery is normally reserved for those patients with whom the cardiovascular risk of surgery is low.[35] Given the widespread availability of percutaneous procedures, guidelines from major cardiovascular societies have recommended an initial percutaneous revascularization, reserving surgery for when arterial anatomy is not favorable for a percutaneous approach.[15,28]

AHA guidelines recommend the following for persons with critical limb ischemia, including endovascular and open surgical treatment for limb salvage:[15,36]

1. For individuals with combined inflow (aortoiliac) and outflow (typically superficial femoral) disease with critical limb ischemia, inflow lesions should be addressed first.
2. For individuals with combined inflow and outflow disease whose symptoms of critical limb ischemia or infection persist after inflow revascularization, an outflow revascularization should be performed.
3. If it is unclear whether hemodynamically significant inflow disease exists, intra-arterial pressure measurements across suprainguinal lesions should be measured before and after the administration of a vasodilator.
4. For patients with limb-threatening lower-extremity ischemia and an estimated life expectancy of 2 years or less or in patients in whom an autogenous vein conduit is not available, balloon angioplasty is reasonable to perform as the initial procedure to improve distal blood flow.
5. For patients with limb-threatening ischemia and an estimated life expectancy of more than 2 years in whom an autogenous vein conduit is available, bypass surgery is reasonable to perform as the initial treatment to improve distal blood flow.

In contrast to chronic limb ischemia, the management of ALI requires emergent evaluation to assess for limb viability. Management of a viable limb includes anticoagulation with heparin and revascularization. Irreversible ALI is managed with amputation.

Prognosis

The overall prognosis of PAD depends on the patient's individual risk factors, the vascular beds that are affected, and the presence of coronary heart disease and other comorbidities.[37–39]

Five-year estimates for limb morbidity in patients with claudication indicate stable claudication in 70–80%, worsening claudication in 10–20%, and CLI in 1–2%.[40,41] In regard to cardiovascular morbidity and mortality, 5-year estimates show nonfatal myocardial infarction or stroke in 20% and death in 15–30%.[42]

Among the 1–2% of patients who develop chronic limb-threatening ischemia, outcomes have improved over time related to improved medical management[43,44] and more liberal use of endovascular interventions.[45] Amputation-free survival has improved even among those without an option for revascularization.[46] Overall, at 1 year 45% of patients will be alive with both limbs, 30% will have undergone amputation, and 25% will have died. At 5 years, more than 60% of persons with chronic limb-threatening ischemia will have died.

These estimates do not apply equally to all patients. Patients with diabetes and lower-extremity PAD tend to have the more aggressive disease compared to nondiabetic patients with PAD because of sensory neuropathy and increased susceptibility to infection. Survival is generally worse in patients with diabetes and end-stage renal disease and those who continue to smoke.[15]

Longitudinal follow-up should include periodic clinical evaluation and assessment of risk factors, limb symptoms, and functional status, and in those who have undergone revascularization, periodic ABI measurement and/or duplex ultrasound.

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Chapter 16

Neurologic Problems

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Introduction

Neurological problems are common reasons for clinician visits among older adults. Neurological issues impact physical health, as well as social and psychological well-being. Management can be challenging because of overlapping symptoms, comorbid conditions, and complex pharmacology in this age group. Familiarity with presentations, diagnostic workup, and treatment help to ensure quick and accurate care for the older patient.

In this chapter, several common neurological complaints of the elderly, their impact, as well as their evaluation and available treatment options will be reviewed. Other conditions are discussed in other chapters, including gait and movement disorders (Chapter 18), dizziness (Chapter 10), delirium (Chapter 11), and dementia (Chapter 12).

Important Considerations

Because of potential cognitive impairment in the older adult, information from collateral sources may be necessary. When assessing ability to perform activities of daily living, or activities such as driving, managing finances, or household activities, family members may under- or overestimate the elderly patient's abilities. With neurological complaints, additional detail should be paid to the impact the symptom has on quality of life and uncovering underlying exacerbating factors.

On examination, changes to the nervous system may be seen with "normal aging." Findings on cranial nerve assessment include changes in visual accommodation, vision distance, diminished pupillary responses and size, and upward gaze impairment.[1] Hearing decreases slowly with age but is a concern when presenting acutely or when associated with symptoms such as vertigo or headache. Normal cognitive changes typically involve decreases in processing speed and efficiency.

Measurable changes of normal aging that affect gait include shortened stride, stooped posture, slower cadence, decreased arm swing, and decreased stride clearance.[1] Slight increases in motor tone and axial and limb rigidity may also be seen with aging. Decreases in muscle mass can lead to muscle weakness.[1] Between the ages of 70 and 79, estimated muscle strength decreases by up to 3% per year.[2] Reflexes may diminish, with an absent ankle reflex being the most common finding.[3] The presence of "frontal release reflexes" in the form of grasp, snout, and suck reflexes may occur in older patients but may also be signs of frontal cerebral damage. Changes in the peripheral sensory system include diminished vibratory, pinprick, and touch sensation, occurring in 33–50% of individuals over the age of 75.[1,3]

Muscle Weakness

Muscle weakness impacts social and physical activities of the older adult. There is a broad range of causes of muscle weakness, with some that are disabling and life-threatening. Unfortunately, symptoms of weakness may be vague, making diagnosis difficult.

Causes of Neurological Weakness

Causes of muscle weakness can be separated into neurological and non-neurological etiologies. Neurological causes of muscle weakness can fit patterns of upper-motor-neuron and/or lower-motor-neuron weakness, while also involving the neuromuscular junction (Tables 16.1 and 16.2).

Multiple Sclerosis

Multiple sclerosis is an idiopathic inflammatory disease, causing demyelination and axonal degeneration in the central nervous system (CNS), described as lesions separated by time and space. It has a wide array of presentations, including neuropathy, neuralgia, diplopia, and urinary and gait disturbances. Muscle weakness from multiple sclerosis is associated with upper- and/or lower-motor-neuron findings. The disease course ranges from a relapsing/remitting course to a progressive one. The disease has a lower incidence in the elderly and more commonly affects younger female adults. However, late-onset multiple sclerosis can

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occur often in a primary progressive form.[4] Disability from the disease may persist into late life if the onset occurs early and carries a higher mortality rate as well.[5]

Treatment consists of disease-modifying agents that decrease relapse rate and slow the accumulation of brain lesions.[6] Exacerbations are treated with glucocorticoids and plasma exchange. A multidisciplinary approach is important when taking care of patients.

Table 16.1 Upper- vs. lower-motor-neuron patterns of weakness

Upper vs. lower motor neuron	Findings	Examples
Upper	Increased motor tone, reflexes, presence of Babinski sign	Central nervous system lesion/mass or stroke, central nervous system injury such as a spinal cord transection. Amyotrophic lateral sclerosis
Lower	Decreased motor tone, reflexes, absence of Babinski sign	Neuropathies, amyotrophic lateral sclerosis neuromuscular junction disorders

Guillain-Barré Syndrome

Guillain-Barré syndrome (GBS) is a demyelinating polyneuropathy typically with ascending paralysis. It is caused by an autoimmune response directed against myelin or nerve axons. Annual incidence varies from 0.4 to 4 cases/100,000 per year affecting every age group and increasing with age.[7,8] The disease causes disability and can be life-threatening. It is often preceded by a respiratory or gastrointestinal illness, most commonly *Campylobacter jejuni* infection. Other infections include cytomegalovirus (CMV), Epstein-Barr virus (EBV), Influenza A, Mycoplasma pneumoniae, and Hemophilus influenzae. An association between hepatitis E and Zika virus is also observed.[9,10] There has been caution over the development of post-vaccination GBS, though recent data shows that previous concerns were overestimated.[11]

Findings usually reveal a flaccid paralysis of the legs with areflexia ascending into the arms. Autonomic involvement can cause respiratory muscle failure and autonomic instability. Cerebrospinal fluid (CSF) examination classically shows albuminocytologic dissociation with elevated CSF protein levels and normal white blood cell count. Diagnosis may be confirmed with electromyography (EMG)/nerve conduction velocity (NCV), but demyelination may not be seen initially. Treatment usually involves supportive care and either plasma exchange or intravenous immunoglobulin (IVIG). Variations

Table 16.2 Neurological causes of weakness and presentation

Neurological causes	Location/etiology	Presentation of symptoms	Additional workup
Guillain-Barré syndrome	Autoimmune disease with antibodies directed at myelin and/or the Schwann cells	Flaccid paralysis	Cerebrospinal fluid studies
Multiple sclerosis	Demyelination and axonal degeneration	Dependent on location of the lesion	Neuroimaging and cerebrospinal fluid studies
Amyotrophic lateral sclerosis	Degeneration involving the upper and/or lower motor neurons	Weakness, hyperreflexia, spasticity, atrophy, fasciculation. Bulbar and ocular symptoms	Electromyography/nerve conduction velocity
Myasthenia gravis	Neuromuscular junction, antibody response at the acetylcholine receptor	Fatigable weakness of the skeletal muscles	Edrophonium (Tensilon) test, acetylcholine receptor antibody (AChR-Ab), muscle specific tyrosine kinase antibody (MuSK-Ab), electrophysiology
Spinal cord pathology, radiculopathies	Spinal cord, nerve roots	Radicular symptoms (weakness, with pain and sensory deficits)	Imaging, electrophysiology studies
Cerebrovascular disease	Cortical, intraparenchymal. ischemic, hemorrhagic etiologies	Weakness, sensory loss, cortical dysfunction in the distribution of the affected area	Neuroimaging studies

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include acute inflammatory demyelinating polyneuropathy, acute motor axonal neuropathy, and the Miller Fisher Syndrome, presenting with cranial nerve involvement, ataxia, and areflexia.

Motor-Neuron Disease

Motor-neuron disease causes degeneration of upper and/or lower motor neurons, leading to disability and increased mortality. Amyotrophic lateral sclerosis (ALS; Lou Gehrig's disease) is the most common. ALS incidence increases with age, with higher numbers in those over 75.[12] ALS affects males earlier on but shifts to females during later years. Patients may present with weakness, and exam findings include hyperreflexia and spasticity (upper motor neuron) along with atrophy and fasciculation (lower motor neuron). Dysphagia, facial weakness, and ocular symptoms occur when cranial nerves are involved. Diagnosis is based on clinical criteria showing progressive upper- and lower-motor-neuron symptoms. EMG and NCV studies can provide supportive evidence. Treatment includes the glutamate antagonist, Riluzole, shown to prolong survival by 3–6 months,[13] and Edaravone, a free radical scavenger, shown to reduce the rate of functional decline.[14] Treatment otherwise consists of supportive care including ventilatory and nutritional assistance.

Additional forms of motor-neuron diseases include spinal muscular atrophies (lower motor neuron), primary lateral sclerosis (upper motor neuron), and progressive bulbar palsy (upper and lower motor neuron).[15] Symptoms typically arise from the neuronal location affected.

Myasthenia Gravis

Myasthenia gravis typically involves fluctuating and fatigable muscle weakness. It is caused by an antibody response directed at the neuromuscular junction acetylcholine receptor. There is a bimodal age distribution to the incidence of disease. The incidence in those over the age of 65 is 52.9 cases a year per million with a predilection for men later in life, while women are typically affected at younger ages.[16,17] Conditions associated with myasthenia gravis include malignancy, thymoma, and autoimmune diseases. Presenting symptoms can involve any skeletal muscle group with transient symptoms with progressive worsening (Table 16.3). Diagnosis is made clinically while additional testing can be done to support and confirm diagnosis.[16,17] See Table 16.2.

Table 16.3 Skeletal manifestations of myasthenia gravis

Ocular weakness (ptosis, diplopia)
Bulbar weakness (dysarthria, dysphagia, weakness with chewing)
Neck extensor weakness
Limb weakness (proximal greater than distal)
Respiratory weakness (can lead to myasthenia crisis)

Table 16.4 Non-neurological causes of weakness

Cause	Example
Cardiogenic	Congestive heart failure, cardiomyopathy, ischemic heart disease, arrhythmia
Pulmonary	Chronic obstructive pulmonary disease, chronic asthma, pulmonary fibrosis
Myopathy/ rheumatologic	Polymyositis, dermatomyositis, inclusion body myositis, vasculitis, arthritic conditions, connective tissue diseases
Endocrine	Thyroid disease, adrenal disease, parathyroid disorder
Infection	Influenza, HIV, bacterial, viral, parasitic, Bell's palsy
Renal	Renal failure, dehydration, infectious etiologies
Electrolyte	Potassium, sodium, calcium, magnesium disturbances
Medications	Glucocorticoids, antibiotics, statins, chemotherapy, immunosuppression
Psychiatric	Mood disorders, psychogenic etiologies

Treatment consists of acetylcholinesterase inhibition (pyridostigmine), and plasmapheresis and IVIG during acute exacerbations. Chronic treatment with immunosuppression therapy includes corticosteroids, azathioprine, cyclosporine, and mycophenolate. The role of surgical thymectomy in the elderly is unclear, given the likelihood of thymic involution.

Other Causes of Muscle Weakness in the Elderly

The list of other neurological and non-neurological causes of muscle weakness in the elderly is otherwise extensive (Tables 16.2 and 16.4).

Evaluation

Obtaining from patients the history, including onset, duration, associated symptoms, and distribution, helps initiate the appropriate workup. A review of medications and supplements as well as social and family history is necessary to identify potential triggers and hereditary risk

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factors. Common medications amongst older adults contributing to muscle weakness include statin and prednisone use. Adjustment in medications may be considered if related to the development of the muscle complaint. "Red flag" symptoms should alert the clinician to evaluate these patients more urgently (Table 16.5).

Important observations include muscle tenderness, possibly implicating an inflammatory myopathy or atrophy suggesting motor-neuron involvement. Cranial nerves, muscle tone, and reflex examination help to localize possible etiologies as well. NCV and EMG studies help clarify the affected site, confirm the diagnosis, and guide muscle biopsy if needed. Muscle biopsy is useful to distinguish between different myopathies, vasculitic, metabolic, and granulomatous disease, or when diagnosis remains uncertain. Care is dependent on determining the correct etiology of the muscle weakness to provide the appropriate treatment, removing triggering factors, and providing supportive care for affected aspects of daily function.

Sensory Disorders

Changes in sensation can affect gait and quality of life in the older adult.[18] Familiarity with common presenting patterns and etiologies can help alleviate some of the confusing presentations and guide workup. Pathology of sensory loss stems from processes that interrupt the normal flow of sensory input (Table 16.6). After synapsing in the thalamus, neurons carrying information from the different modalities terminate in the primary sensor cortex of the parietal lobe, which is arranged somatographically to correspond to the motor homunculus.

Table 16.5 Potential red flags related to muscle weakness

Aphasia
Dysphagia and changes in voice
Vision changes
Respiratory difficulties
Falls
Presence of pain
Alterations in bowel and bladder function
Altered mental status

Causes of Sensory Loss

Decreases in neuronal fibers, nerve conduction, and capacity for axonal reinnervation commonly occur with aging.[19] Exact mechanisms of neuropathic damage are unknown, though the common mechanistic pathways of nerve damage result in Wallerian degeneration of the axon, myelin sheath, and "dying back" of the nerve cell. The causes of sensory loss outside of normal aging are extensive, and it is useful to separate into central and peripheral etiologies (Tables 16.7 and 16.8).

Diabetic Neuropathy

Diabetes neuropathy has a variety of presentations in the older patient (Table 16.9). About 60–70% of people with diabetes have mild to severe forms of nervous system damage, with approximately 20–30% complaining of decreased sensation in the lower extremities and up to 26% of undiagnosed diabetics found to have peripheral neuropathy in one study.[21–23]

The most common presentation is long axon distal sensory polyneuropathy. Symptoms consist of sensory loss and pain with proximal progression forming the "stocking and glove" distribution. Consequences of neuropathy include infections, limb injury, and impairment of other major organ systems. Neuropathic symptoms also reduce participation in physical and social activities, leading to sleep impairment and chronic pain syndromes.

Diabetes also presents as a small fiber neuropathy with painful dysesthesias and loss of thermal and pain perception. Routine nerve conduction studies typically assess large nerve fibers, and additional examination with newer diagnostic tools such as quantitative sensory testing may be needed for small fiber evaluation.[24] See Tables 16.8 and 16.9 for other causes of polyneuropathy and diabetic neuropathy.[24,25] Treatment usually

Table 16.6 Normal sensory pathways

Sensation	Nerve fibers	Crossover	Site of ascension
Pain, temperature, and touch	Small poorly myelinated fibers	Midline of the spinal cord in the anterior commissure	Contralateral anterior spinothalamic tract (touch) Lateral spinothalamic tract (pain and temperature)
Proprioception, vibratory, pressure, and touch	Large myelinated fibers	Medulla (medial lemniscus)	Ipsilateral medial gracile column (lumbothoracic) Lateral cuneate column (cervical)

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Table 16.7 Sensory patterns due to central nervous system pathology (adapted from Zawora et al.[20])

Pattern	Site, laterality	Symptoms/findings	Cause	Examples
Cape distribution	Central cervical cord lesion, spinothalamic tract, bilateral	Decreased pinprick and temperature	Cord compression, demyelination	Tumor, syrinx, demyelinating diseases
Brown-Séquard	One side of spinal cord, ipsilateral and contralateral symptoms	Decreased proprioception, vibration, weakness (ipsilateral), decreased pinprick, temperature (contralateral)	Unilateral spinal cord damage	Tumor, trauma
Brainstem	Brainstem, most well known is Wallenburg syndrome, lateral medulla, ipsilateral and contralateral involvement	Location dependent, Wallenburg = decreased pain and temperature in the ipsilateral face and contralateral limb and trunk, Horner's syndrome, vestibular and cerebella symptoms can be associated	Damage to the brain stem	Vertebral artery disease, dissection, stroke
Thalamic	Thalamus, unilateral	Contralateral sensory loss in all modalities	Damage to the thalamus	Tumor, abscess, lacunar infarct
Cortical	Sensory cortex	Cortical symptoms, associated symptoms, aphasia, neglect	Damage to the cerebrum	Tumor, demyelination

Table 16.8 Sensory patterns due to peripheral nervous system pathology (adapted from Zawora et al.[20])

Pattern	Site, laterality	Symptoms/findings	Cause	Examples
Mononeuropathy	Individual nerve, unilateral	Sensory loss, paresthesias in distribution of nerve	Damage to the nerve, e.g., entrapment, trauma	Carpal and tarsal tunnel syndrome, ulnar nerve entrapment, meralgia paresthetica. Diabetic neuropathy
Radiculopathy	Nerve root, and corresponding dermatome and myotome, unilateral	Sensory loss, "lancinating pain" exacerbated by cough, sneezing, or straining	Damage to the nerve root, e.g., compression, trauma	Disc herniation, spinal stenosis, trauma, infection, malignancy
Axonal neuropathy	Long nerve axons (small and large fiber), bilateral	Stocking, glove distribution of symptoms, distal to proximal progression. More severe disease involves hands (gloves). Can be loss of pinprick and temperature sensation, proprioception, vibratory sense	Damage to long axon sensory nerves, starting distally. Demyelination, inflammatory, vascular etiologies	Diabetes, vitamin B12 deficiency, alcohol, paraneoplastic syndrome, demyelinating neuropathy (GBS), Central cervical cord lesions such as tumor, cervical spondylosis. Infectious (HIV, syphilis)
Sensory neuropathy	Dorsal root ganglion, can be asymmetric	Numbness, paresthesias, lack motor involvement, but can be disabling. Rare	Degeneration, toxic metabolic, autoimmune	Paraneoplastic disease (small cell lung cancer and anti-Hu antibodies), GBS, Sjögren's, chemotherapy

consists of symptomatic treatment with anticonvulsants and antidepressants.

Post-Herpetic Neuralgia

Post-herpetic neuralgia following shingles can cause painful neuropathy. Studies report 8–24% of all age groups developing some degree of neuralgia, with higher occurrences among those over 50.[23] Pain from the dermatomal vesicular rash of shingles typically decreases within months after onset, but some patients will have persistent pain. If cranial nerves are affected, additional complications may arise, including vision loss if the first division of the trigeminal branch is involved, causing herpes zoster ophthalmicus.[26,27]

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Table 16.9 Different forms of neuropathy due to diabetes[22]

Type of diabetic neuropathy	Presenting symptoms
Sensory long axon peripheral neuropathy	Varying degrees of sensory loss, paresthesias, allodynia, pain, stocking and glove distribution
Small fiber neuropathy	Pain, diminished temperature sensations
Autonomic neuropathy	Affects various organ systems: Gastrointestinal: esophageal dysmotility, gastroparesis, constipation or diarrhea, fecal incontinence Genitourinary: neurogenic bladder, sexual dysfunction Cardiovascular: tachycardia, orthostatic hypotension Anhidrosis, heat intolerance
Cranial neuropathy	3rd cranial nerve palsy, double vision, eyelid droop, dysconjugate gaze Facial nerve palsy
Compression and entrapment neuropathy	Median, radial, ulnar and peroneal neuropathies
Trunk mononeuropathy	Dysesthesias of the thoracic dermatomes or radicular thoracic pain
Amyotrophy	Lumbosacral plexus neuropathy, asymmetric, lower limb atrophy and weakness, loss of reflexes. Sudden onset of sharp pain
Mononeuropathy multiplex	Multiple mononeuropathies occurring in the same patient

Diagnosis is clinical, but sometimes viral culture or PCR analysis is necessary for confirmation. Treatment consists of antivirals (acyclovir, valacyclovir) for the initial attack, with symptomatic relief for post-herpetic neuralgia. To reduce the risk of occurrence, two vaccines are available. The recombinant vaccine was shown to have higher efficacy, although no head-to-head trials have been done comparing these vaccines.[28]

Demyelinating Neuropathies

Demyelinating neuropathies include GBS, chronic inflammatory demyelinating polyneuropathy (CIDP), and multiple sclerosis.

CIDP is a heterogeneous sensorimotor neuropathy with variations of proximal and distal muscle weakness along with sensory loss and different paresthesias.[29] The condition has a higher incidence and prevalence in the elderly and is thought to be autoimmune driven.[30,31] The clinical course is variable with relapsing/recurring and chronic progressive presentations. Treatment involves immunotherapy including corticosteroid, IVIG, plasma exchange, and corticosteroid-sparing agents.[29]

Others Causes of Neuropathy

Alcohol is a common etiology of neuropathy and may cause direct toxicity versus contributing to deficiency of vitamins and other nutrients.[32] Alcoholic neuropathy can have different presentations including painful paresthesias and a distal sensory neuropathy. Treatment is aimed at halting progression with reduction/cessation of alcohol and symptomatic treatment.

Vitamin B12 deficiency occurs because of dietary deficiency or malabsorption from atrophic gastritis, chronic use of acid-blocking medications, or *Helicobacter pylori* infection. Vitamin B12 is necessary for optimal nerve functioning, and deficiency can lead to subacute combined degeneration. Laboratory findings include decreased B12 and elevated methylmalonic acid and homocysteine levels. Parenteral therapy is usually required for severe neurological dysfunction.[33]

Monoclonal gammopathy and other plasma cell disorders can present with peripheral neuropathy. Monoclonal protein is suspected to be involved in about 10% of unknown causes of peripheral neuropathy, with a higher association in IgM monoclonal gammopathy.[34] Presentations include paresthesias, pain, and sensory loss in a stocking and glove distribution with proximal progression. Treatment is aimed at the monoclonal gammopathy with immune suppression, along with symptomatic treatment.

In elderly patients as in any age group, an inconsistent sensory exam might suggest a functional or psychogenic cause. Psychological and social stressors must be identified, and this is a diagnosis of exclusion.

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Evaluation

Efforts should be made to accurately describe changes in sensation, the time course, and the sites involved (Table 16.10). Further characterization of the paresthesias helps differentiate small fiber (burning, stinging) from large fiber involvement (tingling, pins and needles). A thorough review of medical problems, medications, and psychosocial history (exposure to toxin, travel, HIV risk factors) is helpful in identifying possible etiologies.

Table 16.11 lists sensory examination techniques in the office. Since common neuropathic complaints involve the long axonal nerves, one can start distally and march proximally to detect the level of the sensory deficit, with attention to the opposing extremity for lateralization of the deficit.

Laboratory and electrophysiologic testing is driven by suspected systemic etiology. EMG and NCV studies help distinguish sensory loss disorders from other myopathies and neuromuscular junction conditions. Lumbar puncture may be required if there is a high index of suspicion for infectious or demyelinating etiology such as GBS.

Treatment

Treatment for sensory neuropathies in the elderly patient involves treating the underlying condition and providing symptomatic relief. Counseling on reducing fall risks, use of an assistive device during ambulation, and instructions on reducing safety risks should be given to the patient and family members. While pain due to neuropathy can be debilitating, treatment with medications is often limited by side effects and drug interactions. Opioids use should be limited in older patients because of side effects.[35,36] See Table 16.12.

Table 16.10 Sensory terms

Sensory term	Definition
Hypesthesia/ hypoesthesia	Decreased ability to perceive sensation
Anesthesia	Inability to perceive sensation
Hypoalgesia	Decreased sensitivity to pain, while analgesia is the complete insensitivity to pain
Hyperesthesia	Increased sensitivity to pain
Allodynia	Pain due to a stimulus that is not normally painful
Dysesthesia	Altered sensation
Mononeuropathy	Single peripheral nerve being affected
Mononeuritis or mononeuropathy multiplex	Multiple single peripheral nerve lesions affected simultaneously because of the same disease process
Polyneuropathy	Multiple peripheral nerves affected, with similar distribution on both sides

Table 16.11 Office sensory examination

Sensory modality	Technique
Pain	Sharp end of cotton swab broken in half
Temperature	Cold can be checked with small plastic bottles of hot or cold water, cool tuning fork, or metal handle of reflex hammer
Vibratory sense	128 Hz tuning fork to bony prominence in lower extremities
Proprioception	Ask the patient to identify movement of the great toe, in various directions. Romberg test integrates proprioception and postural stability (positive, if by closing eyes the patient loses balance – suggests that proprioception is impaired as stability is dependent on the visual system)

Seizure Disorders

Seizures can have a devastating impact on the older adult with risk for physical injury and altering quality of life.[37] Seizures have the highest prevalence amongst children and the elderly but are often difficult to recognize in the older patient.[38]

Background

At least 25% of all new-onset seizures occur in the elderly, while the prevalence rate is approximately 1.5% in the United States compared to 0.5% in younger adults.[38–40] Those living in nursing facilities have seizure and epilepsy rates as high as 9% or higher.[39,41,42] The incidence of epilepsy, defined as recurrent unprovoked seizures without treatment, increases with age as well.[43,44] The most common cause of seizures in the elderly is cerebrovascular disease: 30–40% of epileptic seizures occur in those who have had a stroke, while up to 9% of elderly patients who have had a stroke will suffer a seizure.[39,45] Seizures are more likely to occur with hemorrhagic stroke, larger infarct size, and cortical rather than subcortical involvement.[38,46]

Alzheimer's disease and other dementias prevalent in the elderly population are a risk factor for seizures.[47,48] Up to 10–20% of cases of epilepsy are likely due to dementia.[46] Possible mechanisms include neurotoxicity from

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Table 16.12 Neuropathic pain medications[22,36]

Medications used for neuropathic pain	Advantages	Limiting factors in elderly patients
Tricyclic antidepressants	Low cost, easy dosing, antidepressant, works on sleep	Risk of anticholinergic side effects (dry mouth, constipation, orthostatic hypotension, cardiac toxicity)
Serotonin/norepinephrine reuptake inhibitors (duloxetine, venlafaxine)	Duloxetine efficacy in painful diabetic peripheral neuropathy, depression, fewer cardiac effects Venlafaxine, effective in painful polyneuropathies	Duloxetine – nausea Venlafaxine – cardiac toxicity
Anticonvulsants (gabapentin, pregabalin, carbamazepine)	Efficacy in neuropathic pain; fewer drug interactions CBZ/-trigeminal neuralgia	Dizziness, sedation, limited use with renal insufficiency
Opioids and tramadol	Efficacy in neuropathic pain	Gastrointestinal side effects, nausea, sedation, risk of confusion, risk of abuse Tramadol lowers seizure threshold, risk of serotonin syndrome
Topical lidocaine	Local efficacy for neuropathic pain	Skin irritation

beta amyloid protein, neurotransmitter disruption, and mutations in gamma secretase and amyloid precursor protein genes. These mutations are associated with early-onset familial dementias and seizures.[49] Younger age of dementia onset, advanced disease, and history of antipsychotic use are associated with greater incidence of seizures.[46,47,49,50]

Up to 20% of cases of epilepsy in the elderly are seen with trauma, while 8–45% of seizures are attributable to tumors.[38,46] Other causes include alcohol use and withdrawal, other medical illnesses, medications, and infectious etiologies.

Seizure Presentation in the Elderly

Seizure presentation in the elderly is variable and may not always consist of convulsions. Often symptoms may mimic other conditions; see Tables 16.13 and 16.14.

Status epilepticus is the ongoing occurrence of a seizure or frequent seizure episodes without a return to baseline. Both convulsive and nonconvulsive status epilepticus have a higher frequency in the elderly.[38,51] Convulsive status epilepticus is easier to recognize because of motor symptoms, while nonconvulsive status is difficult to detect because of indistinct presentations. Symptoms include altered mental status, and lethargy in the ambulatory setting. Exacerbations of an existing epilepsy condition, the presence of intracranial pathology, and toxic metabolic disturbances are common causes of nonconvulsive status.[51,52]

Evaluation

Workup up includes obtaining history on presentation and time course of the episode, provoking factors, and examination for deficits and injuries. Associated symptoms, concurrent medical illnesses, and medication review should be gathered (Table 16.15). Postictal confusion can limit history-taking from the older patient, and collateral information is often necessary.

Laboratory studies include evaluating for metabolic disturbance, hepatic and renal function, complete blood count, and thyroid function. Urine tests, cultures, toxicology, and alcohol screening along with syphilis and other infectious screens may be indicated. Concerns for CNS infection and hemorrhage may prompt CSF analysis. Neuroimaging should be checked with new-onset seizures to exclude ischemia, lesions, or inflammatory conditions because of the higher prevalence in this population.

EEG can identify an epileptogenic focus, as well as periodic lateralizing epileptiform discharges (PLEDS) suggesting increased risk for seizures.[39] If concerned for status epilepticus, EEG is useful for confirming the diagnosis. A limitation is that seizure activity may not be present during interictal periods and a normal EEG may not exclude seizure. Other nonspecific EEG findings in the elderly patient such as generalized slowing can occur in a variety of medical conditions and may be seen without seizure activity. Video and ambulatory EEG may provide additional value when available.[53]

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Table 16.13 Differences in seizures between the elderly and the younger patient

	Younger patient	Elderly patient
Type of seizure	Generalized	Focal, partial with secondary generalization
Origin of seizure	Temporal lobe common	Focal areas due to stroke, tumors, head injury, dementia
Symptoms	Prodromal symptoms including déjà vu or olfactory hallucinations, may include facial and motor automatisms	May include nonspecific symptoms, dizziness, paresthesias, confusion Longer postictal period

Table 16.14 Common conditions with similar presentations to seizures

Condition	Workup
Delirium/syncope	Medical workup including cardiac and neurological based on history and exam
Memory loss, transient global amnesia, dementia	Neurocognitive testing, neuroimaging, consideration for cerebrospinal fluid studies in addition to above
Transient ischemic attacks	Neuroimaging with CT/MRI, carotid evaluation, ECG, and telemetry monitoring
Sleep disorders	Sleep testing, polysomnography

Management of Seizures

Initial management of a seizure in the elderly is like that of any age group. The patient is triaged, and basic life support measures are initiated, followed by administering a benzodiazepine and an antiepileptic agent as appropriate.[54]

Identifying provoking causes is important for assessing risk for recurrence and need for maintenance therapy. A seizure caused by a metabolic disturbance, medication, or withdrawal will generally not require maintenance therapy. Seizures due to certain intracranial events, such as a stroke or head trauma, may warrant treatment for a limited period. Neurology consultation can assist in diagnosis and management.

Initiating treatment in recurrent unprovoked seizures in the elderly can be a difficult decision, and is limited by available data. Typically, therapy is started after a second occurrence. However, if risk factors for recurrence such as stroke are present, consideration is often given to initiate antiepileptic therapy after the first seizure.[46] The risks and benefits of initiation of antiepileptic therapy versus no therapy should be discussed with older patients.

Table 16.15 Risk factors for seizures to assess for on history-taking

Cerebrovascular disease history
Previous seizure episodes and provoking factors if known
History of and risk factors for malignancy
Previous infections, especially central nervous system infections
Head trauma
Alcohol and drug use

Aging-related changes in pharmacokinetics, drug metabolism, interactions, and side effects should be considered when choosing a seizure medication. Newer agents are preferred because of the simpler pharmacokinetics and decreased side effects and drug interactions compared to older agents. See Table 16.16.[46,55–57]

Serious but rare adverse effects such as agranulocytosis, aplastic anemia, Stevens Johnson syndrome, hepatic failure, and cardiac arrhythmias can be seen with carbamazepine, phenytoin, valproic acid, and other antiepileptic medications. Osteomalacia and osteoporosis can be associated with certain long-term antiepileptic therapies requiring consideration for bone density monitoring and osteoporosis therapy.

The duration of antiepileptic therapy is controversial. In the elderly, the risk for recurrence of seizures is high because of underlying risk factors. With appropriate treatment, seizure control is attained in 70% or more of older patients.[38] For discontinuing therapy, recommendations on when to start a weaning trial vary from 6 months to 3 years after being seizure-free.[58] The patient should be counseled on risk for recurrence and consequences.

Resection of an epileptic focus and vagal nerve stimulation are additional treatment options for refractory and individualized cases. Other long-term management issues to discuss with older patients surround lifestyle and safety regarding driving, bathing, and swimming. Individual states have different safety laws regarding driving and patients should be counseled accordingly.

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Table 16.16 Seizure medications used in the elderly population (adapted from Zawora et al.[20])

Medication	Adverse effects	Pharmacological considerations	Special considerations in the elderly
Carbamazepine	Many drug interactions, can cause aplastic anemia, agranulocytosis, syndrome of inappropriate secretion of antidiuretic hormone (SIADH), rash, can cause central nervous system depression	High protein binding, enzymatic inducer; hepatic metabolism, urine and feces excretion	Partial seizure use, can cause cardiac conduction abnormalities, contraindicated if using monoamine oxidase inhibitors. Can cause bone loss. Relatively cheap
Eslicarbazepine	Dizziness, somnolence, headache, fatigue, hepatotoxicity	Caution in renal and hepatic impairment	Adjunctive use in partial seizures, hyponatremia, multiorgan hypersensitivity reactions, enzyme induction, drug interactions
Gabapentin	Somnolence and fatigue, weight gain, dizziness, ataxia	Caution in renal insufficiency	Well tolerated for partial seizures. Renal adjust dosing. Limited by multiple dosing, dizziness. Few interactions
Lamotrigine	Aseptic meningitis reported, skin reactions, multiorgan hypersensitivity, sleep complaints	Hepatic and renal, glucuronic acid conjugation	Partial, tonic-clonic seizures, not altered by renal impairment,[38] good tolerability in the elderly.[55]
Levetiracetam	Behavior problems, sedation	Enzymatic hydrolysis, excreted in urine. Dosage adjustment with renal impairment	Fewer drug interactions, partial, tonic-clonic seizures. Can be used in status epilepticus[54] Cognitively benign profile, may be better tolerated in dementia patients[56,57]
Oxcarbazepine	Better tolerability, allergic rash, hyponatremia, ataxia, tremor, fatigue, dizziness a big side effect	Selective enzymatic induction, still with drug interactions. Glucuronidation, excreted in urine	Partial seizures, better tolerance than carbamazepine. Can cause hyponatremia, neurotoxicity, cardiac toxicity. Contributes to vertigo, dizziness, tremor. Potential hematologic adverse effects
Phenytoin/ fosphenytoin	Neuropathy, osteomalacia, gingival hypertrophy, pancytopenia, hepatotoxicity, cardiac arrhythmias, dizziness, psych changes	Heavy protein binding, urine exertion, saturation metabolism, nonlinear kinetics, enzyme induction	Partial, tonic-clonic seizures including status epilepticus, cheap, cognitive effects, osteomalacia and osteoporosis. Many drug interactions (warfarin, tricyclic antidepressants, diabetes, chemotherapeutics)
Pregabalin	Dizziness, somnolence, central nervous system effects	Urine excretion, caution in renal insufficiency	Lower doses needed for therapeutic benefit, adjunctive therapy for partial seizures, useful in other conditions, (neuropathic pain). Renal dosing
Tiagabine	Sedation, dizziness, central nervous system effects	Hepatic metabolism	Adjunctive use in partial seizures, drug interactions
Topiramate	Central nervous system effects, anorexia, weight loss, weakness	Urine excretion, renal adjustments, some liver metabolism, possible decreased clearance with age	Partial and tonic-clonic seizures, can cause cognitive impairment, renal stones. Can cause weight loss
Valproic acid	Weight gain, gastrointestinal upset, hair loss, bruising, tremor dizziness, changes in mood, parkinsonism, osteoporosis, ataxia, hepatotoxicity	High protein binding, longer half-life, drug interactions, hepatic metabolism, not recommended in hepatic impairment, and urine clearance, caution in renal impairment	Can be used in status epilepticus.[54] Partial and complex seizures, broad spectrum, drug interactions, enzymatic induction, drug interactions, can be useful in other conditions (bipolar disorder, migraine prophylaxis)
Zonisamide	Somnolence, dizziness, weight loss	Caution in renal and hepatic impairment. Not recommended in severe renal impairment. Hepatic metabolism	Adjunctive use in partial seizures, limited data for use in the elderly, can cause kidney stones. Drug interactions

Headaches

Primary headaches are defined as headaches without clear anatomical reasons, whereas secondary headaches are due to another underlying condition. In general, new-onset primary headaches decrease with age but still make up a large portion of headaches in this age group.[59] The more common primary headaches are described below. Additional primary headaches that are rare but more common to the elderly patient are listed in Table 16.17. New headaches in the elderly should prompt evaluation for a secondary cause due to potentially serious conditions that increase with age.[60,61]

Primary Headaches

Tension-Type Headache

Tension-type headaches decline with age but still are the most common headache in the elderly, with prevalence ranging between 35.8% and 44.5%.[59,60] Often described as a bilateral headache with tightness or pressing pain, it is usually less disabling and doesn't affect daily activities. Precipitating factors include stress and sleep disruptions, and can be associated with mood disorders. Features such as nausea, vomiting, and photophobia are typically absent, and examination may reveal tenderness of the head, neck, or shoulder muscles.

Patients frequently self-diagnose and treat tension headaches. Simple analgesics such as aspirin, NSAIDs, and acetaminophen generally have good efficacy. See Table 16.18 for treatment options and adverse effects of many analgesics. Additional nonpharmacological treatment with biofeedback, massage, heat, and ice may be useful.

When analgesics are used frequently, a prophylactic medication can be considered (Table 16.19). Considerations for the older patient include starting with low doses and titrating to the lowest effective dose unless adverse effects develop. Prophylactic medications should be given an adequate trial duration, measuring effectiveness by tracking reductions in headache frequency and avoiding additional analgesic use.

Migraines

Migraines are the second most common primary headache syndrome in the elderly and may have different presentations. New-onset migraines in the elderly have an annual incidence of 2%. Some older patients have a preexisting diagnosis continued from a younger age.[60]

Migraines may evolve through four phases: premonitory, aura, headache, and postdromal. The premonitory phase presents with systemic complaints like fatigue and irritability,[65] while auras consists of sensory disturbances, often visual. These may be followed by headaches and a prodromal phase of fatigue and lethargy. Older patients may not experience all four phases of migraines, and duration of each phase can vary. Acute treatment typically involves halting the headache (Table 16.18). Nonspecific analgesics such as NSAIDs and acetaminophen are effective but are limited by side effects. Antiemetic therapy is useful for reducing headaches associated with nausea and vomiting and can be used in combination with simple analgesics. Triptans affect the meningeal blood vessels, the trigeminal nerve endings, and second-order neuron synapses in the trigeminocervical complex.[59] Ergotamines are alkaloids that bind serotonin receptors as well as dopamine and noradrenergic receptors. These medications should not be used in patients with vascular disease and can cause nausea.

Preventive medications (Table 16.19) may be considered if there is increased frequency, disruption of daily activities, contraindications to acute therapy, or associated neurological complications.[59] Treatment options can be chosen based on tolerability and coexisting medical conditions.

Cluster Headache

Cluster headaches are the most common form of trigeminal autonomic cephalalgias (TACs), presenting as a unilateral sharp, stabbing headache in the temporal and orbital regions.[62] Findings include ipsilateral autonomic features of nasal congestion, ptosis, and rhinorrhea, and can occur in short-lived episodes throughout the day that are not relieved with sleep or rest. First-line therapy includes oxygen at a flow rate of 6–12 liters per minute and triptans. Preventive treatment can be used for recurrent attacks and minimizing adverse effects of frequent acute treatments. Options include lithium and high-dose verapamil, which may be limited by adverse effects. Topiramate is an effective add-on to avoid high-dose verapamil monotherapy but is also limited by tolerability.

Secondary Headache Syndromes

New-onset headaches, worsening of preexisting headaches, and presence of "red flag" symptoms should prompt urgent evaluation, as up to 15% of headaches in the elderly have serious underlying causes (Tables 16.20[60] and 16.21).

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Table 16.17 Other primary headache types[59,62,63]

Primary headache types	Characteristics	Treatment considerations in the elderly
Hypnic	"Alarm clock headache," low prevalence. Nocturnal onset lacks migrainous/autonomic features. Presents with diffuse, non-throbbing moderate severity, can be frontotemporal in location. More common in women.	Reported relief with sitting up and pacing. Prophylaxis with lithium or indomethacin, but may be limited due to side effects. Nighttime caffeine or melatonin may also be effective.
Exploding headache syndrome	Rare. Sensation of a loud, painless, explosive noise has occurred in the head. Usually upon awakening, or while falling asleep; some patients also note flashing lights. Triggers include sleep deprivation and stress.	Benign condition. Avoid triggers. Tricyclic antidepressants and topiramate may be useful for recurrent attacks.
Uncommon trigeminal autonomic cephalalgias (TACs)	Short-lasting, unilateral, neuralgiform headache with conjunctival injection and tearing (SUNCT); short-lasting, unilateral, neuralgiform headache with autonomic symptoms (SUNA). Unilateral, sharp, stabbing, severe pain, usually in the orbital or temporal areas. Usually involves V1 distribution of trigeminal nerve. Associated with conjunctival injection and tearing (SUNCT) or cranial autonomic features (SUNA) including nasal stuffiness, rhinorrhea, ptosis. Duration lasting minutes, with high frequency of attacks (separates from trigeminal neuralgia).	Evaluate for secondary causes, including meningioma, cerebellar pontine angle tumors, brainstem infarcts, intraorbital lesions. Oxygen, sumatriptan, verapamil not effective as in cluster headaches, can do trial with anticonvulsants (lamotrigine, topiramate, gabapentin).
Primary cough headaches	Paroxysmal pain, occurring during cough, Valsalva maneuver, usually following upper-respiratory illness. May be due to temporary sensitivity of the carotid baroreceptors to increased intrathoracic pressure. More common in older patients. Secondary causes occur 40% of the time but are more common in younger patients.	Generally resolves following illness. Treatments include indomethacin and lumbar puncture to lower intracranial pressures and reset receptors.

Table 16.18 Acute primary headache treatments

Medication	Indications	Considerations for use in the elderly
Oxygen, high flow	Cluster headache	Relatively safe in the elderly, caution in chronic obstructive pulmonary disease
Caffeine	Migraines, tension-type headaches	Gastrointestinal upset, palpitations, and tachyarrhythmia
Acetaminophen	Migraines, tension-type	Caution in liver dysfunction
NSAIDs	Migraines, tension-type	Risk for gastrointestinal side effects, bleeding. Caution with renal dysfunction
Opioids and barbiturates	Broad range of acute headache	Risk of abuse
Antiemetics (metoclopramide, chlorpromazine)	Migraines with nausea and vomiting	Extrapyramidal side effects, parkinsonism, QT prolongation, sedation, orthostatic hypotension
Triptans	Moderate to severe migraines	Comes in oral, nasal, and parenteral preparations. Caution: limited use in patients with vascular disease
Ergotamines	Migraines	Also available in oral, nasal, and parenteral preparations. Caution: limited use in patients with vascular disease

Table 16.19 Prophylactic treatment for primary headaches

Medication	Indications	Considerations for use in the elderly
Tricyclic antidepressants	Migraines, tension	Anticholinergic adverse effects, sedation, hypotension, QT prolongation, urinary retention, confusion
Venlafaxine	Migraines[64]	Useful for coexisting mood disorder
Valproate	Migraines, cluster	Adverse effects include gastrointestinal symptoms, liver dysfunction, tremor, weight gain, sedation
Topiramate	Migraines, cluster	Central nervous system effects, cognitive side effects, dizziness, sedation, nausea, anorexia. contraindicated if nephrolithiasis is present
Beta-blockers	Migraines	May have benefits if other coexisting conditions (tremor, congestive heart failure). Caution in elderly patients with diabetes, chronic obstructive pulmonary disease, asthma, depression. Adverse effects include hypotension, bradycardia, lethargy, and sedation
Calcium channel blockers	Migraines (weaker data), cluster (verapamil)	High doses may be required, arrhythmia, hypotension, edema, constipation. Drug interactions. Dizziness
Lithium	Cluster, hypnic[59]	Many side effects including tremor, cognitive impairment, weakness, nausea

Headache Induced by Neuralgias

Headache with herpes zoster can occur in the form of a generalized prodromal headache or focal craniofacial pain if the trigeminal nerve is involved. Persistence of burning and/or lancinating pain can lead to debilitating post-herpetic neuralgia. Vaccination helps with prevention, and antiviral medications reduce the severity and duration of acute attacks. Neuropathic pain relief typically includes tricyclic antidepressants and antiepileptic medications.

Trigeminal neuralgia is the most common neuralgia in the elderly. It causes a paroxysmal unilateral headache characterized by sharp or electrical pains in the V2 or V3 divisions of the trigeminal nerve, and less frequently the V1 (ophthalmic) branch. The pain is short-lived but can be recurrent.[60] Triggers include cold air, speaking, chewing, grimacing, or light touch along the affected nerve. Etiologies include demyelination of the trigeminal nerve from multiple sclerosis and compression by structural lesions such as arteriovenous malformations and tumors. Neuroimaging may be required if there are other focal findings on examination.

Treatment generally involves antiepileptic therapy, with carbamazepine the best studied. Additional agents like lamotrigine, gabapentin, phenytoin, and baclofen have varying efficacy. When tolerated, medical treatment is effective in about 70% of patients. Microvascular decompression, rhizotomy, or radiosurgery are treatment options if refractory to medications.

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Table 16.20 Secondary headaches[60,62]

Secondary headache types	Presenting features	Diagnostics and treatment considerations in the elderly
Subarachnoid hemorrhage	Sudden onset of "thunderclap" headache; "worst headache" of life; can be associated with nausea, vomiting, hypertension syncope, neck pain, coma, confusion. Sometimes a sentinel headache in the days prior to the SAH. May present with a normal neuro examination. 40–60% of SAH associated with headache. Etiology includes ruptures aneurysm, arteriovenous malformation, high blood pressure.	Non-contrast CT scan. Lumbar puncture (presence of xanthochromia) if further out from onset of symptoms. Prognosis is worse in older patients (15% of patients >75 returning to independent living at discharge). Early surgical consultation and treatment if possible.
Subdural hematoma	Complaints of mild headache (up to 90% of patients with chronic subdural hematoma). Peak incidence in 6th and 7th decades of life for chronic subdural hematomas (more than 20 days); 80% occurring in elderly men.	Non-contrast enhanced CT scan of the head. Early surgical consultation recommended. Stop blood thinners.
Stroke (cerebral vascular accident)	Described as dull/throbbing, mild to severe. Can be ipsilateral or generalized, associated with nausea and vomiting, and may have a premonitory headache preceding the event by days to weeks. Headache occurs in 17–25% of cases.[60,67]	Non-contrast CT/MRI. Early neurology evaluation, TPA, and antiplatelet therapy.
Neoplasm/mass	Typically described as morning headache that worsens with positional change; associated with nausea and vomiting. Can resemble tension-type headache. May have other neurological deficits. Supportive exam findings include focal deficits, mental status changes, papilledema.	CT/MRI. Incidence of primary and metastatic tumors increases with age.
Meningitis/encephalitis	Initial symptoms may include headache, fever, altered mental status, nuchal rigidity. Additional symptoms can include confusion, nausea, vomiting, altered mental status, photophobia, malaise.	Meningitis: lumbar puncture, with gram stain and culture. 18% mortality in meningitis in all age groups, increases with age. Start empiric antibiotics on suspicion of bacterial meningitis, based on age and health status. Aseptic meningitis usually requires supportive care. Encephalitis: lumbar puncture may show lymphocytic pleocytosis in the cerebrospinal fluid. High mortality, up to 10%. Can result in neurologic damage; herpes is most common (HSV-1). Treat with acyclovir. Intensive monitoring required.
Giant cell arteritis	Classic symptoms are temporal orbital headache, jaw/tongue claudication, and visual changes. Headache occurs in approximately 2/3rds of patients. Can be a continuous or intermittent headache, typically throbbing. Approximately 50% of patients with painful chewing. Permanent loss of vision in up to 20% of patients.	Examination may reveal temporal artery tenderness or nodularity, possible erythema. Absence does not exclude the diagnosis. Check erythrocyte sedimentation rate, C-reactive protein. Temporal artery biopsy may be required. High-dose corticosteroids should be started on suspicion, temporary artery biopsy can still be performed up to 7 days after initiation of corticosteroids

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Acute-angle closure glaucoma	Prevalence increases with age, more common in elderly women. Usually sudden onset of severe pain, with unilateral headache, may also have blurred vision, nausea and vomiting. Exam with fixed mid-position pupil, hazy cornea, "rock hard" globe on palpation of the eye.	Examination of the eye, referral to ophthalmology to measure the intraocular pressure (normal <20 mmHG). Treatment to lower pressures – beta-blockers, acetazolamide, topical parasympathomimetic agents, mannitol, definitive treatment with laser iridectomy.
Hypoxia/carbon monoxide	No characteristic pattern. Associated symptoms: altered mental status, blurred vision, weakness, lethargy. Look for multiple members with similar symptoms. Ask about living situation, furnace, indoor use of grills/gas stoves for heating (may be seen in dementia patients).	Elevated carboxyhemoglobin level on ABG. Requires high index of suspicion Treatment with oxygen, treat underlying cause.
Headache from post- herpetic neuralgia	Following shingles. Burning/lancinating pain; can affect vision if eyes involved.	Shingles vaccination, treatment with antiviral medications.
Trigeminal neuralgia headache	Paroxysmal, unilateral headache, sharp, electrical pains, usually V2/V3 of the trigeminal nerve related. Short pains, recurrent.	Carbamazepine effective, may be limited by side effects. Other agents include lamotrigine, abapentin
Medication-induced headaches	Adverse effects of medication, or from rebound headaches that occur with chronic analgesic use for chronic headache disorder.	Discontinue drugs that trigger the headaches. For medication withdrawal headaches, discontinue the offending agent, and use different agent for symptomatic relief.

Medication-Related Headaches

Some common medications that can cause headaches include antibiotics, antihypertensives, hormonal therapies, H-2 blockers, NSAIDs, sedatives, and stimulants including caffeine.[60,62] Chronic analgesic use can also transform previous episodic headache conditions into chronic daily headaches. Peak prevalence occurs in the late 40s and declines with age, but can still occur in those over the age of 65.[66] Withdrawal or rebound headaches are most common with opioids and analgesics combined with barbiturates and caffeine but can also be seen with triptans and NSAIDs.[60] Withdrawal symptoms include worsening headache, nausea, vomiting, anxiety, and sleep disturbances. Treatment usually requires discontinuing the offending agent. Rescue treatments for withdrawal symptoms include antiemetics, alternative analgesics, intravenous hydration, and other nonpharmacologic approaches.[66] If the patient reverts to baseline and persistent episodic headaches still occur, a prophylactic headache medication can be tried to reduce the frequency of episodes.

See Table 16.20 for additional causes of secondary headache including intracranial masses and cardiovascular disease.

Headache Evaluation

Because of the higher prevalence of secondary headaches in the elderly, evaluation should assess for red flag complaints and underlying causes. History-taking includes assessment for history of malignancy, substance use, medications, head trauma, and uncontrolled medical conditions. See Figure 16.1.

Table 16.21 Potential red flag symptoms related to headaches[68]

Systemic symptoms including fever
History of neoplasm
Presence of new neuro-deficits
Sudden-onset headaches
Older age >50 years
Recent-onset headache or change of pattern, progressive headache or atypical symptoms
Positional headache
Precipitation of headache with cough, sneezing, or exercise
Papilledema
Painful eye with autonomic features
History of immune deficiency (risk of opportunistic infections)
Analgesic overuse or new medication

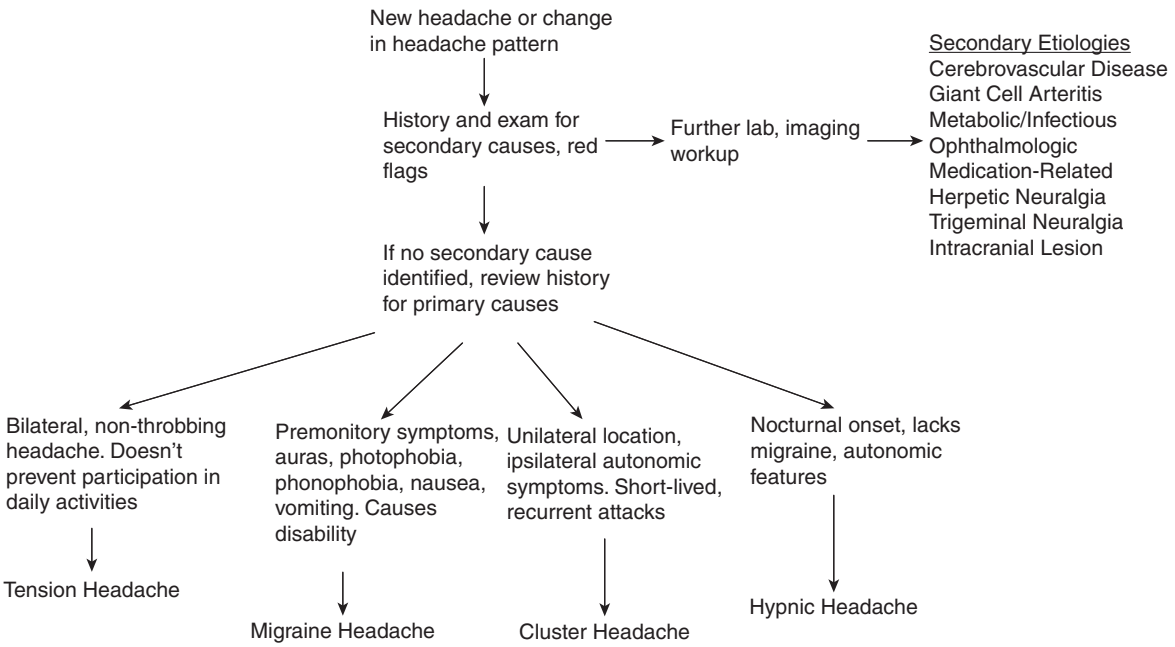

Figure 16.1 Approach to new headaches or a change in existing headache in the elderly.

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Neurological examination includes evaluation for neurological deficits, mental status change, weakness, abnormalities of balance, coordination, and reflexes. Vital signs, cardiopulmonary, eye, head, and neck examination may reveal otolaryngologic, ophthalmologic, and cardiopulmonary causes. If there is a concern for trauma or falls, assessing for injuries should be done.

Laboratory workup is guided by history and exam to identify secondary causes. CT or MRI of the brain should be considered with new-onset headache in the elderly and the presence of red flag signs such as mental status changes, focal neurologic deficits, seizures, post-trauma headaches, and papilledema.

Conclusion

Neurological problems are common in the elderly, a rapidly growing segment of the population. Significant disability can occur if neurological issues are not addressed in the elderly patient, and it is important for the clinician to become familiar with these disorders in order to promptly recognize and manage to help alleviate unnecessary decreases in the quality of life of older patients.

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Chapter 17

Prevention, Diagnosis, and Management of Stroke

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Introduction

Stroke is a leading cause of long-term disability and is the fifth leading cause of death in the United States.[1,2] Strokes can occur at any age; however, advancing age remains a leading nonmodifiable risk factor for first stroke. The risk of having a stroke more than doubles each decade after the age of 55, and up to 75% of all strokes happen in people over the age of 65.[2]

Stroke is defined as an episode of sudden neurological dysfunction, caused by focal ischemia of the central nervous system (CNS). This may include brain, spinal cord, or retinal ischemia that results in infarction. The diagnosis of stroke may be made with clinical, imaging, or pathological data.[3] Thus, the absence of persistent symptoms, but presence of neuroimaging data to support infarction, would fit with this definition of stroke ("silent infarction" or "silent cerebral hemorrhage").

The definition of transient ischemic attack (TIA) was updated in 2009[4] to reflect that TIA is a *transient* episode of neurological dysfunction, caused by focal ischemia of the brain, spinal cord, or retina *without acute infarction*.[4,5]

Epidemiology

Approximately 795,000 people in the USA have a stroke each year,[2] with one third of all strokes occurring in those over age 80. This poses challenges to evidence-based treatment and prevention of stroke, since this age group was largely excluded from many pivotal randomized controlled trials (RCTs) for both stroke treatment and prevention. Despite improvements in stroke prevention, the mean global lifetime risk of stroke increased from 22.8% in 2009 to 24.9% in 2016.[1] In addition to the human cost of stroke, the total financial cost of caring for stroke patients, in the USA alone, according to 2016 US dollar values, was $103.5 billion; 66% of this cost is accounted for by indirect costs from underemployment and death.[6]

In the USA, the age-adjusted stroke mortality for those older than 45 has dropped 77% in the last 50 years. Despite these positive trends in stroke morbidity and mortality overall in the USA, stroke mortality remains approximately 27% higher in the "Stroke Belt," a region of the USA that includes the states of Alabama, Arkansas, Georgia, Louisiana, Mississippi, North Carolina, South Carolina, and Tennessee.[7] Additionally, mortality rates are higher in African Americans.[8] These disparities have existed since at least 1940,[7] the cause of which remains a mystery.

Stroke Symptoms and Initial Assessment

Symptoms of stroke depend on the area of ischemia. However, typically these include the acute onset of symptoms that may include unilateral numbness, weakness, facial droop, speech disturbance (either slurred speech or aphasia), vision loss, vertigo/dizziness, and incoordination or imbalance. Other symptoms that may suggest hemorrhagic stroke include severe headache, altered mentation, and nausea and/or vomiting. Identifying the time when the patient was last seen normal is critical to determine whether they may be a candidate for treatment such as tissue plasminogen activator (tPA), alteplase, or mechanical thrombectomy. If the patient wakes up with stroke symptoms, their last known normal time is typically the time that they went to sleep.

In addition to stabilizing the patient, the initial study of choice for a patient presenting with symptoms concerning for a stroke is a non-contrast computerized tomography (CT) of the head, a very sensitive tool for intracerebral hemorrhage. The CT may be normal early in the course of acute ischemic stroke. In the event of a normal CT, with a high index of suspicion for a large vessel occlusion, immediate imaging of the vessel along with perfusion imaging will help determine whether the patient may be a candidate for mechanical thrombectomy.[9] This can include CT angiogram or magnetic resonance angiography.

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Once stable, echocardiography is a useful tool to identify those with reduced left ventricular (LV) function and intracardiac thrombus, an indication for anticoagulation.

Stroke Types

Stroke is a heterogeneous disorder with various known and unknown etiologies.[3] Approximately 85% of all strokes are ischemic and 15% are hemorrhagic. Hemorrhagic strokes are more likely to be fatal than ischemic strokes.[10] Management of the stroke itself and prevention of future strokes relies on establishing the mechanism of the stroke along with reducing the risk of stroke recurrence. Recurrent stroke rates remain high despite addressing modifiable risk factors, with roughly one out of every four patients suffering a recurrent stroke.[11] Approximately 40% of strokes have unknown etiology (cryptogenic stroke),[12] although these are most likely embolic.[13] Prolonged cardiac monitoring will help identify these patients.[14]

Stroke prevention should target the cause of the stroke. Efforts to understand the underlying cause of stroke have led to multiple classification systems for ischemic stroke. While not interchangeable, these validated classification systems can be helpful in categorizing stroke etiology into large vessel, small vessel, and cardioembolic. The most widely accepted system is the TOAST (The Trial of Org 10172 in Acute Stroke Treatment)[15] (Table 17.1). Other classification systems include the CSS (Causative Classification of Stroke System)[16] and the A-S-C-O (Phenotypic) Classification of Stroke (Atherosclerosis [A], Small Vessel Disease [S], Cardiac Disease [C], and Other [O]).[17] Additional terminology has been proposed to further characterize cryptogenic strokes, including ESUS: Embolic Stroke of Undetermined Source.

Table 17.1 TOAST classification of subtypes of acute ischemic stroke

Large artery atherosclerosis
Cardioembolism
Small vessel occlusion
Stroke of other determined etiology
Stroke of undetermined etiology • 2 or more causes identified • Negative evaluation • Incomplete evaluation

Transient Ischemic Attack (TIA)

A TIA event presents an opportunity for early recognition, assessment, and management of modifiable risk factors, with the goal of preventing further ischemic and potentially more debilitating events. The 90-day risk of recurrent stroke for patients presenting with TIA is approximately 10%.[18]

Risk Stratification for TIA

The ABCD2 score (Table 17.2) is validated to help identify patients more likely to require hospitalization, using clinical data obtained at the initial assessment. Total scores range from 0 to 7 and allow stratification of patients into low-, moderate-, and high-risk categories. A higher score (3 or higher) suggests an elevated risk for stroke in the first 72 hours after the presenting event.[5,19–22]

Clinical trials, such as CHANCE (Clopidogrel With Aspirin In Acute Minor Stroke Or Transient Ischemic Attack), have used the ABCD2 score to identify a subgroup of patients who may benefit from treatment with dual antiplatelet therapy (DAPT) early, further reducing 90-day recurrent stroke risk, as outlined below.[23]

Risk Factors for Stroke

Recurrent strokes account for approximately 25% of all strokes. Risk factors for stroke (outlined in Table 17.3) may either be "nonmodifiable" or "modifiable." Identifying and altering the modifiable risk factors is critical to reducing the risk for stroke recurrence.

Nonmodifiable risk factors include age and genetics. However, there are disparities that may be accounted for

Table 17.2 ABCD2 criteria for transient ischemic attack risk stratification

Criteria	Details	Points
Age	Greater than or equal to 60 years	1
Blood pressure	Initial BP greater than or equal to 140 SBP or 90 DBP	1
Clinical features	Unilateral weakness	2
	Speech disturbance without weakness	1
Duration of episode	10–59 minutes	1
	More than or equal to 60 minutes	2
Diabetes	History of diabetes	1

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Table 17.3 Risk factors for ischemic stroke

Modifiable	Nonmodifiable
Hypertension	Age
Diabetes mellitus	Male sex at birth
Atrial fibrillation	Genetics
Dyslipidemia	
Tobacco use	
Sedentary lifestyle	
Alcohol use	
Sleep apnea	
Diet	
Obesity	

by more than genetics: for example, the higher incidence of stroke in African American men in the USA[24] is also found in ethnic minorities in Europe.[25] There is a higher rate of major cardiovascular events in low-income countries and rural communities, along with higher mortality, despite a lower vascular risk-factor burden.[26] These disparities in stroke outcomes are, in fact, modifiable, and represent future opportunities to improve stroke outcomes.[7]

Risk Factors for Hemorrhagic Stroke

Hemorrhagic strokes represent a heterogeneous group of conditions, accounting for 10–15% of all strokes. This group includes traumatic and nontraumatic hemorrhages, but for the purposes of this chapter, only nontraumatic hemorrhagic stroke is discussed. Hemorrhagic strokes are defined by the location of the blood: intracerebral hemorrhage (ICH), subdural hemorrhage (SDH), epidural hemorrhage, and subarachnoid hemorrhage (SAH). Intraventricular hemorrhage (IVH) is typically rare in adults. These may occur alone or in combination in an individual patient. ICH has an estimated mortality of up to 50% in the first 30 days; of those who survive, only 20% reach functional independence.[27]

Hypertension is the most common etiology for spontaneous ICH, but treatment of hypertension in the elderly patient can present challenges. In particular, hypotension can lead to falls, also a risk for traumatic ICH.[28,29]

During the initial management of acute spontaneous intracerebral hemorrhage, lowering blood pressure (BP) when systolic BP (SBP) is above 220 mmHg may help reduce risk for hematoma size increase.[30,31] However, what the parameters for BP should be and whether lowering BP results in improved outcomes remains unclear, despite several clinical trials to address BP management (INTERACT,[32] INTERACT 2,[33] ATACH,[34] and ATACH 2[35]).

During the initial management of acute ischemic stroke there are at least two considerations. First, if thrombolytic therapy is being considered, BP must be controlled prior to administration of alteplase (SBP <185 and DBP <110 SBP). After alteplase administration, BP must be maintained at SBP <180 and DBP <105 for 24 hours after the infusion, to reduce the risk of hemorrhagic transformation.[36] If thrombolytics are not used, permissive hypertension (SBP >220 or DBP >120) during the first 24–72 hours of acute care may prevent worsening stroke symptoms. Once stable, the patient should resume their prehospitalization antihypertensive regimen or start an antihypertensive regimen if appropriate, since BP is elevated initially after stroke and may return to normal within a few days of the stroke. Guidelines for management of hypertension are beyond the scope of this section.[37]

Medications may contribute to spontaneous ICH, particularly anticoagulants or antiplatelet agents, and may require reversal as outlined below.

Cerebral amyloid angiopathy (CAA) is an established cause for spontaneous hemorrhagic stroke, particularly lobar hemorrhages, in patients older than 55 years. CAA is the result of amyloid deposition in cortical vessels. Advances in biomarkers and MRI have moved this field forward in the last decade.[38–41]

Stroke Prevention

A reasonable strategy for the clinical management of patients presenting with TIA or stroke is to try to identify the stroke type and subtype, followed by a careful analysis of their individual risk factors, targeting modifiable risk factors, as outlined in Table 17.3. In addition to obtaining a detailed clinical history, including drug history (to include prescription and over-the-counter medications, including anticoagulants and antiplatelet drugs), a baseline assessment of fasting lipid profile and glucose will identify potential targets for prevention.

Antiplatelet Therapy for Secondary Stroke Prevention

Aspirin remains the preferred first-line antiplatelet monotherapy for secondary ischemic stroke prevention,

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typically between 75 mg/d and 325 mg/d with no evidence to suggest improved outcomes with a higher dose.[9,42]

Clopidogrel 75 mg daily is also a Food and Drug Administration (FDA)-approved option for ischemic stroke prevention. When initiating clopidogrel monotherapy for stroke prevention, consider genetic testing for variants of CYP gene to identify patients who may not benefit from clopidogrel in the usual dose.[43]

Dual antiplatelet therapy (DAPT) is indicated in patients with minor stroke (NIH Stroke Scale score ≤3) or high-risk TIA ($ABCD^2$ ≥4). This includes aspirin 81 mg for 21 days along with clopidogrel (loading dose of 300 mg on day 1, followed by clopidogrel 75 mg daily for 21–30 days). In patients with intracranial disease, this protocol should continue for 90 days.[23,44,45] Following this time, patients remain on a single antiplatelet medication, selected with a patient-centered evidence-based approach.

Atrial Fibrillation

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia, occurring in 1–2% of the general population. The prevalence of AF in those over age 65 is 5% and doubles (10%) in those over age 80.[46–48] In the setting of cryptogenic stroke or TIA, in patients over age 65, the use of prolonged cardiac monitoring increases the likelihood of identifying AF.[36,49]

Nonvalvular AF confers an increased stroke risk of 2–7 times that of patients without AF, while rheumatic AF confers a risk up to 17 times higher and 5 times higher than nonvalvular AF. Risk stratification provides an estimate of stroke risk without anticoagulants. The CHADS2 score is an acronym for five clinical factors (congestive heart failure, hypertension, age 75 or higher, diabetes, and prior stroke or TIA), the presence of which counts for 1 point, except for stroke or TIA, counting for 2 points. Patients with a CHADS2 score of 2 or higher are considered high risk for stroke. The CHA2DS2-VASc score is an enhanced score that adds female gender, additional age strata, and vascular disease.

Anticoagulation

A patient-centered approach is necessary when deciding to initiate anticoagulant or antithrombotic treatment, to reduce stroke risk as well as determining which drug to start.[50] Combining oral anticoagulation with antiplatelet therapy is not recommended for stroke or TIA prevention, although this combination may be appropriate for other cardiovascular indications.[51,52]

Vitamin K Antagonists: Warfarin

Vitamin K antagonists, such as warfarin, have been used for many years and reduce stroke risk by 68% and overall mortality by 33%,[46] with the recommended target international normalized ratio (INR) of 2.0–3.0. However, warfarin carries significant potential for drug–drug interactions and dietary interactions with a significant burden for monitoring and dose adjustment. Despite these issues, warfarin remains the only anticoagulant indicated for stroke prevention in the setting of valvular atrial fibrillation.[36]

Direct Thrombin Inhibitors and Oral Factor Xa Agents

These drugs are indicated for stroke risk reduction in nonvalvular atrial fibrillation.

Dabigatran is the only oral direct thrombin inhibitor available since ximelagatran was withdrawn from the market because of hepatotoxicity, with twice-daily dosing that does not require coagulation monitoring.[46] Issues that remain under review are overall cost and risk for use in those with renal insufficiency (a major factor in the older adult population), who require lower-dose therapy. Direct factor Xa inhibitors include apixaban, rivaroxaban, and edoxaban.

Several major trials (RE-LY,[52,53] ROCKET-AF,[54] ARISTOTLE,[55] ENGAGE AF-TIMI,[56] ELDERCARE-AF[57]) have established the safety and efficacy of these agents, all of which were noninferior to warfarin and had lower rates of ICH.[58] Selecting an anticoagulant for stroke prevention in older adults is uniquely challenging, given the higher risk for altered pharmacokinetics, polypharmacy, and associated comorbid conditions. The HAS-BLED score (Hypertension, Abnormal renal/liver function, Stroke, Bleeding history, Labile INR, Elderly, Drugs/alcohol concomitantly) provides a practical guide to support decision-making for clinicians, allowing an estimate for the individual 1-year risk of major bleeding.[59–62]

Mechanical Closure

The Watchman implantable device occludes the left atrial appendage, although short-term anticoagulation is still necessary post-procedurally.[63,64]

Timing of Anticoagulation

The ideal time to start anticoagulation after a TIA or stroke event is 4–14 days after the event.[36]

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Other Indications for Anticoagulation in Stroke Prevention

LV thrombus is an indication for anticoagulation for secondary stroke prevention, typically for 3 months. Warfarin is the preferred anticoagulant in the presence of a left ventricular assist device (LVAD).

Anticoagulation Reversal in Hemorrhagic Stroke

Consultation with a pharmacist is recommended in the complex care of these patients. For those on vitamin K antagonists (warfarin), vitamin K, fresh frozen plasma (FFP), and prothrombin complex concentration (PCC) are options. For factor Xa inhibitors (apixaban, rivaroxaban), a modified recombinant factor Xa protein, andexanet alfa, is available. For direct thrombin inhibitors (dabigatran), a monoclonal antibody fragment, idarucizumab, is available.

Lipid-Lowering Therapy

Current guidelines recommend a high-intensity statin for stroke prevention. This may include any of the following: atorvastatin 40 mg, atorvastatin 80 mg, rosuvastatin 20 mg, or rosuvastatin 40 mg. If the LDL-C remains ≥70 mg/dl with good adherence on a maximally tolerated statin, treatment should be escalated to include the addition of ezetimibe followed by the addition of PCSK9 inhibitors, if needed. However, in older adults, moderate- or high-intensity therapy needs to be considered in the context of adverse effects, drug interactions, patient frailty, cost, and personal preference.[65] At this time there is no evidence to suggest discontinuing statins in the setting of ICH.[66]

Glycemic Control

All patients presenting with acute stroke or TIA should be screened for diabetes. In the acute setting, persistent hyperglycemia in the first 24 hours is associated with worse long-term outcomes.[9] Additionally, hypoglycemia should be avoided, as this can worsen stroke symptoms or cause focal neurologic deficits that mimic stroke.

Carotid Artery Disease

Multiple clinical trials have confirmed that carotid revascularization is recommended for patients who present with a vascular event and symptomatic carotid stenosis. The presence of large vessel disease must be confirmed by imaging, which may include (from least to most invasive): ultrasound, CT angiography, MR angiography, and digital subtraction angiography. Available resources and need for clarity will dictate whether a patient may require more invasive testing. Discussion with neurology, vascular neurology, and neuroradiology colleagues can provide important guidance in decision-making. Carotid disease is defined as moderate (50–69%) or severe (70–99%) stenosis of the ipsilateral carotid artery (to the stroke event). The timing of the revascularization procedure is ideally within 2 weeks of the index event, provided there are no contraindications.[36]

The NASCET (North American Symptomatic Carotid Endarterectomy)[67] and the CREST (Carotid Revascularization Stenting versus Endarterectomy for Treatment of Carotid-Artery Stenosis)[68] trials concluded that carotid endarterectomy (CEA) and carotid artery stenting (CAS) were equivalent regarding the primary outcomes of death, stroke, or myocardial infarction (MI). However, the periprocedural stroke rate is higher in CAS, whilst the periprocedural rate of MI is higher in CEA. Thus, it is reasonable to consider patient age when choosing between procedures; specifically, CEA may be associated with improved outcomes compared with CAS for patients older than 70 years.[69]

Intracranial Stenosis

Medical management remains the mainstay of treatment for symptomatic intracranial atherosclerosis. Multiple large, multicenter trials comparing surgical with medical management have failed to demonstrate an advantage for surgical revascularization.[70] The WASID (Warfarin Aspirin Symptomatic Intracranial Disease) trial[71] showed that warfarin offered no benefit over antiplatelet therapy and resulted in an increased risk of adverse events in patients with symptomatic intracranial stenosis.

Other Modifiable Risk Factors

Physical inactivity and poor nutritional status are of concern, particularly in the elderly. All patients should also be screened for tobacco, alcohol, and illicit drug use, regardless of age, and counseled on cessation, as appropriate.

Thrombolytic Therapy in Older Adults with Acute Ischemic Stroke

The NINDS trial that led to the approval of intravenous thrombolytic therapy for the treatment of acute ischemic

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stroke (AIS) in the USA only included 0.5% patients who were age 80 or older, leading to a limitation on use of this medication in older adults.[72] However, there is a growing body of data demonstrating that the use of intravenous thrombolytic therapy is safe and efficacious in those older than 80.

Another limitation to thrombolytic therapy has been the time window. This has been extended from 3 hours to 4.5 hours from onset of symptoms,[9] and in patients who wake up with symptoms, the WAKE-UP trial[73] demonstrated that MRI can help identify candidates for safe use of thrombolytic therapy, even when the onset time is either unclear or beyond 4.5 hours.

Mechanical Thrombectomy

Mechanical thrombectomy can be used within 6 hours of symptom onset, or longer, using advanced imaging modalities to guide the treatment. As with thrombolytic treatment, patients with advanced age were not represented equally in most thrombectomy trials. Despite this lack of evidence, decisions about pursuing mechanical thrombectomy in older adults should be considered, using a patient-centered holistic view of the patient's life goals.

Poststroke Complications

One of the most serious complications after stroke is venous thromboembolism (VTE) due to immobility. Current guidelines recommend intermittent pneumatic compression devices to reduce the risk of VTE, until the patient is fully ambulatory.[74,75] A comprehensive meta-analysis demonstrates that low-molecular-weight heparin or unfractionated heparin are more effective than aspirin or intermittent pneumatic compression in preventing VTE,[76] but may result in increased bleeding complications.[9]

Dysphagia screening is critical for all stroke patients, since dysphagia increases the risk for aspiration pneumonia, which is associated with higher mortality and worse outcomes.[9]

Depression is common after stroke. All patients should be routinely screened for poststroke depression and treated with antidepressants as appropriate.[9]

Stroke Rehabilitation

Early rehabilitation is recommended for stroke patients, and, like all stroke care, it is best undertaken with a team approach.

Caregiving Burden After Stroke

While caregiving of the elderly is a common element in society, the unique nature of stroke is such that the caregiving role is thrust very suddenly upon family members and others. Caregivers of older adults with stroke carry a greater burden, regardless of socioeconomic status, because of factors that may include differences in acute medical care and rehabilitation care, as well as preferences for care on the part of patients and their families.[77]

End-of-Life Decision-Making

Health-care team members, particularly physicians, should serve as an open and evidence-based source of information, providing support to patients and their families and/or caregivers to ensure that active decision-making, rather than passive expectation of death, guides decisions about quality of life and end-of-life care.

Taking care of patients with stroke presents a position of great privilege: we have an opportunity to make a huge difference in their future lives, whether treating the current stroke aggressively to reduce infarct or hematoma size or preventing the next event by using prolonged cardiac monitoring to detect previously unknown AF. This can only be done with a team-based approach, where health-care team members, including neurologists, nurses, physical therapists, occupational therapists, speech and language pathologists, social workers, pharmacists, and geriatricians, maximize communication skills to ensure outcomes are focused on patient-centered care, enveloped in a good dose of empathy and compassion.

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Chapter 18

Movement Disorders

Tsao-Wei Liang

Movement disorders are a heterogeneous group of neurological disorders that lead to significant loss of quality of life and function.[1] Although certain movement disorders require specialized neurological care, the geriatrician has an important frontline role in their recognition, diagnosis, and management. This chapter will highlight the three most common movement disorder subtypes that a geriatrician may encounter in practice. Considering overall community prevalence estimates, a geriatrician could expect to see as many as one in three patients over the age of 65 affected by tremor, Parkinson's disease (PD)/parkinsonism, or gait disorders.[2–4] Each section will include "clinical pearls" to aid in diagnosis and an overview of the approach to treatment.

Tremor

Tremors, defined as involuntary, rhythmic, oscillatory movements that can affect any part of the body, are the most common movement disorder and occur as a result of dysfunction of the basal ganglia, cerebellum, or the brainstem connections between these systems. They are categorized based upon the type of activity that maximizes the tremor. *Action tremors* appear when holding a certain posture (e.g., arms outstretched) or during deliberate targeted movement (e.g., writing, holding a fork) and disappear when the affected body part is relaxed or at rest. A *rest tremor* occurs when the affected body part is supported and completely at rest; for example, when the hands are supported in one's lap or hanging at the side. A rest tremor is temporarily suppressed with action, but it may reemerge with sustained posture-holding. The primary question facing clinicians is to differentiate between PD and essential tremor (ET). Although a rest tremor is nearly pathognomonic for PD or parkinsonism, rest tremor may coexist with ET (Table 18.1).[5]

ET is the most common tremor and movement disorder, with a prevalence of 0.9% in persons over the age of 65.[6] ET is often stable to minimally progressive and for that reason patients often do not seek medical attention for years. However, the condition tends to progress slowly with age and can lead to significant disability. There may be a family history of similar tremor among first-degree relatives since ET has a familial or hereditary component.[7,8] ET most commonly affects the arms and hands, but may also involve the head or voice, either in combination or in isolation. Difficult tasks for a person with ET include writing and holding utensils or drinks. Tremulous handwriting can be demonstrated in the office and used to document responses to therapy. When the head and neck are involved, the tremor can result in horizontal ("No-No") or vertical ("Yes-Yes") bobbing.

The main indications for treatment are social, occupational, or functional disability. Beta-blockers (e.g., propranolol and atenolol) and primidone are considered first-line agents for the treatment of ET.[9] Both decrease tremor amplitude but generally do not abolish the tremor. Second-line agents such as topiramate, gabapentin, benzodiazepines, and phenobarbital should be tried in individuals who are intolerant of or refractory to

Table 18.1 Comparison of essential tremor and Parkinsonian tremor

	Essential tremor	Parkinsonian tremor
Body regions affected	Head, vocal tremor, hands	Jaw/chin, hands, legs
	Often bilateral, symmetric	Often unilateral onset
Associated neurological symptoms	None	Cardinal symptoms of PD
Typical treatment	Primidone or beta-blockers	Dopaminergic and anticholinergic agents
Typical deep brain stimulation target	Thalamus	Subthalamic nucleus or globus pallidus interna

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Table 18.2 First-line agents for treatment of essential tremor

	Propranolol (Inderal, Inderal LA)	Primidone (Mysoline)
Mechanism of action	Nonspecific adrenergic blockade	Metabolized to phenobarbital and phenylethylmalonamide; mechanism of action unknown
Daily dose range	60–320 mg	50–1000 mg
Side effects	Fatigue, impotence, bradycardia, dizziness, depression	Sedation, confusion, dizziness, ataxia, vertigo
Relative contraindications	Atrioventricular block, diabetes, heart failure, asthma/emphysema	Hepatic or renal failure, benzodiazepine/barbiturate use

first-line agents. Occasionally, medications may be more effective in combination (Table 18.2).

Both stereotactic thalamotomy and high-frequency thalamic deep brain stimulation (DBS) can be dramatically effective for medically refractory ET.[10] These procedures require a craniotomy, but fortunately have a very low rate of serious complications (<1–5%). The primary advantage of stimulation compared to thalamotomy is the ability to reverse and optimize response by adjusting electrical stimulation.[11] Focused ultrasound therapy was approved for the treatment of medically refractory tremor in the United States in 2016. The therapy is performed with magnetic resonance imaging (MRI) guidance. A thermal lesion is created by focusing over 1,000 ultrasound waves at a target in the thalamus, heating the target, and lesioning without the need for an incision or craniotomy. Currently, this therapy is approved for unilateral thalamotomy; ongoing study is needed to establish its safety for bilateral treatment.[12]

An *intention tremor* is a specific type of action tremor in which the amplitude increases as the affected limb approaches a target. A true intention tremor results from dysfunction of the tracts exiting the cerebellum, leading to the term *cerebellar outflow tremor*. The term *rubral tremor* may also be used to describe an intention tremor and implies dysfunction of the midbrain red (rubral) nucleus, although intention tremors may arise from lesions outside the midbrain. Multiple sclerosis, head trauma, stroke, and degenerative diseases of the brainstem and cerebellum may produce a rubral tremor.[13] The full spectrum of a rubral tremor may include a rest component, titubation (head and trunk oscillation when the trunk is unsupported), gait ataxia, and a coarse "flapping" quality due to involvement of proximal muscle groups. Rubral tremors are very disabling as they can severely impair basic activities such as feeding, dressing, and hygiene. They are notoriously difficult to treat, and surgical therapies have been attempted with limited success.[14]

Reviewing and reconciling a patient's medication history can help to minimize polypharmacy and determine if a tremor may be related to a medication. The diagnosis of drug-induced tremor is made by exclusion of other causes of tremor, establishing an exposure to a tremor-inducing drug, and identifying a temporal and dose–response relationship. Table 18.3 provides a list of medications commonly associated with tremor. Antipsychotics and other psychotropic medications are often culprits, but other drugs associated with tremor include *albuterol*, *amiodarone*, *prednisone*, and *theophylline*. Chronic treatment with *lithium carbonate* causes a fine postural or kinetic tremor. In overdose or toxicity, lithium can also be associated with a cerebellar or parkinsonian syndrome.[15,16] Withdrawal from benzodiazepines, alcohol, or other central nervous system depressants often results in tremor. If the causative medication cannot be discontinued, treatment with propranolol or primidone may be considered.

Parkinsonism and Parkinson's Disease

General Concepts

Parkinsonism refers to a diverse group of conditions characterized by rest tremor, bradykinesia, rigidity, and postural instability. Although most cases of parkinsonism are sporadic, idiopathic, and neurodegenerative, parkinsonism may also occur as a result of hereditary conditions, structural lesions, drugs, or toxins affecting basal ganglia function. Common secondary causes include vascular parkinsonism, drug-induced parkinsonism, and normal pressure hydrocephalus (NPH).

PD is the second most common neurodegenerative disorder after Alzheimer's disease in the USA. The prevalence of PD is estimated between 100 and 300 per 100,000, and over a million persons are affected in the USA.[17] Although PD can occur on a familial basis, the vast majority of cases are sporadic and occur in those above the age of 65,

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Table 18.3 Medications and toxins that induce or exacerbate tremor

Beta adrenergic agonists
Epinephrine
Isoproterenol
Albuterol
Theophylline
Amphetamines
Lithium
Phenothiazines, butyrophenones
Tricyclic antidepressants
Anti-arrhythmics
Amiodarone
Procainamide
Mexiletine
Antimicrobials
Trimethoprim-sulfamethoxazole
Acyclovir
Antiepileptics
Valproic acid
Carbamazepine
Lamotrigine
Immunosuppressants
Prednisone
Cyclosporine
Tacrolimus
Xanthines (in coffee and tea)
Heavy metals
Mercury
Lead
Arsenic
Bismuth

suggesting a combination of age, environmental factors, and possible genetic susceptibility. The hallmark of the condition is the selective degeneration of midbrain dopaminergic neurons, which leads to the cardinal features of the disorder and the characteristic response to levodopa. Several related degenerative disorders known as Parkinson's plus or atypical parkinsonism are part of the larger complex of parkinsonism, not to be mistaken for PD. Features common to this group of disorders include poor response to levodopa, relatively rapid progression to severe disability compared to idiopathic PD, and additional neurological features that make them atypical for PD.

Clinical Features of Parkinsonism

Movements reduced in speed (bradykinesia), amplitude (hypokinesia), or poverty of movements (akinesia) are the hallmarks of parkinsonism. Passively manipulating joints such as the wrist, elbow, or knee will reveal either a constant increase in tone (leadpipe rigidity) or a ratchety increase in tone (cogwheel rigidity).

Rest tremor is the most recognizable aspect of parkinsonism but may be absent in up to 40% of patients with parkinsonism.[17] The typical tremor of PD tends to present unilaterally at rest and tends to remains asymmetric throughout the course of the disease. Although the hand is most commonly involved, a parkinsonian tremor is unique in that it may affect the legs and lower facial muscles, including the jaw and tongue. Mental concentration (e.g., counting backwards) and walking enhance a parkinsonian tremor. Furthermore, lifting the arms to sustain a posture will dampen the tremor temporarily, but the original amplitude and frequency will return after a delay of 2–3 seconds. This so-called reemergent tremor is an important diagnostic clue to a parkinsonian tremor.

In early disease, the examination may reveal a subtle unilateral tremor, decreased arm swing, or a stiff-appearing leg when walking. Impaired finger dexterity, loss of facial expression (hypomimia), and small handwriting (micrographia) are often the earliest signs of parkinsonism. The voice can be soft and monotone with a loss of diction (hypophonic). The combination of rigidity and bradykinesia leads to difficulty with ordinary tasks such as turning in bed, rising from a chair, or getting in and out of a car. Because of the loss of dexterity and range of motion, dressing and routine hygiene can become laborious.

A characteristic stooped posture develops with flexion of the trunk, neck, elbows, shoulders, and knees. Step height and stride length are reduced, leading to a shuffling gait. The stooped posture and shift in the center of gravity contribute to a tendency to fall forward (propulsion) and an inability to stop (festination). Turning occurs "en bloc," as the head, neck, torso, and extremities no longer rotate independently because of rigidity and bradykinesia. Start hesitation leads to difficulty initiating walking. Patients may describe freezing, as though the feet are transiently stuck or "glued" to the ground. The phenomenon typically occurs in doorways, narrow hallways, or near obstacles. Patients often find that visual cues such as floor or sidewalk markings tend to alleviate freezing. Freezing of gait (FOG) is one of the more debilitating symptoms encountered with parkinsonism, often contributes to falls, and tends to be refractory to dopaminergic therapy.

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Postural stability is tested using the pull test in which the examiner firmly tugs the patient from behind and assesses the ability to maintain an upright stance. Retropulsion or toppling backwards occurs when the ability to adjust or maintain balance is lost.

There are numerous nonmotor features of parkinsonism that lead to further disability, including dysphagia, excess saliva or drooling (sialorrhea), constipation, overactive bladder, erectile dysfunction, seborrheic dermatitis, orthostatic hypotension, pain, paresthesias, and sleep disturbances, including REM sleep behavior disorder (RBD), periodic limb movements of sleep (PLMS), restless legs syndrome (RLS), insomnia, and obstructive sleep apnea. Numbness, tingling, aching pains, fatigue, and weakness are common complaints of PD that may be dismissed early in the diagnosis or mistaken for other neurological or medical conditions. Neuropsychiatric symptoms commonly associated with PD or parkinsonism include cognitive impairment, subcortical dementia, depression, apathy, anxiety, hallucinations, and delusions (Table 18.4).[18]

Differential Diagnosis

The diagnosis of PD or an atypical parkinsonism is made by a careful history and examination. Not all cardinal features may be obvious or present in early stages of the disease. Even in the hands of neurologists, up to 20% of patients have an alternate diagnosis at autopsy, while cases with atypical features may prove to have PD.[19] Since 2011, the dopamine transporter SPECT scan (DaT) has been available in the United States and can aid in the early diagnosis of parkinsonism. The radioisotope ioflupane labels the dopamine transporter found in nigrostriatal neurons. Sensitivity and specificity are high when DaT is used to differentiate degenerative parkinsonism from conditions where the nigrostriatal system is unaffected (i.e., ET, drug-induced tremor, dystonia). However, because nigrostriatal dysfunction is a common feature of PD and atypical parkinsonism, DaT differentiation between these conditions is not often possible.[20]

Rather, the single most important clinical feature differentiating idiopathic PD from secondary and atypical forms of parkinsonism is the response to levodopa. Patients with idiopathic PD display a robust, long-lasting response to levodopa, since the primary issue is a presynaptic loss of dopamine with intact receptors. The lack of response characteristic of other forms of parkinsonism is due to degeneration of postsynaptic receptors and nondopaminergic systems.

Table 18.4 Neuropsychiatric manifestations of parkinsonism

Cognitive impairment and dementia
Visual hallucinations
Delusions
Depression, anhedonia, dysphoria
Anxiety
Akathisia
Apathy or amotivation
Sleep disorders such as insomnia, sleep fragmentation, excessive daytime somnolence, sleep attacks, REM behavior disorder, periodic leg movements of sleep
Impulse control behaviors

Table 18.5 Parkinson's plus syndromes

	First description	MRI findings	primary pathology
Parkinson's disease	1817 James Parkinson	Normal	Nigrostriatal degeneration, Lewy bodies
Progressive supranuclear palsy	1964 Steele et al.	Midbrain atrophy, "hummingbird sign"	Midbrain degeneration, frontal lobe degeneration, Tau-positive neurofibrillary tangles
Multiple system atrophy	1900 Dejerine and Thomas 1960 Shy and Drager 1960 Adams et al. 1969 Graham and Oppenheimer	Lower brainstem and cerebellar atrophy, "hot-cross bun sign," linear putaminal hyperintensity	Striatonigral, olivopontocerebellar, intermediolateral cell column degeneration, glial cytoplasmic inclusions (GCI)
Dementia with Lewy bodies	1996 McKeith et al.	Global atrophy	Diffuse cortical and subcortical Lewy bodies and dystrophic neuritis
Corticobasal ganglionic degeneration	1968 Rebeiz et al.	Asymmetric frontoparietal atrophy	Ballooned neurons

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Red flags suggesting an atypical or secondary form of parkinsonism include: ocular motor palsy, cerebellar signs, early postural instability and falls, prominent cognitive or cranial nerve dysfunction, dysautonomia, or pyramidal signs. These conditions tend to be more rapidly progressive than PD and lack disease-specific treatments.

Progressive supranuclear palsy (PSP) is characterized by an akinetic-rigid parkinsonism with early imbalance and falls. The characteristic vertical gaze palsy may be absent initially but invariably develops over time. Other features include facial expression changes, marked by a prominent stare, with decreased blinking, eyelid retraction and an astonished facial expression, dysarthria, and neuropsychiatric symptoms. Frontal lobe involvement contributes to personality changes and cognitive impairment. The combination of cognitive impairment and lack of insight often leads to issues with medication compliance, social withdrawal, and falls. Microscopically, microtubule-associated protein called tau accumulates, forming neurofibrillary tangles, resembling the pathology of Alzheimer's disease.[21]

Dementia with Lewy bodies (DLB) is a relatively new diagnostic entity that is now considered the second most common cause of dementia after Alzheimer's disease in the USA.[22] DLB presents with prominent mental symptoms that often overshadow the movement disorder. In other words, dementia or progressive disabling cognitive decline is an essential and primary feature of this condition.

Visual hallucinations (VH) are often formed and may be complex and linked to a theme or delusional thought. VH in DLB are nearly identical to those that occur in later-stage PD. Compared to PD, in DLB they tend to occur spontaneously, early (within 2 years of diagnosis), and with little no dopaminergic medication exposure. Delusions revolving around spousal infidelity or neighbors, friends, or relatives stealing from them are also common. Pronounced cognitive fluctuations differentiate DLB from other causes of dementia. The fluctuations may be unprovoked states of altered sensorium or delirium. The most current consensus criteria REM behavior disorder is considered a core clinical feature.[22]

Notable supportive clinical features include (1) repeated falls due to postural instability; (2) syncope and transient loss of consciousness mimicking seizures, stroke, transient ischemic attack, or a cardiac event; (3) sensitivity to neuroleptics – a characteristic feature of DLB – and abrupt worsening of the physical or mental state resulting from use of antipsychotics; and (4) severe autonomic dysfunction, including orthostatic hypotension and incontinence.

Both PD and DLB have the characteristic pathological inclusion called the Lewy body (LB), cytoplasmic inclusions containing the synaptic protein α-synuclein. In DLB, LB are present throughout the brainstem, limbic regions, and cortex, leading to the pathological term diffuse Lewy body disease.

Multiple System Atrophy (MSA) is a clinical term that refers to three formerly distinct conditions: Shy-Drager syndrome (SDS), olivopontocerebellar atrophy (OPCA), and striatonigral denegeration (SND). MSA-A (autonomic form) has replaced SDS; MSA-C (cerebellar form) refers to OPCA, and MSA-P (parkinsonian form) now refers to SND. Besides the hallmark features of parkinsonism, patients may develop either prominent autonomic features (orthostatic hypotension, bowel or bladder incontinence, and sexual dysfunction) or cerebellar dysfunction (dysarthria, ataxia, nystagmus, and limb incoordination). In practice, there is often overlap between the syndromes.[23]

Other suggestive features include REM behavior disorder, which may precede the diagnosis of MSA by many years, stridor, and obstructive sleep apnea due to brainstem respiratory and sleep center dysfunction. Although the response to levodopa is generally not satisfactory, some persons with MSA experience partial benefit and should be tried on levodopa at least once. Another telltale feature of MSA is the development of oromandibular dystonia-dyskinesia as a response to levodopa.

All forms of MSA are linked by a common pathological finding of α-synuclein-laden cytoplasmic inclusions in glial cells.[24] The clinical phenotype is largely determined by the location of pathology. Autonomic centers in the brainstem and cervical and sacral spinal cords are affected primarily in MSA-A. In MSA-C, the medulla, pons, and cerebellum are primarily affected, and in MSA-P, the substantia nigra and the striatum.

Corticobasal ganglionic degeneration (CBD) is a condition that may present with either parkinsonism or dementia. Pathologically, it resembles Alzheimer's disease and frontotemporal dementia rather than Parkinson's disease. Classic corticobasal syndrome consists of asymmetric parkinsonism with prominent limb rigidity, dystonic posturing, and a gait disorder. There may be myoclonic jerks, and cognitive impairment involving frontal lobe function, memory systems, or visuospatial systems. One of the most distinctive features of the syndrome is limb apraxia, or the inability to perform a voluntary movement that is not due to muscle weakness, sensory loss, or ataxia. With time, the so-called alien limb phenomenon may develop in which the limb may assume unusual postures, wander in space, or seem out of the control of the person. Over time, rigidity and dementia predominate.[25]

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In clinical practice, the most common secondary forms of parkinsonism to consider are vascular parkinsonism, drug-induced parkinsonism, and NPH. Lacunar infarctions in the basal ganglia or diffuse white-matter disease may lead to motor symptoms that mimic parkinsonism. The most common pattern of a gait-predominant lower-body parkinsonism may occur as a result of disconnection of frontal gait initiation centers and the basal ganglia. A stepwise pattern of progression is often a clue to recurrent small vessel ischemic events. Levodopa is not typically effective, but should be attempted for both diagnostic and therapeutic purposes. Cerebrovascular risk factors, a history of stroke or transient neurological events, cognitive disturbance, or gait apraxia are often present.[26]

Parkinsonism due to dopamine receptor antagonists is a potentially reversible syndrome that mimics idiopathic PD. The risk of parkinsonism due to antipsychotic medications is dose-dependent and increases in the elderly.[27] With the exception of perhaps clozapine, nearly all antipsychotic medications antagonize dopamine receptors. Other dopamine antagonists include antiemetics such as metoclopramide or prochlorperazine, dopamine-depleting agents such as reserpine or tetrabenazine, and lithium. 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine or MPTP, a compound that causes selective nigrostriatal degeneration, was discovered after it led to acute parkinsonism in a group of college students experimenting with heroin.[28] Other toxins associated with parkinsonism include manganese[29] and carbon monoxide.[30]

Treatment of PD

Treatment Overview

Symptomatic treatment of PD is typically initiated when patients begin to experience disability. Factors to consider when initiating treatment include primary symptom, age, functional status, work status, and comorbid medical conditions. A wide range of medications with proven symptomatic effects exist for PD, including monoamine oxidase inhibitors (MAOIs), anticholinergic agents, amantadine, catechol-O-methyltransferase (COMT) inhibitors, dopamine agonists, and levodopa (Table 18.6).

Both selegiline and rasagiline irreversibly inhibit MAO type B, resulting in modest enhancement of striatal dopamine levels. Side effects may include insomnia and orthostatic hypotension. In the DATATOP (Deprenyl and Tocopherol Antioxidative Therapy of Parkinsonism) study, selegiline treatment was found to delay disability and the need for levodopa therapy.[31] However, the effect was deemed to be at least partly symptomatic, and therefore a clear neuroprotective benefit was never proven.

Table 18.6 Medications for the symptomatic treatment of PD

Medication	Mechanism of action	Typical therapeutic dosage
Selegiline	MAO-B inhibitor	5–10 mg a day
Rasagiline	MAO-B inhibitor	1 mg a day
Benztropine	Anticholinergic	0.5–4 mg a day
Trihexyphenidyl	Anticholinergic	2–8 mg a day
Amantadine	NMDA antagonist	200–400 mg a day
Pramipexole	Dopamine agonist	1.5–4.5 mg a day
Ropinirole	Dopamine agonist	7.5–24 mg a day
Rotigotine	Dopamine agonist (transdermal patch)	1–8 mg daily
Apomorphine	Dopamine agonist (subcutaneous injection)	
Levodopa	Repletes endogenous dopamine deficiency	150–1000 mg a day
Entacapone	COMT inhibitor	200 mg a day

Anticholinergic agents such as benztropine and trihexyphenidyl were among the first medications used for PD. They have mild to moderate effects on tremor, rigidity, and dystonia; however, side effects such as urinary retention, cognitive impairment, delirium, dry mouth, and blurred vision limit their use in the elderly.[32]

Amantadine, originally developed for use against influenza, is a tricyclic amine with multiple putative mechanisms, including enhancing dopamine release, inhibition of dopamine reuptake, antimuscarinic effects, and N-methyl D-aspartate (NMDA) receptor antagonism. In early, mild PD, amantadine has modest effects on tremor and other cardinal motor symptoms. Its anti-dyskinetic properties make it even more useful in advanced patients with motor fluctuations.[33] Amantadine is generally well tolerated, but livedo reticularis, dry mouth, and hallucinations may complicate treatment. Because amantadine is primarily renally excreted, dose adjustments need to be made based on creatinine clearance.

Dopamine agonists (DA) directly stimulate striatal dopamine receptors and are used in early PD as a levodopa-sparing agent and as an adjunct in advanced

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patients with motor fluctuations. DA can be classified into ergot-derived and non-ergot-derived agonists. The ergot-derived agonists, including bromocriptine and pergolide, carry the additional risk of cardiopulmonary and retroperitoneal fibrosis. Ultimately, cardiac valvular abnormalities attributed to pergolide led to its withdrawal from the US market.[34] Ropinirole and pramipexole are oral D2-like receptor agonists that have both shown efficacy in delaying levodopa-induced dyskinesia and motor complications. Rotigotine is delivered through a transdermal patch that provides near-continuous levels of medication over 24 hours. Subcutaneous apomorphine injections have been available in the USA since 2004 for the treatment of refractory off-periods. DA side effects include nausea, lightheadedness, pedal edema, hallucinations, and sedation. Most adverse effects are dose-related and can be avoided by slow titration of the drug. However, shortly after the approval of pramipexole and ropinirole, the phenomenon of impulse control disorders was noted in patients treated with dopamine agonists. These behaviors occurred de novo in some patients or represented an exacerbation of a preexisting tendency for gambling, binge eating, compulsive spending, and hypersexuality.[35,36]

The initial studies of ropinirole and pramipexole monotherapy compared to levodopa have both demonstrated reduced motor fluctuations and dyskinesias compared to levodopa therapy.[37–39] However, a recent longitudinal study suggested that the development of motor fluctuations and dyskinesias is not associated with the duration of exposure to levodopa, but with the duration of disease and dose of levodopa.[40] Longitudinal studies of dopamine agonists reveal that although the rates of motor complications and dyskinesia are reduced compared to levodopa, symptom control and side-effect profile tend to be worse.[41,42] In fact, a recent longitudinal study comparing levodopa to MAO inhibitors to dopamine agonists as initial therapy suggests that when considering a levodopa-sparing strategy, MAO inhibitors may be just as effective and better tolerated than dopamine agonists.[43]

Despite the recent advances in pharmacotherapy, levodopa remains the gold standard and most effective treatment for symptoms of PD. The majority of PD patients will attain significant, long-lasting benefit from levodopa superior to any of the previously mentioned agents. It is well tolerated and effective at a large dose range as long as it is administered with a peripheral decarboxylase inhibitor (e.g., carbidopa). Without such an inhibitor, large doses of levodopa would be required to provide a benefit that in turn would lead to intolerable peripheral side effects of dopamine such as orthostatic hypotension, nausea, and emesis. Carbidopa combined with levodopa in various formulations (10/100, 25/100, 25/250) is marketed as Sinemet®, and allows for flexible dosing. The half-life of Sinemet® is approximately 90 minutes, thus multiple daily doses are necessary. We typically start with half of a 25/100 tablet three times daily, increasing over a few weeks to one to two tablets three times daily. Central nervous system side effects such as sedation, insomnia, visual hallucinations, confusion, or psychosis are more likely to occur in the elderly (>80 years of age) or in later stages of PD (Table 18.7).

Table 18.7 Management of psychosis and hallucinations

- Exclude infection or other intercurrent illness that may cause a delirium
- Obtain detailed medication history with emphasis on recent additions, dosage changes, withdrawals, or possible ingestions
- Withdraw medications in this order:
  - Anticholinergics
  - Amantadine
  - Selegiline/rasagiline
  - Dopamine agonists
  - COMT inhibitors
- If troubling hallucinations persist, then consider decreasing levodopa dosage
- Trial of quetiapine
- Clozapine for refractory psychosis

Medical Treatment of Advanced Disease

Initially, the dose range and therapeutic window of levodopa is wide, and low doses of levodopa have sustained benefits for several years. However, the dosage often needs to be increased (as high as 1,000 to 1,500 mg) to maintain a stable level of function. With chronic treatment, at least half of patients on dopaminergic therapy develop motor complications or response fluctuation.[44] As the disease progresses, levodopa's therapeutic window becomes narrowed, leading to clinically apparent fluctuations from "on" (levodopa is working) to "off" (levodopa is not working). In other words, the dose–response curve in advanced disease mirrors the short half-life of levodopa.[45]

Several types of response fluctuations exist. The duration of the response may progressively shorten (wearing off), responses may become unpredictable (on-off phenomenon), painful limb dystonia may occur when wearing off, and dyskinesias, an overexpression of movement, may develop. In addition, visceral (abdominal and chest pains, shortness of breath),

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autonomic, and neuropsychiatric symptoms (euphoria, dysphoria, panic/anxiety) may occur with response fluctuations. Dosing intervals may need to be shortened to 1–2 hours to maintain symptom control. Strategies at this point focus on maximizing "on" time and minimizing "off" time, dyskinesias, and side effects. The development of fluctuations may be related to a combination of progressive loss of striatal dopamine storage capacity, pulsatile stimulation of striatal receptors, and receptor hypersensitivity.[44] Therefore, it follows that strategies minimizing pulsatile stimulation may prevent the development of motor complications.

Increasing the dosing frequency is the first step in controlling wearing off but may be limited by the patient's ability to handle more frequent and complex dosing. Controlled or Extended release Sinemet (CR/ER)® has a half-life of approximately 3 hours, but with a slower onset of action and decreased bioavailability. Sinemet CR/ER® comes in 25/100 and 50/200 formulations. In early disease, CR/ER® can be dosed twice a day and is often used at bedtime to prevent wearing off upon awakening. Sinemet CR/ER® translates to less regular Sinemet® during conversion from one to the other (100 mg regular Sinemet® is equivalent to 133 mg of CR Sinemet®). Use of Sinemet CR/ER® may prolong "on" time, but unpredictable responses may occur in advanced patients. Direct comparison of Sinemet CR/ER® to regular Sinemet® has shown no major difference in the incidence of dyskinesia and motor complications.[46] Because levodopa absorption occurs in the duodenum through a saturable amino acid transporter, bioavailability may be limited by meals containing a large protein content. To maximize bioavailability, fluctuating patients should be instructed to take levodopa on an empty stomach, i.e., at least 45 minutes before a meal or 1–2 hours after a meal.

In 2015, a capsule form of levodopa (Rytary®) was approved for use in early and advanced PD. This formulation includes immediate-release (IR) and extended-release (ER) beads of carbidopa-levodopa that are absorbed in the gastrointestinal tract at different rates. Pharmacokinetic studies indicate that ER capsules reach an initial peak at 1 hour, very similar to IR tablets, and that the peak is sustained for 4–5 hours, followed by a gradual wearing off over 6+ hours. Clinical trials to date have shown a favorable pharmacologic profile that tends to reduce "off" time and increase "on" time without troublesome dyskinesia because of its improved half-life.[47] In addition, the Cmax of ER capsules is notably lower than an equivalent dose of IR tablets, making it necessary to increase the dose of ER capsules by at least twofold to achieve an "on" response.[48] Levodopa intestinal gel has been available in the European Union for nearly a decade and was recently introduced in the United States as Duopa®. The intestinal gel can be infused continuously up to 16 hours a day through a gastrostomy with a jejunal extension. Studies have shown less intra-subject variability in plasma concentration of levodopa and improved "on" time with similar amounts of dyskinesia.[49,50] The major adverse events surround the placement of the PEG-J tube and PEG site irritation. In addition, there are reports of neuropathy that has been hypothesized to be related to the intestinal infusion and B12 malabsorption.[51]

Dopamine agonists are effective in reducing "off" time, improving "on" function, and reducing levodopa dosages. However, adverse effects such as dyskinesias and hallucinations are more common. Amantadine has been shown to have anti-dyskinetic properties. Catechol-o-methyltransferase (COMT) inhibitors, tolcapone and entacapone, inhibit the breakdown of levodopa in the periphery and central nervous systems and enhance central nervous system availability of levodopa. Since the action of COMT inhibitors is dependent on the presence of levodopa, COMT inhibitors have no effect on parkinsonian symptoms if levodopa is not administered. Entacapone significantly increases the area under the curve and the half-life of levodopa without increasing maximal plasma concentrations. Specific side effects of entacapone include severe diarrhea and an orange discoloration of urine. Both entacapone and tolcapone have been shown to improve motor fluctuations by reducing wearing off and "off" time and increasing "on" time.[52,53] Tolcapone is not commonly used because of rare cases of fulminant hepatitis associated with its use.[54]

A Clinical Algorithm

In summary, the medical treatment of PD has advanced dramatically in the last decade, but is limited by the progression of the disease. To date, no definite disease-modifying therapies exist, and the current state of treatment rests primarily on a symptom-based approach. For patients with mild early symptoms below the age of 65, we typically use a "top-up" approach, beginning treatment of mild PD with a dopamine agonist or MAO-inhibitor either alone or in combination. Mild symptoms are often adequately controlled for several months to years on these agents, and delaying levodopa treatment may delay the onset of response fluctuations and dyskinesias. For patients above the age of 65, the incidence of motor

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complications is low, and side effects of agonists are more common and may outweigh the benefits. In this population, we advocate a "top-down approach" starting with carbidopa/levodopa (25/100 mg), using the lowest effective dose possible. The dose may be increased by half- to whole-tablet increments as needed. There is no fixed dosage ceiling, although 1,000 mg of levodopa is generally considered adequate to control symptoms in the majority of mild to moderate patients.

Mild predictable wearing off is easily addressed by increasing the frequency of dosing. However, compliance becomes an issue when levodopa is dosed every 2–3 hours. Entacapone (200 mg) can be added to each dose of levodopa to increase the duration of the response, thus increasing daily "on" time. A concomitant reduction in levodopa dosage by 15–30% is recommended if the patient is at the high end of the therapeutic window. The response to levodopa may become more unpredictable with advancing disease. Response failures may occur with absent or delayed onset of action or sudden or unpredictable "off." The addition of a low-dose dopamine agonist is often useful in patients with complex or unpredictable motor complications as long as dyskinesias, visual hallucinations, and other levodopa-related side effects are manageable. In a subset of patients, the secondary nonmotor symptoms of PD can become quite disabling. Often increasing dopaminergic therapy is not effective. Table 18.8 outlines some of the most common nonmotor symptoms of PD and the accepted therapeutic strategies.

Surgical Therapy

DBS therapy has been the standard surgical therapy for medically advanced Parkinson's disease since it was approved by the Food and Drug Administration (FDA) in 2003. The procedure was a natural extension of the previous lesioning techniques performed between the 1960s and 1980s. Quadripolar (4-contact) electrodes are stereotactically implanted into one of two locations in the basal ganglia, the globus pallidus interna (GPi), or the subthalamic nucleus (STN). The electrodes are then connected to a pulse generator implanted into the chest wall. The current devices manufactured by Medtronic, Inc., Abbott Laboratories, and Boston Scientific allow for a wide variety of physician and patient controls to provide for optimal symptom control.

More than a decade of experience has defined specific selection criteria and outcomes/expectations of the surgery.[55] DBS is highly effective for patients with advanced PD who continue to respond to levodopa or other dopaminergic agents, but who do not have adequate hours of good function ("on" time) and suffer from prolonged periods of immobility or tremor ("off" time), medication-induced dyskinesia, and other medication side effects. The appropriate candidate should have idiopathic PD with disabling symptoms that are responsive to levodopa, be free of significant cognitive or neuropsychological illness, and have the emotional capabilities and social support to cope with a potentially life-altering surgery and the often frequent and lengthy visits required after surgery. Although no firm age cutoff has been determined, most DBS specialists feel that patients older than 75 years of age will have more difficulties with the rigors of surgery and incur greater risk because of medical comorbidities and cognitive status.[55]

Randomized controlled trials reveal that DBS reduces "off" time, increases "on" time, reduces dyskinesia and medication requirements, and improves not only PD motor scores but also quality-of-life scores.[56–58] Complications unique to DBS include electrical malfunction, lead fracture, battery replacement (every 1–5 years), lengthy programming sessions, hardware infection, skin erosion, intracerebral hemorrhage, or infarct. Although the exact mechanism of DBS is unknown, it is believed that high-frequency electrical stimulation blocks or inhibits the stimulation target, which rebalances the basal ganglia circuitry. Ongoing studies continue to investigate the appropriate timing of surgery, the preferred location (STN vs GPi), and optimal stimulation paradigms, device, and surgical techniques.

Natural History. Since the advent of levodopa, mortality in early PD has been dramatically reduced. The mean duration of disease prior to levodopa discovery was 9.4 years and the mean age at death was 67 years. Hoehn and Yahr reported a mortality rate of 2.9 times that expected for an age-matched population. Since the introduction of levodopa, Hoehn reported a mortality ratio of 1.5, and other studies have found similar rates.[43] It is possible that surgical treatments may further improve mortality; however, there is limited data on how DBS affects the natural history of PD.[59] The mean duration of symptoms is reported to be approximately 13 years, and the mean age of death is approximately 73 years. The DATATOP study found that increased symmetry of parkinsonism at presentation, early gait impairment, and rapid clinical decline portended a worse prognosis.[60] The most common cause of death in PD patients is cardiovascular disease, and the second most common cause of death is pneumonia.[46]

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Table 18.8 Nonmotor symptoms of PD, and therapeutic strategies[56]

Symptom	Nonpharmacologic	Pharmacologic
Constipation	Increasing dietary fiber and fluid intake Regular exercise Discontinue anticholinergics	Osmotic macrogel (polyethylene glycol) Stool softeners (docusate) Stimulant laxative (bisacodyl) Enemas
Drooling	Speech evaluation and therapy	Botulinum toxin injections Peripheral anticholinergic agent (e.g., glycopyrrolate)
Dysarthria/hypophonia	Speech therapy (Lee Silverman technique)	If "off" symptom, increase dopaminergic therapy
Dysphagia	Dysphagia evaluation Soft-mechanical diet Schedule meals with "on" time Gastrostomy	If "off" symptom, increase dopaminergic therapy
Freezing	Physical/occupational therapy for gait training Visual cues	If "off" symptom, increase dopaminergic therapy Droxidopa
Postural instability/falls	Physical/occupational therapy for gait training or home safety evaluation Cane, walker, wheelchair, or other form of assistance Evaluation for orthostasis	If "off" symptom, increase dopaminergic therapy
Male impotence	Review medications Evaluate for diabetes or underlying endocrine disorder Urological evaluation	Sildefanil (Level C) Alprostadil (intracavernous injections or intraurethral suppository)
Orthostasis	Elevate head of bed 10–30 degrees Encourage dietary salt intake Compression stockings	Discontinue potential hypotensive drugs Salt-retaining mineralocorticoid (e.g., fludrocortisone) Pressors (e.g., midodrine, ephedrine)
Overactive bladder	Avoid bedtime fluid intake Exclude infection, prostatitis, or other urological problems	Antimuscarinic agents (oxybutynin, tolteridine, imipramine)
Seborrheic dermatitis		Coal tar or selenium-based shampoos Topical steroids
Excessive daytime sleepiness and fatigue	Review medications (limit anticholinergics and dopamine agonists) Recommend good sleep hygiene	Modafinil Methylphenidate
Insomnia	Review medications	Increase evening levodopa/carbidopa Melatonin
Periodic limb movements of sleep		Increase evening levodopa/carbidopa Trial non-ergot dopamine agonists
REM sleep behavioral disorder		Melatonin Clonazepam
Depression	Review medications	SSRI, SNRI, tricyclic antidepressants ECT
Anxiety	Review medications	Modify anti-parkinsonian drug therapy to limit "off" time Antidepressant or benzodiazepine
Dementia	Rule out reversible causes of dementia Rule out pseudodementia Eliminate anticholinergics, dopamine agonists, amantadine, and selegiline Encourage exercise and social interactions	Donepezil Rivastigmine Memantine

Although PD is associated with a shortened lifespan, the diagnosis is not typically considered "terminal." Uncertainty about when to discuss end-of-life planning exists in part because of the lack of guidelines. Discussions of advance directives, alternate means of nutrition (i.e., PEG), alternate living situations, and caregiver options are highly individual and based on the person's support systems in place, and family/patient's level of comfort. A recent survey study showed that 94% of PD patients wanted to discuss prognosis and treatment options early. Most wanted their family members to be present during these discussions. Approximately half wanted to discuss advanced care documents early in the disease, while the remaining individuals preferred to defer these discussions until their condition progressed.[61] Therefore, health-care providers should ask patients about their preferences and offer discussion periodically, preferably before an acute crisis such as a hospitalization occurs.

Gait Disorders

General Concepts

Gait impairment is a significant cause of morbidity and mortality in the elderly. Fifteen to twenty percent of patients over the age of 65 will suffer from gait impairment, often requiring an assistive device. This number increases with age, with an estimated 40–50% of those over 85 years, and up to 70% in those over 90 years.[2] Falls from gait impairment represent the most common cause of injury in the elderly. Thirty percent of all elderly will have at least one fall annually, with the risk as high as 50% of those over 80 years; 50% of long-term care residents will have at least one fall annually.[2]

Normal locomotion depends on intact motor and sensory systems to maintain equilibrium and balance. The sensory input from vestibular, peripheral nervous, and visual systems is integrated by the cortex, cerebellum, and basal ganglia. The body also uses anticipatory and reactive postural reflexes to maintain equilibrium in response to environmental changes. Disruptions or lesions in any of these structures may result in gait impairment. Historically, slowed gait has been considered a natural part of the aging process; however, this is no longer thought to be true. Some of the common changes seen with advancing age include bent posture, slower pace, shortened stride, increased time in double-support phase (when both feet are on the ground), truncal and limb rigidity, widened base, and en bloc turning. For the majority of older adults, gait speed declines by 12–16% each decade, stride length shortens, and step frequency increases. The time in double-support phase increases from 18% in younger patients to 26% by age 70.[62] What distinguishes the so-called senile gait of the elderly and a true pathological gait is often controversial. In general, a disabling gait (one that causes functional impairment), no matter what the age, should be investigated thoroughly for a potentially reversible cause.

Differential Diagnosis

Most gait disorders develop insidiously and may go unnoticed by the patient. Caregiver reports are often necessary for an accurate history. Patients may describe gait impairment in general terms such as "I'm off balance" or "I feel weak." Caregivers may narrow the differential by reporting that the patient appears "drunk" (suggesting cerebellar ataxia) or that the feet appear "frozen to the ground" (suggesting an extrapyramidal gait disorder). Obtaining associated symptoms and signs are key to localizing the lesion and narrowing the differential. Patients with sensory neuropathy causing ataxia often complain of distal paresthesias and sensory loss. Sensory ataxia is classically associated with more difficulty at night or in the dark, when visual input is limited. Lumbar nerve root compromise generally causes radicular, radiating back or leg pain and would generally not cause gait impairment unless multiple lumbar root levels are affected. Myelopathy or spinal cord compression is often associated with muscle weakness and spasticity unless the pathology affects the dorsal columns, in which sensory symptoms predominate. Concomitant memory and cognitive issues suggest a cerebral cause such as NPH, while dysarthria, depending on its quality, suggests either cerebellar, basal ganglia, or hemispheric disease. Vestibular disorders such as labyrinthitis or Ménière's disease are often recognized more by the disequilibrium or vertigo that they cause rather than the gait impairment. Extrapyramidal disorders may initially lead to a primary gait disorder, often described as shuffling associated with a flexed or stooped posture. Over time, tremor, rigidity, and bradykinesia often emerge. Sudden gait failure may suggest stroke, spinal cord compression, medication toxicity, infection, meningitis, or myocardial infarction (Table 18.9).

Examination

A general gait screen should be incorporated into every encounter and begins by watching a patient enter or exit

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Table 18.9 Localization by associated signs and symptoms

Sign	Diagnoses to consider
Dementia, cognitive impairment	Normal pressure hydrocephalus
Nystagmus	Vestibular, cerebellar, or brain stem dysfunction
Dysarthria	Cerebellar, basal ganglia, or corticobulbar dysfunction
Rigidity, tremor, bradykinesia	Parkinsonism
Hyperreflexia, spasticity with Babinski sign	Corticospinal tract disease
Sensory loss, paresthesias, hyporeflexia	Peripheral neuropathy
Dizziness, vertigo	Vestibular disease

the examination room. Gait features that should be observed include stance, base, initiation, velocity, stride length, cadence, fluidity of movements, and deviation. Patients should be observed walking normally, in tandem, and on heels and toes to assess for distal muscle strength. Romberg testing and pull testing should be performed to assess for postural stability.

A person with a normal gait pattern will hold their body and head upright, feet slightly apart, and swing both arms, freely moving forward. Hips and legs flex with each step, while the ankle dorsiflexes. The heel strikes the ground first, moving smoothly along the sole and pushing off with the toes. Stride length should be equal with each step. Some abnormalities may only be elicited through gait testing with obstacles or distractions, such as walking through a doorway or over objects on the floor. Examination of footwear for pattern of wear may also be useful. Get Up and Go is a timed test in which the patient is observed rising from a chair, walking 3 meters, turning around, and returning to the chair. A score of less than 10 seconds is considered normal, greater than 14 seconds is abnormal, while a time greater than 20 seconds indicates severe gait impairment. Although useful as a screen of functional capabilities, this test cannot distinguish between different causes of a gait disorder (Table 18.10).

Evaluation

In most cases, careful history and simple observation of the patient in motion using the above techniques can help to narrow the differential diagnoses. Laboratory evaluation will depend on the initial presentation and findings on physical exam, and may include complete blood count, metabolic panel, fasting blood glucose, glycosylated hemoglobin, erythrocyte sedimentation rate (ESR), rapid plasma reagin (RPR), thyroid-stimulating hormone (TSH), vitamin B12, folic acid levels, and brain imaging with computed tomography (CT). Additional testing if indicated may consist of MRI to evaluate structural degeneration or lesions, and electromyography (EMG) and nerve conduction study (NCS) to evaluate the peripheral nervous system. Specialized gait labs staffed by neurologists, physical medicine specialists, or physical and occupational therapists may help to define a gait disorder and clarify its etiology (Table 18.11).

PD and parkinsonism lead to a slow gait with shortened stride length and low step height, often described as shuffling. When trying to walk faster, patients with parkinsonism tend to increase step speed out of proportion to stride length or step height. In the earliest stages of PD, gait impairment is very subtle with patients noting dragging of one leg or difficulty getting in and out of a car or low seat. The typical posture of a patient with PD consists of flexion of the neck, elbows, waist, and knees. Hesitation and freezing may occur upon initiating gait, turning, and maneuvering through a doorway. Festination refers to the short, accelerating steps that occur when the center of gravity is ahead of the patient, leading to forward propulsion, and the need for the legs to race to catch up with the upper body. When making a turn, the upper and lower body move as a unit, with decreased arm swing and hip rotation, so-called en bloc turning. Loss of postural stability is generally a late manifestation of idiopathic PD (>10 years symptom duration), but an early sign in atypical and secondary forms of parkinsonism (<5–10 years symptom duration).

Frontal lesions lead to a gait pattern similar to a parkinsonian gait. However, in contrast to a parkinsonian gait, it often appears clumsy, ataxic, and unsteady, with difficulty initiating gait. Patients with frontal lesions tend to hold their trunk upright, appear stiff, have a wide-based gait, and show prolonged time in double-support phase. Patients are prone to falling backwards. A frontal gait may also appear magnetic, referring to the appearance that the feet are stuck to the ground. The lower body is predominantly involved, so that arm swing is preserved and may even be exaggerated when a patient attempts to "release" the legs from the ground. Gait initiation fails, but patients show improvement with continued walking. The term "gait apraxia" is used to describe the fact that gait is impaired despite preserved sensation, muscle strength, and leg movements not related to walking.

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Table 18.10 Gait patterns and classification

Type of gait	Description	Associated signs	Causes
Parkinsonian	Short-stepped, shuffling, with hips, knees, and spine flexed, festination, en bloc turns	Bradykinesia, rigidity, postural instability, rest tremor, reduced arm swing	Parkinson's disease, and atypical or secondary forms of parkinsonism
Frontal gait disorder (gait apraxia)	Magnetic, start and turn hesitation, freezing, "marche petit pas"	Frontal lobe signs, dementia, incontinence	Normal pressure hydrocephalus, multi-infarct state, frontal lobe degeneration
Sensory ataxia	Unsteady, worse without visual input and at night	Romberg sign present, impaired position and vibratory sensation, distal sensory loss	Sensory neuropathy, neuronopathy, dorsal column dysfunction
Cerebellar ataxia	Wide-based, staggering	Dysmetria, dysarthria, dysdiadochokinesia, postural instability, Romberg sign present, nystagmus, titubation, impaired check, rebound, intention tremor	Cerebellar degeneration, stroke, drug or alcohol intoxication, thiamine and B12 deficiency, multiple sclerosis
Vestibular ataxia	Unsteady gait, falling to one side, postural instability	Vertigo, nausea, unidirectional nystagmus, normal sensation, reflexes, strength	Acute labyrinthitis, Ménière's disease
Steppage gait	Resulting from foot drop, excessive flexion of hips and knees when walking, short strides, slapping quality, tripping	Atrophy of distal leg muscles, loss of ankle jerk, distal sensory loss, weakness, and foot drop	Motor neuropathy
Waddling gait	Wide-based, swaying, toe-walk, lumbar lordosis, symmetric	Proximal muscle weakness of lower extremities, hip dislocation, use arms to get up from chair	Myopathy, muscular dystrophy
Antalgic gait	Limping, unable to bear full weight, limited range of motion, slow and short steps	Pain worsening with movement and weight-bearing	Degenerative joint disease, trauma
Hemiparetic	Extension and circumduction of weak and spastic leg, flexed arm	Face, arm, and leg weakness, hyperreflexia, extensor plantar response	Hemispheric or brainstem lesion
Paraparetic	Stiffness, extension, adduction, scissoring both legs	Bilateral leg weakness, hyperreflexia, spasticity, extensor plantar responses	Spinal cord lesion or bilateral cerebral lesions
Dystonic	Abnormal posture of foot or leg, distorted gait, foot dragging, hyperflexion of hips	Worse with the action of walking, may improve when walking backwards	Inherited, acquired (i.e., poststroke), or idiopathic
Choreic	Irregular, dance-like, slow and wide-based, spontaneous knee flexion and leg rising	Choreoathetic movements of upper extremities	Huntington's disease, levodopa-induced dyskinesia
Cautious gait	Wide-based, careful, slow, "walking on ice," arms and legs abducted, en bloc turns	Associated with anxiety, fear of falling or open spaces	Post-fall syndrome, visual impairment, deconditioning
Psychogenic	Bizarre and nonphysiologic gait, rare fall or injury, lurching, "astasia abasia"	Give-way weakness, absence of objective neurological signs	Factitious, somatoform disorders, or malingering

Associated frontal lobe signs, such as primitive reflexes (e.g., snout, grasp, or suck), cognitive impairment, or disinhibited behaviors, may be present. Frontal gait impairment is thought to arise because of a disconnection between frontal, basal ganglia, brainstem, and spinal cord gait centers. A wide variety of pathological conditions lead to frontal lobe dysfunction, including NPH, diffuse cerebrovascular disease, and frontal lobe degenerative conditions such as frontotemporal dementia (FTD) or progressive supranuclear palsy (PSP).

Communicating or normal pressure hydrocephalus is an often misunderstood and potentially underdiagnosed entity. Early recognition of the condition is imperative, since symptoms may reverse with prompt treatment.

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Table 18.11 Gait patterns and select etiologies

Maneuver/condition	Finding	Implication
Sitting	Unable to sit upright	Profound imbalance and/or weakness
	Titubation (truncal or head tremor)	Cerebellar disease
	Leaning to one side	Hemiparesis or basal ganglia disorder
Rising from chair	Unable to rise without using arms to push off	Proximal muscle weakness (myopathy), arthritis, or basal ganglia disorder
Standing	Wide-based stance	Cerebellar disease, dorsal column dysfunction
	Stiff neck and head, avoiding motion	Vestibular disease, pain
	Unstable with sternal nudge	Back problems or neurologic problems
Walking on toes and heels		Peripheral neuropathy
Gait	Freezing or start hesitation	Parkinsonism
	Reduced arm swing	Parkinsonism
	Involuntary movements	Huntington's disease, basal ganglia disease
Turning	Widened base, multiple short steps without pivoting	Cerebellar disease, hemiparesis, reduced proprioception
	En bloc turns	Parkinsonism, cautious gait, frontal lobe gait
Romberg sign	Sway/instability with eyes closed	Impaired proprioception

Classically, NPH is associated with the triad of gait impairment, urinary incontinence, and dementia. However, the full triad is not always present. Furthermore, the course may be relatively chronic and progressive, mimicking a neurodegenerative disorder; acute or subacute, mimicking a vascular insult; or static. Gait impairment is usually the first symptom that develops, with memory and urinary symptoms following later. The gait is often wide-based, unsteady, with small, short steps, and feet barely lifting off the floor. The diagnosis of NPH relies on high clinical suspicion and the finding of enlarged ventricles on brain imaging (CT or MRI). It is important to differentiate between ventricular enlargement due to hydrocephalus and so-called hydrocephalus ex vacuo resulting from cerebral atrophy. This latter entity is characterized by both ventricular enlargement and increased sulcal size, and occurs as a result of neurodegenerative diseases and aging. In addition, communicating hydrocephalus needs to be differentiated from obstructive hydrocephalus, which is caused by obstruction of the ventricular system.

Confirmatory testing includes a large-volume lumbar puncture (the so-called tap test), which involves removal of 30–50 mL of spinal fluid with careful examination of gait and cognitive function before and after the procedure. A patient with NPH may respond briskly and dramatically to the procedure. Although a positive response to the tap test confirms the diagnosis, in practice, issues that may confound the test include inability to remove a sufficient volume of spinal fluid, a clinical exam that is not sensitive enough to detect subtle improvements, delayed improvement, and placebo response.[63] Patients with an indeterminate response to the tap test may benefit from observation with a 3-day lumbar drain placed by a neurosurgeon. The procedure increases the ability to detect responders and often helps to confirm the potential benefit of controlled drainage of fluid before resorting to a ventriculoperitoneal shunt.

Cerebellar lesions cause gait ataxia in combination with a wide variety of signs and symptoms. The gait is jerky, clumsy, and unsteady; the stance and base are broad-based, and there is often truncal sway when sitting or standing. Patients are aware of the imbalance and take great effort to ambulate. Lurching of the body may occur as the patient overcompensates to maintain balance. Stepping, direction, distance, and timing are irregular. Step height and stride length are reduced. Tandem gait is impaired because of improper response to postural sway. Patients with cerebellar dysfunction demonstrate dysmetria with past-pointing on finger-to-nose testing. Speech becomes ataxic or scanning with variations in rhythm, volume, and pitch. Rapid alternating movements display impaired rhythm or dysdiadochokinesia, which is demonstrated by rapid tapping

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of the hand or foot. Acute cerebellar ataxia requires an immediate imaging study, since a vascular insult is most likely. Subacute ataxia may be related to infection, nutritional deficiency, or an autoimmune disorder.

In the geriatric population, acute or subacute ataxia without a structural lesion on MRI is suggestive of paraneoplastic cerebellar degeneration (PCD). In that population, chronic progressive ataxia is likely due to a neurodegenerative process such as multiple system atrophy (MSA-C). Hereditary spinocerebellar ataxias (SCAs) are marked by an autosomal dominant pattern of inheritance, onset in the third to fifth decade, and progressive loss of function. Corticospinal tract lesions anywhere from the cerebrum to spinal cord will cause weakness and spasticity, defined as increased tone to passive range of motion. Bilateral corticospinal tract lesions caused by spinal cord injury or cerebral palsy will lead to a spastic or "scissoring" gait. A spastic gait is characterized by extension, adduction, and internal rotation at the hips.

Hemiparetic gait is caused by a unilateral lesion of the corticospinal tract, most commonly occurring after a stroke. The weak leg is spastic, stiff, and extended and circumducts or makes an arc so that the foot can clear the ground. The weak arm is usually flexed at the elbow and pulled toward the chest. Waddling gait is associated with weakness of hip girdle muscles, leading to the dropping of the pelvis toward the swinging leg and compensatory lean toward the standing leg. Sensory ataxia occurs with dorsal column or large fiber sensory loss. In addition to loss of distal position or vibratory sensation, Romberg sign may be present. To test for Romberg sign, have the patient stand with feet close together and evaluate postural stability with eyes open, then closed. A positive Romberg sign is defined as excessive sway or loss of balance when visual input is removed (eyes closed). Steppage gait occurs as the knee and hip flex excessively to compensate for foot drop. Slapping of the feet to the ground may occur as well. Cautious gait is commonly observed in the elderly who have suffered a fall, usually a severe accidental fall resulting in injury. Acute anxiety develops surrounding the risk of falling again. Physical activity is often decreased, and agoraphobia may develop. The gait mimics the gait of a normal person walking on ice. The arms are tense, stance is wide-based, and the body turns en bloc. Once a patient has found physical support, the gait is improved.

Treatment

Treatment of gait disorders involves addressing reversible causes and treating chronic medical conditions that exacerbate the gait problems. Physical and occupational therapy can improve strength, balance, and confidence. Medications should always be evaluated with special attention to psychotropic medications and anticholinergic or antihistaminergic agents. Other treatment modalities include assistive devices, adaptive equipment training, driver's training, and nutritional counseling. Although not rigorously studied, exercise programs such as Pilates, yoga, and tai chi would intuitively improve balance and gait.

Besides dopaminergic agents for Parkinson's disease and ventriculoperitoneal shunting for NPH, there are few specific pharmacological agents for gait disorders. Amantadine, buspirone, and acetazolamide have shown mixed results in patients with cerebellar ataxia.[64]

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Chapter 19

Sleep Disorders

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Introduction

Sleep disorders in late life are often unrecognized, underdiagnosed, and poorly treated. Epidemiological evidence suggests that over 50% of elderly people suffer from one of several different sleep disorders, many of which carry serious negative physical, mental, and social consequences.[1,2] This chapter will focus on the two most common sleep disorders in older patients – insomnia and sleep apnea. Both disorders will be described in terms of epidemiology, diagnosis, and treatment management. Contextual factors that complicate the diagnosis and management of sleep disorders in late life will be reviewed with the aim of providing practical information for the medical professional working with older patients.

Healthy Sleep

Healthy sleep can be characterized as sleep that is regular, of good quality, highly efficient, and of appropriate duration.[44] While disturbed sleep is a common complaint, sleep disorders are not an inevitable event in late life, and many older adults maintain good sleep into the last decades of life. There are, however, noticeable and normal changes in sleep timing, sleep architecture, and sleep quantity that occur throughout the lifespan.[3,4] Older adults often display an advanced circadian tendency, exhibiting an earlier sleep initiation and an earlier wake-up time. Sleep architecture changes with advanced age and includes spending an increased proportion of time, compared to younger adults, in Stages N1 and N2 sleep (i.e., the lighter stages of sleep) and a decreased proportion of time in Stage N3 sleep (i.e., a deeper stage of sleep) and rapid eye movement (REM) sleep. These architecture changes reflect a relative decrease in deep, restorative sleep and an increase in light, transitory sleep. Lastly, older adults tend to spend slightly less time asleep than their younger counterparts. These changes are considered normal in late life and do not indicate a sleep disorder.

Insomnia

Insomnia is the subjective complaint of difficulty initiating sleep, maintaining sleep, or early morning awakenings that must occur at a minimum of three nights per week for three months and be related to significant daytime impairments such as difficulty concentrating, mood disturbances, or fatigue (*International Classification of Sleep Disorders*, 3rd edition[5]). Key concepts in the definition are the subjective nature of both the sleep complaint and the subsequent daytime consequences. The insomnia complaint need not occur in isolation from other clinical disorders. Insomnia is commonly comorbid with other health conditions and/or other sleep disorders, and this co-occurrence does not preclude an insomnia diagnosis or treatment.

Current estimates suggest the prevalence of insomnia is as high as 60% in older adult community-based samples, and higher still in institutional settings. Prevalence rates tend to be higher in older individuals with multiple physical and psychiatric conditions, and higher among older women than men.[3,6] There is evidence to suggest that the higher prevalence rates of insomnia among older adults are a direct consequence of the physical health and mental health comorbidities of aging, rather than a consequence of aging itself.[7] Whether or not insomnia develops as a result of common disorders of aging or the aging process, it still necessitates an independent diagnosis and focused treatment plan.

In older adults, insomnia is most often experienced as a chronic condition (i.e., 3 months or longer), with the average length of insomnia complaints in older adults lasting several years.[11] Negative consequences of insomnia in late life include: decreased quality of life, risk for falls, cognitive difficulties, psychological and physical difficulties, vast economic and social costs, risk for nursing home placement, and mortality.[8–10,45]

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Diagnosis

Given the high prevalence rates of insomnia in older adults and the serious negative consequences of poor sleep, the routine examination of older patients by their primary care physician or geriatrician should include questions aimed at determining satisfaction with sleep. Asking about sleep satisfaction and sleep-related daytime impairment could highlight the need for additional inquiry. Formal insomnia diagnosis requires a structured clinical interview.

The clinical interview should focus on probable predisposing factors, precipitating factors, and perpetuating factors. Predisposing factors include those that may increase an older adult's likelihood of experiencing poor sleep, such as poor physical and mental health, family history of insomnia, or low socioeconomic status. Precipitating factors include recent life events (e.g., depressive episode, hospitalization, loss of a loved one, moving residences) that may initially hasten an insomnia disorder. Perpetuating factors include all contextual, emotional, and behavioral influences that may sustain an insomnia disorder.

Did you know . . .?

The 3P Model[9] of insomnia is very useful for the conceptualization, diagnosis, and treatment of late-life insomnia.

- Predisposing factors are all things that increase an older adult's likelihood of experiencing insomnia.
- Precipitating factors are all things that may initially cause an insomnia disorder to occur.
- Perpetuating factors are all things that sustain an insomnia disorder.

Perpetuating factors are the focus of psychological treatment approaches, while pharmacotherapy tends to target symptoms alone.

Further tools to aid in insomnia diagnosis include validated self-report questionnaires. Examples include the Insomnia Symptom Questionnaire,[46] Insomnia Severity Index,[13] and the Pittsburgh Sleep Quality Index.[14] These questionnaires should take less than 5 minutes to complete, and could be implemented within routine medical visits. While these instruments have the potential to aid in insomnia diagnosis, some older patients may find the retrospective recall of sleep to be onerous. A prospective self-report measure of sleep (i.e., a daily sleep diary) can capture very useful information regarding sleep timing, quantity, and quality across consecutive days. However, the use of a sleep diary requires daily monitoring by the patient and a return visit to their provider in 1–2 weeks. As such, the use of a daily sleep diary may not be practical for the clinical diagnosis of late-life insomnia by a primary care physician or geriatrician. More expensive, objective measures of sleep, including wrist actigraphy and polysomnography, are not indicated for routine use in the diagnosis of insomnia in older adults.

Treatment

Treatment of late-life insomnia can involve either pharmacological agents, psychological strategies, or both. Given that psychological treatment is safe and has outperformed pharmacotherapy as well as combined psychological/pharmacotherapy in head-to-head trials,[15,16] psychological techniques should be considered as initial treatment. In fact, the American College of Physicians released a Practice Guideline that suggested all adult patients with insomnia receive Cognitive Behavioral Treatment for Insomnia (often termed CBTI).[47]

Psychological

Psychological interventions for insomnia encompass a variety of different techniques, including: sleep education, cognitive therapy, sleep hygiene, relaxation strategies, stimulus control, sleep restriction, and multicomponent treatment packages (i.e., CBTI). Sleep is largely behaviorally regulated, with a strong homeostatic biological drive and circadian component. Interventions with a foundation in behavioral theory and practice have been proven quite effective in the management of insomnia.[12]

Stimulus control is a behavioral strategy based on classical conditioning principles that aim to increase the response of sleep associated with the stimulus of the bed and bedroom. There is strong evidence in support of stimulus control for insomnia in late life.[4,17,18] Sleep restriction is a behavioral strategy aimed at increasing the homeostatic sleep drive and strengthening the circadian signal strength through more closely aligning time spent in bed to actual time spent asleep. There is strong evidence in support of sleep restriction for insomnia in late life.[17,18] Sleep education (e.g., providing older patients with information regarding normal age-related changes in sleep), cognitive therapy (e.g., examining the evidence for commonly held dysfunctional beliefs and attitudes about sleep), and sleep hygiene (e.g., avoiding and/or limiting caffeine, nicotine, and alcohol consumption)

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have little empirical evidence to support their use as standalone treatment options for insomnia in older adults. These three techniques are better suited for use in combination with other psychological treatment strategies. Relaxation strategies may prove beneficial in isolation in improving the sleep of older adults. Appropriate relaxation techniques include: passive muscle relaxation, autogenic strategies, visualization techniques, and breathing exercises. Because of the high prevalence of pain conditions in late life, progressive muscle relaxation (PMR) should be used cautiously, as the contract-relax method of PMR may inadvertently exacerbate existing pain conditions. However, like the above-mentioned strategies, relaxation techniques may be better suited for use in combination with other psychological treatments.

Did you know . . .?

- Sleep hygiene recommendations are the most commonly used nonpharmacological treatment approach to the management of insomnia.
- There is no evidence to support use of sleep hygiene alone for insomnia management.
- In fact, sleep researchers often use sleep hygiene recommendations as the control or placebo condition in psychological intervention research.

CBTI has the strongest empirical grounding of all available behavioral treatment options for insomnia in older adults.[17,18] CBTI is a combination treatment for insomnia typically consisting of stimulus control, sleep restriction, and sleep education. CBTI has been demonstrated to result in large improvements in perceived sleep in older adults with insomnia, and older adults prefer CBTI to sedative hypnotics. The typical delivery of CBTI involves one-on-one, face-to-face delivery in four to six weekly (or biweekly) sessions, each lasting between 30 minutes and 60 minutes. CBTI has been successfully delivered in as little as one to two 30-minute sessions, in group formats, and over virtual communication lines. While commonly delivered by specially trained clinical psychologists, CBTI has been successfully administered by supervised nurse practitioners, mental health nurses, non-specialist sleep coaches, and over web-based platforms.[19,49] The overwhelming confluence of the evidence leads to CBTI as the recommended frontline treatment for insomnia in older adults. See Table 19.1 for a comprehensive listing of psychological treatment options for insomnia in late life.

Pharmacotherapy

Although sedative hypnotic medications are the most commonly prescribed treatment approach for insomnia in older patients, medication treatment for late-life insomnia should be used in the minority of patients. The fact that older adults are over twice as likely to be prescribed medication treatment for insomnia as younger adults[20] is particularly concerning, given the increased risk for drug side effects, drug interactions, tolerance and dependence, and lack of empirical evidence supporting long-term use in older patients. Short-term pharmacotherapy may be indicated in situations of acute insomnia, but in older adults with chronic insomnia, sedative hypnotics should be used with great caution. When the decision is made to prescribe a sedative hypnotic to an older patient, the smallest effective dose with the lowest risk of adverse effects should be prescribed for the shortest duration of time. Discontinuation of sedative hypnotic medications can be difficult. A promising technique in older adults is masked tapering.[48]

The commonly used sedative hypnotic medications can be broadly grouped into three categories: (1) short-, intermediate-, and long-acting benzodiazepines, (2) nonbenzodiazepines or "z-drugs," and (3) sedating antidepressants. See Table 19.2 for a detailed listing of specific drugs. In general, long-acting benzodiazepines should not be used with older adults because of increased risks of daytime sedation, falls, and confusion.[21] Short- or intermediate-acting benzodiazepines are preferable for patients with a primary complaint of sleep maintenance difficulties. The nonbenzodiazepines are a relatively newer group of sedating hypnotic medications. The nonbenzodiazepines have a shorter duration of action than the benzodiazepines, are believed to carry a lower side effect profile, and appear to be well tolerated in late life; however, there is a limited amount of evidence pertaining directly to use of most nonbenzodiazepines specifically in older patients. Sedating antidepressants are often used off-label for their sedative hypnotic effects, though very little empirical evidence supports the use of sedating antidepressants as hypnotic agents in older adults. Additionally, agents with anticholinergic side effects should be avoided.

Sleep Apnea

Sleep apnea is a breathing disorder characterized by a reduction (hypopnea) or absence (apnea) of airflow during sleep. When this phenomenon is secondary to a reduced respiratory effort, it is termed central sleep apnea (CSA). More frequent is obstructive sleep apnea

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Table 19.1 Psychological treatment approaches for insomnia in older adults

Technique	Level of support
• Sleep education Information regarding normal sleep changes with age. Aimed at normalizing current sleep, improving expectations, and reducing anxiety.	Low[1]; not an evidence-based practice[2]; not a recommendation.[3]
• Cognitive therapy Maladaptive thoughts, beliefs, and attitudes can negatively impact sleep. Challenging these thoughts can help promote sleep through a reduction in sleep-disruptive thoughts and emotions.	Low[1]; not an evidence-based practice[2]; not a recommendation.[3]
• Sleep hygiene Instruction to avoid or limit sleep-disruptive substances and behaviors, including caffeine, alcohol, nicotine, exercising, and heavy meals at night.	Low[1]; not an evidence-based practice[2]; not a recommendation.[3]
• Relaxation strategies Active or passive relaxation techniques all aimed at reducing physiological or mental arousal that may be interfering with sleep.	Moderate[1]; not an evidence-based practice[2]; standard recommendation.[3]
• Stimulus control Behavioral technique based on classical conditioning principles. Instructs an individual to limit use of the bed to sleep and sex, and to limit the amount of time spent awake in bed.	Strong[1]; not an evidence-based practice[2]; standard recommendation.[3]
• Sleep restriction Behavioral strategy aimed at matching the amount of time one spends in bed with the actual amount of time asleep. A consistent sleep schedule and time in bed is collaboratively prescribed and adjusted as needed.	Strong[1]; evidence-based practice[2]; guideline recommendation.[3]
• Multicomponent treatment packages Combine several individual components into a treatment package. Usually consist of stimulus control, sleep restriction, sleep education. Sometimes include cognitive therapy, relaxation techniques, or sleep hygiene recommendations.	Strong[1]; evidence-based practice[2]; standard recommendation.[3]

Notes: [1] Based on authors' critical review of empirical evidence and clinical practice with older adults.

[2] Criteria for an intervention to be considered evidence-based include: 50% of the outcome measures must demonstrate significant treatment effects with between-group effect sizes of at least 0.20.[14]

[3] American Academy of Sleep Medicine (AASM) Practice Parameters.[15]

(OSA), in which the respiratory effort persists during the episodes of hypoventilation.

The prevalence of sleep apnea increases with age, with rates up to 40% being reported in people older than 65 years old.[22] Sleep apnea is more common in men and in patients with multiple comorbidities, especially dementia and obesity. In CSA the absence of respiratory effort is secondary to neurological conditions (e.g., stroke), heart failure, or drugs and substances that depress the central nervous system (e.g., opioids). In OSA, the repetitive obstructions of the upper airway are secondary to anatomical factors such as obesity and/or reduced activation of the dilatory muscles of the airway (e.g., under the effect of alcohol or sedative drugs). Patients incur frequent episodes of hypoxia and hypercapnia during sleep resulting from these breathing pauses.

Associations between sleep apnea and medical and neuropsychiatric conditions are abundant, and include hypertension, coronary artery disease, depression, car accidents, cognitive impairment, stroke, and mortality.[23,24] A healthy older adult with sleep apnea usually complains of headache (typically in the morning), daytime sleepiness, irritability, fatigue, and impotence in men. Medically compromised patients who perform few daytime activities or those with cognitive impairment may present with subtle manifestations or may even be asymptomatic. Bed partners should be interviewed, as they can describe snoring, gasping, choking, apnea episodes, or irritability. Although common findings at the physical exam include hypertension and obesity, the latter is less frequent in older patients.[25] A crowded oral pharynx may also be seen.

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Table 19.2 Medications commonly used for insomnia

Generic name	Drug class	Initial dosage (mg)	Usual dosage (mg)	Half-life (in hours)	Comments
Eszopiclone	Short-acting nonbenzodiazepine	1	1–2	6	Approved for long-term use (not specific to older patients); may cause unpleasant taste and headache
Zolpidem	Short-acting nonbenzodiazepine	5	6.25	3	Little daytime carryover or rebound insomnia; risk of nocturnal behaviors
Zaleplon	Short-acting nonbenzodiazepine	5	5–10	1	Little daytime carryover or rebound insomnia
Temazepam	Intermediate-acting benzodiazepine	7.5	7.5–15	8.8	Psychomotor impairment; risk of falls
Ramelteon	Melatonin receptor agonist	8	8	2.6	Little rebound insomnia or withdrawal; dizziness, myalgia, and headaches
Tasimelteon	Melatonin receptor agonist	20	20	1.3	Headache, increased liver enzyme, nightmares and abnormal dreams, FDA approved for non-24-hour sleep-wake disorder only
Doxepin	Sedating antidepressant	3	3–6	15.3	Should not be taken within 3 hours of eating; anticholinergic effects
Trazodone	Sedating antidepressant	25	25–150	2–4	Off-label use; insomnia + depression; low anticholinergic effects; orthostatic hypotension
Mirtazapine	Sedating antidepressant	7.5	7.5–45	31–39	Off-label use; insomnia + depression; increased appetite, weight gain, headache; akathisia
Suvorexant	Orexin receptor antagonist	5	5–20	8–19	Next-day somnolence and impaired performance (such as driving); cataplexy-like symptoms also reported. Contraindicated in patients with narcolepsy.

Diagnosis

Clinical findings are neither specific nor sensitive enough to diagnose sleep apnea. Some clinical prediction rules are useful for screening purposes (e.g., STOP-BANG[26]). The gold standard for the diagnosis of sleep apnea is in-laboratory polysomnography. The American Academy of Sleep Medicine recommends the measurement of the sleep stages (electroencephalogram), muscular activity, nasal airflow, oxygen saturation, electrocardiogram, snoring, body position, and limb movements.[27] A more convenient and feasible diagnostic alternative is the use of portable monitors at home, most of which record nasal airflow, respiratory movements, and arterial oxygenation. These tests have shown a high correlation with polysomnography, especially for moderate and severe cases, but are not recommended in patients with multiple comorbidities or when CSA or other sleep disorders are suspected. The diagnosis is based upon the average number of hypopneas and apneas per hour (apnea hypopnea index, or AHI). Sleep apnea is classified as mild (AHI 5–15), moderate (AHI 16–30), or severe (AHI >30). Coexisting sleep apnea and insomnia may be common in older adults. (See Figure 19.1.)

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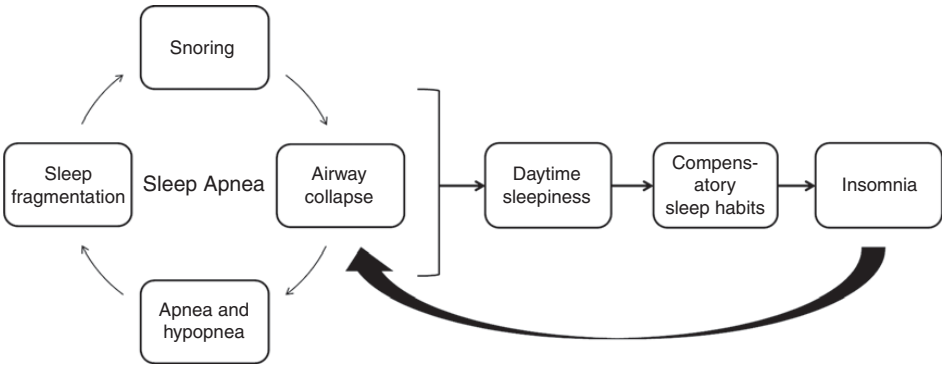

Figure 19.1 Hypothesized relationship between sleep apnea and the development of insomnia. The model illustrates that sleep apnea can cause daytime sleepiness, which in turn may relate to an individual engaging in compensatory sleep habits, which can lead to insomnia symptoms. Poor sleep can feedback into a weakening of the musculature of the upper airway, potentially worsening sleep apnea.

Did you know . . .?

Sleep apnea can be easily screened for during routine clinical care using the STOP-BANG[18] questionnaire:

1. Do you SNORE loudly (louder than talking or loud enough to be heard through closed doors)?
2. Do you often feel TIRED, fatigued, or sleepy during daytime?
3. Has anyone OBSERVED you stop breathing during your sleep?
4. Do you have or are you being treated for high blood PRESSURE?
5. Body mass index (BMI) more than 35?
6. AGE over 50 years old?
7. NECK circumference >15.75 inches?
8. Male GENDER?

≥3 yes answers: High risk for OSA

<3 yes answers: Low risk for OSA

Treatment

General treatment recommendations for older patients diagnosed with sleep apnea include avoidance of sedative drugs and alcohol; exercise; and the consumption of a hypocaloric diet to reduce weight and control blood pressure. In some cases, a short nap before long driving periods may be recommended if excessive daytime sleepiness is present.[28,29] Positive airway pressure (PAP) used to keep the airway open during sleep episodes is the first-line therapy for patients with moderate or severe sleep apnea. This therapy has been demonstrated to improve sleep quality, daytime symptoms, blood pressure, ventricular ejection fraction, and cognitive function.[28,50] Observational studies have reported that PAP reduces the risk of stroke, cardiovascular disease, and mortality in older patients.[30,31] Good adherence to PAP therapy (defined as >4 hours of use per night on 70% of nights) may be a challenge to older patients.

Continuous PAP (CPAP) is the most commonly used type of PAP device. CPAP provides a fixed pressure of air during the breathing cycle. There are more advanced devices (e.g., BiPAP or APAP) that can provide different levels of pressure and may be better tolerated by some patients. Other measures that can improve patient compliance to PAP therapy are the use of humidifiers, proper fitting of a correct interface (e.g., nasal or facial mask), and concomitant psychological cognitive behavioral therapy. A multidisciplinary approach is vital, especially at the beginning of the treatment. Long-term adherence is best predicted by use during the first week of therapy.[32]

There are several alternatives for patients who reject or do not adhere to PAP therapy. Oral appliances (i.e., mandibular advancement devices) move the jaw forward with the goal of reducing obstruction of the airway. Nose valves attempt to increase airway pressure through closing during expiration. Surgical procedures (e.g., uvulopalatopharyngoplasty) remove excess tissue in the upper airway. The effectiveness and availability of these options is scarce, and most empirical studies have excluded older adults.[28] Hypoglossal nerve stimulation, or upper-airway stimulation, is a newer treatment for OSA that has shown promise in treating older patients.[51]

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Contextual Factors

Hospitalization, institutionalization, and dementia are common in late life and are associated with increased rates of sleep disorders when compared to that seen in community-dwelling older adults. Sleep disorders in these contexts are reviewed below.

Sleep Disorders in the Hospital

Sleep disorders are frequent and generally underrecognized in the hospital. Research has shown that patients tend to underestimate their total sleep time in hospitals, while nurses overestimate how long patients sleep.[33] Abnormalities in electroencephalogram patterns and plasma levels of melatonin have been observed among hospitalized patients. These alterations may occur during sepsis or inadequate light exposure, but the underlying mechanisms behind these observations are not completely understood.[34] Some consequences of poor sleep in the hospital are poor pain management, increased risk of delirium, and worse metabolic control.[52]

There are many potential factors leading to the increased prevalence of insomnia in hospitalized older adults. Acute pulmonary and cardiac diseases may interfere with the normal breathing cycle, generate dyspnea and cough, and lead to subsequent problems during sleep. Other symptoms that are also common in the hospital setting and may disrupt sleep are pain, delirium, and anxiety. Hospital-related environmental factors that interrupt sleep are noise, light, an unfamiliar bed, administration of medications, and the measurement of vital signs during the night. These disruptors are much more frequent in intensive care units. Multicomponent interventions can effectively improve the sleep of older patients in the hospital.[53] Some simple, safe, and effective recommendations are the use of eye masks and earplugs, reductions in nighttime light and noise, encouragement of daytime physical activity, and minimization of sleep interruption by health-care providers.

The overall prevalence of sleep apnea in the hospital setting is unknown; however, in specific populations such as stroke patients it is as high as 60%.[35] Adverse outcomes are more frequent in patients with sleep apnea and include intra- and postoperative complications, prolonged length of stay, and possibly increased mortality.[36,37] Screening questionnaires can be used to assess the risk of undiagnosed sleep apnea in hospitalized patients. Some recommendations for patients at risk are to reduce the doses of neuromuscular blockers, avoid opioids, assume a semi-upright sleeping position in bed, peripheral oxygen monitoring, oxygen supplementation, and the use of PAP therapy. Empirical evidence to support these strategies is limited. Those patients who have been previously prescribed PAP therapy should use it during the hospital stay.

Sleep Disorders in the Nursing Home

Sleep disorders are more frequent in nursing home residents than in community-dwelling older people. Sleep/wake patterns are altered in most residents. A typical sleep presentation includes several episodes of napping during the day, coupled with fragmented sleep at night. Factors associated with these findings are older age and presence of medical and psychiatric diseases. Institutionalized older people present with a high prevalence of comorbidities and geriatric syndromes, such as frailty, depression, heart failure, urinary incontinence, and dementia. As in the hospital, environmental factors (e.g., lack of exposure to sunlight and physical activity, increased amount of time spent in bed during the day, and the use of medications with effects on the central nervous system) also contribute to the occurrence of sleep disorders. Some of the negative consequences ascribed to sleep disorders in this population are poorer self-rated quality of life, reduced involvement in social activities, and increased mortality.[38,39]

A multidimensional intervention should be considered to treat sleep disorders in nursing homes. Nonpharmacological measures such as an increased light exposure (e.g., bright light boxes) and physical activity participation may have modest benefit. Nighttime reductions in noise and light should be attempted. Adherence of nursing home staff to these recommendations can prove difficult. Cognitive behavioral strategies have been demonstrated to improve the subjective sleep quality of older adults in nursing homes.[40] Sedative hypnotic medications may have a small benefit in improving sleep of residents,[41] but considering the side effects, risks, and complex metabolism of these drugs in fragile older adults, they should be avoided if possible.

Sleep Disorders and Dementia

Sleep disorders are frequent and have great impact in patients with dementia. Sleep disorders are associated with loss of function, cognitive impairment, and an increased burden in caregivers.[42] Disturbed sleep is a frequently cited reason for institutionalization.

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Sundowning is a particularly salient sleep-related disturbance in older adults with dementia. Sundowning is an altered behavioral state (e.g., delirium, anxiety, agitation, wandering) of a patient with dementia, in which the symptoms are characteristically more intense during the evening and at night. Biological and environmental factors have been suggested to explain this phenomenon. Conversely, some authors have proposed that sundowning is the result of more exhausted caregivers' perceptions at the end of the day.[42]

Did you know . . .?

In addition to insomnia and sleep apnea, older adults may present with the following sleep disorders:

1. Advanced phase disorder: a systematic shift in sleep timing to an earlier sleep initiation and rise time.
2. REM behavior disorder: movement or acting out of dreams during sleep. Much more common in Parkinson's disease.
3. Restless legs syndrome: uncomfortable sensations in the legs that occur with rest/inactivity.

In a patient with dementia, a comprehensive evaluation of not only the patient but also the environment should be performed to identify the potential causes of the sleep disorder. Special attention is necessary regarding the use of caffeine, alcohol, or any medication that may interfere with sleep. Common clinical findings in healthy older adults could be subtle or absent in patients with dementia. For example, delirium could be the only clinical manifestation of sleep apnea in dementia patients and should trigger further evaluation in patients with risk factors mentioned above. Pain and mood disorders could also be precipitating factors of the sleep disorder and may be more difficult to evaluate in patients with cognitive impairment.

Identifying and treating the underlying causal factors of the sleep disorder in conjunction with sleep-focused interventions could be effective in improving sleep patterns in older adults with dementia. Light exposure, physical exercise, stimulus control, sleep restriction, and behavioral activation/social activities should always be considered as first-line therapy. A recent meta-analysis of sleep medications for patients with Alzheimer's disease reported that trazodone in low doses (50 mg) improves total nocturnal sleep time and sleep efficiency, while there was no benefit for melatonin or ramelteon. There is a lack of evidence for use of most sedative hypnotic drugs in patients with Alzheimer's disease (e.g., benzodiazepines, nonbenzodiazepines hypnotics, other antidepressants, and antipsychotics agents).[43] Given the increased risk of falls and cognitive decline with benzodiazepines and increased mortality with antipsychotics agents in patients with dementia, a thoughtful risk/benefit evaluation should be performed and discussed with the patient and caregivers before considering these drugs.

Conclusions

Sleep disorders are a very common occurrence in late life. These sleep disorders are associated with serious negative physical, mental, and social consequences. Insomnia and sleep apnea are particularly important conditions in older adults, and may coexist. While good sleep is often taken for granted, poor sleep can be very deleterious to the general health and quality of life of older patients. Recognition and appropriate management of sleep problems in older adults is essential.

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Chapter 20

Clinical Geropsychiatry

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While most older people are mentally healthy, persons over age 65 are vulnerable to the same spectrum of psychiatric disorders as are younger people. Community epidemiologic studies indicate that prevalence rates for major depressive disorders, panic disorders, and substance use disorders are lower in the elderly. However, the prevalence of phobic disorders does not change with age, and the prevalence of cognitive disorders and their associated psychiatric morbidity sharply increases with age.[1,2]

Psychiatric problems in the elderly are more common in certain settings. For instance, anxiety and depressive disorders are common among patients in medical clinics, while confusional states (delirium) are seen in approximately 30–60% of hospitalized patients on medical and surgical services.[3] In nursing homes and long-term care facilities, more than 50% of residents have been found to suffer from some sort of psychiatric problem, most commonly dementia, and behavioral problems and depression are common.[4] Individuals with developmental disabilities, including intellectual disability and autism spectrum disorder (ASD), are at increased risk of psychopathology compared to the general population, and clinicians ought to be comfortable in treating this aging population.[5] In all, there is a need for careful attention to psychiatric symptoms in the elderly, since compassionate and appropriate treatment improves both overall functioning and quality of life.

Evaluation

History

The evaluation of the older adult with a possible mental disorder begins, as does any medical evaluation, with a careful history. If the patient is accompanied by family members, it is helpful to meet with them also, to facilitate obtaining a complete history and database. The history should focus on a thorough assessment of the reason for the appointment, including a careful determination of when symptoms first appeared, how they have progressed over time, and accompanying features. In addition, the complete history should include the following:

1. *Family psychiatric history.* The clinician should inquire whether any blood relatives, especially first-degree relatives, have ever suffered from a mental disorder, suicide, or alcoholism or have been hospitalized in a psychiatric facility.
2. *Psychiatric history of the patient.* This should include any prior contact with psychiatrists or therapists, prior psychiatric hospitalizations, or previous treatment by any medical professional for mood problems or bad nerves.
3. *Medical history.* It is important to detail all prior hospitalizations and surgeries and any current medical conditions that continue to be a focus of treatment.
4. *Medications.* This should be a complete list of all medications, both prescription drugs and over-the-counter medications being taken by the patient, including dosages. Because many medications prescribed for a variety of medical conditions have psychiatric side effects, it is helpful to inquire about the length of time the patient has taken the medication and to pay particular attention to changes in medications prescribed shortly before the onset of the presenting psychiatric symptoms.
5. *Personal history.* This includes information about the patient's family of origin, siblings, childhood history, schooling (especially level of education), work history, adjustment to retirement, sexual history, marital history, and children. It is also important to inquire about the patient's living situation, including with whom he or she lives and the type of home (i.e., house or apartment, rented or owned). This is also a good time to ask about any structural aspects of the home that may pose problems for the patient, such as stairs, second-floor bathrooms, tub, and showers.

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6. *Patterns of alcohol and drug use.* Problems of alcohol use occur in the elderly, as in younger persons, and may underlie symptoms of anxiety, depression, irritability, memory loss, sleep disturbance, sexual dysfunction, and paranoia. It is necessary to obtain information on the type of alcohol consumed, how frequently, and how much and to inquire about early-morning shakes, blackouts, alcohol-induced seizures, and prior episodes of detoxification and treatment. In addition, members of the "baby boomer generation" use illicit drugs at higher rates than previous cohorts of older people, highlighting the need for clinicians to inquire about marijuana, cocaine, heroin, and other illicit drug use in the elderly.[6,7]

Mental Status Examination

The heart of the psychiatric evaluation is the mental status examination, the here-and-now data-gathering equivalent of the physical examination. It allows a systematic examination of the major aspects of the patient's mental state. Depending on the nature of the presenting complaint and the cooperativeness of the patient, certain areas of the mental status examination may be emphasized, while others may be only touched on briefly. The complete mental status examination, however, always includes attention to the following areas:

1. *General appearance.* This includes observation of neatness and personal hygiene; eye contact during the interview; any abnormal movements, tremors, tics, or unusual behaviors; and the individual's posture and gait upon entering and exiting the office, noting any need for assistive devices.
2. *Speech.* This refers to the motor and linguistic forms of the patient's verbal language. It includes attention to the rate, rhythm, and loudness of the patient's speech and whether the patient's use of language is coherent, goal-oriented, logical, and easy to follow. Does the patient seem to jump from one idea to another with little connection between ideas? This is described as loose association and in an extreme form may be called flight of ideas. Some patients may have trouble sticking to the topic at hand and exhibit a tendency to wander off track (tangentiality) or can be redirected to the issue being discussed. Obsessional patients may be inclined to be over-inclusive in detail (circumstantiality), sometimes losing sight of the forest for the trees. Aphasic patients have word-finding difficulty, paraphasias (made-up words), and non-fluent or fluent but content-free speech.
3. *Mood.* The assessment of mood involves both ascertainment of the patient's subjective description of his or her mood state and the clinician's objective observations of the patient's mood. Some depressed elderly patients report that they do not feel depressed, yet use words such as "sad," "bewildered," or "drained" and appear tense, anxious, or withdrawn.
4. *Suicidal ideation.* It is important to ask any patient with a sad mood about suicidal thoughts. Contrary to popular myth, asking about suicidal thoughts does not increase the likelihood that a patient will follow through on such ideas. We distinguish between passive suicidal thoughts (i.e., wishing one were dead or would die) and active suicidal ideation (i.e., planning self-harm). Many depressed patients express passive wishes for death but are adamant that they would never attempt suicide for personal, religious, or family reasons. Religious beliefs and sense of responsibility toward one's family can be protective factors against suicide.
5. *Abnormal thought content*.
    a. Hallucinations are sensory experiences that are perceived in the absence of a sensory stimulus. Auditory and visual hallucinations are most common, but tactile and olfactory hallucinations also occur in some disorders.
    b. Delusions are idiosyncratic, fixed, false beliefs that are not culturally determined or shared. Paranoid delusions and delusions of persecution are most common. Manic patients may have grandiose delusions about themselves and their abilities. Other types of delusions that may develop in older patients are delusional jealousy (falsely believing one's spouse has been unfaithful) and delusions of parasitosis (believing one's skin to be infested with worms or insects). Often the delusion seems plausible until further medical or social investigation reveals it to be unfounded. A distinguishing feature of delusions is that the patient cannot be persuaded that the belief is false despite evidence to the contrary.
    c. Obsessive thoughts are intrusive, repetitive, unwanted ideas that a person cannot stop from coming to mind.
    d. Compulsions are intrusive, repetitive, unwanted behaviors that a person cannot stop, although they recognize them as unnecessary, excessive, or foolish. Some examples are compulsive hand-washing and checking behaviors.

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e. Phobias are excessive specific fears that cause a person to avoid the dreaded situation.

6. *Cognitive assessment.* Every psychiatric evaluation of the older patient should include an assessment of cognitive functioning. Depending on the nature of the initial presenting complaint and the cooperativeness of the patient, this assessment may be fairly brief or detailed and focused. A basic cognitive screening should include: level of alertness, attentiveness, orientation, short- and long-term memory, attention and concentration, naming ability and language comprehension, and abstract reasoning. If significant cognitive impairment is detected in one or more of these areas, further neuropsychological testing and/or laboratory testing may be warranted.

Specific Conditions

Anxiety Disorders

Anxiety is feelings of tension and distress that are distinct from sadness and that usually lack a stressful stimulus of such severity as to explain the feeling. It often has both somatic (physical) and psychological components. *Generalized anxiety disorder* is a condition marked by excessive worry and anxiety persisting for 6 months or more. It is accompanied by signs and symptoms of motor tension, including muscle aches or soreness; a feeling of restlessness; a feeling of shakiness; and reports of easy fatigability. In addition, there are feelings of being on edge, having difficulty concentrating and falling asleep, and being unusually irritable. At least three of these additional symptoms of motor tension must be present along with the subjective distress of constant worry to make the diagnosis of generalized anxiety disorder. Generalized anxiety disorder should be distinguished from a patient's report of feeling "anxious." The new development of a complaint of "anxiety" in an older person is most commonly caused by major depression. Generalized anxiety disorder, on the other hand, is usually lifelong, not episodic, and associated with the somatic and psychological accompanying symptoms described above. *Panic disorder* is diagnosed when the patient reports discrete episodes (attacks) of intense fear and somatic anxiety symptoms that are both unprovoked and unexpected, with accompanying anticipatory worry about the attacks or avoidance of situations that may precipitate attacks. The associated somatic symptoms include palpitations, sweating, trembling, shortness of breath, chest discomfort, lightheadedness, and abdominal distress. It is common for panic attacks to occur repeatedly in certain circumstances, e.g., in a grocery store. Specific phobias are clearly delineated fears of objects or situations that a person realizes are unrealistic but nevertheless cannot resist. They sometimes occur in concert with panic attacks.

The anxiety disorders are among the most common psychologic problems identified in mental health surveys. Nonpharmacologic and pharmacologic therapies are usually used to treat them. Desensitization (gradually exposing the patient to the source of distress) coupled with relaxation is often effective. The most effective pharmacologic therapy is the use of antidepressants. There is no evidence that one antidepressant is better than another. Selective serotonin reuptake inhibitors (SSRIs), such as escitalopram, sertraline, and fluoxetine, and selective noradrenergic/serotoninergic uptake inhibitors (SNRIs), such as venlafaxine and duloxetine, are effective for treating anxiety disorders. SSRIs are better tolerated with fewer side effects than tricyclic antidepressants.

Benzodiazepine compounds are also effective for anxiety disorders, but, because of their addictive potential and side effects, are generally not prescribed as a first-line therapy. Short-acting benzodiazepines (e.g., alprazolam) have more abuse and addiction liability than longer-acting compounds (e.g., clonazepam), but the longer-acting compounds are more likely to accumulate and lead to sedation, functional impairment, and drowsiness. Buspirone is non-addicting but appears to be less effective in the treatment of anxiety than benzodiazepine or antidepressants. If symptoms are severe and immediate results desirable, the clinician may choose to initiate treatment with both an antidepressant and benzodiazepine and taper the benzodiazepine several weeks after the antidepressant begins to work.

Mixed Anxiety and Depression

Symptoms of anxiety and depression frequently co-occur. The clinician should make an effort to determine which is primary and to focus treatment on that set of symptoms. In our experience, depression is more frequently the primary disorder, but this is controversial. Features in the history suggesting that depression is primary include a history of episodic (prior) depressive episodes, a family history of depressive episodes, diurnal mood variation (i.e., a tendency for symptoms to be worse in the morning), self-blame, guilt, difficulty staying asleep in contrast to falling asleep, hopelessness, and mental somatization.

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While anxiety disorders can begin de novo in late life, it is much more common for a depressive episode to appear for the first time in an older person. Because antidepressants are effective in treating both anxiety disorders and major depression, they should be the first-line treatment when the clinician is unsure which is primary.

Mood Disorders

Mood disorders are the most frequently clinically diagnosed and the most treatable psychiatric disorders in older people.[8,9] They encompass a spectrum of disorders ranging from adjustment disorder (in which an identified psychosocial stressor provokes a mild depressive reaction that impairs functioning) to psychotic major depression with hallucinations and/or delusions to mania.

Major depression is characterized by a persistent diminution in three spheres of functioning: (a) mood, (b) vital sense (a sense of one's well-being and energy), and (c) attitude toward oneself (self-confidence). Depressed patients tend to have a more negative self-assessment than is usual for them, may be self-blaming, or can have excessive feelings of guilt, regret, or worthlessness. Patients with major depression experience loss of energy, disturbed sleep (usually insomnia and early-morning awakening), diminished appetite and weight loss, difficulty thinking and concentrating, and a loss of interest and pleasure in activities that they once enjoyed. Ruminant thoughts of death and suicidal thoughts may occur during the course of a major depression. Elderly patients who are depressed often complain of physical rather than psychologic distress. Up to a third of older people who suffer from major depression do not describe their mood as depressed. Rather, they focus on feelings of weakness, lack of energy, and lack of motivation. Somatic complaints, including headaches, gastrointestinal disturbances, and body aches, are common. Occasionally, hallucinations and delusions occur. Such hallucinations and delusions tend to have a depressive theme and are consistent with low mood, e.g., the persecutory delusion that one deserves punishment; the delusion that one has no money, clothes, or insurance; and the delusion that one has a terrible illness that doctors cannot find.

Major depression can first occur at any point in the lifespan. It may occur as a single episode, but recurrence is common. The causation of major depression is complex, involving genetic, neurochemical, and psychologic factors. While genetic transmission is poorly understood, it is clear that affective (mood) disorders tend to run in families and that there is a higher prevalence of affective disorders among the first-degree relatives of depressed individuals. The neurochemistry of depression is an active area of research focusing on abnormalities in adrenergic and serotonergic neurotransmitters in the brain. Many commonly prescribed medications, including steroids, reserpine, methyldopa, antiparkinsonian drugs, and β-adrenergic blockers, can cause depression. Depression is especially common in diseases of the brain. For example, 30–60% of poststroke patients have a clinically significant episode of depression within 6 months to 2 years of the stroke.[10,11] The incidence of poststroke depression has been found to be greatest among patients with strokes affecting the left anterior cerebral hemisphere, particularly during the first 6 months following a stroke.[10,11] While major depression can occur in the absence of any precipitating event, psychologic issues such as recent loss (i.e., job, independence, social supports) and chronic medical illness play a contributing role in many cases.[12,13] Regardless of whether psychological factors provoke a depressive episode, they clearly can affect its course and outcomes. Supportive psychotherapy is an important part of the treatment of depression in conjunction with appropriate pharmacotherapy.

The psychopharmacologic treatment of major depression has advanced considerably in recent years, and many effective antidepressant medications are available.[14] Tricyclic antidepressants are older drugs with well-established efficacy. Older persons do best when given antidepressant drugs with the least anticholinergic activity. Therefore, nortriptyline and desipramine are the tricyclics that are favored for older people. SSRIs, including fluoxetine, sertraline, and escitalopram, are well tolerated by older patients. They have minimal anticholinergic effects and are not associated with blood-pressure and heart-rate changes. However, SSRIs can impair sleep even when taken in the morning. If this occurs, adding trazodone at bedtime can improve sleep. Nausea and loose stool, other common side effects, may be dose related. Monoamine oxidase inhibitors can be given to older patients if prescribed cautiously. They may be indicated for difficult cases when other medications have failed.

Other antidepressants include: buproprion, which has mild central nervous system-activating effects, minimal anticholinergic effects, and few cardiovascular side effects, but a higher risk of inducing seizures at higher doses; venlafaxine and duloxetine, which inhibit both norepinephrine and serotonin reuptake and have a side effect profile similar to those of the SSRI agents except

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that they can increase blood pressure and have an uncomfortable discontinuation syndrome if stopped abruptly; and mirtazapine, which has noradrenergic and serotonergic pharmacologic properties, and is sedating and stimulates appetite.

All antidepressants must be taken for a minimum of 6–8 weeks at appropriate dosages before efficacy can be determined. To prevent relapse, they should be prescribed for a minimum of 6–12 months once the right dose and therapeutic response have been achieved.

Another effective treatment for severe depression is electroconvulsive therapy (ECT).[15,16] ECT may be the first-line treatment of choice if the patient cannot eat or is refusing to eat and is at risk for dehydration. It may be used as a second-line treatment after one or two antidepressant trials have failed to improve symptoms adequately. There is no age limit to ECT, although several medical conditions are relative contraindications that must be evaluated case by case. Those include brain tumor, recent myocardial infarction, coronary artery disease, hypertensive cardiovascular disease, bronchopulmonary disease, and venous thrombosis. The only absolute contraindication for ECT is increased intracranial pressure, since ECT causes a rise in cerebrospinal fluid pressure that may lead to herniation. Relapse of major depression after ECT is high, and therefore it must be followed by maintenance antidepressant treatment.

Bipolar disorder is a lifelong recurrent disorder characterized by one or more manic episodes. There is often at least one prior episode of major depression. Recurrence can take the form of either mania or depression. While most patients with bipolar disorder have their first episode of illness before age 50, late-onset mood disorder does occur.[17] Most patients with late-onset mania have had at least one episode of major depression, often 10–15 years earlier. There is a tendency for patients with late-onset bipolar disorder to have a lower incidence of positive family history of mood disorders. In addition, a number of studies of late-onset mania reveal a high rate of secondary mania, in which there is an association between onset of mania and known brain injury, especially affecting the right side of the brain, or another medical problem such as thyrotoxicosis or hypercortisolemia.

Patients having a manic episode usually have little need for sleep, are talkative, and may have loose associations in their speech. Hyperactivity, hypersexuality, overspending, and involvement in foolish or unwise endeavors are frequently seen. Patients usually have inflated self-esteem and an increased sense of well-being but may also be irritable and demanding to those around them. Frank, grandiose delusions may develop, such as believing oneself to be chosen by God for a special mission.

The mainstay of treatment for mania is lithium pharmacotherapy, sometimes in combination with low-dose antipsychotic medication. For patients who cannot tolerate lithium because of sensitivity to side effects or impaired renal function, divalproex sodium and carbamazepine are alternative mood-stabilizing agents. While many patients enjoy long periods of remission, it is typical for episodes of illness to become more frequent with age. In addition, complicated clinical conditions such as mixed episodes, in which symptoms of mania and depression coexist, and rapid cycling, in which four or more mood episodes occur within 12 months, may develop. Because of the high recurrence rate, patients with bipolar disorder often require lifelong pharmacologic treatment and regular psychiatric monitoring.

Chronic depression is a persistent depressive condition lasting 2 years or longer and marked by a persistently low mood more days than not and at least two of the following: appetite change, insomnia, low energy, low self-esteem, poor concentration or difficulty making decisions, and hopelessness. It may be a milder depressive disorder than major depression in severity of individual symptoms, but the chronicity of the depressive symptoms can be disabling and demoralizing to the patient and may contribute to lowering functional capacities.[18,19] In addition, some patients with persistent depressive disorder, or dysthymia, go on to develop a major depressive episode. In older people, dysthymic disorder often develops in the setting of physical disability, multiple medical problems, isolation, and loneliness. Many patients with a dysthymic disorder respond to treatment with an antidepressant. Supportive psychotherapy is a vital component of treatment, the goals being to increase social contacts and activity level and to improve self-esteem and outlook through an empathic therapeutic relationship.

Grief is not a mental disorder, and depressive symptoms are considered to be part of the normal bereavement process. While persons vary in their response to losing a loved one, there are common predictable phases to grieving.[20] The initial response, which lasts several days, is characterized by shock, disbelief, and emotional numbing. This is often followed by prominent emotions, including anger and frustration, that evolve into periods of fluctuating despair, mourning, and wishing to be with the deceased. During the first 3–6 months following the death of a loved one, insomnia is common, as are

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frequent episodes of tearfulness, anxiety, and a loss of interest or pleasure in activities once enjoyed. Usually the intensity of symptoms begins to remit after the first 6–12 months; strong feelings of loss and mourning continue for 1–2 years, longer for some people. In addition, intense emotional feelings tend to return on the anniversary of the loved one's death and birthday and at holiday times. Transient hallucinations or a sense of presence of the deceased are common early in grief and normal.

It is unclear at what point a bereaved person should be referred for professional help or counseling. The support of family, friends, and clergy is sufficient to help most bereaved persons through the grieving process. Widow and widower support groups can also be helpful in readjusting to life without a spouse and increasing social contacts. Often a physician may prescribe short-term medication for treatment of insomnia during the early days and weeks following the death of a loved one. When the bereaved person is overwhelmed by grief and unable to begin to return to usual activities or if grief is complicated by panic attacks, delusions, or suicidal thoughts, referral for psychiatric evaluation is indicated. Grief may trigger a full major depressive episode. While sadness, disruption of sleep, and loss of motivation and interest may be part of an uncomplicated grief syndrome, feelings of guilt, worthlessness, and hopelessness are not part of grief and should signal concern that a major depression has developed and should be treated as outlined earlier.

Suicide

Suicide is the third leading cause of death due to injury among persons over age 65, after unintentional falls and motor-vehicle accidents. Age-specific rates for suicide have consistently been higher among the elderly than for any other age group.[21] Of particular concern is data from the National Center for Health Statistics (NCHS) of the Centers for Disease Control and Prevention that indicates that after decades of declining rates, the period from 1980 to 1992 saw a marked increase in the rate of suicide among persons aged 65 and older.[22] Of persons 65 and older, men accounted for 81% of the suicides. Rates were higher for divorced or widowed men. Other risk factors include depression, alcoholism, chemical dependency, physical illness, and social isolation. Older persons make fewer attempts per completed suicide than other age groups and tend to use violent means of suicide. Indeed, firearms were the most common method of suicide by men over age 65 and the second most frequent means of suicide by women over age 65.[21]

While suicide cannot be predicted with complete accuracy, the potential for suicide must be considered seriously by all health providers who care for the elderly. Particular attention must be paid to patients who are despondent, overwhelmed by the burdens of physical illness or disability, lack social support, drink alcohol excessively, or have made previous suicide attempts. Clinicians must become comfortable asking their older patients about suicidal ideation and should not hesitate to seek psychiatric consultation for any patient who seems at high risk.

Personality Disorder

Personality is the set of enduring traits that make each person unique. Traits are universally shared characteristics on which persons differ. They include patterns of perceiving and relating to one's self and one's social environment. For example, all people can be rated on their tendency for tidiness. People vary widely in this tendency, but for each person a certain degree of tidiness or lack thereof is characteristic. Personality disorders are diagnosed when a person falls at the extreme end of a normal distribution on a set of traits that commonly occur together. Personality disorders reflect enduring, inflexible, and maladaptive patterns of experience and behavior. For example, *dependent personality disorder* is characterized by an excessive need to be taken care of by others, which leads to difficulty in making independent everyday decisions. These persons lack confidence in their own judgment or abilities to do things on their own, and they often go to excessive lengths to obtain reassurance or support. *Antisocial personality disorder* is associated with repeated illegal actions, impulsivity, frequent lies, consistent irresponsibility, lack of remorse, and lack of concern toward others. *Obsessive-compulsive personality disorder* is characterized by extreme perfectionism, rigidity, emotional inexpressiveness, excessive preoccupation with rules and details, and inflexibility. To satisfy a diagnosis of personality disorder, problems in these realms must be lifelong. Thus, in the elderly a diagnosis of a personality disorder must reflect a pattern of behavior that has been present throughout adulthood and has caused problems for the person throughout his or her life.

Personality disorders complicate the care of the medically ill. Persons with *narcissistic personality disorder*, for example, are more likely to clash with health professionals, sometimes to the detriment of their well-being. Conversely, patients with obsessive-compulsive personality disorder

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may underreport symptoms, have very high expectations of their physicians, be inflexible, be unable to make decisions, and have difficulty accepting the lack of clear guidelines that sometimes occurs in medical conditions.

The physician who is aware that a personality disorder is underlying a patient's problematic behavior can avoid or alleviate problems by considering the patient's predispositions. Patients with prominent obsessional traits often need detailed discussions of proposed procedures and an extensive and specific discussion regarding the steps that are to be taken, the order in which they are to be taken, and the implications of the most likely outcomes. While all patients need options and clear descriptions, patients with narcissistic features often do best when information is presented in a reassuring, calm tone, a concise description of alternatives, a direct acknowledgment of emotional distress ("I know this is upsetting, but let me present the alternatives before we discuss them"), and frequent short visits. Patients with dependent personality disorder have difficulty following through with recommendations on their own and do better if important persons in their social support network are part of the treatment process.

Psychotic Symptoms

Hallucinations (perceptions without a stimulus occurring in any of the five senses) and delusions (false ideas that are unshakable and persistent) occur in many medical and psychiatric disorders. The first step in their assessment is to determine whether a cognitive impairment (delirium or dementia) is present. The importance of this step is twofold. First, cognitive disorder is a common cause of hallucinations and delusions, and second, this recognition leads to the appropriate medical evaluation.

Hallucinations and delusions can also be caused by depression, schizophrenia, and delusional disorder. As previously noted, some grieving individuals report vivid visual or auditory experiences of the deceased person. These are normal. After a primary cognitive disorder has been ruled out, the next step is to assess for mood disorder. Self-deprecation, self-blame, hopelessness, loss of interest in usually enjoyed activities, somatic preoccupations, and complaints of sad mood all suggest the possibility that major depression is the cause of the psychotic symptoms.

Schizophrenia and *delusional disorder* are uncommon conditions, occurring in less than 1% of the population. They can present to medical practitioners with isolated somatic delusions (e.g., belief that someone is sending an electrical shock into the body or belief in a physical illness for which there is no evidence). By definition, a *delusional disorder* is characterized by a single delusion occurring in the absence of cognitive impairment, mood impairment, and other psychiatric symptoms. *Schizophrenia* is an illness in which symptoms are present for at least 6 months, hallucinations and social dilapidation are predominant, and mood disorder criteria are not met. While schizophrenia most commonly begins in early adulthood, it can begin in late life. Patients with late-onset schizophrenia frequently have paranoid delusions, social isolation, and hearing impairment.[23] Very-late-onset schizophrenia-like psychosis occurs in individuals who are 60 years and older, with women at higher risk compared to men.

Lifelong schizophrenia has an associated cognitive decline (reflected in its original name, dementia praecox) characterized by progressive loss of social, occupational, and interpersonal skills. The cognitive impairment of schizophrenia typically impairs frontal-executive abilities; that is, reasoning, abstraction, planning, and adaptation to changing circumstances. The decline is usually slow. Prominent decline in cognition or function over a 6–12-month period, new-onset language impairment such as paraphasic errors, new-onset apraxias (inability to do motor tasks with intact strength) or agnosias (not recognizing familiar people or places), or a noticeable decline in memory over 6–12 months should trigger an evaluation for a cause of cognitive decline other than schizophrenia. A dramatic decline in alertness or cognition should always trigger an evaluation for delirium.

Psychiatric symptoms may arise from toxic effects of prescribed medication, such as carbidopa with levodopa (Sinemet) or steroids. In addition, isolated visual hallucinations (i.e., not accompanied by delusions, cognitive impairment, or mood disorder) sometimes occur in patients with a wide variety of visual disorders, such as glaucoma, cataracts, and retinal degeneration.[24] Finally, hallucinations and delusions may develop in the course of several neurologic diseases such as dementia, Huntington's disease, and Parkinson's disease.

The treatment of psychotic symptoms depends, in part, on the diagnosis of the underlying disorder. If the patient is delirious, all attempts should be made to correct the underlying abnormality and to avoid pharmacotherapy unless there are clear indications. So-called neuroleptic, psychotropic, or antipsychotic drugs are the treatment of choice when protection from harm makes pharmacotherapy necessary. Antipsychotic drugs have been found to reduce psychotic symptoms in patients with very-late-onset schizophrenia, a group of individuals often undertreated.[25] Benzodiazepines should be avoided if possible, unless the

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source of delirium is withdrawal from alcohol. In dementia, reorientation and activity therapy should be tried first. Pharmacotherapy is appropriate when these symptoms increase the likelihood of harm to the patient or others or cause emotional distress to the patient. For mood disorder, several studies demonstrate that delusional major depression responds better to the combination of an antidepressant and neuroleptic than to an antidepressant alone. For the treatment of Parkinson disease psychosis, the Food and Drug Administration (FDA) has approved a selective serotonin receptor inverse agonist, pimavanserin, that lacks the dopamine receptor-blocking properties and in a few studies has been found to be partially effective.[26]

Psychiatric Treatment of Irreversible Cognitive Disorders

Dementia and delirium are discussed in detail in Chapters 11 and 12. Up to 60% of patients with dementia – 40% of patients with Alzheimer's – have psychotic symptoms sometime in the course of a dementing illness.[27,28] These noncognitive symptoms, which can interfere with the quality of life of the patient and caregiver, are often amenable to treatment. Nonpharmacologic, environmental therapy is most desirable because of the side effects of drugs, including a 60–100% increase in mortality. Nondrug treatments include providing a structured environment, stimulating the patient at an appropriate level, redirection, and providing the level of care that the person needs. When hallucinations and delusions interfere with function, become distressing to the patient, or are dangerous to others, cautious low-dosage neuroleptic therapy is appropriate.

Some 15–30% of patients with dementia also suffer from depression that interferes with function. Antidepressant drugs with low anticholinergic properties (e.g., nortriptyline, SSRIs) are recommended, although clinical trials have shown mixed results. Emotional support for both patient and caregiver is indicated in all cases. The physician should play an important role in educating the family, in managing specific behavioral and noncognitive symptoms, and in helping the families address their social, legal, and financial concerns.

Overview of Treatment Issues

Pharmacotherapy

While older patients can benefit from the same psychopharmacologic agents as younger ones, the clinician must be aware both of changes in physiology and pharmacokinetics with age and potential interactions with other medications. Prescribing psychotropic medication for older patients requires special considerations discussed in detail in other sources.[29] However, some important principles are outlined here.

Perhaps the most familiar axiom in prescribing for older adults is "start low and go slow." This means that for just about every medication, be it an anxiolytic, antipsychotic, or antidepressant, one should start at a low dose and titrate the dose up to a therapeutic dose slowly and gradually. A good rule of thumb is to allow at least 5 days between each dosage increase. This allows the patient to adjust to a new medication and to report any troublesome side effects before they become problematic.

Older patients are more sensitive to the anticholinergic effects of medication and therefore more likely than younger patients to develop delirium, constipation, urinary retention, dry mouth, and orthostatic hypotension. For these reasons, medications with the least anticholinergic effects are preferred when a choice of several agents is available.

Another important principle is to choose medications with shorter half-lives. Because of the changes in hepatic metabolism that occur with aging, the half-lives of most pharmacologic agents are prolonged in older people. This increases the likelihood that psychologically active metabolites will accumulate over time and cause toxicity. Obviously the problem is worsened if the original drug and/or its active metabolite or metabolites have long half-lives to begin with. Among benzodiazepines, for instance, lorazepam (half-life 16 hours) and oxazepam (half-life 8 hours) are better tolerated in older people than is diazepam (half-life 3–4 days). If a longer-acting benzodiazepine is required to manage severe anxiety or withdrawal from benzodiazepines, clonazepam (half-life 1–2 days) is useful.

Lithium carbonate deserves special mention, since it is nearly totally excreted in the kidneys. Because glomerular filtration rate and creatinine clearance decrease steadily with age, older patients are likely to develop lithium-induced tremor and delirium at low doses. Furthermore, recent studies seem to indicate that the therapeutic effects of lithium occur at lower blood levels in older patients than in younger ones. For all of these reasons, older patients require lower doses of lithium than younger patients, usually 150 mg daily to 300 mg twice a day.

In general, the lowest dose of antipsychotic medication needed to control symptoms should be prescribed. In addition to their extrapyramidal and anticholinergic side

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effects, neuroleptics are likely to cause tardive dyskinesia in the elderly.

For most antidepressants, on the other hand, patients do best if the medication is within the therapeutic range regardless of age. Low-dose antidepressant treatment is likely to be inadequate to treat a major depressive episode. Because of wide variations in older persons' hepatic metabolism, it is impossible to predict the dose of antidepressant needed to achieve a therapeutic level, but often it is the same as for much younger persons. It is important to give an antidepressant an adequate trial length (4–6 weeks minimum) at a therapeutic dose before deciding that the medication trial was a failure and changing to another antidepressant. Indeed, there is evidence that for some antidepressants, such as fluoxetine, a longer trial period (6–8 weeks) may be necessary to establish maximum therapeutic benefits. Furthermore, to prevent relapse it is important that full-strength antidepressant therapy continue for 6–12 months once a therapeutic response has begun. Long-term antidepressant therapy is indicated for patients with recurrent depression.

Finally, as with all medications, it is important to consider drug interactions. Fluoxetine, for example, increases serum levels of digoxin, warfarin, and other protein-bound drugs. Tricyclic antidepressants and neuroleptics have hypotensive effects that can compound the effects of antihypertensive medications. Nonsteroidal anti-inflammatory drugs and some diuretics and antihypertensives increase the plasma level of lithium and put an older person at risk for lithium toxicity. Thus, older patients must be carefully monitored while being treated with psychotropic medications to avoid both undertreatment and toxicity. Monoamine oxidase inhibitors should not be prescribed concomitantly with SSRIs or venlafaxine, and to avoid serotonin syndrome there should be a minimum 2–5-week washout period after one agent has been discontinued and the other type of antidepressant started. Serotonin syndrome is a serious, sometimes fatal, condition that is characterized by hyperthermia, rigidity, myoclonus, autonomic instability, and mental status changes.

Psychotherapy

Individual Psychotherapy

Contrary to many prevailing myths, older patients do benefit from psychotherapy in the treatment of a variety of disorders.[30] For patients with depression, anxiety, and bereavement, psychotherapy is an important part of treatment even when pharmacotherapy is indicated. Through psychotherapy, older people can improve significantly in self-esteem, self-awareness, adaptation, and personal satisfaction. No one psychotherapeutic method works best with older people. We recommend a pluralistic approach that emphasizes life review and focuses on specific issues of concern. Many persons benefit from a focus on the development of problem-solving skills. Some patients benefit from a return to an active, creative life. Also, patients with anxiety disorders and phobias may benefit from a more cognitive-behavioral approach that stresses the importance of positive problem-solving and teaches relaxation techniques.

Marital Therapy

Marital or couples therapy is helpful for older people in several circumstances. Retirement and late-life illnesses can dramatically alter the dynamics of a marriage or long-term partnership. Spouses who were used to busy but relatively independent work lives may find it an adjustment to be home together most of the time. Roles may change as one spouse does more or less of the cooking, shopping, and housekeeping. If one spouse is unable to drive because of health problems, this can put limitations on the lifestyles of both partners. Retirement also means living on a fixed income for most people, and these new financial constraints may pose an additional burden. In short, the reality of living "the golden years" often does not meet the expectations for this time of life. This may result in disappointment or resentment, especially if one spouse blames the other for preventing the fulfillment of the retirement dream. In addition, problems can develop between widowed or divorced elders involved in new relationships. Issues such as whether to live together or marry, how to combine finances, and how to deal with each other's adult children can put a strain on the relationship. While couples' issues may be the presenting focus for treatment, usually they are not. Rather, these issues may emerge as the patient is beginning treatment for depression or anxiety. Short-term marital therapy can be very useful in defusing stressful situations, improving communication between partners, and fostering a more healthful adaptation to the couples' changing way of life.

Family Assessment

Often other family issues come to light during the course of assessment and treatment of the older patient. For some, decreased functional abilities or illness raise dependence on adult children. This may necessitate moving in with adult

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children or moving closer geographically. When an elderly person develops dementia and/or other disabling medical illness, the spouse or adult child may become a primary caregiver and have to assume responsibilities for the impaired person's personal and financial care. Alternatively, older patients may find that their grown children need them in new or different ways, e.g., because of illness or divorce on the part of the adult children.

These and other situations can produce family conflict and stress. It is very helpful to meet with all involved family members at least once to assess how various family members relate to one another, solve problems, deal with their changing family dynamics, and address the needs of the impaired elder person. These meetings can also be useful for teaching the family about the impaired older person's medications and illness, for mobilizing family and community resources, and for identifying others in the family who need support or counseling.

Barriers to Treatment

There are many reasons why older people often do not get the psychological treatment they need.[31] One common reason is that older people themselves are reluctant to see a psychiatrist because of embarrassment and negative attitudes. While education is slowly changing society's outlook on mental illness and mental health care, older people who grew up in the Depression may still believe one should solve one's own problems and "pick oneself up by the boot straps." To such persons, seeking help for psychologic problems is viewed as a sign of personal weakness. Other contributors to elders not receiving care for emotional problems include the negative attitude of their physician; the focus by patient, family, or physician on medical issues; and lack of transportation. These issues are best overcome by discussing them openly and reviewing the reasons a person is reluctant to seek help.

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Chapter 21

Substance Use Disorder

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Introduction

In the year 2000, there were just over 35 million adults in the USA aged 65 and over, making up about 12% of the US population. By 2018, the population of older adults rose to more than 52 million people and comprised more than 16% of the US population.[1] Compared to preceding generations of older adults, the large, aging baby boomer generation has higher rates of alcohol and substance use, and the prevalence of substance use disorder (SUD) in older adults is currently increasing. According to the Substance Abuse and Mental Health Services Administration (SAMHSA) of the US Department of Health and Human Services, the rates of lifetime use, past year use, and past month use of alcohol and all categories of illicit psychoactive substances are increasing among older adults.[2] Even though SUDs (which include alcohol use disorder and use disorders of the other psychoactive substances) present at reduced rates among seniors compared to the general population, the incidence and prevalence of these diagnoses are increasing.[1] Thus, clinicians must remain vigilant in screening, diagnosing, and treating their patients for alcohol and SUDs as they age.

Definitions and Epidemiology

Alcohol and substance use disorders among older adults are an escalating health problem in the USA. According to reports, approximately 4–6 million older adults in the USA would benefit from SUD treatment in 2020, a substantial increase from the 1.7 million older Americans who obtained SUD treatment in 2000–2001.[3] However, because of the social stigma surrounding SUDs, this could be an underestimation of the affected population.[4] As a result, the full extent of this health problem remains unknown, and those affected by it are likely undertreated.

Much of the problematic substance use in older adults is due to unsafe combinations or dosages of prescription medications, taking medications together with alcohol, or obtaining medications from several providers in order to treat common complaints among older adults, such as anxiety, pain, and insomnia.[5] It is estimated that one in four older adults use psychoactive medications that have potential for harmful use or could be involved in the development of a SUD.[1] Additionally, improper use of prescription medications affects up to 23.5% of older adults.[3]

Using a dataset with >12,000 respondents age 65 and older, researchers found the prevalence of SUD to be 6.1% in the 55–64-year-old group, 2.6% in the 65–74-year-old group, 1.7% in the 75–84-year-old group, and 0.15% in the 85+-year-old group, with men having a prevalence of at least four times that of women in all the age groups.[6] There are several established risk factors for the development of SUD in later life, although most of these risk factors are based on studies of alcohol use. The risk factors associated with SUD in older adults are:[7]

- Demographic and general health risk factors
  - Male sex (for alcohol), female sex (for prescription drugs)
  - European ancestry
  - Chronic pain
  - Physical disabilities or reduced mobility
  - Transitions in care/living situations
  - Poor health status
  - Multiple chronic physical illnesses
  - Polypharmacy
- Mental health risk factors
  - Avoidance coping style
  - History of problems related to alcohol
  - Previous diagnosis of SUD
  - Previous or concurrent psychiatric illness
- Social risk factors
  - Affluence
  - Bereavement
  - Unexpected or forced retirement
  - Social isolation (living alone or with non-spouse individuals)

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Alcohol

The most commonly used substance among the population age 65 or older is alcohol. Data demonstrates that between 2001 and 2013, there was a large increase in both alcohol use and alcohol use disorder (AUD) among older Americans. During that 12-year time period, the number of individuals who drank any alcohol within the past year increased from 45% to 55%. Furthermore, the prevalence of AUD increased from 1.5% to 3.1% in the total population studied, with the rate of AUD rising from 3.2% to 5.6% among older adults after excluding those who never drank alcohol.[8]

For individuals aged 65 years and older, guidelines from the National Institute on Alcohol Abuse and Alcoholism (NIAAA) recommend drinking no more than seven standard drinks (i.e., 12 oz beer, a 4- to 5-oz glass of wine, 1.5 oz 80-proof liquor) per week and no more than three drinks on any day.[9] *Binge drinking* is defined as consuming five or more drinks on the same occasion for men and four or more drinks on the same occasion for women. According to the US National Survey on Drug Use and Health from 2015 to 2017, 10.6% of Americans aged 65 years or older were estimated to be binge drinkers, which is an increased rate compared to prior surveys.[10] *Hazardous* or *risky drinking* is generally defined as drinking alcohol of sufficient quantity or frequency to be at risk for future alcohol-related harm while not meeting formal criteria of AUD. There are many formal definitions of *hazardous* or *risky drinking* in the 65 years and older population, including the definition used in one large observational study – a score of 8 or greater on the Alcohol Use Disorders Identification Test (AUDIT), a screening test, or the consumption of 16 or more standard drinks per week for men and 12 or more standard drinks for women.[11]

Benzodiazepines

Benzodiazepine use is common in the elderly, with rates of new and continuing benzodiazepine use being higher in older adults than all other age groups in the United States.[12] A study from 2015 found that the prevalence of benzodiazepine use in the United States increases with age and that 8.7% of adults aged 65–80, and even higher rates among adults aged 80+ years, were prescribed benzodiazepines in the previous year.[13] Of these older patients prescribed benzodiazepines, approximately 31% of the prescriptions were for "long-term use" (≥120 days) and less than 5% of the prescriptions for benzodiazepines were from a psychiatrist. The greatest source of prescriptions for benzodiazepines in the elderly population is from the individual's primary care provider, and less than 1% of the visits for a new benzodiazepine prescription resulted in a referral for psychotherapy.[14] These findings are concerning, and suggest that benzodiazepines may continue to be prescribed without clear indication of an ongoing psychiatric need.

Use of benzodiazepines is of particular concern in elderly patients because of its association with falls, fractures, motor-vehicle accidents, cognitive impairment, and dementia.[14] As such, the 2019 American Geriatrics Society (AGS) Beers Criteria for Potentially Inappropriate Medication Use in Older Adults gives a strong recommendation to avoid prescribing benzodiazepines for older patients in almost all circumstances, including insomnia and agitation.[15] Older adults have increased sensitivity to benzodiazepines and decreased metabolism of the longer-acting agents. However, the AGS reports that benzodiazepine use may be appropriate treatment for seizure disorders, generalized anxiety disorder, periprocedural anesthesia, and benzodiazepine or alcohol withdrawal treatment.

Opioids

Opioid use disorder (OUD) has a troublingly high prevalence within the older adult population in the USA. A 2017 report from the Substance Abuse and Mental Health Services Administration (SAMHSA) indicated that the second most common reason for adults aged 65 years or older to seek SUD treatment was for OUD, second only to seeking treatment for AUD.[16] Older women are particularly prone to risky opioid use, with the US Food and Drug Administration Adverse Events Reporting System (AERS) showing that 10.4% of adverse drug events related to opioids occurred in women aged 65 years and older.[17]

While risky opioid use among older adults has certainly been a longstanding issue, the scale of the problem has compounded within recent years. Hospitalizations for opioid-related illnesses among older adults increased 54.4% between 2010 and 2015.[16] Additionally, Centers for Medicare & Medicaid Services (CMS) reported in January 2017 that Medicare recipients (aged 65+ or disabled) had among the highest and fastest-growing rates of OUD, at 6 per 1,000 individuals.[18]

The increasing prevalence of OUD in older adults appears to be largely due to the number of opioid prescriptions written for older adults by their providers. Of adults aged 65 years or older who misused prescription opioid medications, 70% were prescribed the

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medication by one or more of their providers.[16] Older adults frequently begin using opioid medications because of painful chronic conditions. As the length of time increases that older adults are treated with opioids, their risk of development of an OUD increases. Data from the National Health and Nutrition Examination Survey (NHANES) found that between 2007 and 2012, 7.9% of US adults aged 60 years or older used opioids for analgesia each month, including 8.6% of women and 6.9% of men.[19] Furthermore, NHANES showed that individuals aged 65 and older made up 25.4% of long-term (90+ days) users of opioids.[20] Thus, the high rate of long-term opioid prescriptions in older adults helps to explain the prevalence of OUD within this population. From 1995 to 2010, prescriptions for opioid medications to adults aged 65 years and older increased almost ninefold.[21] This increasing rate of prescriptions led to a growth of OUD among older adults in recent years. However, there exist measures that have been shown to reduce the overuse of opioids within this population. Prescription drug monitoring programs (PDMPs) result in a reduction in opioid use, as measured by monthly total number of prescriptions and monthly total opioid volume, in Medicare beneficiaries age 65 or older.[22]

Screening

The goal of substance use screening is to identify individuals who have, or are at risk of developing, SUDs. Valid screening tests are used to detect patients who need further diagnostic evaluations to identify those with SUDs. There are several validated instruments used to screen patients for SUD in a primary care setting. Most of these screening tools are specifically geared toward screening for AUD, but they may be adapted to screen for drug use disorders as well. However, few of these tools have been developed or validated for an older population. Screening older adults for alcohol or other SUDs is often omitted from their periodic and routine health examinations,[23,24] limiting the opportunity to detect SUD among geriatric patients and to offer counseling and treatment as indicated.

Screening tools most commonly used with older adults include:

- Single-Item Screening Questions (SISQs)
- CAGE Questionnaire
- Alcohol Use Disorders Identification Test (AUDIT)
- Alcohol, Smoking and Substance Involvement Screening Test (ASSIST)
- Short Michigan Alcoholism Screening Test-Geriatric version (S-MAST-G).

Each of these tests has advantages and disadvantages to their routine use, including the sensitivity and specificity of the screening instrument, the ease of administration, and patient acceptance.[25] See Table 21.2 for summary information on these tests.

Below is an overview of each of these screening tests:

Single-Item Screening Questions (SISQs)[26]

- Screening for alcohol use disorder: "How many times in the past year have you had X or more drinks in a day?" (*X=5 for men, X=4 for women*)
- Screening for other substance use disorder: "How many times in the past year have you used an illegal drug or used a prescription medication for nonmedical reasons (for example, because of the experience or feeling it caused)?"

Any positive response is considered a positive result on this screening test. This screening test is quite easy to use, it can be provider- or self-administered by patients, and it can be completed quickly during a busy clinic session. This test, however, is not validated for individuals >65 years old.

Test operating characteristics[26]

- Sensitivity of 87% and specificity of 74% for alcohol use disorder
- Sensitivity of 85% and specificity of 87% for drug use disorder.

CAGE questionnaire[27]

- Have you ever felt you should cut down on your drinking?
- Have people annoyed you by criticizing your drinking?
- Have you ever felt bad or guilty about your drinking?
- Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover (eye-opener)?

Scores are assigned based on the number of positive responses. This screening test is quite easy to use, it can be provider- or self-administered, and it can be accomplished quickly during a busy clinic session. However, it is less likely to detect problematic drinking in an older adult due to binge drinking. The operating characteristics below are derived from a single site study of individuals >60 years old. This screening test has also been validated in a wide variety of other patient populations.

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Test operating characteristics[27]

- Sensitivity of 86% and specificity of 78% using a cutoff score of one
- Sensitivity of 70% and specificity of 91% for a cutoff of two.

Alcohol, Smoking, and Substance Involvement Screening Test (ASSIST)[28]

The Alcohol, Smoking, and Substance Involvement Screening Test (ASSIST) is a screening instrument developed and validated by the World Health Organization (WHO) to be useful in a variety of cultural settings, and to be administered by primary health-care professionals. The test is utilized to screen for many substances including tobacco, alcohol, and drug use. The test consists of an interview, which can take quite some time depending on the number of substances used by the patient within the past 3 months. The ASSIST, although commonly used in both research and clinical practice, has not been validated in older adults, although some authors consider it acceptable and valid for use in elderly patients in a general health-care setting.[29] The ASSIST consists of eight general questions for nine types of substance known to have the potential for harm or the development of a use disorder.[28] The ASSIST is administered to screen for use of:

- tobacco
- alcohol
- cannabis
- cocaine
- amphetamines/stimulants
- inhalants
- sedatives
- hallucinogens
- opioids.

For each substance ever used in a patient's lifetime, the following questions are asked, with patients choosing responses ranging from "never" to "daily" and scored accordingly.

For each substance ever used in a patient's lifetime, specific questions are asked about how often during the past 3 months the individual has used the substance, has had a strong desire or urge to use, has had health, social, legal, or financial problem due to use, has failed to do what was expected due to use, has had a friend or relative express concern about drug use, and has tried unsuccessfully to cut down on use.

A helpful element of ASSIST is the guidance provided for interpretation of the test results. Each response is given a specific score, and the sum of the scores for each drug/substance corresponds to three recommended outcomes: no intervention, brief intervention recommended, intensive treatment recommended.[28] These recommended options, and their focus on physician interventions, closely correspond to the Screening, Brief Intervention, and Referral to Treatment (SBIRT) model (see section on SBIRT) of treatment.

ASSIST is time-consuming and requires a clinician or other trained professional to administer. Additionally, ASSIST consists of 8–57 questions and utilizes a complicated scoring system. There are no reported operating characteristics of ASSIST in an elderly population, making its routine use of uncertain benefit. ASSIST may, however, prove useful in elderly patients using multiple substances, or to provide a recommended course of action (no intervention, brief intervention recommended, intensive treatment recommended) when uncertainty persists or patients express an interest in objective evidence of the need for treatment.

Operating characteristics of ASSIST in a general population[30]

- Sensitivities of 54–97% depending on which substance use being screened
- Specificities of 50–96% depending on which substance use being screened.

Alcohol Use Disorders Identification Test (AUDIT) and its several varieties[31]

- AUDIT is a 10-question screening test with the first three questions addressing alcohol consumption, and the remaining seven questions asking about negative consequences of drinking alcohol.
- The AUDIT-C consists of the first three questions of the AUDIT.
- The AUDIT-QF includes the first two questions, which address quantity consumed and frequency of drinking.
- The AUDIT-3 consists of only the third question from the original AUDIT, regarding binge drinking.

AUDIT questions are based upon

1. Frequency of having a drink containing alcohol.
2. Quantity of drinks containing alcohol on a typical day when patient is drinking.
3. Frequency of having five or more drinks on one occasion.
4. Inability to stop drinking once the patient had started.

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5. Frequency of failing to do what was normally expected of the patient because of drinking.
6. Frequency of needing a first drink in the morning to get going after a heavy drinking session.
7. Frequency of feelings of guilt or remorse after drinking.
8. Frequency of being unable to remember what happened the night before because of drinking.
9. History of injuries as a result of drinking.
10. Whether a relative, a friend, a doctor, or another health worker has been concerned about the patient's drinking or suggested the patient cut down.

Scores are assigned based on the specific answers to the questions, with each answer being assigned scores between 0 and 4.

The AUDIT, like the ASSIST, was developed and validated by the WHO to be useful in a variety of cultural settings, and to be administered by primary health-care professionals. The AUDIT can be cumbersome to administer and score, but the screening tests derived from the AUDIT (AUDIT-C; -QF, -3) are easier and less time-consuming to use. Unfortunately, the AUDIT-QF and the AUDIT-3 lacked adequate sensitivity and specificity in elderly populations with all cutoffs evaluated.[31] The original AUDIT and the AUDIT-C also lacked sensitivity and specificity to be adequate screening tests for AUD,[27] but some state that the operating characteristics are helpful in screening for hazardous or risky drinking among the elderly if unconventional cutoff scorers are used.[31]

Test operating characteristics[31]

- AUDIT: ≥5 is 86% sensitive and 87% specific for hazardous or risky drinking among the elderly
- AUDIT-C: ≥4 is 95% sensitive and 80% specific for hazardous or risky drinking among the elderly.

Short Michigan Alcoholism Screening Test-Geriatric (S-MAST-G)[32,33]

The Short Michigan Alcoholism Screening Test-Geriatric Version (S-MAST-G) is a validated short form of the 24-question MAST-G, containing only 10 items. S-MAST-G was developed to account for patterns of alcohol misuse, which can be different in older adults compared with younger adults. The focus of the individual questions is:

1. Potential to underestimate how much you actually drink?
2. Skipping a meal because you didn't feel hungry after drinking?
3. Drinking to help decrease shakiness or tremors?
4. Does alcohol ever make it hard for you to remember parts of the day or night?
5. Drinking to relax or calm your nerves?
6. Drinking to take your mind off your problems?
7. Increasing your drinking after experiencing a loss in your life?
8. Having a doctor or nurse be worried or concerned about your drinking?
9. Making rules to manage your drinking?
10. Drinking to help with loneliness?

≥2 "yes" answers is considered a positive screen on the S-MAST-G

The S-MAST-G is specifically designed for use in older patients. It is not as brief as some of the other tests (SISQ, CAGE), but it may be administered by staff in a busy clinic setting. It is highly sensitive and specific for AUD in an older population.

Test operating characteristics[33]

- Sensitivity of 85% for AUD among an elderly population
- Specificity of 97% for AUD among an elderly population.

In summary, there are several screening instruments that can be used in the process of screening older adults for at-risk behaviors or use disorders concerning alcohol or drugs. Although all the tests cited here are designed to screen for alcohol use, only the SISQ and ASSIST are also validated screening tools for drug use. However, the AUDIT and CAGE screening tools have been studied in outpatient substance abuse treatment populations, and this work supports their use as substance abuse screening instruments in primary care populations. The decision of which test to use among the screening tools presented is not nearly as vital as the decision to screen older patients for SUDs. At this time, too many older patients are never screened for these important conditions.[34] Overall, increased screening and earlier detection is an important means of improving safety for older persons with SUDs.[35]

Screening, Brief Intervention, and Referral to Treatment (SBIRT)

There is increasing interest in the SBIRT paradigm to identify patients with risky alcohol or drug use, offer brief counseling to those who have (or are at risk of developing) SUDs, and refer to treatment patients who

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require more intensive care or fail to respond adequately to brief interventions. An SBIRT program was implemented and studied in a project called Florida BRITE (BRief Intervention and Treatment of Elders). The study lasted 5 years and enrolled 85,001 individuals aged 55 years and older. Of those individuals enrolled in Florida BRITE, 77% screened negative for SUD or at-risk drug/alcohol use, 19% screened positive and received brief intervention, and 4% screened positive and were referred to specialty treatment.[36]

The patients in Florida BRITE were initially screened for alcohol use with questions from the National Institute on Alcohol Abuse and Alcoholism's (NIAAA's) clinician's guide, and other questions concerning drug use.

1. On average, how many days a week do you drink alcohol?
2. On a typical day when you drink, how many drinks do you have?
3. What is the maximum number of drinks you had on any given day in the past month?

According to the NIAAA scoring criteria, responses to the first two questions are multiplied (days/week × drinks/day). A score of 7 or more indicated a positive alcohol screen. A positive screen was also indicated if the maximum number of drinks per day exceeded three.[36]

For drug use, the following initial screen was used:

1. In the last year, have you tried to cut down on the drugs or medication that you use?
2. In the last year, have you used prescription or other drugs more than you meant to?

A positive response to either question was considered a positive screen for drugs. Any person with an initial screen scored as positive was then administered a more detailed screening instrument: Alcohol, Smoking, and Substance Involvement Screening Test (ASSIST).

Following a positive screening test using ASSIST or another detailed screening instrument, patients in Florida BRITE received a brief intervention to lessen the potential harm of continued at-risk alcohol or drug use. Only patients who likely had a high risk of short-term harm were referred to treatment at this point in the SBIRT process.

The Substance Abuse Prevention Older Americans Technical Assistance Center and the Substance Abuse Mental Health Services Administration (SAMHSA) recommend using the FRAMES model in conducting brief interventions:[37]

- **F**eedback is given to the individual about risk.
- **R**esponsibility for change is placed with the individual receiving the intervention.
- **A**dvice for changing behavior is given by the clinician.
- **M**enu of alternative options for change is offered.
- **E**mpathic and motivational style is used by the intervener.
- **S**elf-efficacy or empowerment is promoted.

In a clinical setting, a brief intervention for a patient with risky or problematic drinking habits includes direct comments and personalized feedback about the patient's alcohol or drug use and recommendations that the patient reduce or stop drinking or using drugs. This should be followed up within 1–2 weeks with a brief appointment or telephone call to measure progress. Sometimes, however, a more intensive conversation is warranted. In this case, the brief intervention should take approximately 30–45 minutes spaced over several sessions and include the elements below.[37]

1. Identification of future goals for health, work, activities, hobbies, relationships, and finances
2. Summary of health habits like drinking/drug use, exercise, nutrition, safe sex, etc.
3. Discussion of standard drinks (e.g., one standard drink = 12 oz. of beer or ale; 1.5 oz. of distilled spirits; 4–5 oz. of wine; 4 oz. of sherry or liqueur)
4. Discussion of the norms for alcohol/drug use in the population and where the older adult's use fits into the population norms for his/her age group
5. Potential consequences of at-risk and problem drinking and drug use
6. Reasons to quit or cut down on use
7. Negotiated agreement for alcohol and/or drugs
8. Coping with risky situations, like social isolation, boredom, pain, and negative family interactions
9. Summary of the session, with a plan for a follow-up appointment within 6 weeks.

This intervention should be conducted in a supportive and nonconfrontational manner to aid in its efficacy.[38] The use of the term "drugs" in the brief intervention may not align with the clinical situation or the patient's view of their behavior. As such, the term "psychoactive medication" or another appropriate term may be substituted for "drug." Finally, "Referral to Treatment," the last element of SBIRT, denotes an ability to provide access to specialty SUD assessment and care, if needed by a patient. It would be beneficial to refer patients to counseling or treatment facilities that have experience in caring for older adults in

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need of specialty assessment and care, if such facilities can be identified.

Diagnosis

Most clinicians and researchers use the *Diagnostic and Statistical Manual of Mental Disorders*, 5th edition (DSM-5) as the reference standard for classifying and diagnosing SUDs.[39] Unlike the earlier DSM-4, which categorized SUDs into two groups (abuse and dependence), the DSM-5 combined the criteria for substance abuse and substance dependence, added the symptom of craving, removed the symptom of legal problems, and formed the new mental health diagnostic category of Substance Use Disorders.[40] The diagnosis of SUD is made when an individual meets two or more of the 11 criteria within a 12-month period (Table 21.1). The severity of the SUD can be further classified depending on the number of symptoms present over the past year: mild (presence of two to three symptoms); moderate (presence of four to five symptoms); and severe (presence of six or more symptoms). Many less technical definitions of SUDs are available from various medical organizations. These definitions usually classify SUD as a brain disease presenting with a loss of control over use of the substance and continued use of the substance despite the presence of negative consequences.[41]

Diagnosing SUD in elderly patients, based on the DSM-5 criteria, may be challenging, as illustrated in Table 21.1. This difficulty arises because older adults may have coexisting cognitive impairment, experience more social isolation, or engage in a more limited set of social and recreational activities and may be likely to be retired from regular employment. Also, older adults can experience harm from substance use at lower amounts than used by younger people, and without escalating use of substance. Together, these factors make it challenging to assess for loss of personal control over substance use or the presence of negative consequences from substance use.[42] A frequent challenge for clinicians caring for older adults is distinguishing among the diagnoses of alcohol or drug-related neurological sequelae, dementia, and other forms of brain dysfunction. These conditions all result in a decrease of cognitive abilities, and may cause similar deficits on neuropsychiatric testing and even have similar appearances on brain imaging.[38] Frequently, substantial abstinence from all substances combined with serial examinations and neuropsychological assessment of patients is required to differentiate among the various causes of impairment in brain functioning.[43]

Table 21.1 Diagnosis of substance use disorder in elderly patients

DSM-5 criteria for SUD*	Considerations for older adults
A substance is often taken in larger amounts or over a longer period than was intended.	Cognitive impairment can prevent adequate self-monitoring. Substances themselves may more greatly impair cognition among older adults than younger adults.
There is a persistent desire or unsuccessful efforts to cut down or control substance use.	It is the same as the general adult population.
A great deal of time is spent in activities necessary to obtain the substance, use the substance, or recover from its effects.	Consequences from substance use can occur from using relatively small amounts.
There is craving or a strong desire to use the substance.	It is the same as the general adult population. Older adults with entrenched habits may not recognize cravings in the same way as the general adult population.
There is recurrent substance use resulting in a failure to fulfill major role obligations at work, school, or at home.	Role obligations may not exist for older adults in the same way as for younger adults because of life-stage transitions, such as retirement. The role obligations more common in late life are caregiving for an ill spouse or family member, such as a grandchild.
There is continued substance use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance.	Older adults may not realize the problems they experience are from substance use.
Important social, occupational, or recreational activities are given up or reduced because of substance use.	Older adults may engage in fewer activities regardless of substance use, making it difficult to detect.

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Table 21.1 (cont.)

DSM-5 criteria for SUD*	Considerations for older adults
There is recurrent substance use in situations in which it is physically hazardous.	Older adults may not identify or understand that their use is hazardous, especially when using substances in smaller amounts.
Substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance.	Older adults may not realize the problems they experience are from substance use.
Tolerance is developed, as defined by either of the following: 1) A need for markedly increased amounts of the substance to achieve intoxication or the desired effect 2) A markedly diminished effect with continued use of the same amount of the substance	Because of the increased sensitivity to substances as they age, older adults will seem to have lowered rather than increased tolerance.
Withdrawal, as manifested by either of the following: 1) The characteristic withdrawal syndrome for the substance 2) The substance or a close relative is taken to relieve or avoid withdrawal symptoms	Withdrawal symptoms can manifest in ways that are more "subtle and protracted." Late-onset substance users may not develop physiologic dependence; or non-problem users of medications, such as benzodiazepines, may develop physiologic dependence.

* SUD is defined as a medical disorder in which two or more of the aforementioned listed symptoms are occurring in the last 12 months.

Adapted from Barry KL, Blow FC, Oslin DW. Substance abuse in older adults: review and recommendations for education and practice in medical settings. *Subst Abus.* 2002; 23(Suppl. 3):105–131; and data from American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*, 5th edition. Arlington, VA: American Psychiatric Publishing, 2013, p. 491.

Treatment

The goals of SUD treatment in older adults are to improve overall health, prevent cognitive impairment, repair or maintain family, professional, and social relationships, and improve overall quality of life. Individuals aged 65 years and over are increasingly entering treatment for SUD, especially AUD, and are referred for this treatment from a variety of sources. Roughly one third of older adults in SUD treatment initiate the care on their own (self-referral), approximately one third of patients are referred by a physician, and about one third are referred via a legal process for driving under the influence of alcohol (DUI).[35] This differs from a younger population (age 30–49 years), who are most likely referred for SUD treatment by the criminal justice system for non-DUI offenses. Another notable feature among older people in treatment (age 65–74 years) is their likelihood of being married (almost one half), which is almost double the rate of the younger cohort, aged 30–49 years. Fortuitously, older age (age 65–74 years) is associated with a greater rate of treatment success and better long-term outcomes compared to younger adults (age 30–49 years).[35,44] Even though a large majority of older individuals seeking treatment for SUD do so because of their alcohol use, a growing number of admissions are being seen for cocaine, cannabis, and opioids.[4]

Alcohol Withdrawal

Although alcohol withdrawal is treated similarly in older and younger patients, there are important considerations unique to the care of older individuals. As in younger patients, older patients in alcohol withdrawal usually exhibit two or more of the following signs and symptoms: anxiety, tremor, sleep disturbances, anorexia, vivid dreams, headache, nausea, tachycardia, hyperactive reflexes, sweating, elevated blood pressure, and hyperthermia. However, because older adults are more likely to have comorbid illnesses and may have less physiologic reserve, they are at higher risk of complications related to alcohol withdrawal. For instance, tachycardia and tachyarrhythmias are more likely to cause myocardial ischemia and heart failure in older adults, and older adults may be less able to tolerate the nausea and anorexia of withdrawal and be more likely to develop dehydration, further exacerbating their withdrawal. Additionally, older adults with alcohol withdrawal are more likely to

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Table 21.2 Screening tests for at-risk or substance/alcohol use disorder in older adults

	Assesses amount used	Assesses symptoms	Number of items	Sensitivity%/Specificity%
SISQ	++		1	87/74 (alcohol) 85/87 (drugs/ medication)
CAGE		++	4	86/78
ASSIST	++	++	8–57	54–97/50–96
AUDIT	++		10	86/87
AUDIT-C	++		3	94/80
S-MAST-G		++	10	75–85/69–97

Adapted from Kuerbis A, Sacco P, Moore A, et al. *Alcohol and Aging: Clinical and Public Health Perspectives.* Springer International Publishing. 2017; 1–283. doi: 10.1007/978-3-319-47233-1.

experience delirium and falls than younger patients. As such, older adults with alcohol withdrawal should be closely supervised while undergoing detoxification, and hospitalization is generally recommended.[45]

As in younger patients, the use of benzodiazepines remains the mainstay of therapy for alcohol withdrawal. However, the accumulation of active metabolites of benzodiazepines, especially in older patients or patients with liver disease, may occur and complicate the treatment of older patients by causing oversedation, respiratory depression, or delirium. These risks may be lessened by using symptom-triggered treatment rather than a fixed-dose schedule of benzodiazepines, with symptoms monitored using a validated alcohol withdrawal severity scoring instrument, like the CIWA-Ar.[46]

Treatment and Medication Management of Alcohol Use Disorder

Despite studies demonstrating efficacy for psychosocial treatments for AUD,[47] only a minority of patients engage in these activities. The most common sources of treatment include involvement in cost-free self-help groups, like 12-Step programs (i.e., Alcoholics Anonymous) and cognitive-behavioral therapy-based programs like SMART Recovery (Self-Management and Recovery Training). Additionally, multiple other types of treatment are available from medical and nonmedical practitioners, including abstinence-based group and individual counseling.

Four medications are approved by the US Food and Drug Administration (FDA) for the treatment of alcohol use disorder: disulfiram, oral naltrexone, long-acting injectable naltrexone, and acamprosate. Non-FDA-approved medications used to treat AUD include nalmefene, baclofen, gabapentin, and topiramate.

Disulfiram is indicated to sustain abstinence, but not to reduce drinking, because of its interactions with alcohol, and is most efficacious when medication adherence is supervised.[48] Disulfiram works by inducing flushing, headache, and nausea with alcohol consumption, causing drinking alcohol to be quite an unpleasant experience. There is no specific data on the use of disulfiram in older patients, but disulfiram may be contraindicated in patients with a history of diabetes, hypothyroidism, seizures, cerebrovascular disease, severe coronary artery disease, and peripheral neuropathy, all of which are common in older populations.

Naltrexone is indicated for the treatment of AUD, either as a daily oral tablet or a monthly intramuscular injection. Its mode of reducing alcohol consumption is not completely understood, but on a cellular level, naltrexone acts as an opioid receptor antagonist via competitive inhibition. Naltrexone has been shown to mildly reduce the probability of a return to drinking and to reduce the number of days with binge drinking. Caution must be exercised when initiating naltrexone, as it may cause precipitated opioid withdrawal in patients taking chronic opioids.[49] A small randomized clinical trial comparing disulfiram and oral naltrexone in elderly patients with AUD showed some differences between the two treatment groups. Disulfiram was found to be superior to naltrexone in preventing and delaying

relapses of alcohol use, but naltrexone was superior in preventing cravings for alcohol.[50]

Acamprosate has been shown to reduce the risk of drinking among abstinent patients but not reduce binge drinking, despite its association with a reduction in craving for alcohol.[51] Acamprosate's mechanism of action is likely through its modulation of central NMDA and GABA receptors. It must be taken three times daily to be effective, but has a very low risk of toxicity or side effects beyond diarrhea, so its use in elderly patients is considered safe.

Benzodiazepine Withdrawal

Symptoms of benzodiazepine withdrawal are similar to symptoms of alcohol withdrawal and commonly include tremors, confusion, anxiety, and insomnia. Occasionally more severe symptoms including large rises in blood pressure as well as seizures or delirium occur. Many of the relatively mild symptoms of benzodiazepine withdrawal occur less frequently in the elderly compared to younger patients, as plasma levels of benzodiazepines decrease at a slower rate than in younger patients.[52] This may explain why older patients tolerate gradual benzodiazepine taper better than younger patients.[53] The exception is delirium, which is more likely to occur in elderly patients. Additionally, as would be expected, discontinuation of benzodiazepines with a prolonged half-life are less likely to result in withdrawal symptoms than medications with a short half-life.

Benzodiazepine withdrawal, like alcohol withdrawal, can be quite dangerous and even result in death. As a result, proper management of benzodiazepine withdrawal is vital. This is best accomplished by substitution and taper. In this method, the benzodiazepine is replaced by a pharmacologically equivalent dose of a long-acting benzodiazepine or a barbiturate, usually phenobarbital, and gradually reduced. A taper of 10–25% of the daily dose every 1–2 weeks is generally used in an outpatient setting,[54] but a more rapid taper is sometimes used in an inpatient setting.

Treatment and Medication Management of Benzodiazepine Use Disorder

Treatment of benzodiazepine use disorder (BUD) has three main goals: ease the withdrawal itself; promote further abstinence; and treat the underlying disorder.[54] There are no FDA-approved medications for the treatment of BUD, so medications are generally selected to treat the various underlying psychiatric conditions that accompany BUD. These include antidepressants, especially selective serotonin reuptake inhibitors (SSRI) for mood disorders, anxiety disorders, and insomnia. Non-benzodiazepine anxiolytics are the preferred agents to treat anxiety symptoms. In the case of mood disorders with chronic insomnia, the sedating antidepressants, like trazodone, mirtazapine, or doxepin, may be useful.[54] Brief interventions (e.g., SBIRT) may be useful as initial treatment of BUD, and cognitive-behavioral therapy has also been shown to be an effective treatment for BUD.[55]

Opioid Withdrawal

Opioid withdrawal is a syndrome of cravings for opioids together with somatic symptoms that develop in response to a decrease or discontinuation of opioid use in individuals with prior chronic opioid use. The symptoms can develop within hours to days after the reduction or cessation of opioid use. The symptoms usually include two or more of the following: dysphoria/anxiety, abdominal cramps, diarrhea, vomiting, hypertension, tachycardia, rhinorrhea, lacrimation, diaphoresis, piloerection, yawning, and myalgias. Symptoms of opioid withdrawal can be reduced through medical treatment. The WHO recommends tapered doses of full or partial opioid agonists, methadone or buprenorphine, to safely and effectively aid patients through the withdrawal process.[56] Other medications found to be useful in alleviating symptoms of opioid withdrawal are the alpha-2 adrenergic receptor agonists, clonidine and lofexidine.[56] However, methadone and buprenorphine are more likely to be helpful in lowering the symptoms of opioid withdrawal, retaining patients in withdrawal treatment, and supporting the completion of the opioid withdrawal time period.[46]

Some experts recommend buprenorphine over methadone in older adults because of the higher risk of methadone toxicity in older patients. The risk of respiratory depression from methadone, the risks associated with administering methadone to patients taking benzodiazepines or other sedating drugs, the higher risk of prolonged QTc in the elderly, and the potential lower level of opioid tolerance in the elderly all make buprenorphine a safer choice.[57] Opioid withdrawal treatment via an ultra-rapid opioid detoxification (UROD) is not recommended in older patients because of the high risk of adverse events or death.

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Treatment and Medication Management of Opioid Use Disorder

OUD in older adults is best treated by combining psychosocial counseling, especially cognitive-behavioral therapy, with medication management. Depending on the severity of the OUD and the patient's preferences, the treatment setting for OUD may be in a general outpatient clinic, an addiction specialty clinic, an intensive outpatient treatment (IOP), or a residential addiction treatment facility. The choice of medications available to patients frequently depends on the setting of the care. Opioid treatment programs (OTPs) offer patients methadone, and occasionally buprenorphine. Office-based opioid treatment (OBOT) practices are not permitted to prescribe methadone for OUD treatment, but frequently offer buprenorphine. The third medication approved for the treatment of OUD, naltrexone, can be prescribed by any clinician. Methadone as treatment for OUD can only be prescribed and dispensed by OTPs certified by SAMHSA. However, there are some limitations to using methadone, a full opioid agonist medication, in older patients, including the risk of respiratory depression, especially in less opioid-tolerant individuals.[16] OTPs are mandated by federal and state regulations to utilize counseling sessions, toxicology screens, and same-day screening requests, which can be prohibitive for individuals as they age. Additionally, there are several pharmacologic characteristics of methadone that may limit its utility in older patients. Methadone is known to prolong the QTc in some patients, especially those with structural heart disease and in individuals treated with other QTc-prolonging drugs. Methadone serum levels can be affected by renal disease and by drugs that impact the cytochrome P-450 system, which may cause some variability in its bioavailability.[58] Despite these possible impediments to its use, some have called for a decrease in the barriers to methadone use by adding OTP and methadone coverage to Medicare.[59] The most common side effect of methadone is constipation, which can be quite profound in older patients.

Buprenorphine is a partial opioid agonist that suppresses cravings and withdrawal symptoms from opioids, and reduces illicit opioid use.[60] In order to prescribe buprenorphine in the outpatient setting, physicians, nurse practitioners, and physician assistants must obtain a waiver from SAMHSA and the Drug Enforcement Administration. Some have argued that eliminating this waiver requirement to prescribe buprenorphine in outpatient practices may possibly incentivize a greater number of clinicians treating older patients to screen for and treat OUD.[59] Buprenorphine is usually administered sublingually to avoid its high first-pass effect, although a newer, long-acting injectable formulation is now available as well. As a partial opioid agonist, buprenorphine is safer than methadone, has fewer drug interactions than methadone, and does not usually prolong the QTc interval. Buprenorphine may be safer than methadone for individuals with severe cardiac and especially respiratory illnesses, as it is much less likely than methadone to cause respiratory depression.[58] Additionally, it may be challenging for older adults to routinely access methadone clinics, whereas buprenorphine is covered through Medicare Part D and may be prescribed at regular office-based practices, making buprenorphine a popular treatment among elderly patients with OUD.[16] Unfortunately, there are no well-conducted studies of buprenorphine for treating OUD in the geriatric population, but studies in younger patients with OUD demonstrate a decrease in nonfatal overdose and a decrease in mortality.[61]

Naltrexone is an opioid receptor antagonist used to prevent relapse in OUD by blocking opioid receptors and the subsequent effects of opioid compounds. As such, there is little to no risk of overdose or diversion with the use of naltrexone. Another benefit of naltrexone is that it can be prescribed by all prescribers without limitations or the need for waivers. Oral naltrexone needs to be taken either daily or three times a week (at a higher dose) but can also be given as an intramuscular injection to improve convenience. However, as oral naltrexone has a high first-pass effect and as adherence may be an issue in elderly patients, the injectable formulation is usually preferable. Unfortunately, studies of naltrexone have not shown the same benefits as treatment with methadone or buprenorphine. Naltrexone, to date, has not been shown to decrease nonfatal overdose or mortality in patients with OUD, although specific data in older patients is lacking.[16]

Conclusion

In summary, substance use and substance use disorders are an important and growing problem for older adults. The management of these conditions is frequently more complicated in older patients compared to younger adults for a variety of reasons. Older adults are more likely to suffer from physical and mental health comorbidities, to be taking multiple medications with a greater number of potentially hazardous effects, and some have suffered from the consequences of substance use for a greater length of time. All of these elements must be taken into

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consideration when delivering medical care to older adults in need of medical management of their problematic use of psychoactive substances.

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Chapter 22

Pulmonary Issues

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Introduction

Older adults are disproportionately affected by many chronic lung diseases including chronic obstructive pulmonary disease, bronchiectasis, and idiopathic pulmonary fibrosis. Numerous normal physiologic changes occur in the lungs with age, from reduced parenchymal elastic recoil to thoracic cage distortion. These changes impact pulmonary pathophysiology and disease diagnosis. Clinicians should be cognizant of geriatric issues that can impact diagnosis, treatment, and the occurrence of adverse events secondary to treatment. For example, multimorbidity, the co-occurrence of multiple comorbidities, is more common with increasing age. Multiple comorbidities, such as heart failure or sleep-disordered breathing, must be evaluated and treated in order to appropriately manage chronic lung diseases. Additionally, people with chronic lung diseases have a higher burden of geriatric syndromes, such as frailty, functional impairment, falls, and social isolation.

Older adults are at increased risk of severe morbidity from acute lung conditions such as pneumonia and pulmonary embolism. Treatment of older patients in the intensive care unit requires special attention to geriatric issues (called "age-friendly care") that will improve the quality of their care. This chapter will review the natural history of pulmonary system aging, discuss the most commonly encountered chronic lung diseases with aging, and briefly examine special issues with caring for older adults in a critical care setting.

Physiologic Changes with Aging

Numerous changes to the respiratory system occur naturally with aging (Table 22.1).[1,2] The chest wall stiffens because of degenerative joint disease of the spine, kyphoscoliosis, and calcifications of the costal cartilages and chondrosternal junctions.[3] These changes lead to almost a one third decrease in chest wall compliance.[4] With age, airway size decreases as elastic tissue is replaced by collagen.[5] Small airway collapse at end inspiration increases, leading to air trapping that can mimic obstructive lung disease. The diaphragm and other respiratory muscles become less efficient as well,[6] which may result in increased respiratory effort (e.g., increased respiratory rate and smaller tidal volumes). Given these normal changes with age, the interpretation of pulmonary function tests requires caution to avoid the overdiagnosis of obstructive lung disease in older adults in the absence of clinically meaningful signs and symptoms of respiratory disease.[7,8]

The alveolar-arterial oxygen difference increases in older persons because of a variety of factors, including loss of alveolar surface area and an increase in ventilation-perfusion mismatch. Despite these changes, older adult patients should not be hypoxic at sea level in the absence of disease.[9] A significant increase in alveolar hypoventilation resulting in elevations of arterial pCO_2 is not a part of expected pulmonary system aging.[10] Either of these findings should prompt investigation into an underlying disease process.

Finally, geriatric patients can undergo a number of physiologic changes that decrease their natural pulmonary mechanical defenses against inhalational injury, such as infection, chemical irritation, or foreign bodies. The cough reflex and mucociliary clearance can decline with age.[11] Older adult patients can also be susceptible to dysfunction of the swallowing mechanism from central nervous system disorders, primary swallowing dysfunction, or sedating medications, thus increasing the frequency of aspiration events.[12]

Chronic Lung Diseases: Chronic Obstructive Pulmonary Disease, Bronchiectasis, Sleep-Disordered Breathing, and Interstitial Lung Disease

Chronic Obstructive Pulmonary Disease

Obstructive lung diseases, including chronic obstructive pulmonary disease (COPD) and asthma, are diseases

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Table 22.1 Normal physiologic pulmonary system changes with age

Anatomical region	Changes	Clinical observation
Thorax	• Chest wall stiffening due to kyphosis, and/or rib cage/cartilage calcification • Decrease in chest wall compliance	• Restrictive pattern on PFTs (reduced TLC, FVC) • Vertebral compression fractures/osteoporosis contribute to thoracic distortion
Respiratory muscles	• Weakening (sarcopenia) of diaphragm, intercostal and other accessory muscles	• Reduced maximal inspiratory or expiratory pressure (MIPs/MEPs) • Ineffective cough/airway clearance
Lung parenchyma	• Parenchymal/alveolar distortion • Reduced elastic recoil	• Obstructive changes (e.g., reduced FEV_1 and FEV_1/FVC ratio) on PFTs

TLC: total lung capacity; FVC: forced vital capacity; MIP: maximum inspiratory pressure; MEP: maximum expiratory pressure; FEV_1: forced expiratory volume in 1 second; PFT: pulmonary function test

frequently encountered in the geriatric population. Approximately 12% of Medicare beneficiaries age 65 years and older have COPD.[13] Asthma is often underrecognized in older adults, as the obstructive deficit often becomes more "fixed" and less reversible with bronchodilators, partially due to the natural history of the disease and also due to a diminished response to β-agonists with aging.[14,15] Many older adults experience asthma-COPD overlap syndrome (ACOS), with obstructive lung disease features of both asthma and COPD that results in more dyspnea, wheezing, and reduced physical activity compared to non-overlap COPD patients.[16,17]

The Global Initiative for Chronic Obstructive Lung Disease (GOLD) defines COPD as "a common preventable and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases and influenced by host factors including abnormal lung development."[18] COPD is currently the fourth leading cause of death in the world and an important source of morbidity and hospitalizations.[19] It is a significant contributor to health-care costs and utilization as well. In 2010, the estimated cost in the USA due to COPD was $32.1 billion, and estimated to be $49 billion in 2020.[20]

COPD should be suspected in people with the chronic respiratory symptoms of cough, dyspnea, and/or sputum production, and spirometry should be obtained in order to evaluate for airflow obstruction that is central to the diagnosis.[18,21] Tobacco smoking is the most common risk factor for developing COPD, but nonsmokers may also have COPD for a variety of reasons including air pollution or impaired lung development in childhood.[21–23]

The diagnosis is confirmed by spirometry with a postbronchodilator ratio of forced expiratory volume in 1 second (FEV_1) to forced vital capacity (FVC) of <70% (using absolute values). However, as previously outlined, this definition introduces significant risk of overdiagnosis in the older adult, as normal physiologic changes of lung aging can lead to a reduction in the FEV_1/FVC ratio. For this reason, appropriate clinical context (e.g., symptoms, risk factors) are key to diagnosis. The GOLD criteria further classify COPD severity according to degree of obstruction based on predicted values:[18] stage 1 is an FEV_1 ≥80% of predicted; stage 2 is an FEV_1 50%–<80% predicted; stage 3 is an FEV_1 30%–<50% predicted, and stage 4 is an FEV_1 <30% predicted. The terms chronic bronchitis and emphysema both represent COPD. While either may be used to describe this lung disease, COPD is more accurate and many people have features of both.

Management of Stable COPD

Treatment of COPD centers on chronic symptom control, mortality reduction, and managing acute exacerbations.[22] Management of COPD requires special considerations in the geriatric population. Determining the appropriate COPD treatment plan is largely based on three factors: severity of obstruction, impact of symptoms (as measured by common tools such as the modified Medical Research Council dyspnea scale [mMRC] or COPD assessment test [CAT]), and frequency of exacerbations (Table 22.2).[12] Inhaled medications are recommended for symptom control in a stepwise manner with addition of medications as the disease severity increases.

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In 2011, the GOLD guidelines were updated with an evolved classification scheme called the "ABCD" assessment tool, which takes a more nuanced approach in evaluating COPD severity beyond spirometric values alone. This classification system stratifies patients by symptoms (either the mMRC or CAT) and frequency of exacerbations (Table 22.2).[18] The mildest form of COPD is GOLD category A disease, characterized by mild symptoms (mMRC 0–1 or CAT <10) and 0–1 exacerbations in the past year. The initial management recommendation for category A disease is a short-acting bronchodilator such as ipratropium or albuterol used as needed. It is important to note that short-acting bronchodilators are recommended for use as needed in all categories of COPD severity. For persistent symptoms, a long-acting anti-muscarinic (LAMA) inhaler can be added. Category B disease is characterized as having either moderate or severe symptoms (mMRC ≥2 or CAT ≥10) and 0–1 exacerbations in the past year. Category B patients benefit from a LAMA and long-acting beta agonist (LABA) (combination inhalers can help reduce drug cost) as well as pulmonary rehabilitation.

Patients with category C disease have mild symptoms (mMRC 0–1 or CAT <10) but ≥2 exacerbations per year (or ≥1 leading to hospital admission). They should also use a LAMA or LAMA/LABA or LAMA/inhaled corticosteroid (ICS). Category D disease is the most severe and is characterized by moderate to severe symptoms (mMRC ≥2 or CAT ≥10) and ≥2 exacerbations per year (or ≥1 leading to hospital admission). These patients benefit from LAMA/LABA/ICS combination therapy as well as pulmonary rehabilitation. Inhaled corticosteroids are frequently overused, and should be reserved for people with a history of hospitalizations for COPD exacerbations, ≥2 hospitalizations per year, blood eosinophils >300 cells/uL or asthma overlap.[18]

Mortality reduction is achieved chiefly through smoking cessation, pulmonary rehabilitation, use of supplemental oxygen in individuals who have resting hypoxemia, immunizations to prevent fatal viral diseases, and lung cancer screening in appropriate populations. Smoking cessation is paramount and is a critical non-pharmacologic intervention for improved COPD management, since smoking leads to a more rapid decline in FEV_1.[23] Cessation can be achieved with nicotine replacement therapy, bupropion, varenicline, or counseling. The highest success is seen with a combination of counseling and medications.[24] It is important to note that both bupropion and varenicline have significant potential side effects. Bupropion can lower the seizure threshold. Varenicline can lead to neuropsychiatric changes in some patients and may increase the risk of adverse cardiovascular events in patients with known cardiovascular disease.[25] There are several free national and state-led phone counseling options (National Cancer Institute: 1–877-44U-QUIT; smokefree.gov: 1–800-QUITNOW; Veterans Smoking Quitline: 1–855-QUITVET).

Pulmonary rehabilitation should be prescribed for all patients with category B disease or greater, as it has been shown to improve symptoms, decrease mortality, and

Table 22.2 COPD classification and management

Disease class	Diagnosis requirements	Suggested treatment
A	Mild symptoms (mMRC 0 or 1/CAT <10) and <2 exacerbations in past year	Short-acting bronchodilator as needed (e.g., ipratropium or albuterol) Add LAMA if persistent symptoms
B	Moderate or severe symptoms (mMRC ≥2 /CAT ≥10) and 0–1 exacerbations in the past year	LABA or LAMA daily Short-acting bronchodilator as needed Pulmonary rehabilitation
C	Mild symptoms (mMRC 0 or 1/CAT <10) and ≥2 exacerbations per year or COPD hospitalization	LAMA or LAMA/LABA or LABA/ICS Short-acting bronchodilator as needed
D	Moderate or severe symptoms (mMRC ≥ 2 /CAT ≥10) and ≥2 exacerbations per year or COPD hospitalization	LAMA, LABA and ICS Short-acting bronchodilator as needed Pulmonary rehabilitation

mMRC: modified Medical Research Council dyspnea assessment; CAT: COPD assessment test; LAMA: long-acting muscarinic agent; LABA: long-acting beta-2 agonist; ICS: inhaled corticosteroid

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decrease health-care utilization.[26–31] Unfortunately, pulmonary rehabilitation centers are inequitably distributed and there are few programs in rural regions.[32] Ideally, more virtual pulmonary rehabilitation programs will help address this disparity in the future.[33,34] Live Better is a resource for finding a pulmonary rehabilitation program (http://livebetter.org). Patients with more severe disease who are hypoxemic at rest should be prescribed long-term supplemental oxygen therapy, which improves survival rates in these patients. Severe hypoxemia is defined as a PaO_2 ≤55 mmHg or a SaO_2 ≤88% at rest.[35]

Annual influenza vaccinations should be given to all patients with COPD; most studies have demonstrated that influenza vaccination reduces COPD exacerbations and hospitalizations as well as all-cause and respiratory mortality.[36] The pneumococcal polysaccharide vaccine should also be offered to all patients with COPD.

Lung cancer screening, with yearly low-dose CT scan, deserves consideration in appropriate populations. The US Preventive Services Task Force (USPSTF) recommends screening in adults aged 55–80 with a 30-pack-year smoking history who are still smoking or who have quit in the last 15 years.[37] Decision aids should be used in conversations with patients, to explore the potential risks (e.g., additional studies, procedures, and anxiety) as well as expected benefits of screening; however, decision aids are infrequently used.[38] One such decision aid is the AHRQ Lung Cancer Screening Decision Aid: https://effectivehealthcare.ahrq.gov/decision-aids/lung-cancer-screening/patient.html. Clinicians can seek additional decision support through tools such as University of Michigan's "Should I Screen" website: https://shouldiscreen.com.

Acute Exacerbations of COPD

An acute exacerbation of COPD is defined as an acute increase in typical respiratory symptoms such as increased cough, sputum production, or dyspnea.[18] Most exacerbations are managed on an outpatient basis but may require emergency department utilization and hospitalization for impaired gas exchange or increased work of breathing. Approximately 75% of exacerbations are attributable to viral or bacterial infection.[39] Treatment goals include resolving the underlying cause and optimizing lung function, which can become greatly compromised during exacerbations. After hospitalization for a COPD exacerbation, the 5-year mortality rate is approximately 50%.[40]

Noninvasive positive pressure ventilation (NPPV) should be utilized in acute respiratory failure due to COPD exacerbations, as this has been shown to decrease need for intubation, reduce hospital length of stay, and improve survival.[31] If the patient's respiratory failure persists in spite of NPPV or there is a contraindication to NPPV (e.g., altered consciousness), invasive ventilation is indicated if in congruence with the patient's advance directives.

COPD exacerbation pharmacologic interventions center on improving lung function. Inhaled short-acting bronchodilators (beta-2-agonists with or without anticholinergic agents) are the primary intervention. Systemic glucocorticoids for 5–7 days improve symptoms and decrease hospital length of stay.[41] Oral medications can be used if the patient can take them. If the exacerbation is so severe that the patient cannot tolerate oral medications, IV medications should be used initially. Finally, antibiotics with good respiratory coverage, such as a respiratory fluoroquinolone, should be used if the exacerbation is severe, e.g., in ICU patients.[42]

Special Considerations for the Geriatric Population

There are several important considerations for the management of COPD in older adult populations.[43] Many of the standard treatments for stable COPD and COPD exacerbations may have higher rates of adverse events in part related to the systemic absorption of inhaled medications. Systemic absorption can lead to the same adverse reactions observed from oral or intravenous drugs in the same classes. For example, tiotropium may have as much as 25% systemic bioavailability.[44] As an anticholinergic agent, tiotropium and other medications in this class require careful monitoring because of the risk of urinary retention. Older adults with lower urinary tract symptoms secondary to benign prostatic hyperplasia are at increased risk of this side effect.[45]

Use of short-acting bronchodilators has not been demonstrated to increase the risk of acute myocardial infarction.[46] However, tachycardia is a known side effect of these medications, and caution is especially important in patients with known arrhythmias. Inhaled corticosteroids may increase the risk of pneumonia.[47] In the treatment of acute exacerbations, hyperglycemia severe enough to require intervention can be observed with the usage of systemic glucocorticoids.[48]

Inhaler use may be more challenging for older adults because of reduced coordination or arthritis in the hands, both increasing the risk of incorrect use. An error rate of over 40% for metered-dose inhalers has been observed, though dry powder inhaler use is slightly better. Common inhaler errors include poor coordination or no

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post-inhalation breath hold.[49] Repeated inhaler use assessment should be performed, and use of spacers can help reduce errors associated with poor coordination. The COPD Foundation offers helpful videos and tips for patients in the correct use of inhalers (www.copdfoundation.org). In some patients who cannot effectively use inhalers, such as those with severe cognitive impairment, nebulized medications should be substituted.

Inhalers are costly and their expense is associated with nonadherence in up to 31% of people. Inhaler cost over $20/month is associated with cost-related nonadherence. Out-of-pocket cost is highly variable given the wide range of medication coverage plans, including Medicare Part D, which covers inhalers.[50,51]

Frailty is much more common among older adults with COPD. In the Rotterdam study, a population-based cohort study, frailty prevalence was 10.2%, 95% CI 7.6–13.5% in those with COPD compared to 3.5%, 95% CI 2.6–4.4% in those without COPD.[52] High-quality care for adult patients with COPD should focus on a comprehensive assessment of functional impairments and need for durable medical equipment and community resources to support well-being. Integration of geriatrics and palliative care principles into care of older adults with COPD is likely to be very beneficial.[53]

Advance directives should be discussed with all patients with severe COPD. A helpful resource for advance care planning is Prepare For Your Care (https://prepareforyourcare.org). It is particularly critical to discuss advance directives with patients who have had acute exacerbations requiring hospitalization, as this is associated with a marked increase in mortality.[54] Patients on oxygen therapy should also be a particular focus for receiving advance directive education, as they typically have more advanced disease and therefore are at an increased risk of respiratory failure from an exacerbation of their chronic lung disease or reparatory infection. Prognostication can be quite challenging in people with COPD. Two prognostic tools, the body mass index, airflow obstruction, dyspnea, and exercise capacity index (BODE) and the age, dyspnea, obstruction (ADO) tool, can be used, but they predict mortality over years, which is not useful for immediate prognostication for hospice referral.[55,56]

Bronchiectasis and Nontuberculous Mycobacterium

Bronchiectasis is a chronic lung disease characterized by distortion of airways resulting in mucous trapping and airway colonization with bacteria that can lead to a vicious cycle of airway inflammation, infection, and damage.[57] Bronchiectasis is also referred to as non-cystic fibrosis (CF) bronchiectasis, as it shares features of cystic fibrosis, and many treatment principles are extrapolated from CF management.

Diagnosis

Bronchiectasis is frequently misdiagnosed as COPD, as patients with bronchiectasis typically have obstructive lung disease on PFTs and the most common presenting symptoms are dyspnea and productive cough. Bronchiectasis is also underrecognized as its diagnosis must be confirmed by CT scan, and many people with these symptoms have not had a CT scan for diagnosis. In fact, the prevalence of bronchiectasis increased significantly from 2000 to 2007, but this could be due to increased recognition of an underrecognized disease.[58]

A review of the US Bronchiectasis Research Registry (BRR) in 2017 demonstrated that most enrolled patients were women (79%), white (89%), and never-smokers (60%).[59] Bronchiectasis can be caused by a wide variety of insults, including previous pulmonary infections, genetic diseases such as CF, and primary ciliary dyskinesia or autoimmune conditions.[57] A common cause particularly in the geriatric population is recurrent aspiration, which causes airway damage.

Treatment

Because there is little strong data to guide bronchiectasis management, patients should ideally see a pulmonary specialist for evaluation of the underlying cause and recommendations for treatment. Mucous clearance strategies are typically regarded as the mainstay of therapy, with the goal of disrupting mucous stasis and bacterial overgrowth.[60,61] Obtaining sputum cultures is beneficial to guide antibiotic choice when patients experience an exacerbation of bronchiectasis symptoms (e.g., increase or change in sputum production or cough/dyspnea symptoms). Patient education is critical in management of bronchiectasis, as most patients have not heard of this disease entity. The American Thoracic Society Patient Education Information series has two resources for such education: 1. "What Is Bronchiectasis?" (www.thoracic.org/patients/patient-resources/resources/bronchiectasis-pt1.pdf) and 2. "Treating Bronchiectasis" (www.thoracic.org/patients/patient-resources/resources/bronchiectasis-pt2.pdf).

In some people with bronchiectasis, nontuberculous mycobacterium (NTM) infection leads to additional airway damage and morbidity. In the US BRR, 63% of those

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enrolled in the registry had a history of or current infection with NTM.[59] NTM is a group of bacteria highly prevalent in the environment (e.g., soil and water) that typically does not cause pathogenic disease in people with normal lungs but can cause progressive pulmonary infection in those with underlying lung disease like bronchiectasis or COPD.[62] The most common NTM bacteria is *Mycobacterium avium complex* (MAC). Infection with NTM can be indolent and symptomatic or highly pathogenic resulting in constitutional symptoms (e.g., fatigue, weight loss) and progressive pulmonary illness. Two-year prevalence has been estimated at 20 per 100,000 people in those over age 55. Median age of incidence is 66 years old, with an increased prevalence in females.[63] Imaging findings can range from tree-in-bud opacities to pulmonary nodules to cavitary lung lesions. Diagnosis requires a combination of pulmonary symptoms, radiographic changes, and a positive culture from at least two separate sputum samples or from one bronchial lavage sample.[62]

The decision to initiate treatment for NTM infection is complex, as treatment is long, requires multiple antibiotics for efficacy, and has a high frequency of side effects, especially for older adult patients who may be taking multiple other medications. Decisions should be based on disease severity (e.g., evidence of cavitary lung disease and/or positive acid-fast bacilli sputum smears, which indicate more severe disease), symptom burden, and patient wishes according to risks and benefits of therapy based on comorbidities and life expectancy.[62] Patients with chronic indolent disease, mild symptoms, or significant other comorbidities, such as malignancy, may choose to forgo treatment. For patients who do decide to undergo treatment, antibiotic susceptibility testing should be performed on respiratory isolates, and a pulmonologist and/or infectious diseases clinician should be involved in treatment planning. Treatment should continue until sputum cultures are consecutively negative for 1 year, if possible.

Interstitial Lung Diseases

Interstitial lung diseases (ILDs) are a heterogenous group of diseases that ultimately result in restrictive lung disease from chronic lung inflammation and/or scarring. The diseases affect people of various ages; some causes are identifiable (e.g., due to autoimmune disease or an environmental exposure), while others are idiopathic. At one center, approximately 24% of all ILD patients were over age 70, and idiopathic pulmonary fibrosis (IPF) is the most common ILD in the older adult population.[64] The prevalence of IPF in patients aged 75 years or older has been estimated at 227 per 100,000 compared to 4 per 100,000 in the 18- to 34-year-old demographic.[65]

Diagnosis

In a patient with ILD, pulmonary function tests reveal a restrictive pattern, which means that static and dynamic lung volumes are reduced (e.g., total lung capacity and forced vital capacity) and there is typically a disruption in the alveolar-capillary unit due to inflammation or scarring, resulting in a reduction in the diffusing capacity for carbon monoxide (DLCO). Severity of restriction correlates with worse disease.[66] Fine crackles are frequently heard on lung exam,[67] and clubbing may be seen in later stages of disease.[68]

Idiopathic pulmonary fibrosis (IPF) is the most common ILD among older adults. Patients with IPF typically have dyspnea on exertion that has progressed over several years with an associated non-productive cough. Radiographic studies are vital for diagnosis, particularly a high-resolution chest CT scan (HRCT). The classic features of IPF on HRCT are supportive of a usual interstitial pneumonia (UIP) pattern if a lung biopsy was obtained. A "definite UIP pattern" consists of subpleural and bibasilar reticular opacities with honeycombing with or without peripheral traction bronchiectasis.[69] People with ILD should be referred to a specialty center, ideally with transplant services, for evaluation by a multidisciplinary team consisting of pulmonologists, pathologists, and radiologists. Accurate diagnosis of ILD subtype is critical to management, and these specialists will determine if surgical lung biopsy or serologic tests are indicated and set forth an appropriate treatment plan.

Treatment

Treatment of ILD depends on the specific diagnosis of ILD, and as outlined above there are numerous ILD diagnoses. As IPF is the most common ILD among older adults, this summary will focus on treatment of IPF. IPF is a serious illness with a mean survival after diagnosis of 5.6 years.[70] In the last decade, two antifibrotic agents, pirfenidone and nintedanib, have been demonstrated to slow progression of lung function decline and are now widely used in the USA.[71–73] Pirfenidone was used in Europe for many years before approval in the USA.

Additional treatment recommendations are based on consensus guidelines and largely consist of supportive

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care with oxygen therapy, pulmonary rehabilitation, and careful consideration of the role of comorbidities.[74] Data does not support the use of corticosteroids or other immunosuppression agents in people with IPF, and in fact these treatments increase mortality.[75] Given the chronic progressive nature of IPF and its unpredictable course, patients with IPF should be referred upon diagnosis to lung transplant, if this is within their treatment preferences.

Special Considerations for the Geriatric Population

Follow-up analyses have demonstrated that pirfenidone adverse drug reaction that resulted in interruption of therapy or dose reduction is much more common in adults aged 80 or older (32.7%) compared to patients younger than 65 (18%).[76]

Transplant centers vary regarding the age at which they will no longer consider patients for lung transplantation, so this should be determined by the tertiary center. Frailty assessment may provide important morbidity and mortality risk information for those awaiting transplant, as it has been demonstrated to be associated with an increased risk of delisting or death before transplant.[77] Advance care planning discussions are paramount in all patients with IPF. Guidelines recommend against mechanical ventilation due to respiratory failure in most patients with IPF, as this disease is progressive and irreversible, but patient preferences should drive this decision.[70] Clinicians should prioritize disease-related education, advance care planning, and consider co-management or referral to a palliative care specialist.

Sleep-Related Breathing Disorders

Sleep-related breathing disorders, such as obstructive sleep apnea (OSA), central sleep apnea, and obesity hypoventilation syndrome, result in either an abnormal respiratory pattern like apnea, or reduction in gas exchange during sleep. Between 12 and 18 million adults in the USA are affected with these disorders.[78]

Obstructive sleep apnea is the most common of these disorders, with an estimated prevalence of 17% in men and 9% in women aged 50–70.[79] In addition to age and male gender, obesity is a significant risk factor, with obese individuals suffering up to a sixfold increased risk for OSA when compared to nonobese patients.[79]

Diagnosis

Presenting symptoms of OSA include daytime sleepiness, snoring, and witnessed apneas by a bed partner. A useful screening tool is the STOP-BANG questionnaire, in which snoring, tiredness, observed apnea, high blood pressure, body mass index, age, neck circumference, and male gender all increase likelihood of OSA diagnosis.[80] Obstructive sleep apnea is a risk factor for systemic hypertension and multiple adverse cardiac effects, including coronary artery disease and arrhythmias.[81–83]

Diagnosis is made via overnight polysomnography. Diagnosis is made when there are 15 or more apneas, hypopneas, or respiratory-effort-related awakenings per hour of sleep in asymptomatic patients or five or more events per hour in a patient with symptoms of disturbed sleep.[84] Diagnostic evaluation should be encouraged, as the test is relatively safe and treatment has large potential benefits on quality of life. Home sleep apnea testing is becoming increasingly available. Although it may be less informative than conventional polysomnography, it is less expensive and more convenient for patients.

Treatment

The treatment of choice for OSA is positive airway pressure therapy delivered via either continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BIPAP) devices. Although improved morbidity (e.g., improved blood pressure) and symptoms (e.g., better quality of life) have been demonstrated with these therapies, no randomized controlled trial to date has demonstrated an improvement in mortality.[85] The pressure is titrated to the required setting during polysomnography either by splitting the night into diagnosis then treatment phases or with a second visit to the sleep laboratory. As with home diagnostic testing, out-of-center treatment with auto-titrating positive pressure therapy is becoming available for patients with high pretest probability of uncomplicated, moderate OSA.

Acute Pulmonary Conditions: Pneumonia and Pulmonary Embolism

Pneumonia

Pneumonia is undoubtedly an important clinical entity, leading to significant morbidity among older adults. Pneumonia is the eighth leading cause of death in the USA,[86] and the 30-day mortality rate for

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community-acquired pneumonia (CAP) approaches 10%.[87] Further, older adult patients with pneumonia frequently require hospital and even ICU admission.[88]

Diagnosis

Based on the 2016 American Thoracic Society (ATS) and Infectious Diseases Society of America (IDSA) clinical practice guidelines, pneumonia can be divided into three different classifications based on the location that pneumonia is acquired. Treatment largely depends on this classification (Table 22.3).[89,90] Hospital-acquired pneumonia (HAP) is not present on hospital admission but occurs ≥48 hours after admission. Ventilator-associated pneumonia (VAP) is a subclass of HAP that occurs ≥48 hours after intubation and mechanical ventilation.[41] Finally, community-acquired pneumonia (CAP) is a pneumonia that lacks any of these predisposing exposures; an updated ATS/IDSA clinical practice guideline addressing CAP management was published in 2019.[90] The 2016 guidelines removed the concept of "healthcare-associated pneumonias."

Suspicion for pneumonia is increased in a patient who has cough, pleuritic chest pain, and/or dyspnea. Examination findings include tachypnea, tachycardia, hypoxia, crackles, and/or evidence of consolidation by egophony and percussion.[91] An important caveat is that in patients who take beta-blocker medications, tachycardia may be blunted. Plain film chest X-ray is recommended to confirm diagnosis if there is clinical suspicion for pneumonia. Diagnosing pneumonia can be difficult in the older adult patient, as "classic" symptoms such as fever may be absent and nonspecific symptoms like altered mental status may be present. Additionally, older adult patients may lack characteristic X-ray findings, and additional imaging with CT chest may be required.[90]

Treatment

Antibiotic treatment for CAP depends on several factors, including severity of illness (e.g., if patient is being treated as an outpatient, in the general medical ward, or in the intensive care unit), likely pathogens, risk factors for antibiotic resistance, and medical comorbidities (Table 22.3). Healthy outpatients with no comorbidities may be treated with amoxicillin or macrolide or doxycycline. Patients with comorbidities (chronic lung, heart disease, etc.) should be treated with either a respiratory fluoroquinolone, or a cephalosporin or amoxicillin/clavulanate plus a macrolide or doxycycline.[90] Hospitalized patients are typically treated with an anti-pneumococcal beta-lactam and macrolide, while patients with methicillin-resistant Staphylococcus aureus (MRSA) risk factors also receive either linezolid or vancomycin.

Empiric treatment of HAP and VAP requires coverage for MRSA, pseudomonas, and other gram-negative bacilli organisms. Double-coverage antibiotic therapy (two antibiotics with different mechanisms of action) for treatment of gram-negative bacilli, particularly pseudomonas, is controversial and there is not strong evidence to support this practice. This practice should be reserved for those patients with a high risk of

Table 22.3 Classifications and suggested treatments of pneumonia

Pneumonia type	Predisposing exposures	Antibiotic treatment
Community-acquired pneumonia (CAP)	Pneumonia contracted outside of the hospital	Patients with minimal comorbidities: amoxicillin OR macrolide OR doxycycline Patients with comorbidities: Respiratory fluoroquinolone OR amoxicillin/clavulanate or cephalosporin AND macrolide or doxycycline
Hospital-acquired pneumonia (HAP)	Occurs ≥48 hours after hospital admission	Empiric coverage for S. aureus (if no risk factor for MRSA, prescribe antibiotic with activity against MSSA) plus coverage for Pseudomonas/other gram-negative bacilli (e.g., vancomycin + 4th-generation cephalosporin, carbapenem or piperacillin- tazobactam) Consider dual coverage for Pseudomonas if resistance expected
Ventilator-associated pneumonia (VAP)	Occurs ≥48 hours after intubation/mechanical ventilation	Empiric coverage for S. aureus (choose coverage depending on need to cover for MRSA vs MSSA) PLUS coverage for Pseudomonas/other gram-negative bacilli (e.g., vancomycin + 4th-generation cephalosporin, carbapenem or piperacillin-tazobactam) Consider dual coverage for pseudomonas if resistance expected

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antimicrobial resistance and should take into account the local resistance patterns via a hospital's antibiogram. Empiric antibiotic choices should be narrowed based upon culture and sensitivity of respiratory tract specimens.

The decision to admit patients to the hospital for treatment of pneumonia is based upon severity of illness. Validated clinical scoring rules offer guidance on hospital admission based upon risk of death from pneumonia. One such scoring tool, called CURB-65, identifies morbidity risk based on confusion, blood urea level, respiratory rate, blood pressure, and age.[92] In the simplest terms, any patient aged 65 or over presenting with CAP should be admitted to the hospital if they present with confusion, have a respiratory rate ≥30, or are hypotensive. Other risk factors that increase the consideration for hospital admission in older adult patients include associated hypoxemia or acidosis, hyperglycemia, hypernatremia, anemia, pleural effusion, and severe underlying comorbidities.[92,93] Finally, for nursing home patients with pneumonia and advanced dementia, goals of care should be clarified, as antimicrobial treatment has been shown to modestly prolong survival but not increase comfort.[94,95]

Acute Pulmonary Embolism

Acute pulmonary embolism (PE) is a common malady among older persons, with an approximate prevalence of 400 per 100,000 patients who are ≥80 years old.[96] It carries a mortality rate of 30% if untreated.[97] Risk factors for PE include comorbidities that increase in prevalence with age, such as malignancy, immobilization, stroke, paresis, chronic heart disease, recent central venous catheter or pacemaker placement, autoimmune disease, and a history of prior venous thromboembolism (VTE).[98,99] Additionally, obesity and hypertension have been identified as risk factors in women.[100]

Diagnosis

Because of its associated mortality, accurate and early diagnosis of PE is important. However, this is not always easily achieved, since the signs and symptoms are nonspecific and current diagnostic tests are imperfect. Symptoms most commonly include the acute onset of dyspnea, pleuritic chest pain, and cough; and less commonly, hemoptysis. Signs include tachypnea, inspiratory crackles, and tachycardia.[101]

Multiple studies of laboratory and imaging modalities have been performed in an attempt to improve accuracy of PE diagnosis. Overall, the current data supports utilizing a combination of clinical probability, laboratory tests, and radiographic imaging. One widely used algorithm utilizes the modified Wells criteria to stratify patients into high or low risk for PE. For those deemed low risk for PE, with a Wells score <4, a D-dimer should be checked. A negative D-dimer (<500 ng/mL) virtually excludes PE. A positive D-dimer should prompt further imaging with either CT or V/Q scans. A Wells score ≥4 suggesting high pretest probability should proceed to imaging without checking a D-dimer.[102]

Pulmonary angiography is the "gold standard" for diagnosis of PE, but there is an approximately 5% morbidity associated with this procedure and it is rarely performed for PE diagnosis.[103] For this reason, two other imaging modalities, spiral (helical) CT with IV contrast and the ventilation-perfusion (V/Q) scan, are routinely used in the diagnosis of PE. The V/Q scan has a high sensitivity for detecting PE in patients with a high pretest probability and a high-probability scan. It also has a high specificity to exclude PE in patients with a low pretest probability and low-probability scan.[104] Spiral CT has similar performance characteristics in that it has good sensitivity in those with high pretest probability and a positive CT, and good specificity for those with low pretest probability and a negative CT.[105] Both scans are much less accurate in intermediate-probability patients.

Treatment

Anticoagulation is the mainstay of treatment and results in a decrease in mortality.[106] Hemodynamically unstable patients should be hospitalized to initiate treatment and determine need for thrombolytic therapy or an embolectomy. Select, very low-risk patients may initiate anticoagulation therapy in the outpatient setting, but most patients are hospitalized as they initiate anticoagulation. The 2016 American College of Chest Physicians' guidelines on antithrombotic therapy for VTE disease recommend, in patients with VTE and no cancer, antithrombotic therapy with dabigatran, rivaroxaban, apixaban, or edoxaban over vitamin K antagonist therapy (e.g., warfarin).[107] These agents have the advantage of not requiring routine blood testing. Patients with cancer should receive low-molecular-weight heparin therapy. Duration of therapy is dependent on the presence of previous VTE history, risk factors for a recurrent embolic event, and whether or not these risk factors are modifiable. In a patient without any of these risk factors, treatment duration is typically 3 months.

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Special Considerations in the Older Adult

Diagnosis of PE can be complicated in older adult patients, as D-dimer is a nonspecific marker for inflammation and is frequently elevated in the older adult because of comorbidities. This can decrease specificity of the D-dimer test.[108] Renal function decreases with age, so caution must be exercised when using IV contrast for CT scans in older patients. Low-molecular-weight heparin must also be used with caution in this group for the same reason, since it is renally metabolized. Finally, patients >65 years old and those at risk for falls are at an increased risk of bleeding while on anticoagulation, so great caution should be exercised with usage of these drugs.[107]

Critical Care in the Older Adult

Older adult patients account for over 40% of patients in ICUs.[109] Age can impact triage decisions, whether by explicit rationing decisions in resource-limited settings or by implicit bias (ageism). This is inappropriate for many reasons, including that ICU mortality benefit may be greater in older adults[110] and age itself is less predictive of mortality risk than acute illness severity and degree of underlying comorbidities and disability.[111] In fact, in a longitudinal study of the pre-ICU functional trajectories of older adults, mild to moderate disability and severe disability were associated with more than double and triple the risk of death, respectively. within 1 year of admission compared to those without pre-ICU disability.[112]

There are many important issues that should be considered when caring for older adult patients in the ICU. Delirium is common, with approximately 70% of older adult patients suffering from delirium during a hospitalization requiring ICU care.[113] (See Chapter 12.) Delirium is defined as an acute change in mentation that results in inattention and either disorganized thinking or an altered level of consciousness. Delirium is an independent predictor of both increased in-hospital mortality and prolonged hospitalization.[114] Further, more days of delirium is associated with increased risk of 1-year mortality among adults age 60 and older.[115] Delirium diagnosis in the ICU can be easily performed using convenient bedside tools such as the Confusion Assessment Method-ICU (CAM-ICU) assessment tool (Table 22.4).[116,117] Given the association of delirium with increased mortality, prevention is incredibly important. Potential strategies include reorientation protocols, environmental modifications such as dimming the lights at night in order to preserve the sleep-wake cycle, and early mobilization. Minimization of long-acting sedatives such as lorazepam has also been proven to be an effective prevention strategy.[118] Antipsychotics do not treat delirium, but there may be a role for their use in management of severe agitation.[119] These interventions may not always be possible in the critically ill patient but should be implemented when possible.

Table 22.4 Confusion Assessment Method for the ICU (CAM-ICU)

Level of consciousness	Patient must be conscious and, at maximum, moderately sedated, in order to assess delirium (can assess via Richmond Agitation-Sedation scale (RASS), must be ≥-3)
Feature 1: acute onset or fluctuating course	Based on provider assessment: present if provider assesses mental status different than baseline or fluctuating over last 24 hours
Feature 2: inattention	Read list of 10 letters to patient, patient squeezes provider's hand with prespecified letter (e.g., "A"), present if inaccurately performed
Feature 3: altered level of consciousness	Present if RASS anything other than zero
Feature 4: disorganized thinking	Present if patient incorrectly answers series of abstract questions and is unable to accurately follow commands. See reference for details
Scoring	Delirium diagnosed if feature 1 + 2 AND either 3 OR 4 are present

Adapted from Dr. Wes Ely's Confusion Assessment Method for ICU (CAM-ICU): The Complete Training Manual (http://tetaf.org/wp-content/uploads/2016/03/CAM_ICU_training.pdf)

Pre-ICU cognitive impairment is associated with increase in post-ICU disability over the 6 months following critical illness, and moderate cognitive impairment doubles the likelihood of a new nursing home admission.[120] Many older adult survivors of critical illness are left with significant functional impairments and newly acquired cognitive impairment.[121]

Severe sepsis is one of the most common diagnoses leading to admission to critical care units, and older adult patients are at higher risk for hospitalization with severe sepsis than the general population. The yearly incidence of severe sepsis for patients over 80 years old is 26.2 per 1,000 compared to 3 per 1,000 overall. The incidence of severe sepsis in the older adult is expected to increase in the coming years as patients live longer with multiple

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comorbid conditions that increase the likelihood of sepsis, such as end-stage renal disease, COPD, cirrhosis, and malignancies.[122] Early appropriate antibiotics are essential to good outcomes.

Geriatric patients are at higher risk for prolonged mechanical ventilation after acute illness.[123] Although recent strategies to improve mechanical ventilation liberation have improved the rates of prolonged ventilation, the most effective approach is still to avoid intubation when possible. Judicious use of noninvasive positive pressure ventilation in patients presenting with respiratory failure due to COPD or congestive heart failure reduces the incidence of mechanical ventilation and the risk of mortality.[124]

Multiple studies have demonstrated that treatments provided to older patients with critical illness are often incongruent with their care preferences, including a multicenter prospective cohort study of hospitalized patients over age 80 that demonstrated that 70% of patients who died preferred comfort-focused care but the majority (63%) received one or more life-sustaining treatments before death.[125] Eliciting individual care preferences from older patients and their health-care proxies in order to ensure that the treatments received are aligned is a vital component of delivering exceptional critical care. Therefore, the most important factor in whether an older adult person should be treated in an ICU setting is whether ICU treatment would be congruent with their wishes. This can be difficult to ascertain if the patient does not have advance care planning (ACP) documentation or if that documentation is ambiguous. These situations are best averted by being proactive with ACP in the outpatient setting.[126]

Conclusion

Many chronic lung diseases, such as COPD, IPF, and bronchiectasis, are much more common with increasing age. Given normal pulmonary system aging and the increasing prevalence of multimorbidity, older persons have numerous special diagnostic and treatment considerations for managing chronic lung diseases. Older adults account for a large proportion of patients in ICUs. Practitioners should focus on utilizing the most effective therapies while minimizing potential harm. Further, one should make every effort to ensure that diagnosis and treatment strategies are in congruence with patient wishes.

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Chapter 23

Gastrointestinal Disorders

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Aging leads to progressive deterioration of physiological function and predisposes to pathological processes. Common geriatric syndromes (such as dementia, mobility impairment, and osteoporosis) along with age-related impairment in appetite, absorption, and food intake affect nutrition, symptom presentation, and response to therapy of common gastrointestinal (GI) disorders in the elderly. Age-associated changes in drug metabolism and polypharmacy can result in potential interactions and side effects of drugs used in the treatment of GI diseases, which in turn complicates their management. Polypharmacy, which is common in elderly, can also exacerbate digestive symptoms. Elderly patients with neurocognitive decline often have atypical presentation of their GI disorders. These factors can make the diagnosis of GI diseases in the elderly more challenging and therefore require different management approaches. In this chapter, we will discuss the common GI disorders that affect the elderly with special focus on age-related pathophysiology and clinical implications.

Disorders of Esophagus

Gastroesophageal Reflux Disease (GERD)

GERD refers to symptoms and/or histopathological changes and/or complications due to reflux of gastric contents into the esophagus. This contrasts with physiological asymptomatic reflux that can happen during or after a meal. GERD can have a broad spectrum of presentation from mild heartburn to various complications including ulcers, strictures, and bleeding. GERD is very common in the elderly population, with a reported prevalence of about 23%.[1,2] High prevalence in the elderly population may be, in part, related to multiple factors that are specific to old age, including reduction in salivary gland production, changes in motility, higher prevalence of hiatal hernia, and inability to stay in an upright position after a meal. The symptoms may decrease with advanced age partly because of decreased visceral sensitivity; however, the severity of esophageal mucosal injury and its associated complications are higher in geriatric patients, as the elderly may have late and atypical presentation compared to younger patients (see Table 23.1).

Clinical Presentation

The diagnosis of GERD poses unique challenges in the elderly population. The typical symptoms of GERD, including heartburn, sour regurgitation, and epigastric pain, are less common in the elderly. The elderly may present with atypical symptoms, including pulmonary symptoms such as chronic cough, bronchitis, and recurrent asthma, and laryngeal symptoms such as hoarseness and chronic laryngitis. Often elderly patients with GERD present with nonspecific complaints including vomiting, weight loss, and anemia, which makes the diagnosis challenging. They may be completely asymptomatic before presenting with complications of GERD such as dysphagia or odynophagia due to esophageal stricture.

Diagnosis

Since elderly patients often present with atypical or nonspecific symptoms, upper endoscopy (esophagogastroduodenoscopy, EGD) should be considered early in elderly patients suspected of having GERD. EGD helps in determining grade of esophagitis, presence of hiatal hernia, and evaluation of potential complications such as ulcer, stricture, and Barrett's esophagus (BE). A barium swallow study should be performed prior to EGD in patients who present with dysphagia to evaluate for strictures and aspiration in cases of patients with recurrent pulmonary symptoms. Esophageal pH monitoring should be performed in patients when EGD is negative but the patients continue to have symptoms suggestive of GERD and prior to anti-reflux surgery. Esophageal manometry may be considered in patients suspected to have motility disorder of the esophagus contributing to GERD symptoms.

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Table 23.1 Gastroesophageal reflux disease (GERD) characteristics in the geriatric population

High prevalence in elderly of up to 23%
Late and atypical presentation more common in elderly
Typical symptoms: heartburn, sour regurgitation, and epigastric pain
Atypical symptoms (dysphagia, odynophagia); pulmonary symptoms (chronic cough, bronchitis, recurrent asthma); laryngeal symptoms (hoarseness and chronic laryngitis) more common in elderly
Complications (Barrett's esophagus, severe esophagitis, ulcerations, strictures) more common in elderly
Treatment with lifestyle modification, H2 blockers (caution with cimetidine), and PPI (caution with long-term use – concern for osteoporosis, *Clostridium difficile* colitis, pneumonia)
Laparoscopic anti-reflux surgery and transoral incisionless fundoplication is safe and effective in the elderly

Treatment

Lifestyle Modification

This includes avoidance of certain foods, smoking and alcohol cessation, weight loss, reduction in caffeine intake, and appropriate body position. Citrus and other acidic foods, coffee, peppermint, nicotine, and alcohol are believed to counteract the body's natural anti-reflux defense mechanisms. Studies have shown that weight loss and elevation of the head of the bed can help reduce GERD. Certain medications such as aspirin, NSAIDs, bisphosphonates, and ferrous sulfate, which can cause direct esophageal mucosal injury, should be used with caution, and if needed they should be taken in an upright position with a full glass of water. This is especially relevant in elderly patients who may have challenges maintaining the upright position. Other medication such as benzodiazepines, calcium channel blockers, theophylline, tricyclic antidepressants, and anticholinergics, which reduce lower esophageal sphincter (LES) pressure, should be used carefully in elderly patients with GERD.

Pharmacological Therapy

Antacids can be used to provide symptomatic relief in patients with mild symptoms. Elderly patients may be more susceptible to the side effects of antacids including constipation, hypercalcemia, salt retention, and interference with absorption of other drugs.

H2 receptor blockers can also be used and are usually well tolerated. Cimetidine, one of the H2 blockers, should be used with caution in the elderly because of its tendency for potential drug interactions and delirium. The mainstay of therapy is proton pump inhibitors (PPIs), which provide a more complete relief of GERD symptoms, are considered superior to H2 blockers, and are often considered first-line therapy for GERD. Long-term use of PPIs requires caution, especially in the elderly, as they are associated with risk of osteoporosis, *Clostridium difficile* infections, pneumonia, and interstitial nephritis. Omeprazole is a commonly used PPI, inhibits CYP2C19 cytochrome P-450 enzymes, and interacts with clopidogrel, reducing its efficacy. Therefore, it should be used with caution in patients on clopidogrel.[3]

Surgery

Anti-reflux surgery such as laparoscopic Nissen fundoplication may be considered in patients refractory to medical therapy or with complications of GERD. More recently, newer procedures such as transoral incisionless fundoplication (TIF) have been used as a minimally invasive endoscopic procedure and could be an attractive option in elderly patients.[4]

Barrett's Esophagus

BE is a complication of longstanding GERD and refers to replacement of normal squamous epithelium in the distal esophagus with metaplastic columnar epithelium (columnar-lined esophagus or CLE).[5] CLE is a precursor for the development of high-grade dysplasia and adenocarcinoma, which is its most feared complication. The incidence of adenocarcinoma in patients with CLE is about 0.5% per year.[6] Increasing age is a risk factor for development of advanced dysplasia and adenocarcinoma. The mean age of diagnosis of CLE is in the 60s and hence it is prevalent in the geriatric population. Routine surveillance of patients with CLE with endoscopy for dysplasia can lead to early detection of advanced dysplasia and potentially curable adenocarcinoma, but the benefits of early detection need to be weighed against the risk of the procedure (which is higher in very elderly patients with comorbidities), low incidence of adenocarcinoma, and limited life expectancy in some elderly patients. In a cross-sectional study of Veterans Affairs population, it was noted that one third of the elderly patients at the time of the diagnosis of CLE had limited life expectancy. The authors suggested the potential harms from surveillance in these elderly patients with limited life expectancy are likely to exceed the benefit from early detection of cancer, and hence recommended avoiding BE surveillance in those with limited life expectancy to help reduce unnecessary procedures and complications.[7] The decision to perform surveillance endoscopy in elderly patients with CLE needs to be

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individualized, considering patient preferences as well as their life expectancy.

Dysphagia

Dysphagia is common in elderly population and is divided into oropharyngeal/initiation dysphagia and esophageal/transit dysphagia. Oropharyngeal dysphagia results from difficulty in passage of food from the mouth to upper esophagus, manifesting as the sensation of difficulty chewing and difficulty in initiation of swallowing. Several factors in the elderly predispose them to oropharyngeal dysphagia, including painful or diseased teeth, dry mouth, ill-fitting dentures, and the higher prevalence of neuromuscular disorders affecting the soft palate, pharynx, tongue, and upper esophageal sphincter such as stroke, Parkinson's disease, Alzheimer's dementia, and multiple sclerosis, as well as muscular disorders such as myasthenia gravis and polymyositis. It can also be brought on by local causes such as pharyngeal tumor or stricture. Patients present with symptoms such as a cough with swallowing, nasal regurgitation, and food stuck in the throat. Oropharyngeal dysphagia is common in the elderly population, with a reported prevalence of up to 13% in patients aged 65 years or older, and has been reported in almost 50% of nursing home residents.[8,9] Elderly people present a challenge, as they may not report it until they develop complications including aspiration pneumonia, malnutrition, or dehydration.

Esophageal dysphagia results from difficulty in passage of food from the upper esophagus to the stomach and presents as a sensation of food being stuck in the chest. Possible etiologies include mechanical obstruction (tumor, stricture), compression with outside structure (vascular compression or mediastinal masses), neuromuscular disorders such as scleroderma, esophageal spasm, and achalasia, and inflammatory/infectious disorders of esophagus such as eosinophilic esophagitis and candidiasis.

Odynophagia refers to painful swallowing and is usually secondary to infection or malignancy. Common infections that can result in odynophagia include cytomegalovirus, candidiasis, and herpes simplex virus. A study by Lasch et al. showed that elderly patients often have reduced pain sensation to graded intraesophageal balloon inflation compared to their younger counterparts, providing evidence of diminished visceral pain with aging.[10] This may be connected to age-related reduction in the number of myenteric neurons in elderly patients. As a result, the elderly often present late and the disease may be more advanced at presentation.

A detailed history is important in elucidating the differential diagnosis of dysphagia. Dysphagia to solid foods first often indicates mechanical obstruction in contrast to difficulty swallowing both solids and liquids, which is suggestive of motility/neuromuscular problem. Patients who take steroid inhalers because of chronic obstructive pulmonary disease (COPD)/asthma are at risk for candidiasis. A long history of smoking predisposes to squamous cell cancer of the esophagus. In addition, weight loss may be indicative not only of the severity of the problem but also an underlying malignancy. Patients with dysphagia, who have a long history of heartburn or chest pain related to GERD, may have a stricture or adenocarcinoma secondary to development of BE.

Diagnosis

Barium esophagogram can help confirm the diagnosis as well as potentially diagnose the underlying cause. Most patients will need EGD for diagnosis, biopsies, and potentially treating some causes such as stricture where endoscopic dilatation can be done. Esophageal manometry is indicated if the above workup is negative and motility disorders are suspected as the potential etiology of dysphagia.

Treatment

The goals of treatment for dysphagia include treating the underlying condition as well as maintaining nutrition and preventing complications such as aspiration. Modifications of the diet to allow for food with less aspiration potential as well as maintenance of appropriate posture can help patients with oropharyngeal dysphagia. Dysphagia due to stricture may respond to endoscopic dilatation. Dysphagia due to achalasia/increased lower esophageal sphincter tone may benefit from LES dilation, botulinum injection, or laparoscopic Heller's myotomy. More recently, peroral endoscopic myotomy (POEM), a minimally invasive endoscopic procedure to treat achalasia, has been introduced, which may be preferred in the elderly population because of its minimally invasive nature. Patients with diffuse esophageal spasm may benefit from drugs that reduce smooth muscle contractions such as calcium channel blockers, nitrates, and anticholinergic drugs; however, they should be used with caution in the elderly because of potential side effects and drug interactions. In some cases, patients need artificial modes of feeding such as a peg tube or gastrojejunal tube, and

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such decisions need to be individualized and should be made with patient participation.

Disorders of the Stomach

Age-related alterations in gastric microbiome, motility, blood flow, and protective mechanisms contribute toward changes in gastric physiology,[11] which can influence the presentation as well as management of gastric diseases in this population. The common gastric ailments in elderly are described below.

Peptic Ulcer Disease (PUD)

PUD is defined as a break in the lining of the stomach or duodenum where it can manifest as an ulcer. There has been a decline in the incidence and prevalence of PUD, but the incidence of complicated PUD remains high in the elderly population and is associated with higher hospitalization and mortality rates. This may be a result of a high prevalence of unrecognized *H. pylori* infection in the elderly population, higher use of medications like NSAIDs, antiplatelet agents, and anticoagulant medication, as well as an age-related decrease in the cytoprotective mucus-bicarbonate barrier and cell proliferation in the gastric wall.[12] Approximately 50% of the world population is affected by chronic *H. pylori* infection. It is more common in the elderly in developed countries (and in younger age groups in developing countries), likely because of the cohort effect of earlier generations in developed countries with poor sanitation.[13]

Clinical Features

PUD commonly presents as epigastric abdominal pain, nausea, vomiting, hematemesis, coffee ground emesis, and melena. The clinical presentation in the elderly can be subtle and nonspecific, meaning the PUD may go unnoticed and continue to progressively worsen, leading to perforation and chemical peritonitis.

Hemodynamic resuscitation including transfusion of packed red blood cells, endoscopic evaluation (preferably within 12 hours of clinical presentation), and management with endoscopic therapies (heater probe, laser coagulation, or injection of vasoactive drugs) should be considered for bleeding PUD. The endoscopic treatments significantly reduce the rebleeding risk as well as the need for surgical interventions. In cases of recurrent bleeding after endoscopic therapy, imaging guided embolization of the artery feeding the bleeding ulcer can be considered prior to surgical management.

PPIs are recommended for 8–12 weeks. Endoscopic healing is the gold standard for confirming healing of ulcers, and a surveillance repeat endoscopy to confirm healing and rule out malignancy in case of persistent ulcer changes is recommended. Elderly patients on chronic NSAIDs or antiplatelet agents and who have an ulcer are at high risk of rebleeding; continued longer-term PPI use can be considered, as the benefits may outweigh risks.

Testing of *H. pylori* by histological evaluation of biopsies taken during endoscopy is important. One needs to be careful when selecting the biopsy site, as *H. pylori* may be more common in gastric body and fundus and not the antrum in patients on PPI. It is also reasonable to consider that there will be a lower prevalence of *H. pylori* in the gastric biopsy of an elderly patient with chronic atrophic gastritis. Thus, if there is a high index of clinical suspicion for underlying *H. pylori* infection, a second modality of *H. pylori* testing with rapid urease test or *H. pylori* stool test should be performed. In some cases, if the elderly patient is from a geographic area of high prevalence of *H. pylori* infection, empiric treatment may be considered, despite a negative test.

Treatment options for *H. pylori* include triple therapy with PPI and combination of antibiotics. Low patient compliance and antibiotic resistance are the two main reasons for failure of treatment. It is advised to avoid repeating same antibiotics if used recently for any reason, including failed *H. pylori* treatment. Retreatment in such cases can be guided by culture and sensitivity results from a biopsy specimen obtained endoscopically. Rifabutin-containing regimens are reserved for patients who have failed more than three antibiotics regimens.

The treatment for *H. pylori* is not considered complete unless eradication is confirmed with a follow-up noninvasive method or histological evaluation with repeat endoscopic biopsy (if an endoscopy is clinically indicated). A surveillance test should be performed at least 1 month after completion of antibiotic therapy, and patients need to be off PPI for 4 weeks to minimize the chances of false-negative test results. Serology (IgG antibodies to *H. pylori*) cannot be used for surveillance, as antibodies, once formed, remain positive for life. A urease breath test has slightly higher sensitivity and specificity for hospitalized frail patients than a stool test while on antisecretory medications or antibiotics.

A comprehensive geriatric assessment for risk, based on comorbidities, is crucial in evaluating the bleeding risk of an elderly patient. In such geriatric patients, a low-dose

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NSAID or selective inhibitors of Cyclo-oxygenase-2 (Coxibs) with short plasma half-life, which are less disruptive to the protective mechanism for gastric mucosa, can be considered. Slow-release formulations with augmented ulcer risks should be avoided in the elderly population. A conscious effort toward a more mindful approach while prescribing NSAIDs and discontinuing the prescription as soon as the short-term clinical objective is met improves clinical outcomes by reducing GI bleeding related to PUD in the elderly.

Atrophic Gastritis (AG)

AG is chronic inflammation and thinning of the gastric mucosa with a decrease in glands, leading to achlorhydria and hypochlorhydria. It can sometimes be associated with change in the morphology of the epithelial cells known as metaplasia. Metaplasia can be of two types: environmental metaplastic atrophic gastritis (EMAG) and autoimmune metaplastic atrophic gastritis (AMAG). Both overlap clinically and resemble each other histologically. AMAG exhibits reduced pepsin and acid production and is associated with loss of intrinsic factor, leading to pernicious anemia. It is associated with other autoimmune disease processes like type 1 diabetes, vitiligo, and autoimmune thyroiditis. EMAG is associated with environmental factors like *H. pylori* and other factors, including diet practices like high salt intake, cigarette smoking, alcohol consumption, and chronic bile reflux.[13] Patients with underlying EMAG changes have a 10- to 15-fold higher risk for gastric cancer (GC). In elderly patients with nonspecific dyspepsia symptoms, upper endoscopy with biopsy can be considered. At endoscopy, extensive atrophy, flattening of rugal folds, and pseudopolypoid mucosa is noted. Most patients may be asymptomatic but may manifest with nonspecific dyspepsia. The elderly commonly manifest with symptoms of vitamin B12 deficiency, which can often be vague and nonspecific (fatigue, irritability, cognitive decline, subtle neurologic-paresthesia, numbness and gait problems, cognitive and psychological changes), and often go unnoticed, or symptoms can be misdiagnosed with dementia. A high index of suspicion is needed, since if recognized, it is a treatable, reversible condition with vitamin B12 supplementation. These atrophic changes are also associated with osteoporosis in the elderly due to compromised calcium absorption. Treatment of underlying *H. pylori* infection is important to decrease the risk for gastric adenocarcinoma. The risk for gastric neuroendocrine tumors is also increased with underlying atrophic gastritis. Close surveillance with upper endoscopy is recommended for patients with intestinal metaplasia (every 2–3 years with no family history of GC, and every 1–2 years in patients with a family history of GC).

Gastric Cancer

GC is more common in the elderly. Half of the patients diagnosed with GC are over the age of 65 years. The World Health Organization has listed *H. pylori* as group 1 or definite carcinogen for GC. There are some known environmental risk factors like high-salt or salt-preserved foods such as salted fish, cured meat, and salted vegetables. In animal models, it has been shown that high-salt foods cause damage to stomach mucosa and increase the risk for carcinogenesis. Other major contributors to the risk for GC are Nitroso compounds from some cured meats, cheeses, and tobacco. Diets high in fried food, processed meats, and alcohol and low in fiber have been associated with increased risk of GC in several epidemiological studies. A meta-analysis of cohort studies of >9,000 GC cases showed increased risk of GC in patients with body mass index (BMI) >25 kg/m^2 (odds ratio OR 1.22, 95% CI 1.06–1.41).[14]

The most common form of GC is gastric adenocarcinoma (95%), followed by lymphoma (4%). Endoscopy is indicated if there are red flag signs in the history such as overt GI blood loss, weight loss, family history of gastric cancer, physical signs of abdominal mass, or lymphadenopathy. Once pathology confirms the diagnosis, further staging may need imaging studies such as CT abdomen and endoscopic ultrasound (EUS), based on which chemotherapy, neoadjuvant chemotherapy, radiation, and surgical management can be pursued.

Disorders of the Small Intestine

Celiac Disease (CD)

CD is a chronic autoimmune inflammatory disease in which genetically predisposed individuals develop intestinal and extraintestinal manifestations in response to dietary gluten protein. Typical symptoms suggest malabsorption syndrome and include diarrhea, weight loss, failure to thrive, and nutritional deficiencies. Many patients present with atypical CD with only extraintestinal manifestations, while some can be asymptomatic (silent CD). It is traditionally thought to be a disease of young people; however, the incidence in older patients is increasing.[15] The increased incidence in older patients may in part be related to delayed diagnosis with onset at

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a younger age; however, true onset for the first time in older age can occur in patients who tolerated gluten in the past.[16] Up to 34% of newly diagnosed CD patients are older than 60 years[17] and 20% of celiac disease patients in high-prevalence areas of the world (such as Europe and North America) are over age 65.[18] As with other autoimmune diseases, celiac disease is more prevalent in women, especially in young women; however, its incidence gradually decreases in women and increases in men after the age of 65 years.[16] GI symptoms are often less prominent in elderly patients with CD, who often present with micronutrient deficiency.[19] Iron deficiency anemia is seen in up to 80% of the patients.[20] Abdominal symptoms in the elderly are often vague and nonspecific such as abdominal bloating, flatulence, and abdominal discomfort, making diagnosis challenging. Calcium and vitamin D deficiency can develop, which can lead to metabolic bone disease, and this predisposes elderly people to fracture given their tendency to fall. Liver involvement in the form of celiac hepatitis and manifesting as hypoalbuminemia, elevated liver function tests, and ascites is seen in up to 20% of patients and is not autoimmune mediated.[21,22] This contrasts with cholestatic liver diseases such as primary biliary cirrhosis (PBC), primary sclerosing cholangitis (PSC), and autoimmune hepatitis (AIH), which are immune mediated and are more common in CD as well. Interestingly, unlike celiac hepatitis, which usually responds to a gluten-free diet, cholestatic liver disease has a less favorable response to gluten cessation.

Some patients with CD present with neurological manifestations of celiac disease such as dementia, ataxia, and neuropathy, which makes diagnosis challenging because of wide range of differential diagnosis of these symptoms in the elderly and the lack of awareness among providers about atypical presentation of CD. In the geriatric population, dementia is often attributed to Alzheimer's or vascular disease and gait disturbances are thought to be due to advanced age and deconditioning, when in fact they may have atypical CD. Lymphoma of the small intestine can develop in patients who have untreated celiac disease for years, who have not been compliant with a gluten-free diet, or who develop celiac disease in older age, and can present as an ulcer or a tumor.

Management of CD involves avoidance of gluten in the diet. This can be particularly challenging in the elderly, as they may find complying with a strict gluten-free diet more difficult. This may be related to social factors such as residence in an assisted living facility, difficulty in precisely reading and interpreting the ingredients while shopping, and the fact that they may find that long-term eating habits are often hard to break. Identifying some of these barriers and having a detailed discussion with the elderly patient is important. Some elderly patients may be highly motivated to adhere to a gluten-free diet given that advantages are widespread, including improvement in symptoms, correction of nutritional deficiencies, prevention of osteoporosis, improvement in mood, appetite, fatigue, and quality of life, and reduced risk of intestinal lymphoma.

Mesenteric Ischemia (MI)

MI is due to obstruction in the celiac axis arterial branches. It can be acute or chronic. It can be caused by arterial occlusion due to embolism (which is more prevalent in superior mesenteric artery), thrombosis, or hypoperfusion (usually in the setting of hypovolemia or hypotension). The blood flow in the splanchnic circulation decreases with age, making the elderly particularly vulnerable to nonocclusive mesenteric ischemia due to hypoperfusion, especially in the setting of clinical states that predispose to it such as cardiopulmonary bypass, hypotension due to sepsis, dehydration, or hypovolemia.

Patients with chronic mesenteric ischemia (CMI) have a classic presentation of postprandial angina, which refers to abdominal pain occurring after meals. This may result in avoidance of meals, decreased food intake, and weight loss. This contrasts with acute mesenteric ischemia (AMI) where patients typically present with acute abdominal pain, which is usually out of proportion to physical signs of acute abdomen; however, in the elderly, abdominal pain may be vague and minimal and sometimes absent. MI in the elderly can have vague symptoms such as tachypnea, confusion, nausea, vomiting, and diarrhea. The insidious onset of symptoms and nonspecific symptoms may make establishing the correct diagnosis of MI difficult in the elderly population.

AMI is a more common cause of acute abdomen than ruptured abdominal aortic aneurysm and appendicitis in patients older than 75 years.[23] Early diagnosis is somewhat challenging as the initial CT scan may be negative for the presence of occlusion/thrombus in the artery. A high index of clinical suspicion in elderly patients with multiple risk factors predisposing them to MI is important. Risk factors such as the presence of peripheral arterial disease, atrial fibrillation, and a prior history of myocardial infarction or stroke, along with suggestive laboratory findings such as leukocytosis, elevated lactic

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acid, and metabolic acidosis, and radiological findings such as thrombus in artery, increased bowel enhancement, and pneumatosis, can help make a diagnosis of AMI. It can be treated with endovascular therapy and open surgical revascularization, with endovascular therapy being preferred in the geriatric population because of the high risk of open surgery in view of advanced age and comorbidities.

Small Intestinal Bacterial Overgrowth (SIBO)

SIBO refers to a condition where there is an excessive presence of bacteria in the small bowel aspirate (more than 10^5–10^6 organisms/ml).[24] SIBO is noted to be more common in the elderly healthy population with a reported prevalence of 15.6% vs. 5.9% in healthy young adults.[25] Several factors predispose to development of SIBO, including achlorhydria, slow small bowel transit, alteration in gut immune system, small intestine dysmotility, and anatomical abnormalities such as the presence of fistula, anastomosis, or bowel resection. Achlorhydria may be related to increased PPI usage or *H. pylori* infection and leads to passage of more bacteria through the stomach, which overpopulate the small intestine, contributing to SIBO. Similarly, small bowel dysmotility leads to a slower rate at which the bacteria are swept away from the small bowel to colon, leading to SIBO. Even though the small intestinal motility is little affected by age itself, older patients often have slower small bowel transit related to polypharmacy and medication side effects as well as the presence of coexisting diseases such as diabetes and associated diabetic neuropathy. Diabetes, portal hypertension, scleroderma, polymyositis, and chronic renal failure are other risk factors for the development of SIBO.

SIBO presents as chronic diarrhea, malabsorption, weight loss, and secondary nutritional deficiencies.[24] Patients with SIBO often present with symptoms of diarrhea, nausea, and vomiting, although the elderly may not have a classic presentation, so that symptoms can be subtle and often nonspecific such as bloating, flatulence, abdominal distention, and poorly localized discomfort, which makes diagnosis challenging. The elderly sometimes present with complications including signs of nutritional deficiencies. Unlike vitamin K and folic acid, which are produced by gut bacteria, vitamin B12 deficiency is often noted in patients with SIBO because of the competitive uptake of vitamin B12 by the bacteria.

SIBO may be diagnosed by small bowel aspirate bacterial analysis or more commonly with glucose or lactulose breath test. Treatment involves modification of diet with less carbohydrate, use of prokinetic agents to increase GI motility, and use of antibiotics to help reduce bacterial overgrowth. Prokinetic agents due to their drug interactions should be used with caution in elderly patients who often are already taking multiple drugs. Antibiotics do carry the risk of C. difficile colitis, which has high morbidity and mortality in the elderly population, making its use for SIBO in this population challenging. The use of prokinetic agents and antibiotics in the elderly should be individualized and should only be done after having a detailed discussion with patients regarding their risks and benefits.

Disorders of the Colon

Constipation. By Rome criteria, constipation is described as decreased frequency of bowel movements to three or fewer per week. It has been reported that there is a growing prevalence of constipation in up to 40% in the elderly population.[26] There has been conflicting evidence, with some studies suggesting that with growing age, because of the neurodegenerative process, there may be a slowing of colon motility, leading to constipation, while other studies suggest constipation is not a consequence of physiologic aging. There is an emerging discussion about other contributory factors such as age-related decreased mobility, diet changes, cognitive impairment, use of anticholinergic medications, polypharmacy, and other medical comorbidities, which play an important role.

Constipation is classified as primary or secondary. Primary constipation can be further divided into normal transit or functional constipation; slow transit constipation; or due to anorectal dysfunction. Evaluation of colonic transit is typically done with the Sitz marker test. Sluggish colon transit can be treated with medications that help trigger colon motility, like Linaclotide, prucalopride, and Misoprostol. Anorectal dysfunction is connected to age-related changes in anorectal physiology like pelvic dyssynergia or sphincter tears from urologic and gynecological procedures. Secondary constipation can be multifactorial and needs a comprehensive evaluation. In elderly patients, medications like opioids, calcium channel blockers, antacids, and iron supplementation, as well as comorbidities like hypothyroidism, hypercalcemia, Parkinsonism, spinal cord injury, scleroderma, and amyloidosis, need to be considered as possible etiologies of constipation.

It is common for the elderly to present to the emergency room with fecal impaction, which can be noted on

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rectal exam. However, in cases with proximal impaction it may not be felt on digital exam and can be diagnosed on imaging. It needs to be addressed promptly as it can often lead to stercoral ulcer and spontaneous colonic perforation, which can be associated with significant morbidity and mortality in the elderly population. One needs to consider a prompt and cautious approach with suppositories or water enemas, followed by polyethylene-based solution ingestion and continued use of medications like Senna and magnesium citrate to maintain a bowel regimen. Phosphate-based enemas should be avoided in patients with underlying kidney conditions to avoid complication with phosphate nephropathy. Chronic constipation may also present as overflow incontinence, which can be a counter-intuitive presentation with diarrhea caused by liquid stool getting around the hardened impacted stool.

Diverticulosis. This involves herniation of colonic mucosa through the submucosa of the colonic wall due to increased intracolonic pressures during straining related to constipation. It is often an incidental finding during colonoscopy and is noted in half of the patients by the age of 50 years and two thirds of patients by the age of 80 years.[2] Its pathophysiology is based on age-related colonic wall connective tissue change, together with an interplay of change in colon microbiome and colonic motility, which triggers colonic inflammation. There has been a change in thought process from its being an acute disease to a chronic medical disease process like irritable bowel syndrome, presenting with flares. Diverticulitis is one of the most common complication-related presentations and can be associated with bleeding, abscess, and micro-perforation. Younger individuals more commonly present with acute diverticulitis than the older population.

Inflammatory bowel disease (IBD). This chronic inflammatory condition of the GI tract is of two types: Crohn's disease and ulcerative colitis. IBD in the elderly can either be a manifestation of disease acquired at a young age (with a continued disease progression with aging) or can manifest itself for the first time at an age >60 years. Even though IBD is considered a disease of young age, the prevalence of IBD is around 10–30% in the elderly population.[27]

IBD happens in genetically predisposed individuals and is triggered by an abnormal immune response to varied gut antigens. Aging-related decline in T and B lymphocyte immune function triggers changes in microbiota, thereby altering the gut immune function. Genetics tend to play a more pivotal role in IBD in younger age groups than in the elderly (family history of IBD seen in 16% vs. 7%, respectively). The diagnosis is challenging in the elderly related to atypical manifestations, polypharmacy altering bowel function, and comorbidities. In the elderly with IBD, rectal bleeding is more common than diarrhea and is often perceived to be due to etiologies like hemorrhoids. As opposed to younger patients who have more structural and penetrating/fistulous disease, older-onset disease more frequently has an inflammatory phenotype. Older-onset CD often presents as isolated colon involvement and perianal fistula. Older-onset disease is usually less aggressive and progresses more slowly with less inflammation and fewer signs of disease, making diagnosis even more challenging.

The goals of IBD treatment include inducing and maintaining remission, minimizing complications, and improving quality of life. The mainstay of treatment for mild to moderate disease is mesalamine, which is used topically as suppository to address proctitis, enema for inflammation involving the sigmoid colon, and a combination of oral and topical therapies for quick response for acute presentation of disease. Some challenges with use of mesalamine in the elderly include high rates of nonadherence (related to pill size, frequent dosing, and GI side effects), higher rates of fecal incontinence, and a higher risk of nephrotoxicity related to a higher prevalence of chronic kidney disease. In patients with moderate to severe disease, corticosteroids are effective in inducing remission but are associated with myriad side effects like osteoporosis, diabetes, glaucoma, and hypertension, which is especially relevant in elderly patients. The immune-modifying agents are often underutilized in the elderly, and the side effects from these agents are not increased in the elderly compared to younger patients. Failure of medical therapies often leads to surgical management. Given complex management with these immunosuppressive medications, it is recommended to refer to a gastroenterologist for specialized care.

Clostridium Difficile (C. Difficile) Colitis (CDC)

CDC refers to neutrophil-predominant inflammation of the colon caused by *C. difficile* infection. *C. difficile* is an anaerobic gram-positive, spore-forming bacteria that generates toxin A and toxin B and damages the colonic epithelium, leading to its hallmark manifestation of pseudomembranes. Eighty percent of CDC is seen in patients over 65 years.[28] The elderly are not only particularly

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vulnerable to it but also have higher morbidity and mortality related to it. CDC is related to the use of antibiotics, which alter the gut microbiota, predisposing it to colonization by C. difficile. Patients who can mount an early immune response to C. difficile become a carrier, while patients with compromised immunity often develops symptoms that can vary from mild diarrhea to more severe presentation leading to dehydration, acute kidney injury, and potentially life-threatening toxic megacolon and sepsis. Elderly patients as well as nursing home and hospitalized patients are particularly vulnerable to developing severe forms of CDC and its complications. Age is not only a risk factor for severe forms of disease but also predisposes to recurrent CDC. Age over 65 years, severe comorbidities, and additional antibiotic use after initial treatment have emerged as the three major clinical risk factors to predict the recurrence of CDC.

Treatment of CDC involves discontinuation of inciting antibiotics, intravenous hydration, and initiation of anti-C. difficile antibiotics including oral vancomycin or oral fidaxomicin or metronidazole. Traditionally, both oral vancomycin and metronidazole were believed to be equally effective; however, in the elderly, increased treatment failure is noted with metronidazole compared to vancomycin. Vancomycin is now believed to be superior, especially in the elderly with severe disease, and is recommended as an "initial" empiric therapy for suspected CDC. Another advantage of oral vancomycin is that it is not systemically absorbed unlike metronidazole, which because of its systemic absorption can cause side effects such as dysgeusia, nausea, seizures, peripheral neuropathy, or encephalopathy. Having a better side-effect profile is especially important in elderly who are more vulnerable to drug toxicities and interactions. Infection control measures such as placing patients on contact precautions is essential to prevent spread of infection. Patients with recurrent CDC despite multiple antibiotic treatments can be treated with fecal microbiota transplant (FMT). Elderly patients who have FMT to treat recurrent CDC have a higher recurrence rate of about 9.3% compared to younger counterparts, who have a much lower recurrence rate with FMT of about 4.6%.[29]

Irritable bowel syndrome (IBS). This is one of the functional disorders triggered by altered gut motility, gut microbiome, psychosomatic factors, and visceral hypersensitivity, which can manifest itself with abdominal pain associated with diarrhea or constipation with no underlying organic etiology. Diagnosis is based on Rome criteria. The prevalence of IBS is equal in both elderly and young age groups.[30] However, the incidence is higher in adolescents, and it is rarely diagnosed for the first time in patients over 65 years of age. Diagnosis is challenging in the elderly, as they have myriad vague symptoms related to their comorbidities, polypharmacy, and coexistent depression. Alarm features include unintentional weight loss (which may also be related to depression, leading to a lack of interest in eating and change in eating habits and metabolism) and GI blood loss (which may also be common in the elderly related to anorectal etiology like hemorrhoids). Alarm symptoms need a well-balanced diagnostic approach between accurate assessment, avoiding misdiagnosis, and minimizing unnecessary invasive procedures with related anesthesia risks in elderly patients. Serotonin is a neurotransmitter that has also been recognized as a key player in the pathophysiology of IBS. Increased concentrations of serotonin can lead to diarrhea, while decreased concentrations can drive symptoms of constipation in an IBS patient. Keeping this in mind, treatment strategies with selective serotonin reuptake inhibitors can be used. Other treatment options like tricyclic antidepressants help with neuromodulation to assist with symptom control. Antispasmodics and antidiarrheal agents are also helpful in patients with abdominal cramps and diarrhea. In the elderly population, one needs to be mindful of potential worsening of urinary retention, closed-angle glaucoma, and cognitive impairment with use of medications with anticholinergic side effects. The role of brain–gut dysfunction, triggered by a close interplay of psychological conditions like stress, anxiety, and depression, particularly needs to be addressed in the elderly who may have inadequate social support systems. It is important to discuss relaxation techniques and refer the elderly patient for cognitive-behavior therapies, which would empower them to combat psychological factors driving IBS-related GI symptoms.

Malnutrition in the Elderly

Many elderly patients are at risk of malnutrition. The presence of malnutrition in the elderly is associated with increased mortality, increased risk of hospitalization, increased health-care costs, and decreased quality of life. Various factors influence nutritional adequacy in the elderly, which may be physiological (such as decreased sense of taste or smell, delayed gastric emptying, dysregulation of satiation), pathological (such as poor dentition, dysphagia, diseases such as cancer, congestive heart failure, and COPD, which are more prevalent in the

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elderly, polypharmacy, and dementia), sociological (such as lack of ability to shop for food or prepare food, need for feeding assistance, strained financial status, impaired activities of daily living), and psychological (such as depression, anxiety, loneliness, and grief).[31] Depression is one of the most important causes of weight loss, and malnutrition may be the presenting symptom of depression in the geriatric population. Elderly patients with malnutrition should be screened for subclinical depression.

The diagnosis of malnutrition in the elderly involves assessment of dietary history, body weight, weight trend, and evaluation of muscle wasting as well as laboratory data such as hypoalbuminemia, hypocholesterolemia, low pre-albumin, and the presence of other nutritional deficiencies such as vitamin B12 or folate, or low lymphocyte count. Albumin and pre-albumin in hospitalized elderly patients can be low because of inflammatory disease and may not always be a good nutritional measure in the setting of acute illness. Various screening tools such as the Mini Nutritional Assessment can be used to screen the elderly for the presence of malnutrition.

Treatment of malnutrition in the elderly needs a multidisciplinary approach, which not only treats the pathology but also addresses the nutritional, social, and psychological factors. Percutaneous tube-feeding (peg tube) is not recommended routinely in elderly patients with advanced dementia. It is associated with agitation, increased use of mechanical and pharmacological restraints, and higher risk of pressure ulcers in this population. Supervised oral hand-feeding is preferred and is believed to be as good as percutaneous tube-feeding in terms of outcomes such as death, patient comfort, aspiration risk, and functional status. Environmental changes should be made to facilitate supervised oral hand-feeding, and individualized patient-directed approaches should be adopted to enhance oral feeding in older patients with advanced dementia. It is important to promote informed decision-making and honor patient preferences when making decisions about tube-feeding.

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Chapter

24 Serious Infections

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Introduction

While modern medicine has significantly reduced early death due to infection, diseases caused by infectious pathogens remain a major cause of illness and death among elderly persons.[1] Many of the most serious infectious diseases have a predilection for those at the extremes of age – individuals with relatively deficient immune function. In addition, infections common to persons of all ages can be devastating when they occur in those of more advanced age. Elderly individuals also are now commonly found in environments, such as hospitals and nursing facilities, where antibiotic-resistant organisms are prevalent, and catheters and wounds breach the protection offered by an intact integument. At the other end of the functionality spectrum, many older individuals are active and may spend their postretirement years traveling to locales where they are exposed to exotic organisms.

The Elder Host

Age-related changes have been described in various components of the immune system. Part of the increased burden of infections in elders may be due to changes in the adaptive and innate components of immunity: a negative correlation between age and immunity is observed in most studies.[2–4] Changes in innate immunity include decreases in T-cell activation by dendritic cells, cytotoxicity by natural killer cells, CD80 upregulation and TLR1/2 function.[5–8] The most generally accepted changes in the adaptive immune system are thymic involution and a resultant decrease in the output of naïve T-cells, and the accumulation of memory and CD28-null T-cells in the peripheral blood of elderly individuals.[2–4,9,10] Vaccine responses become muted along with diminished delayed hypersensitivity in older persons.[11,12] The important barriers to infection, such as skin and mucosal surfaces, also weaken. Skin becomes thinner and glandular secretions decrease and, along with age-related immune deficits, raise the risk for soft tissue infection and/or systemic spread.

A number of conditions associated with aging also enhance the risk of infectious diseases. These include diabetes mellitus, malignancies, chronic obstructive pulmonary disease, and bladder-emptying disorders. As we age, the need for prosthetic joints and organ/tissue transplantation increases. Malnutrition secondary to comorbid disease, poverty, poor dentition, or other causes of inadequate caloric intake further reduces host defenses against infection.

The Environment

Approximately 1.2 million individuals aged 65 years and older live in a nursing home.[13] In addition, many elderly individuals regularly attend clinics or require hospitalization.[13] These environments and health-care encounters increase the risk of contact with infectious pathogens – including organisms with resistance to antimicrobials, outbreaks of endemic infections, and nosocomial infections. The illnesses that are common with advancing age often lead to instrumentation, catheterization, and surgery, each of which carries a risk of infection. Table 24.1 summarizes factors related to increased vulnerability to infection during aging.

Among more functional older patients, it is mandatory to take a complete travel and sexual risk behavior history. Older Americans comprise a significant proportion of those traveling domestically and internationally. Sexual activity may diminish as we age, but substantial numbers of persons in their later decades of life have sex, especially if their health is relatively good. New partnerships can result in sexually transmitted infections, and the clinician should never discount the possibility of such an infection based solely on the age of the patient. The Centers for Disease Control and Prevention and the US Preventive Disease Task Force recommend that human immunodeficiency virus (HIV) testing should be ordered at least once for all persons seeking health care who are

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Table 24.1 Factors increasing vulnerability to infectious diseases during aging

Host	Environment
Immunological	Nursing home residence
↓ Naïve T-cells	Hospitalization
↓ CD8 + cells	Instrumentation
↓ IgM memory B-cells	Exposure to drug-resistant organisms
↓ Toll-like receptor function	Exposure to endemic infectious diseases
↓ NK cell cytotoxicity ↓ Dendritic cell function	
Mechanical Skin thinning and breakdown	
↓ Glandular secretions	
↓ Cough reflex Poor dentition	
Systemic Cognitive impairment	
Malnutrition	
Tobacco use	
Poverty	

aged 13–65 years.[14] Many experts believe that testing should continue to be offered to those older than 65 years of age if they remain sexually active and at risk of acquiring HIV infection.

Approach to the Elderly Patient with Suspected Infectious Disease

The diagnosis of infectious diseases in elderly patients can be challenging. Classic features of infection such as fever and leukocytosis may be absent in older individuals even during fulminant infection, dangerously delaying diagnosis.[15] Approximately 40% of older adults may not mount a febrile response to serious infection.[16] Therefore, small elevations in temperature above individual baseline should be concerning, and fevers of 38.3°C are significant.[17] Localizing symptoms of infection may be subtle, and impaired cognition because of dementia or as a manifestation of the infection may render the patient unable to describe symptoms accurately. Infections among older patients can present atypically with vague aches, anorexia, or confusion as the only indication that an acute illness is present, and delirium is common.[18] A high degree of suspicion for underlying infectious processes is necessary when older individuals present with subtle changes. Failure to consider the possibility of infection in the elderly patient and overreliance on indicators of infection that are more common among younger patients can lead to tragic misdiagnosis of potentially treatable conditions.

Major Infectious Diseases

Urinary Tract Infections

Infection of the urinary tract is the most common bacterial infection in older adults and the major source of bacteremia in this population. Urinary catheters – urethral, condom, and suprapubic – greatly increase urinary tract infection (UTI) risk.[15] Furthermore, host factors including neurogenic bladder, prostate enlargement, vaginal atrophy, and increased vaginal pH can foster bacterial colonization that predisposes to UTI.[15] Frequent bladder emptying is protective against urinary infection but often impaired in older patients. Factors that lead to poor bladder emptying in the elderly include an increased volume of urine required to sense a need to void, as well as reduced urinary flow because of poor fluid intake, obstruction, and/or decreased bladder contractility.[19]

Clinical Manifestations/Diagnosis

Classic symptoms of UTI include dysuria, urinary frequency and urgency, suprapubic tenderness, costovertebral angle tenderness, and fever. An atypical presentation of UTI can manifest as symptoms of nausea, vomiting, dehydration, and delirium. Urinalysis of a clean catch specimen demonstrating pyuria, increased leukocyte esterase, and nitrite is highly suggestive in the setting of the aforementioned clinical presentations. Urine culture is recommended to provide guidance for antibiotic therapy. Blood culture should be obtained in patients with acute illness in order to assess for sepsis. In addition, a catheter-associated UTI in patients with indwelling urethral, indwelling suprapubic, or intermittent catheterization is defined by the presence of classic symptoms of UTI and a urine sample showing 1,000 CFU/mL or greater of 1 bacterial species from a catheterized specimen or a midstream specimen if the catheter has been removed within the past 48 hours.[20]

Asymptomatic Bacteriuria

Quantitative clean catch urine culture revealing 100,000 CFU/mL or greater in persons without genitourinary

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symptoms of UTI is considered evidence of asymptomatic bacteriuria. Asymptomatic bacteriuria is common in cognitively and/or functionally impaired older adults. In addition, up to 50% of women and 30% of men in nursing facilities can have asymptomatic bacteriuria.[21] Current evidence supports careful observation of these patients who have bacteriuria with non-localizing symptoms (such as behavioral changes) and no systemic signs of infection.[22] Observational and randomized studies do not show a significant impact on behavior, functional recovery, or mortality with antimicrobial administration in older adults with asymptomatic bacteriuria.[23–25]

Management

Gram-negative organisms are the most commonly cultured UTI pathogens, and empiric therapy should be directed toward these organisms until culture results return.[18] The recommended treatment duration for uncomplicated cystitis is 3–5 days, with a longer duration of 10–14 days recommended for complicated cystitis and pyelonephritis.

Patients with indwelling urinary catheters are at increased risk for less common pathogens such as *Enterococcus.* In patients with this gram-positive organism, cephalosporin resistance is common, and coverage with appropriate antibiotics such as ampicillin is necessary. Detection of *Staphylococcus aureus* in the urine raises concern for endovascular infection because this organism is often hematogenously spread to the urine. Blood cultures and echocardiography are indicated when *S. aureus* is retrieved from clean catch urine specimens.

Candida is sometimes encountered in the urine of elderly patients, especially those treated with broad-spectrum antimicrobials or with indwelling urinary catheters. Treatment is typically unnecessary as this represents colonization and not active infection.[26] In contrast, in patients who are immunocompromised or will receive urological instrumentation, treatment of candiduria should be considered.[27,28] Reducing risk factors for candiduria by removing indwelling urinary catheters when feasible is strongly recommended.

Bacterial Pneumonia

Respiratory infections are a leading cause of infectious-disease-associated deaths among older individuals and can be acquired in the community or at nursing/medical facilities.[1] A number of factors conspire to raise the risk of pneumonia in the elderly, including a decline in pulmonary function, diminished cough reflex, reduced mucociliary transport, and decreased lung elasticity.[29–31] These mechanical factors lead to trapping of air, diminished ability to clear oral secretions, and colonization of the pharynx with pathogenic bacteria.[31] Aspiration of secretions is a major cause of pneumonia among elderly patients with impaired swallowing and/or cognition and is exacerbated by poor dentition. Prior or current smoking and its sequelae further enhance the risk of respiratory infections among older patients.

Clinical Manifestations/Diagnosis

As with other infections in the elderly, pneumonia may not be heralded by the usual signs and symptoms. Cough may not be prominent, fever can be absent or mild, and shortness of breath subtle. Nonspecific symptoms of confusion or other mental status change, lethargy, and falling may be the initial indications that something is amiss.[32,33]

A chest radiograph should be considered when the physician is confronted with such changes and has clinical reason to suspect respiratory infection. X-rays often reveal an infiltrative process, but the absence of an infiltrate on the film does not preclude pneumonia, as dehydration may minimize radiographic evidence of infection.[34] Imaging findings should thus be correlated with respiratory signs and symptoms, as well as compared to prior films. Sputum analysis, although valuable in the identification of causative organisms, is rarely obtainable, as older individuals may be unable to cooperate with specimen collection or expectorate. When respiratory secretions can be obtained, Gram stain and routine bacterial culture should be performed. Blood cultures may also yield an organism associated with pneumonia. Other noninvasive or minimally invasive studies to be considered include multiplex polymerase chain reaction (PCR) for respiratory viruses on nasopharyngeal cells, obtained by either swabbing the nasopharynx or performing lavage, and urinary antigen testing for *Streptococcus pneumoniae*. In select cases, testing for *Legionella pneumophila* and/or *Histoplasma capsulatum* should be considered by urinary antigen testing. Viral and other atypical pneumonias, malignancy, and pulmonary embolus should also be considered in the differential diagnosis of the older patient in whom pneumonia is suspected.

Management

Treatment of bacterial pneumonia should be guided by sputum gram stain and culture. However, given the difficulty of establishing a specific bacterial cause of

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Table 24.2 Causes of pneumonia in the elderly

Community acquired	Nursing facility associated	Hospital associated
S. pneumonia	Enteric gram-negative bacilli	Enteric gram-negative bacilli
H. influenzae	Oral aerobes and anaerobes	Oral aerobes and anaerobes
Enteric gram-negative bacilli	*S. aureus*	*S. aureus*
S. aureus	*S. pneumonia*	*S. pneumonia*
Legionella pneumophila	*H. influenzae*	*Legionella pneumophila*
Mycoplasma pneumoniae	*Moraxella catarrhalis*	*Moraxella catarrhalis*
Chlamydia pneumoniae	Influenza	*Pseudomonas* spp.
Influenza	Other respiratory viruses	*Acinetobacter* spp.
Respiratory syncytial virus		*Stenotrophomonas* spp.
Other respiratory viruses		Influenza
Pneumocystis jiroveci		Other respiratory viruses

pneumonia via sputum analysis and the seriousness of such infections in elderly patients, empiric therapy directed at the likely culprits is prudent and recommended.[35,36] In community-acquired pneumonia, *Streptococcus pneumoniae*, *Hemophilus influenza*, enteric gram-negative bacilli, influenza, and other respiratory viruses are most common; however, *S. aureus* and atypical organisms such as *Mycoplasma*, *Legionella*, and *Chlamydia* also occur.[37] Patients residing in nursing homes also experience pneumonia caused by the enteric gram-negative organisms, oral aerobes and anaerobes, and *S. aureus*.[29] These organisms (see Table 24.2) are responsible for the major share of pneumonia in the hospitalized patient, but more unusual organisms (including *Acinetobacter* and *Pseudomonas*) may also cause disease. Patients who are more ill and those unable to tolerate oral intake may require inpatient care and intravenous administration of antibiotics if consistent with their goals of care.

Antibiotic choice must be guided by host and environmental factors such as concomitant illnesses, risk of aspiration, and the setting in which the patient resides. Broader coverage – taking into account drug-resistant organisms and anaerobes – is typically indicated in institutionalized patients compared with those who live at home who may be able to be treated initially with an anti-pneumococcal fluoroquinolone, third-generation cephalosporin, or macrolide, depending on local drug susceptibility patterns. Treatment of most bacterial pneumonia lasts for 5–7 days, with duration predicated upon multiple factors including clinical improvement and microbial etiology. Longer durations for complicated infections may be necessary.[35,38]

Detection of a specific organism can lead to the narrowing of antibiotic therapy. Failure to detect improvement during therapy may indicate that the selected therapy is suboptimal and that a change in antibiotics is required. In such cases, the presence of underlying immunodeficiency, such as that from HIV infection, and atypical infections caused by fungi or *P. jiroveci* should be considered.

Patients with pneumonia who are moderately to severely ill should be considered for hospitalization. Findings associated with poor prognosis in elderly patients with community-acquired pneumonia include PaO_2 <60 mm Hg, O_2 saturation <90%, altered mental status, heart rate higher than 125 beats/minute, respiratory rate higher than 30/min, hypo- or hyperthermia, leukocytosis or leukopenia, anemia, hyponatremia, hyperglycemia, multilobar infiltrates, and pleural effusion.[15] In addition, older patients with pneumonia and significant comorbid diseases such as malignancy, immunodeficiency, renal or hepatic insufficiency, or cardiovascular disease may also require inpatient monitoring.

The risk of pneumococcal pneumonia can be reduced by vaccination. The Advisory Committee on Immunization Practices (ACIP) recommends a single dose of pneumococcal polysaccharide vaccine-23 (PPSV23) for all adults 65 years of age and older. Patients who are naïve to the pneumococcal conjugate 13-valent (PCV13) and have immunocompromising conditions, cerebrospinal fluid leak, or cochlear implants should also receive a single dose of the 13-valent pneumococcal conjugate vaccine (PCV13). For other adults over 65 years of age, shared decision-making is recommended regarding PCV13,

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which if administered should be given at least 1 year prior to PPSV23.[39]

Influenza

Influenza is a cause of viral pneumonia occurring generally in the winter months in the United States. Data suggests that the virus thrives in cool temperatures with limited humidity, accounting for its seasonality.[40] Community and institutional acquisition occur under these conditions because this is a highly infectious virus with an incubation period of only 2–3 days. The infection and its complications can be lethal in elderly individuals.

Clinical Manifestations/Diagnosis

Elderly patients with influenza may present atypically with the triad of cough, fever, and acute onset less evident than mental status alteration, generalized malaise, and other nonspecific complaints.[41,42] Among patients living in institutional settings, the report of a similar illness or diagnosed influenza among other residents or staff is an important epidemiological clue. Secondary bacterial infection with streptococci or staphylococci occurs and typically manifests as a period of worsening of disease after an initial improvement. The chest radiograph may demonstrate bilateral infiltrates, suggesting viral pneumonia. Definitive diagnosis is made on nasopharyngeal samples through rapid antigen or PCR testing. The benefit of point-of-care rapid antigen testing must be seriously weighed against a decreased sensitivity.[43]

Management

Antiviral drugs active against influenza include the adamantine derivatives, amantadine and rimantadine, neuraminidase inhibitors such as oseltamivir and zanamivir, and polymerase inhibitor baloxavir marboxil.[44–46] The adamantine derivatives are only active against influenza A and do not cover influenza B strains. They are of limited use, given the widespread resistance found in circulating strains of the most recent influenza seasons and their association with reversible adverse effects involving the central nervous system. Baloxavir marboxil has activity against both A and B strains; however, it has not been studied in adults over 65 years, and its efficacy and safety profile in this population are unknown.[47]

Neuraminidase inhibitors are the drug of choice to treat influenza.[44] Starting treatment early after the onset of symptoms is key to maximum efficacy. However, in elderly patients who are ill or need to be hospitalized, treatment should not be withheld even if several days have passed since symptom onset. Neuraminidase inhibitors can also be used as prophylaxis in institutionalized elderly patients in outbreak situations. When two institutional residents manifest signs and symptoms of influenza-like illness within 72 hours of each other, testing for influenza should occur.[46] When influenza viruses are circulating in the community, even one positive laboratory result in conjunction with other compatible illnesses on the unit indicates that an outbreak of influenza is occurring. In these outbreaks, all residents should receive chemoprophylaxis, regardless of vaccination status. Quarantining of cases does not appear to be a useful strategy.

The most effective method of preventing influenza and reducing its impact on morbidity and mortality of older adults is vaccination. The influence of aging on the immune system is associated with a decreased response to the standard-dose inactivated influenza vaccine. This decreased response has led to the development of high-dose quadrivalent inactivated vaccines, recombinant vaccines, and adjuvanted vaccines, all of which demonstrate increased immune responsiveness in older adults.[48–50] Any of these vaccines are recommended annually for those 65 years and older and younger individuals with chronic medical conditions or who reside in confined settings.[51] Additionally, individuals such as healthcare workers who have frequent contact with high-risk older adults should also be vaccinated annually to further reduce the transmission of influenza to vulnerable older adults.

Pulmonary Tuberculosis

More than half the cases of tuberculosis (TB) in the United States are diagnosed in individuals 65 years of age or older.[52] Age-related waning of cellular immunity, comorbid conditions, immunosuppressive conditions, and malnutrition increase the risk of reactivation of latent TB in the elderly. Primary acquisition of TB also occurs among the elderly, and transmission of TB within nursing facilities is well documented.[52]

Clinical Manifestations/Diagnosis

Pulmonary TB presents generally with insidious onset of weight loss, fever, night sweats, cough, and hemoptysis. Nonspecific constitutional complaints may mask the more classic symptoms, and the diagnosis of TB should be considered in elderly persons with "failure to thrive." Chest films may reveal an area of infiltration – often in the upper lobes – or a cavitary lesion, but patterns similar to bacterial pneumonia can also be seen.[53] The diagnosis

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is made microbiologically with the culture of sputum for *Mycobacterium tuberculosis*. Given the difficulty of establishing a quick diagnosis, empiric therapy is considered when suspicion of TB is high, such as in a patient with a classic chest film and a positive tuberculin skin test. Rapid tests for TB are now available and are generally used to confirm the presence of TB in respiratory specimens that reveal acid-fast bacilli (AFB).[54]

The United States Preventive Services Task Force (USPSTF) recommends screening for latent tuberculosis infection in persons who are born in or are former residents of countries with increased tuberculosis prevalence, as well as in persons who live in high-risk congregate settings.[55] Tuberculosis screening is required on admission to long-term care facilities. Tuberculin skin testing is useful for determining prior exposure to TB. As the test relies on cellular immune responses, the false-negative rate of the test increases during advanced age. In some individuals, skin testing itself can boost immune responses to tuberculin such that repeated testing will become positive.[56] Bacillus-Calmette-Guérin (BCG) vaccination may also produce skin reactions to tuberculin. For those who received the vaccine as a child, the cross-reactivity to the skin testing should wane by adulthood and not be a significant factor in elderly individuals. Those who receive BCG vaccination as adults should have skin testing done several months after vaccination to establish a baseline test reaction size.[57]

Interferon-gamma release assays (IGRAs) are also used to detect latent TB and can be used in all circumstances in which tuberculin skin testing is performed.[58–60] These assays are not affected by BCG vaccination status and are useful for evaluation of latent tuberculosis in BCG-vaccinated individuals.[57]

Management

The therapeutic management of pulmonary TB in the elderly patient is not different from management in younger patients.[61] Drug therapy typically consists of four drugs (isoniazid, rifampin, pyrazinamide, and ethambutol) administered for 2 months. Provided drug susceptibility testing indicates sensitivity to isoniazid and rifampin, treatment can be simplified to these two agents for the remainder of the course. As polypharmacy is common in many elderly patients, care must be taken to avoid drug–drug interactions between anti-TB and other medications. Monitoring for drug toxicity, especially changes in hepatic transaminases, is prudent.

Latent TB should also be treated in elderly individuals. More recent data indicates the risk of isoniazid-related hepatitis is rare, with only one case per 1,000 persons; however, the incidence was not analyzed according to age.[62] Patients should be counseled to abstain from alcohol during treatment with isoniazid to decrease the risk of isoniazid-related hepatitis. In addition, peripheral neuropathy from isoniazid can be prevented with pyridoxine supplementation.[63]

Herpes Zoster

Herpes zoster, or shingles, is a common condition associated with advancing age. The causative organism is the varicella zoster virus (VZV), which causes chickenpox in younger persons and, like all herpes viruses, remains latent within the dorsal root ganglion following the initial infection. With diminished immune function subsequent to aging, drugs, or illness, the virus can be activated and lead to the clinical syndrome known as shingles.[64]

Clinical Manifestations/Diagnosis

As opposed to the indistinct presentations of other infectious diseases in the elderly, herpes zoster almost always announces itself with a constellation of classic symptoms. The illness starts with a 2–7-day prodrome of tingling and pain at the site where soon thereafter an erythematous rash emerges in an area restricted to one or two adjacent dermatomal regions and does not usually cross the midline.[64] The rash matures quickly, first becoming papular and then vesicular coincident with an increased intensity of pain and burning. Within 2 weeks, the lesions crust and begin to fade; however, they may leave permanent scars. Serious systemic illness is rare, although fever, weakness, and anorexia can occur. While the clinical presentation is usually sufficient to establish the diagnosis of herpes zoster, laboratory confirmation can be achieved by testing of the vesicle fluid for the presence of VZV, either via viral culture, polymerase chain reaction, or Tzanck smear.

Unusually, herpes zoster can involve the eye, either externally or at the retina. Zoster lesions at the tip of the nose are an indication of involvement of cranial nerve V. Myelitis and encephalitis are rare but serious complications. The most common complication of shingles is postherpetic neuralgia (PHN), with continued pain and hypersensitivity at the area of the rash that can last up to a year.[65]

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Management

A number of oral antivirals – including acyclovir, valacyclovir, and famciclovir – are active against VZV and can be used to reduce the duration of illness.[64] These are most effective in reducing the time to lesion crusting and acute pain resolution when initiated within 72 hours of the onset of the rash. Treatment with antivirals should continue until crusting of all lesions occurs – generally 7–10 days. Persons with active lesions are infectious and can transmit VZV to those who have not previously been exposed to or vaccinated against the virus. Crusting of the lesions is associated with a marked reduction in infectiousness. Patients with suspected or confirmed VZV should be placed on airborne and contact precautions and only health-care providers with evidence of immunity to varicella should care for patients with this infection.

The management of PHN is often difficult. Topical therapies (including anesthetics) and capsaicin can be used; however, systemic therapy with narcotics, anticonvulsant drugs (e.g., gabapentin) or antidepressants (e.g., nortriptyline, amitriptyline, desipramine, and sertraline) may be required.[66]

Vaccination

Prevention of herpes zoster is possible with both the herpes zoster live attenuated vaccine (known as the zoster vaccine live or ZVL) and the herpes zoster recombinant subunit vaccine containing VZV glycoprotein E and an adjuvant (AS01B) (known as the recombinant zoster vaccine or RZV). Although the ZVL and RZV have not been compared head to head in a clinical trial, efficacy trials show superiority of the RZV vaccine for both preventing shingles and reducing the incidence of post-herpetic neuralgia.[67–70] ACIP thus preferentially recommends use of the RZV vaccine since it is 90% effective in immunocompetent adults over 50, whereas the ZVL is only 50% effective.[71] However, an advantage of the ZVL is that it only requires administration of one dose, whereas the RZV requires two doses at least 2 months apart. Serious systemic side effects including malaise, myalgias, headache, chills, and fever are reported in approximately 1% of patients receiving the ZVL and over 10% in the RZV.[67,68,72]

Antibiotic Resistance and Stewardship

Antibiotic resistance is a growing threat worldwide.[73] The impact of resistance is magnified in health-care settings, disproportionately affecting the most vulnerable such as those in congregate living settings largely populated by older adults. Infection with multidrug-resistant pathogens contributes to longer length of stay for hospitalizations and is associated with higher mortality, and while health-care-associated infections have received attention, there is also concern for rise in resistant infections in the community.[73–76] There is likely a spectrum of risk for drug-resistant bacterial infection spanning from living at home to assisted living facilities to nursing homes and skilled nursing facilities, and the attendant risk with each setting may influence selection of treatment. In general, the level of independence for activities of daily living as well as the exposures to the health-care system and specifically antibiotic exposure will determine the risk for multidrug resistance.

While patients should always be treated with antibiotics when indicated, it is also important that antibiotics for medical treatment, as well as animal health and agriculture, are used for the appropriate indication and duration to limit development of resistance. Antibiotic stewardship programs to optimize the use of antibiotics, along with infection prevention and control efforts, are effective in decreasing the number of infections and deaths caused by antibiotic-resistant pathogens.[77]

Methicillin-Resistant *Staphylococcus Aureus*

Methicillin-resistant *S. aureus* (MRSA) remains a major cause of nosocomial infections.[78] The spread of MRSA in health-care settings has had a significant impact on both clinical outcomes and health costs. Risk factors associated with hospital-acquired MRSA include prolonged hospitalization, preceding antimicrobial therapy, presence in an intensive care unit or burn unit, hemodialysis, surgical site infection, and proximity to a patient colonized or infected with MRSA.[78] As many, if not most, hospital inpatients are elderly, MRSA can be considered a major infection in this population. Furthermore, there is a great potential for the spread of MRSA within nursing facilities, given the high rates of colonization with the organism among residents of such facilities. Community-acquired (CA) MRSA infections have also become more common. Risk factors associated with CA-MRSA infection include African-American race, HIV infection, antibiotic therapy within the past 6 months, and skin trauma.[79]

Clinical Presentation/Diagnosis

MRSA colonizes the nasal mucosa and oropharynx but may also be found on the skin. Colonization itself does

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not cause illness, but it does increase the risk of subsequent disease from the organism. The clinical manifestations of MRSA infection are protean and include skin and soft tissue infections, septicemia, pneumonia, endovascular infections, joint infections, and infections of indwelling devices and prosthetic implants.[80,81] CA-MRSA frequently presents as a boil or skin abscess that is painful and erythematous. Clinically, it is impossible to distinguish between MRSA and other bacterial causes of these infections. Culture of the organism from infected tissue or blood is the basis of diagnosis. Given the aggressive nature of many staphylococcal infections, a high level of suspicion should be maintained in the presence of predisposing conditions such as defects in phagocytic function and diabetes mellitus.

As mentioned previously, a common error in the management of endovascular infections caused by *S. aureus* is the assumption that detection of this organism in the urine indicates only a UTI. Isolation of this organism in the urine should prompt evaluation for the presence of endovascular infection, as this organism commonly enters the genitourinary system hematogenously.

Management

Antibiotics and drainage of infected material are the mainstays of therapy. Skin and soft tissue abscesses require surgical drainage and adjunctive antibiotic therapy. Nonsurgically managed MRSA infections require appropriate antibiotic therapy. There are different drug susceptibility profiles for MRSA seen in hospital compared with community-acquired isolates.[82] There is also a difference in patterns of drug resistance depending on geography. It is, therefore, important to tailor treatment according to regional antibiotic susceptibility patterns (see Table 24.3).

In general, vancomycin is active against MRSA. There are reports of some strains of *S. aureus* with reduced susceptibility to vancomycin, but these are rare, show only intermediate resistance to the drug, and retain susceptibility to the newer classes of anti-staphylococcal antibiotics.[83] These alternatives are typically used when vancomycin is not tolerated.

Clostridioides (Formerly *Clostridium*) *difficile*

Clostridioides difficile is an anaerobic, toxin-producing bacteria that can manifest as asymptomatic carriage or active infection. The major risk factors for infection include antibiotic exposure (primarily penicillins, fluoroquinolones, cephalosporins, and clindamycin), advanced age, immunosuppression, and inflammatory bowel disease. Infections are associated with health-care facilities and community spread. Older adults are particularly susceptible to *C. difficile* infection (CDI) because of their increased contact with hospitals and long-term care facilities, antibiotic exposure, and underlying medical comorbidities.[84]

Clinical Presentation/Diagnosis

Patients typically present with copious watery diarrhea, and severe cases can result in sepsis, ileus, and toxic megacolon. Testing is recommended for individuals with ≥3 unformed stools in 24 hours unexplained by alternative diagnosis.[85] Available diagnostic tests include enzyme immunoassays for *C. difficile* glutamate dehydrogenase (GDH) and *C. difficile* toxins A and B, and the nucleic acid amplification test (NAAT). Since the NAAT detects the gene encoding for the toxins rather than the actual toxins, it cannot reliably differentiate asymptomatic carriage from active infection and is not recommended for use alone. A multistep approach to diagnosis – first using the enzyme immunoassay to test for GDH and toxins, then using the NAAT to adjudicate an indeterminate result – is recommended.[84–86]

Management

Treatment for an initial episode of CDI is 10 days of daily oral vancomycin or twice-daily oral fidaxomicin, including if the patient meets criteria for severe CDI (defined as a white blood cell count greater than 15,000 per microliter or creatinine greater than 1.5 milligrams per deciliter). Patients presenting with fulminant CDI, defined by presence of hypotension, shock, ileus, or toxic megacolon, should be treated with oral vancomycin 500 mg four times daily. Both rectal vancomycin and intravenous metronidazole should be added if ileus is present.[85]

The risk of CDI recurrence is approximately 20% after a first episode and 60% after multiple prior episodes.[87] A second CDI episode should be treated with oral vancomycin in a pulsed and tapered regimen or fidaxomicin. For further CDI recurrence, fecal microbiota transplant (FMT) should be considered.[85] FMT involves the transfer of stool from a healthy individual to an individual with recurrent CDI through various modalities including capsules, retention enemas, and endoscopic procedures. Small randomized controlled trials of FMT in patients with recurrent CDI have shown resolution of *C. difficile* diarrhea in 80–90% of those receiving FMT versus 25–30% of those receiving antibiotic treatment.[88,89]

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Table 24.3 Antibiotic options for MRSA

Antibiotic	Route	Indications	Routine dose	Major side effects
Trimethoprim-Sulfamethoxazole (Septra, Bactrim)	PO, IV	Skin and soft tissue infections. Not specifically FDA approved for infections resulting from MRSA.	2 double-strength tablets (160 mg TMP/800 mg SMX) po bid	Anemia, neutropenia, rash, pruritus, Stevens-Johnson syndrome. Not recommended during the third trimester of pregnancy.
Minocycline (Minocin) and Doxycycline (Doryx)	PO	Skin and soft tissue infections. Not specifically FDA approved for infections resulting from MRSA.	100 mg po bid	Photosensitivity, rash. Not recommended for use during pregnancy.
Clindamycin (Cleocin)	PO, IV	Skin and soft tissue infections, bone infections. Not specifically FDA approved for infections resulting from MRSA.	300–600 mg po tid-qid	Rash, *Clostridium difficile* colitis
Rifampin (Rifampicin)	PO	Should not be used as a single agent. May be used in combination for treatment and eradication of MRSA.	600 mg po qd	Rash, liver inflammation. High frequency of drug–drug interactions.
Vancomycin (Vancocin)	IV	Endocarditis, bacteremia, bone/joint infections.	1000 mg q12 h	Hypersensitivity reactions, red man syndrome.
Quinupristin – Dalfopristin (Synercid)	IV	Skin and soft tissue infections.	7.5 mg/kg q8–12 h	Arthralgias, myalgias.
Linezolid (Zyvox)	IV, PO	Skin and soft tissue infections, pneumonia.	600 mg q12 h	Bone marrow suppression. NOTE: not recommended for routine oral use because of potential for inducing resistance and/or toxicity, and high cost.
Daptomycin (Cubicin)	IV	Skin and soft tissue infections. Right-sided endocarditis, bacteremia.	4–6 mg/kg qday	Myopathy. NOTE: not active in pneumonia.
Ceftaroline (Teflaro)	IV	Skin and soft tissue infections, pneumonia.	600 mg q12 h	Rash, diarrhea, nausea.
Tigecycline (Tygacil)	IV	Skin and soft tissue infections. Intra-abdominal infections, pneumonia.	100 mg loading dose followed by 50 mg q12 h	**Black box warning for increased mortality.** Nausea, vomiting, headache, liver inflammation.

Summary

The diagnosis and management of infectious diseases in older persons can be challenging. Clinicians must be familiar with the differences in clinical presentation among older and younger patients. In addition, it is important that subtle indicators of serious infection and impending clinical decompensation such as hypothermia or leukopenia are not missed. Timely therapeutic intervention when infection is suspected is essential, as a delay in appropriate treatment – even for a few hours – can have devastating consequences. As the population in the United States continues to age, familiarity with the clinical presentation, diagnosis, and management of the major serious infections of elderly individuals becomes an increasingly critical component of general medicine and primary care.

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Chapter 25

Human Immunodeficiency Virus

David Alain Wohl and Christina Prather

Introduction

The human immunodeficiency virus (HIV) pandemic began in the USA among young men but has matured from a progressive and usually fatal illness into a chronic disease that predominately, and increasingly, affects older men and women. Of all People Living with HIV (PLH) in the USA, more than half are over age 50, and this proportion is growing; in the 5 years between 2013 and 2017, the number of HIV-infected individuals aged 65 or older nearly doubled from 53,000 to 92,000, according to the Centers for Disease Control and Prevention (CDC).[1,2] The graying of the domestic HIV epidemic largely reflects the dramatic increase in the life expectancy of HIV-positive individuals following advances in HIV therapeutics, but also newly acquired infections among older men and women.[1,2]

Aging for those living with HIV has been well described to differ in many respects from the expected trajectories most follow as they get older.[3–6] Conditions associated with advancing age, including cardiovascular disease, bone demineralization, and cognitive impairment, have been observed to develop at a relatively younger age in PLH, leading some to postulate that HIV causes "accelerated" or "premature" aging.[3–7] Even during the SARS-CoV-2 pandemic, PLH hospitalized with COVID-19 tended to be younger age than those without HIV.[8] Whether there is a biological acceleration of the aging process due to HIV infection or its therapies is a question driving much research, but an answer is confounded by numerous individual-, community-, and societal-related factors that individually or in concert provide an alternative explanation for early and multiple comorbidities among aging PLH.[9] Regardless of the mechanisms, clinicians must be attuned to the evolving and special needs of HIV-infected patients as they age.

Epidemiology of HIV in Older Adults

Of the 1,040,000 people in the USA estimated to be living with HIV in 2018, 51% are over 50 years of age; the prevalence rate is highest among those aged 50–54 who comprise 15% of all persons diagnosed with HIV (Figure 25.1).[1,2] Advances in HIV treatment, particularly the advent of potent combination antiretroviral therapy (ART), have led to dramatically increased survival and quality of life for PLH. Clinical cohort studies from the USA and Western Europe find that life expectancy for those infected with HIV has approached that of the general population.[10,11] Older persons may also acquire HIV, typically via unprotected sex, further increasing the pool of HIV-positive persons in this age category. In 2018, one in six new diagnoses of HIV infection were among persons aged 50 and older.[2] Given this, and an overall decline in new infections, the proportion of PLH who are in the later decades of life will continue to grow.

Screening and Diagnosis

Missed Opportunities

Detection of HIV infection is the obvious starting point for management. However, older individuals are less likely to be screened for HIV infection compared to younger people.[12] Older persons may underestimate their risk for HIV acquisition, and health-care providers may be uncomfortable or unskilled in risk assessment in this population.[12,13] As a consequence, older PLH are more likely to present later and with more advanced disease than younger individuals.[2,14]

As HIV infection may be asymptomatic or mimic normal aging or other medical problems that are common in older individuals, it is essential that clinicians include routine screening for HIV in their practice and not exclude HIV infection in the differential diagnosis of these conditions simply because of a patient's age.

Risk Assessment

A good assessment of sexual health and substance use is good medical care and is the starting point for detecting

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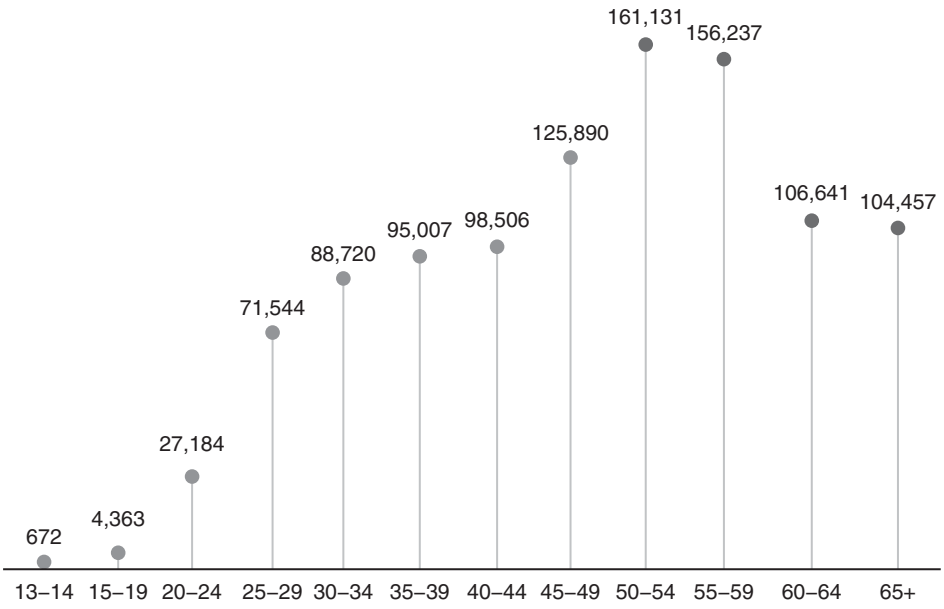

Figure 25.1 Adults and adolescents with diagnosed HIV in the USA and dependent areas by age, 2018.
Source: CDC. Diagnoses of HIV infection in the United States and dependent areas, 2018 (updated). *HIV Surveillance Report* 2020; 31.

risk for and presence of HIV and other communicable infections. There are various techniques for sexual history-taking. Some have been developed by the CDC, the Health Resources and Services Administration (HRSA), and others that can be used to efficiently gather data during clinical encounters.[15,16] In general, these techniques share the use of nonjudgmental and unassuming questions and emphasize the routine nature of the assessment. Health-care providers may be uncomfortable with asking sexual questions of older patients, but data suggests that many patients appreciate the opportunity to discuss these issues, which can also include erectile dysfunction, loss of libido, and dyspareunia. Substance use risk can be detected using one of a number of screening tools that have become commonplace in many primary care settings.[17] Injection drug use has dramatically increased in recent years, and outbreaks of HIV and hepatitis C in many parts of the USA have detected multigeneration use and sharing of equipment such as needles, syringes, and cookers that can transmit blood-borne pathogens.[18,19] These assessments are critical not only for triggering appropriate screening tests but also for opportunities to intervene with counseling, and biomedical preventive interventions such as vaccination (e.g., hepatitis B) and, as described below, HIV pre-exposure prophylaxis (PrEP) even when infection is not detected.

HIV Testing

The CDC and the United States Preventive Services Task Force recommend routine, voluntary opt-out HIV screening at least once for all adults aged 13–64, regardless of risk factors.[20,21] The frequency of subsequent testing is dependent on risk, further highlighting the importance of a sexual and substance use history. It is notable that these recommendations do not include routine testing in persons 65 years of age or older. However, many experts (these authors included) strongly recommend extending routine HIV screening beyond the age of 65 years. Clinicians must appreciate that people remain sexually active throughout their lives and that substance use is pervasive.[22]

HIV testing relies on detection of antibodies to HIV, the virus itself, or both.[23] The fourth-generation assay that is now recommended by the CDC for universal use is a combination HIV antibody and antigen (p24 antigen) assay that detects antibodies to both HIV-1 and HIV-2. It is commercially available and in wide use. This test reduces the window period from acquisition of HIV to

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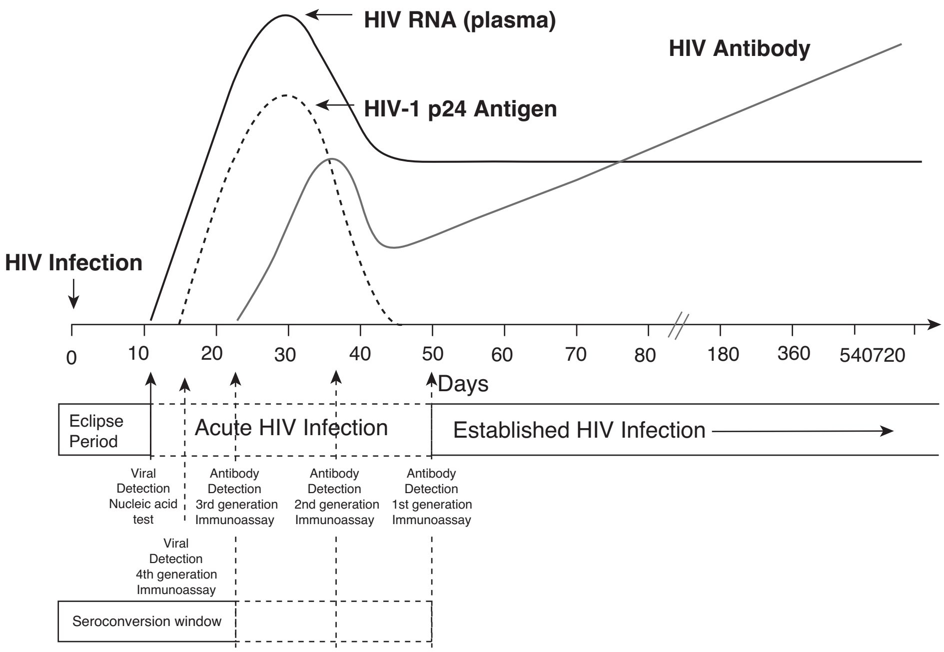

Figure 25.2 Sequence of appearance of laboratory markers of HIV-1 infection.[23]

laboratory detection of infection to 4–12 days (Figure 25.2). Negative results can be considered conclusive unless a potential exposure to HIV occurred within the 2 weeks prior to the test. If the antibody/antigen screen is positive, follow-up testing including a nucleic acid test for HIV RNA is performed to determine whether the infection is very recent. The presence of HIV RNA in the absence of antibody is diagnostic of acute HIV infection.

Acute HIV infection is symptomatic in most cases and may present as fever, generalized lymphadenopathy, viral exanthem, and severe fatigue. As these are nonspecific symptoms, especially during "flu" season, a careful history should be taken to assess for possible HIV exposure. While the fourth-generation HIV Ag/Ab test reduces the window period between infection and test positivity, when acute HIV is suspected, an HIV RNA test (i.e., PCR) is most appropriate.

At diagnosis, older adults present with characteristics of more advanced or severe HIV disease compared with younger adults.[2] They consistently have a lower median CD4+ cell count at the time of diagnosis, and a greater proportion have an acquired immune deficiency syndrome (AIDS)-defining diagnosis at or within 3 months prior to presenting for HIV care.[14] As mentioned, delayed testing due to a low clinical suspicion for HIV infection contributes to advanced presentation at the time of diagnosis. HIV testing often follows the diagnosis of opportunistic conditions that are well associated with AIDS such as pneumocystis pneumonia, toxoplasma encephalitis, Kaposi's sarcoma, cryptococcal meningitis, etc. More challenging is the consideration of HIV infection with the presentation of less obvious consequences of advancing immunosuppression like oral candidiasis, multi-dermatomal or severe herpes zoster, cognitive impairment, unintended weight loss, and recalcitrant seborrhea dermatitis, which are not uncommon and can also have alternative etiologies. Clinical suspicion is key to recognizing potential HIV infection.

Clinical Management

The mainstay of the management of the older PLH is the prevention and treatment of comorbid conditions. While many PLH have their routine medical and HIV care needs attended to by their HIV health-care provider, a substantial proportion look to primary care clinicians to manage their non-HIV health care. These clinicians should be aware of the potential impact of HIV infection on aging and the increased risk that PLH have for multimorbidities, polypharmacy, stigmatization, social isolation, and mental health disorders. Potential drug–drug interactions between antiretrovirals and other medications also need to be considered

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when prescribing medication. Online tools such as www.HIV-druginteractions.org can be useful to avoid potentially hazardous interactions. Communication between primary and specialty care providers is the essence of coordinated, patient-centered care but is challenged by incompatible electronic medical record (EMR) systems and time constraints. Efforts should be made to overcome such barriers so that care can be aligned.

Accelerated Aging?

There is growing concern that PLH experience aging differently than those without the virus.[3–7] A number of studies document comorbidities and frailty rates that are higher among HIV-infected men and women compared to uninfected controls.[6,24–27] In a comparison of a large cohort of HIV-infected and uninfected patients receiving care at two Boston hospitals, those infected with HIV had an almost twofold greater risk of acute myocardial infarction than uninfected patients, with the difference between the groups increasing at older ages.[24] Other studies have demonstrated an increase in carotid artery intima-media thickness, coronary artery calcium deposition, and noncalcified coronary plaque (increased risk of rupture compared to noncalcified plaque) in HIV-infected persons.[28,29] HIV-infected men and women also have relatively high rates of diminished bone density and nontraumatic fractures, as well as neurocognitive impairment.[30,31] An analysis of data collected during the Men's AIDS Cohort Study (MACS), a large multicenter longitudinal observational study of HIV-infected and uninfected men who have sex with men (MSM) in the USA, found significantly higher rates of frailty (defined as having ≥3 of the following: low grip strength; slow 4-meter walking speed; low physical activity; exhaustion; and unintentional weight loss of >10 pounds) among the HIV-positive men at each decade of life past 50 years of age.[32]

Some of these and other studies implicate biological processes such as immune activation and subsequent inflammation as drivers of excess comorbidity among PLH. Markers of immune activation tend to be elevated in HIV-infected patients, as are those that assess inflammation.[5,33–35] Such markers of inflammation, including C-reactive protein (CRP), interleukin 6 (IL-6), and D-dimer, have been associated with mortality and non-AIDS morbidity in HIV-positive cohorts.[34] Similarly, higher rates of altered gut integrity have been demonstrated in PLH, leading to microbial translocation – an additional contributor to the inflammatory state.[35] Common coinfections in HIV-infected patients, such as hepatitis C virus and cytomegalovirus, may also play a role by triggering additional immune activation and inflammation.[5,36]

However, that PLH experience conditions associated with aging earlier and at rates that exceed those uninfected should not be a surprise, given the higher rates of smoking, substance abuse, poverty, depression, and similar contributing factors among those living with the virus.[37] Studies that adjust for such confounders typically find that the associations between HIV and comorbidity are attenuated.[29]

The role of HIV therapies in comorbidity development must also be considered. Modern ART has led to profound improvements in survival and quality of life but may have long-term consequences. LDL cholesterol and triglyceride levels are increased by some antiretrovirals; abacavir and protease inhibitors have been associated with an increased risk of cardiovascular disease (CVD).[38] Renal complications develop in a small but significant minority of patients treated with tenofovir disoproxil fumarate (TDF); it can also reduce bone density. The commonly used integrase inhibitors dolutegravir and bictegravir have been linked to excessive weight gain – a risk for frailty and glucose intolerance.[37,38] In some reports, older PLH may rarely develop neuropsychiatric problems immediately after starting these and other drugs of the integrase class. Despite these concerns, accumulated data, including examination of treatment interruptions, make clear that uncontrolled HIV itself presents a greater risk for CVD, renal, hepatic, and other end-organ diseases, trumping any contributions by antiretroviral therapy.[39,40]

Multimorbidity

As the age of PLH increases, so too does their risk for multiple comorbid conditions and polypharmacy. A recent analysis of CDC-collected data on PLH treated with ART projects a dramatic increase over the next 10 years in the burden of multimorbidity, defined as more than one of the following: anxiety, depression, chronic kidney disease, hyperlipidemia, diabetes mellitus, hypertension, cancer, myocardial infarction, or end-stage liver disease.[41] During this time period, the population of PLH aged 70 and older will soar, as will their projected prevalence of two or more physical comorbidities – from 58% at present to 69% (Figure 25.3).

Several select conditions of particular importance in the management of the aging HIV-infected patient are

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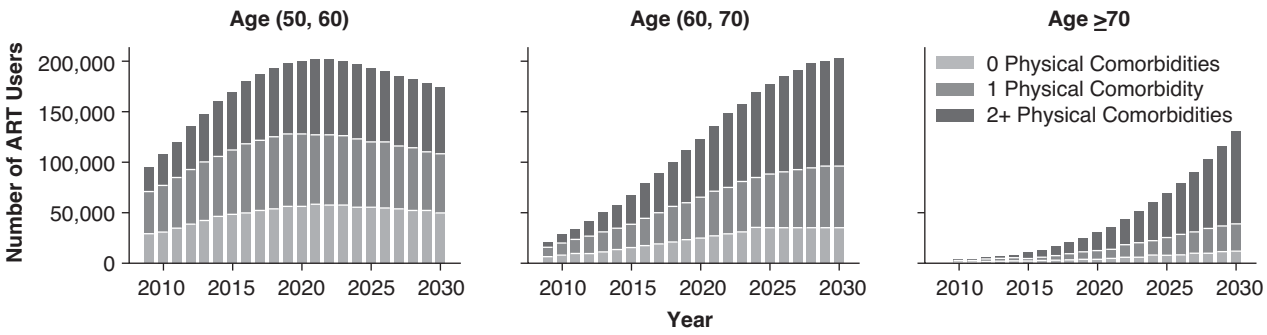

Figure 25.3 Projected burden of multimorbidity by age.[41] (A black-and-white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

discussed below. These, in addition to more general health maintenance of older patients such as cancer screening and immunizations, must not be neglected.

Cardiovascular Disease

As described above, PLH are at increased risk for CVD, including heart disease and stroke, and have increased subclinical CVD related to age-matched controls.[24,25,29,43] The etiology for this increased burden of CVD among PLH is complex. Data clearly supports a role for HIV itself as a major risk for CVD, particularly when viral replication remains uncontrolled – provoking immune activation, inflammation, and CD4 cell depletion.[43–46] Indeed, rates of CVD are reduced with effective HIV therapy, especially when initiated early.[47,48] While the role of inflammation in the pathophysiology of CVD among PLH is likely strongest in those without viral suppression, an association between the presence of low-level inflammatory markers and subclinical CVD has also been reported,[49,50] suggesting the protective effects of control of CVD vis-à-vis ART are not complete.

The effects of ART on CVD can be double-edged, with some antiretrovirals also potentially contributing to risk.[43] Many antiretrovirals, most notably protease inhibitors and their pharmacological boosters, can impact lipids, often raising levels of triglycerides as well as LDL cholesterol. In large observational studies of PLH, members of this ART class have been linked to CVD.[51] More directly, the nucleoside analogue abacavir has been associated in several studies with CVD independent of any impact on lipid levels.[52] In contrast, TDF has been observed to lower lipid subsets (LDL and HDL cholesterol) and increases in HDL cholesterol levels are often seen with the non-nucleoside reverse transcriptase inhibitor (NNRTI) efavirenz. Interestingly, the protease inhibitor atazanavir has been observed to possibly lower CVD risk, putatively because of its tendency to cause increases in indirect bilirubin, which may be cardioprotective.[53]

The concern for HIV-related contributors to CVD should be considered within the context of the prevalence of traditional risk CVD factors among many PLH whose rates of smoking, substance use, metabolic disease, hypertension, and depression exceed those of the general population.[27,43,54] Effective management that addresses these traditional risks is predicted to have a greater impact on CVD in PLH than manipulation of the timing or composition of ART.[27] The management of these common risk factors for CVD can reduce clinical disease incidence for HIV-infected patients. A study from investigators at Kaiser Permanente in California found that rates for stroke and myocardial infarction among HIV-positive patients have approached those of uninfected members over time – a reduction attributed to more aggressive risk factor management and earlier initiation of HIV therapy to control viremia.[42] Preventive measures for CVD that apply to the general population apply to PLH. Specifically, so-called lifestyle modifications that lead to smoking cessation, adoption of healthy eating habits, and regular physical activity may be particularly beneficial to aging PLH.

The recommended pharmacological intervention for primary CVD prevention for PLH follows that for those without HIV infection, although some HIV clinicians favor a more aggressive approach. Monitoring of lipids and calculation of CVD risk should be routine.[55] Lipid-lowering therapies should be prescribed following general population guidelines, but the clinician must be mindful

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of the potential for drug–drug interactions, especially between statins and certain ART. The pharmacological boosters ritonavir and cobcistat are potent inhibitors of the hepatic cytochrome P450 system responsible for clearance of many drugs. Therefore toxic levels of some statins, including lovastatin and simvastatin, can result with co-administration. In general, atorvastatin and pitavastatin can be used with these boosters but at reduced doses. In contrast, some NNRTIs, including efavirenz, can induce metabolism of statins, requiring an upward adjustment in dosing.

Renal Disease

Chronic kidney disease (CKD) is common among PLH and, once present, is more likely to progress to end-stage renal disease (ESRD) compared to the general population.[56,57] Whereas earlier in the HIV pandemic, renal disease was often a consequence of opportunistic infections or HIV nephropathy, currently more traditional factors are driving kidney injury among PLH including hypertension and diabetes mellitus.[27,57] As in the general population, effective management of these comorbidities can help to prevent their effects on renal function.

HIV-associated nephropathy (HIVAN) is a late complication of advanced HIV infection and typically presents with nephrotic-range proteinuria and progresses rapidly to ESRD. Potent ART can improve renal outcomes in those with HIVAN.[56] Among antiretrovirals, TDF is a standout for its association with renal toxicity, as it can uncommonly cause a Fanconi's syndrome characterized by proteinuria, glycosuria, and phosphate wasting.[58] This adverse effect accounts for less than 5% of cases of acute kidney injury in HIV-positive patients and is usually reversible.[59] TDF should be avoided in those with an eGFR <60 mL/min and should be discontinued in those experiencing a decline in renal function during treatment. There has been a shift from TDF to tenofovir alafenamide (TAF), which has much less renal and bone exposure.[60]

Bone Health and Fractures

Low bone mineral density (BMD) is prevalent in HIV-infected persons[61,62], and fracture rates (overall and fragility fractures) in the HIV-infected population are estimated to be 35–60% higher than the general population.[63] Bone loss may be due to ART (e.g., TDF, especially when co-administered with ritonavir or cobicistat), as well as traditional osteoporosis risk factors such as low levels of vitamin D, low body mass, alcohol use, corticosteroids, hypogonadism and proton pump inhibitors.[61,62]

ART initiation is associated with an immediate sharp decline in BMD;[64] patients may experience a decrease in BMD from 2% to 6% over the first 2 years after initiation of ART, a decline that is similar to that experienced as a result of menopause and is independent of the ART regimen selected. This drop in BMD is likely a consequence of activation of bone remodeling cells.[65] TDF is strongly associated with greater declines in BMD than other antiretrovirals, possibly as a consequence of hypophosphatemia secondary to drug-induced proximal renal tubular dysfunction. Therefore it should be avoided in those with or at greater risk of low BMD, such as many older PLH. TAF has been found to have minimal impact on BMD.

The Infectious Disease Society of America (IDSA) and the European AIDS Clinical Society (EACS) recommend dual-energy X-ray absorptiometry (DXA) screening for osteoporosis in HIV-infected postmenopausal women and men age ≥50 years.[61,66–68] The Osteo Renal Exchange Program (OREP) recently added to these recommendations, suggesting DXA screening for HIV-infected adults aged 40–49 years with a Fracture Risk Assessment Tool (FRAX) score of >10% and for adults with major fragility fracture risk factors.[69]

Treatment recommendations for bone loss are consistent with those published by the National Osteoporosis Foundation.[70] Briefly, patients who have a T-score at the hip, femoral neck, or lumbar spine less than or equal to –2.5 or have a history of fragility fracture should be considered for pharmacologic treatment. Patients should be re-evaluated every 2–5 years based on the proximity of their results to treatment thresholds. Fracture risk should be calculated for all patients with osteopenia, using FRAX to further determine potential candidates for pharmacologic treatment. As with all patients with severe bone disease, rheumatologists and endocrinologists can play a significant role in co-managing these patients.

Psychiatric Illness

Depression, anxiety, substance use, and other mental health disorders are prevalent among PLH and, like most comorbidities in this population, multifactorial.[71–75] Among older PLH psychiatric issues may be more common – a consequence of diminishing functional status, loneliness, stigmatization, cognitive impairment, and other losses.[74] Data suggests untreated depression is a predictor of nonadherence to treatment

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and care engagement and portends adverse effects on overall morbidity and mortality.[72,73]

Screening for depression, anxiety, and substance use disorders is becoming routine in clinical HIV practice and can identify opportunities for intervention. Patient-reported outcome measures can be valuable in detecting and reacting to "red flags" such as anhedonia, insomnia, anorexia, suicidality, and self-medicating with alcohol and illicit substances.[76]

The approach to the management of psychiatric disorders in older PLH largely follows that of other older persons.[77] Again, the potential for drug–drug interactions must be considered, and coordinated care between HIV and mental health-care providers is ideal.

Cognitive Impairment, HAND (HIV-Associated Neurocognitive Disorder), and Peripheral Neuropathy

Nearly 50% of HIV-infected adults demonstrate some degree of impaired performance on neuropsychological testing consistent with cognitive impairment.[78,79] Only a quarter of these patients endorse symptoms, and only half of symptomatic patients will meet diagnostic criteria for HIV-associated dementia (HAD).[78] While few patients meet the criteria for HAD, many demonstrate functional impairment due to cognitive dysfunction and meet the criteria for HIV-associated neurocognitive disorder (HAND). HAND and HAD predominantly affect subcortical processes, which manifest through impaired executive and frontal lobe functions. Patients typically endorse difficulty completing tasks, maintaining attention, and learning new material, as well as difficulty with balance and motor coordination.[79] On cognitive testing, HAND manifests as impaired attention and concentration, psychomotor slowing, executive dysfunction, and impaired recall and learning. Visuospatial and semantic abilities are spared.[80] Patients may have depression, impaired manual dexterity, gait disturbance, and impaired information retrieval. Patients with HAND demonstrate difficulty with ART adherence, making screening and identification of cognitive impairment imperative for successful treatment. HIV patients with advanced disease may develop progressive multifocal leukoencephalopathy, which requires management modalities familiar in geriatric medicine, including physical and occupational therapy, incontinence supplies, safety evaluations, and advance directives.

Currently, there are no established consensus recommendations for particular screening tools to utilize in this population. The Montreal Cognitive Assessment fares well given its evaluation of executive and other higher cognitive abilities; however, validation studies are lacking. The Mini-Mental Status Exam fails to address domains primarily impaired in HAND and should not be used for screening. Both the HIV Dementia Scale and International HIV Dementia Scale are well validated to detect HAND but are limited by their inability to detect clinically significant impairment that is not severe.[81,82] Valcour et al. extensively reviewed screening instruments specific for use in the HIV population.[83] Regardless of the screening method utilized, providers should recognize that cognitive impairment in HIV is prevalent and presents with changes in behavior, motor, and cognitive domains.

Both HIV infection and some of the older antiretroviral medications rarely in use today can cause peripheral neuropathy. This may manifest as numbness of the distal extremities, similar to diabetic sensory neuropathy, or be painful with burning and tingling. Treatment of peripheral neuropathy in the setting of HIV is similar to that of diabetic neuropathy and ranges from topical treatment such as capsaicin or lidocaine to antiepileptic medications such as gabapentin and pregabalin, although the evidence base for these medications is scant. In some cases, careful administration of narcotics is required.

Liver Disease

Liver disease in PLH is often due to viral hepatitis, alcohol, or nonalcoholic fatty liver disease (NAFLD). Hepatitis C co-infection is not uncommon among PLH who have injected drugs and can be sexually transmitted among MSM during sexual activities that can lead to mucosal tears and bleeding.[84,85] Hepatitis B infection is much less common in the USA but can also be transmitted via shared injection equipment or sexually. Treatment of hepatitis C has evolved to become highly efficacious and can be curative. Drug interactions between hepatitis C therapies and ART are possible and must be managed. Both TDF and TAF are highly active against hepatitis B.[86]

Alcohol use is common among PLH; 27% of patients receiving care at university-based clinics in the USA screened positive for unhealthy drinking on a standardized survey.[87] Alcohol use can directly cause liver injury and hastens hepatic fibrosis among those with viral hepatitis.[88]

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NAFLD ranges from steatosis to nonalcoholic steatohepatitis (NASH) to end-stage liver disease and is a leading cause of morbidity and mortality among PLH. An estimated 20–60% of PWH have NASH, and of these, 14–63% have NASH with fibrosis.[89] Optimal management of NAFLD has yet to be determined and lifestyle modification, especially weight loss, remains a mainstay recommendation.[90]

Functional Decline

HIV-infected adults experience functional limitations earlier than HIV-negative adults.[27,91] In addition to high rates of multimorbidity, HIV-infected older adults often lack social and familial support at the time of functional decline. Fragile social networks are a limitation for many older adults who need assistance in order to maintain independence. Many older PLH live alone and are estranged from family and friends due to their HIV/AIDS diagnosis.[92] Many live in poverty. As the population of HIV-infected adults ages and experiences complications of multimorbidity, there is a pressing need for public health and health-care system officials, community advocates, and others to deeply consider how to prevent and manage what some have termed the "silver tsunami" of older PLH in need of care and services.[93]

ART Adherence

Older PLH demonstrate better adherence to ART compared with the younger PLH.[94] However, risk of cognitive impairment increases with age and is a legitimate concern when managing HIV-positive adults because of its potential impact on treatment adherence. Older patients with detectable viral loads are twice as likely to have cognitive impairment as similarly aged patients with undetectable viral loads; this may serve as an indicator to clinicians that patients need to be evaluated for treatment adherence.[68] Providers should also be aware that patients with depression and other psychiatric disorders are at increased risk for nonadherence and should be evaluated to assess compliance with treatment. Other ways that older PLH tend to differ from younger PLH are listed in Table 25.1.

Table 25.1 Characteristics of older (≥50 years) adults relative to younger adults with HIV

- More severe disease course
- Less desirable health indices at diagnosis, including lower CD4+ counts and higher HIV RNA levels
- Shorter AIDS-free intervals and greater risk of opportunistic infections
- Earlier development of certain malignancies
- Higher daily medication and pill burden
- Higher rates of HIV treatment response
- Greater rates of HIV therapy adherence and persistence

Prevention

Minimizing transmission remains a core tenet of HIV education and patient care; however, elderly patients remain at risk for new infections, in part because of a lack of patient and provider education. Although older adults engage in behaviors that place them at risk for HIV infection, lack of perceived risk can lead to failure to adopt safer practice. Sexual activity among older adults is consistently underestimated. While sexual activity does decrease with age and is less commonly reported by women, a study of 3,005 US adults aged 57 to 85 emphasizes the importance of recognizing ongoing sexual activity.[22] Overall, 73% of respondents aged 57 to 64, 53% of respondents aged 65 to 74, and 26% of respondents aged 75 to 85 reported sexual activity in the preceding 12 months. Sexual activity in this sample correlated with overall health and self-reported well-being.

Among older men diagnosed with HIV infection in 2018, 66% were MSM and an additional 3% were MSM and injection drug users.[1,2] Because of stigmatization, many older men avoid self-identification with their sexuality and may underappreciate their increased risk of HIV infection. Among women aged 50 and older diagnosed with HIV infection in 2018, 86% reported heterosexual contact and 14% intravenous drug use (Figure 25.4).[2]

In addition to risk factors related to sexual activity, older adults are at increased risk for infection because of a variety of physical and psychosocial factors,[95–97] including:

- Less knowledge about HIV
- Less education about HIV prevention
- Denial regarding their own risk factors for HIV infection
- A sense of false security about risk because they are in monogamous or "nearly monogamous" relationships
- Decreased screening due to lack of patient and provider awareness of HIV risk factors and prevalence
- Decreased screening due to provider discomfort asking about sexual activity and habits
- Increased risk of stigmatization and lack of social support in the community once diagnosed with HIV

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Figure 25.4 New HIV diagnoses among people aged 50 and older in the USA and dependent areas by transmission category and sex, 2018.[2]
Source: CDC. Diagnoses of HIV infection in the United States and dependent areas, 2018 (updated). *HIV Surveillance Report* 2020; 31.

- Increased susceptibility to new infection due to age-related immune compromise
- Increased transmission risk in postmenopausal women due to age-related vaginal wall dryness and thinning from estrogen loss
- Decreased condom use due to lack of concern for pregnancy
- Decreased condom use due to erectile dysfunction.

HIV prevention and health education materials targeted to older persons are lacking and must counter the challenge of stigmatization and lack of social support that elders may encounter. Older adults self-identify stigmatization due to HIV status more often than younger adults and may be less likely to disclose their HIV status to relatives, partners, friends, or neighbors. Further barriers to educating older adults about HIV may exist at the level of the medical provider, with many PLH uncomfortable discussing these issues with their health-care providers without the conversation being initiated by their provider. Physician recognition of HIV risk factors and the existence of social supports are essential for individualized HIV care and acceptance of HIV-infected patients in the community; these factors play an important role in minimizing exposures to new infection.

Pre-Exposure Prophylaxis (PrEP)

PrEP is a biomedical HIV prevention method that is safe and highly effective at protecting against sexually and injection drug-associated HIV infection in both men and women.[98–102] TDF and TAF are each co-formulated with emtricitabine (FTC) and Food and Drug Administration (FDA)-approved as HIV PrEP. Like TDF, TAF is a pro-drug that delivers tenofovir, a nucleotide analogue, to the cell where it acts as an inhibitor of viral replication. However, unlike TDF, limited tenofovir is released in the circulation following ingestion of TAF, reducing exposure and toxicity to renal and bone cells. Given the higher prevalence of renal disease and low bone density in older people, TAF/FTC may be generally preferred. It is notable that the approved indication for TAF/FTC as PrEP does not include women who can be exposed to HIV via vaginal sex, as there is limited efficacy data in this population. Regardless, many experts would use TAF/FTC in older women given likely efficacy and greater safety than TDF. There is data, albeit limited, supportive of TDF preventing infection among injection drug users who share equipment.[102]

PrEP is indicated for those who are at risk of exposure to HIV.[98] Both TDF/FTC and TAF/FTC are single tablets that should be taken every day. Inadvertent initiation of PrEP alone in a patient with HIV infection can lead to viral resistance. Therefore, prior to the start of PrEP, patients must undergo testing to ensure that they are HIV-negative. TDF/FTC is approved for those with an eGFR of 50 ml/min or greater, while TAF/FTC can be taken by those with eGFR of 30 ml/min or greater. Therefore, a creatinine should be obtained prior to initiation. Patients should be screened for HIV infection and sexually transmitted infections approximately every 3 months. Renal function monitoring is generally recommended every 6–12 months but can be more frequent based on the clinical circumstance, especially if TDF/FTC is being used. Adherence to the medication must be stressed, as pauses in PrEP followed by exposure, infection, and then re-initiation of PrEP can lead to viral drug resistance. Excellent guidance for the initiation and management of PrEP has been issued by the CDC and the Department of Health and Human Services.[98]

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Uptake of PrEP has generally been slow, although direct-to-consumer marketing has increased awareness. Yet, there remain many who are candidates for PrEP but are not receiving it. Barriers to PrEP include lack of knowledge and acceptance not only on the part of the patient but also the provider. The cost of PrEP, clinical evaluations, and laboratory tests can also be an obstacle. Manufacturer co-pay assistance may not be able to be used for those with Medicare coverage. Again, provider risk assessment is critical to identifying those who can benefit from PrEP.

Summary

1. The population of older adults with HIV in the United States and worldwide is increasing, due to aging of the HIV-infected population as well as new infections in adults 50 years and older.
2. Older adults have more advanced HIV at the time of presentation compared to younger patients. An inappropriately low suspicion for HIV infection contributes to delayed diagnosis.
3. Older adults are less knowledgeable about HIV compared with younger adults and are less likely to ask providers about sexual health or HIV. Older adults also tend to lack community support and may be stigmatized because of HIV diagnosis.
4. Routine HIV screening as recommended by the CDC and US Public Health Task Force should be extended to those aged 65 and older.
5. HIV infection should be considered when patients present with conditions that may indicate immunosuppression.
6. People with HIV infection have higher rates of comorbid conditions and experience them earlier than those without HIV infection.
7. The risk of multimorbidity and polypharmacy increases with age.
8. ART should be prescribed to older adults. Awareness of drug metabolism should guide drug selection, monitoring for adverse effects, and consideration of avoidable drug interactions.
9. HIV prevention applies to older people, many of whom remain sexually active or suffer from substance use disorders and share injection equipment. PrEP is safe and effective for both men and women.

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Chapter 26

Kidney Disease

C. Barrett Bowling and Nicole DePasquale

Introduction

Kidney diseases include a spectrum of disorders characterized by abnormalities of kidney structure or function that have implications for health.[1] The two major categories of kidney disease are (1) chronic kidney disease (CKD), defined as persistent impairment in kidney function, and (2) acute kidney injury (AKI), defined as a sudden loss in kidney function (Table 26.1). The prevalence of both CKD and AKI increases with aging, and more than half of all patients with kidney disease are over the age of 65 years.[2,3] While CKD is often asymptomatic early in the course of disease, later stages are associated with health implications related to the loss of key kidney functions: filtration and urine production, maintaining fluid and electrolyte balance, acid-base homeostasis, calcium and vitamin D metabolism, and stimulation of red blood cell production. In addition to morbidity related to direct loss of kidney function, among older adults kidney disease has been linked to higher rates of mortality, cardiovascular disease (CVD), and geriatric conditions.[4,5]

This chapter first describes the burden of kidney disease among older adults. We next review the clinical practice guideline recommendations for evaluation and management of CKD, with an emphasis on the challenges that arise when these guidelines are applied in older populations. Finally, we describe how a person-centered approach to care, including better engagement of family caregivers, may help address these challenges.

Epidemiology

Chronic Kidney Disease (CKD)

CKD is defined as abnormalities of kidney structure or function present for at least 3 months. The Kidney Disease Improving Global Outcomes (KDIGO) Clinical Practice Guideline recommends that CKD be classified and staged based on three components: (1) cause, (2) glomerular filtration rate (GFR) category, and (3) albuminuria.[1] Among older adults, common causes of CKD are comorbidities including hypertension, diabetes mellitus, vascular disease, and chronic urinary obstruction. However, intrinsic causes of CKD, including glomerular nephritis, nephrotic syndrome, vasculitis, and multiple myeloma, are also possible.

Guidelines recommend estimating GFR (eGFR) using a standardized estimating equation that includes age, race, gender, serum creatinine, or newer markers of kidney function such as serum cystatin-C.[1] Albuminuria is an indicator of the protein albumin in the urine measured on a random, spot collection. GFR and albuminuria categories are displayed in Table 26.1. The combination of GFR and albuminuria category can be used to assign a CKD stage. Staging of CKD provides an estimate of prognosis and is used to guide management decisions, with higher stages being associated with a higher risk of kidney failure and CKD complications. For example, a patient with an eGFR of 20 ml/min/1.73 m^2 and albuminuria level of 500 mg/g would be assigned G4 A3, which corresponds to an elevated risk for adverse kidney disease outcomes. Kidney failure is defined as an eGFR <15 ml/min/1.73 m^2, or the need to initiate renal replacement therapy. In the USA, end-stage renal disease (ESRD) is an administrative term defined by the Centers for Medicare and Medicaid Services (CMS) as permanent kidney failure requiring long-term dialysis or a kidney transplant.

The prevalence of CKD in the United States is estimated to be 15%, affecting approximately 37 million people. CKD is more common among non-Hispanic Black US adults compared to non-Hispanic White adults, and among Hispanic compared to non-Hispanic adults.[6] The prevalence of CKD is 7%, 13%, and 38% among US adults aged 18–44, 45–64, and 65 years and older, respectively. Among US adults aged 80 years and older, the prevalence of CKD has increased over the past 20 years.[2,7] If these trends continue, projections indicate that there may be nearly 16 million US adults aged 80 years and older with CKD by the year 2050.[8]

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Table 26.1 Key kidney disease terms

Term	Description	Definition
Chronic kidney disease (CKD)	Abnormalities of kidney structure or function persisting for at least 3 months with implications for health	Decreased GFR (<60 ml/min/1.73 m^2), or Urinary albumin-to-creatinine ratio (≥30 mg/g)
Kidney failure	Advanced kidney disease	eGFR <15 ml/min/1.73 m^2, or need for renal replacement therapy
End-stage renal disease (ESRD)	Administrative term based on Medicare benefit for those with kidney failure	Need for renal replacement therapy for >90 days or having received a renal transplant
Acute kidney injury (AKI)	A sudden loss in kidney function characterized by change in serum creatinine or urine production	Increase in serum creatinine by ≥0.3 mg/dl within 48 hours, increase in serum creatinine by 1.5 times baseline within the previous 7 days, or urine volume <0.5 ml/kg/h for 6 hours
Glomerular filtration rate (GFR) category	G1: Normal or high G2: Mildly decreased G3a: Mildly to moderately decreased G3b: Moderately to severely decreased G4: Severely decreased G5: Kidney failure	≥90 ml/min/1.73 m^2 60–89 ml/min/1.73 m^2 45–59 ml/min/1.73 m^2 30–44 ml/min/1.73 m^2 15–29 ml/min/1.73 m^2 <15 ml/min/1.73 m^2
Albuminuria category	A1: Normal to mildly increased A2: Moderately increased A3: Severely increased	<30 mg/g 30–300 mg/g >300 mg/g

While the prevalence of CKD is in the many millions, only around 125,000 individuals start treatment for ESRD annually.[9] For those who do progress to higher stages of CKD, there are high rates of mortality, CVD, and geriatric conditions such as functional decline, falls, and cognitive impairment. By the time older adults progress to ESRD, nearly 25% have limitations in activities of daily living (ADL), and survival is limited (median survival 13 months for 80-year-old initiating dialysis).[10] Predicting who will progress to ESRD is challenging, especially among older adults who often experience nonlinear patterns of progression with episodes of AKI and recovery.[11] This highlights the major epidemiologic challenge in older adults: while many older adults have CKD, a small proportion will have a predictable progression, but those who do have high needs and high healthcare costs.

Acute Kidney Injury (AKI)

AKI is defined as a sudden loss in kidney function characterized by a change in serum creatinine or urine production (Table 26.1). The current staging system for AKI spans small changes in kidney function to acute kidney failure with no urine production (i.e., anuria).[12] The incidence of AKI increases with older age from approximately 500 per 100,000 person-years among adults aged 50 to 59 years, to 1,100, 2,800, and 4,900 per 100,000 person-years at ages 60 to 69 years, 70 to 79 years, and ≥80 years, respectively.[3] This definition requires that serum creatinine or urine output be measured prior to a change. Therefore, this is most applicable to hospitalized patients and likely underestimates the incidence of AKI among older adults. Community-acquired AKI (CA-AKI) is a more recently developed term used to describe kidney injury among non-hospitalized patients.[13] While older adults may be vulnerable to CA-AKI, formal definitions of CA-AKI have not been agreed upon, thereby limiting the ability to estimate the incidence.

The high incidence of AKI with aging is due in part to the increase in AKI risk factors among older adults. As with multifactorial geriatric syndromes, AKI risk factors can be categorized as either predisposing or precipitating.[14] In addition to CKD, which is a major predisposing factor, other risk factors that make older adults more susceptible to AKI include multimorbidity, polypharmacy, prostate and bladder dysfunction, reduced fluid intake, and decreased thirst sensation. Common precipitating factors among older adults include acute infection or sepsis, dehydration, acute urinary obstruction, heart failure with decreased renal perfusion pressure, and new use of nephrotoxic medications.

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Evaluation for Kidney Disease

History and Physical Exam

The history and physical exam focuses on identification and assessment of the severity of kidney disease risk factors and complications. Obtaining a history of diabetes mellitus including retinopathy and neuropathy, as well as hypertension, vascular disease, urinary obstruction, renal stones, or surgical removal of a kidney, may be helpful for identifying potential causes of kidney disease. Obtaining a history of new prescription or over-the-counter (OTC) medications is very important, including the dose, duration, and start date. A review of systems may be helpful for identifying fatigue, lower-extremity edema, fractures, decreased urine output (which may be related to anemia), fluid retention, kidney-related bone and mineral disease (BMD), and urinary obstruction. Early in the course of CKD, symptoms are more likely to be attributable to other conditions rather than CKD. Symptoms of kidney failure include fatigue, edema, shortness of breath, deceased appetite, and pruritus.

Laboratory and Imaging Tests

Laboratory evaluation is necessary for confirmation of CKD, staging, and identification of CKD complications.[1] As described above, serum creatinine for eGFR and ACR should be obtained. Because of challenges obtaining a 24-hour urine collection, a random urinary ACR should be performed. When possible, reviewing or graphing the change in eGFR over time is helpful for evaluating the rate of progression or distinguishing between CKD and AKI. Urgent or emergent evaluation of sudden change in kidney function should be done when AKI is suspected. Additional laboratory tests to obtain include potassium, bicarbonate, calcium, phosphorus, glucose, hemoglobin, and hemoglobin A1c among diabetic patients. Urine microscopy is necessary for identifying red blood cell casts or other sediment abnormalities. A renal ultrasound, as well as a postvoid residual, are commonly conducted in older adults. Further testing to identify less common causes of CKD or advanced CKD complications such as a 24-hour urine collection, serum protein electrophoresis, urine protein electrophoresis, human immunodeficiency virus, markers of vasculitis, parathyroid hormone (PTH), or a kidney biopsy are most often done in collaboration with a kidney disease specialist.

Referral to Specialists

The purpose of referral is (1) to aid with diagnosis, especially when less common causes are suspected, (2) to help develop treatment plans, and (3) to prepare for renal replacement therapy. Accordingly, guidelines recommend referral to nephrology for the following reasons: AKI, eGFR <30 ml/min/1.73 m^2, persistently high albuminuria, CKD progression, urinary red cell casts, refractory hypertension, persistent hyperkalemia, recurrent kidney stones, or when there is a family history of hereditary kidney disease.[1] Clinical practice guidelines recommend that patients at high risk for progression based on a kidney failure risk of 10–20% at 1 year be referred for education and planning for renal replacement therapy. Validated prediction models for kidney failure, such as the Kidney Failure Risk Equation, take into account a patient's level of kidney function and age.[15] Recommendations to refer pre-kidney failure patients to nephrology are based on observational studies showing that late referral is associated with poor health outcomes among those who initiate dialysis. However, these studies are limited by survival bias and confounding. Therefore, the ideal time for referral to improve health outcomes is not known and should be individualized based on the recommended guidelines and the patient and family's health goals.

Challenges to Evaluation Among Older Adults

There are several challenges to evaluating CKD in older adults related to their exclusion from studies used to develop GFR estimating equations and the physiology of aging and loss of muscle mass.[16] Initially, studies used to develop and validate GFR estimating equations were limited to young and middle-aged adults or had broad exclusions such as an inability to participate or a life-limiting illness that reduces the generalizability of study findings and the accuracy of eGFR results in clinical practice. More recent studies have included a higher percentage of older adults, but are still limited to very few participants aged 80 years and older.[17]

There is also uncertainty regarding the clinical ability to distinguish between reductions in kidney function related to normal aging versus disease.[18] Cross-sectional studies have shown that eGFR is lower at older age, starting in the fourth decade of life. Therefore, some kidney disease specialists have argued that eGFR cutoffs should be adjusted at older age to avoid labeling normal aging as a disease. Others have pointed to (1) longitudinal studies showing that many older adults, particularly those

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without hypertension, have no loss in GFR over time, and (2) large population-based studies showing higher mortality among those with reduced eGFR to support the use of current eGFR cutoff points to define CKD.[2] Adding to the uncertainty around eGFR-based diagnosis in older adults is the higher prevalence of sarcopenia, or loss of muscle mass with aging, which can lead to lower serum creatinine and an overestimate of GFR. Studies have also shown that kidney disease terminology and the purpose of blood tests to evaluate and monitor kidney function are often not explained to patients, which may delay referral and limit kidney disease evaluation.[19]

Management of CKD

The three main goals of management of CKD are to prevent kidney failure by slowing the progression of loss of eGFR, identifying and treating CKD-related complications, and preparing for renal replacement therapy.[1] The specific recommendations for achieving these goals may change over time as new evidence is published and may differ from one guideline to another. For example, recent evidence from clinical trials supports intensive blood pressure (BP) treatment in hypertension for some patients.[20] However, different societies have published guidelines with different BP treatment goals.[21] To avoid providing conflicting recommendations, in this section we provide a broad overview of the management approach, but encourage readers to consider updated guidelines for setting specific treatment goals. It is also important to recognize that many of the over 100 guideline recommendations for CKD management, such as lowering BP, require ongoing self-management on the part of the patient. Therefore, identifying potential barriers to CKD self-management is important when developing a treatment plan.[22]

Slowing Progression

Prior to the first Clinical Practice Guideline for CKD management developed by the National Kidney Foundation in 2001, most kidney disease care focused on the management of patients with ESRD. Because of the overall poor prognosis and high costs associated with ESRD, the goal of clinical practice guidelines was to shift the focus of care to earlier in the disease course to prevent progression. The primary approach to prevention is to identify the main cause or causes of CKD and treat these conditions.

The two most common causes of kidney disease are hypertension and diabetes, and management of these conditions is key to slowing progression. Treatment of hypertension focuses on lowering systolic and diastolic BP. The most recent CKD Clinical Practice Guidelines recommend BP ≤140/90 mm Hg for all patients with CKD and ≤130/80 mm Hg for those with diabetes or albuminuria >30 mg/g (i.e., A2 or higher).[1] However, recent hypertension clinical practice guidelines recommend first estimating CVD risk and then determining BP goals based on this risk estimate. Based on this approach, the goal for almost all older adults with CKD would be ≤130/80 mm Hg, regardless of diabetes status. The KDIGO guideline development group is currently preparing an update to their hypertension guidelines. In addition to specific BP goals, Renin-Angiotensin-Aldosterone System (RAAS) blockage with an angiotensin-converting enzyme inhibitor (ACE-I) or angiotensin II receptor blocker (ARB) is recommended in patients with CKD and diabetes and/or albuminuria. Among patients with diabetes, guidelines recommend a hemoglobin A1c near 7.0%, but suggest that higher A1c levels may be allowed to avoid hypoglycemia or among patients with limited life expectancy. Recently updated guidelines for diabetes management in CKD recommend using a sodium-glucose cotransporter-2 inhibitor (SGLT2i) among patients with eGFR ≥30 ml/min/1.73 m^2 to reduce the risk of CKD progression as well as CVD. Among patients who have not achieved individualized A1c goals despite use of an SGLT2i and metformin, guidelines recommend including a long-acting glucagon-like peptide receptor agonist (GLP-1 RA) as part of their treatment. However, more research is needed to determine the potential risks and benefits of these new recommendations among older adults.

Additional recommendations for slowing the progression of CKD focus on healthy behaviors include lowering sodium intake, encouraging physical activity, and smoking cessation.[1] Avoiding nephrotoxic medications in order to prevent AKI is another strategy to reduce the risk of CKD progression. For some patients, additional recommendations to reduce protein intake or take bicarbonate supplementation when serum bicarbonate levels fall below 22 mmol/l may be helpful to slow CKD progression. However, decisions about these treatment approaches are best made in collaboration with a kidney disease specialist.

Managing CKD Complications

The second management goal of CKD is to identify and treat CKD complications, specifically anemia, CKD-MBD,

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electrolyte disorders, acidosis, and CVD.[1] In general, the management of CKD complications should be done in collaboration with a kidney disease specialist. Dietary restrictions and modifications are first-line management for many of the complications, and referral to a dietician is often necessary. Current recommendations for identification of complications include measuring hemoglobin in all CKD patients at least annually, and measuring serum levels of calcium, phosphate, PTH, and alkaline phosphatase at least once. Routine evaluation for hyperkalemia should also be performed. Anemia is defined as a hemoglobin <13.0 g/dl in men and <12.0 g/dl in women.[23] Evaluation for and correction of iron deficiency is an early step in the management of anemia. For patients seen by nephrology, erythrocyte stimulation agents (ESA) may be appropriate when the hemoglobin falls between 9.0 and 10.0 g/dl, with the goal hemoglobin on treatment to not exceed 11.5 g/dl. Early management of CKD-MBD includes lowering of serum phosphate toward the normal range, first through dietary restrictions and if necessary with phosphate binders.[24] The choice and dosing of phosphate binders or treatment of abnormal PTH levels should be decided in conjunction with a kidney disease specialist. Vitamin D supplementation is often necessary, but some patients may be at risk for hypercalcemia, highlighting why the management of CKD-MBD may be best done by a specialist. For primary care physicians and geriatricians, a key role in management of CKD is identifying and addressing risk factors for CVD. Specific recommendations for addressing CVD risk factors are described in the Slowing Progression section above, as well as in Chapters 13 and 15.

Preparing for Kidney Failure

In order to understand the treatment decisions that patients, their families, and providers face, it is important to consider the multiple steps necessary to start renal replacement therapy. These include identifying patients at high risk for progression to ESRD, referring patients early to nephrology, providing education about treatment options, eliciting treatment preferences, choosing a treatment option, scheduling surgery to develop a dialysis access, finding a treatment center for those choosing in-center hemodialysis, and finally, initiating treatment. Therefore, rather than a single treatment decision, there is a series of complex decisions about who and when to refer, what treatment to choose, when and what type of dialysis access to place, and when and where to start treatment. Sometimes these decisions take place over time, but often kidney failure is precipitated by AKI, and patients and their families may have to make difficult decisions while dealing with a health crisis. Additionally, as patients' health status and health-care goals change over time, decisions at each step may also change over time.

An overarching decision that patients face is whether or not to pursue renal replacement therapy. The term "conservative management" is used to describe care for patients with kidney failure that focuses on symptom management and quality of life without renal replacement therapy.[25] For patients who do choose renal replacement treatment, options include renal transplant and dialysis. Dialysis treatment options, or modalities, are categorized as peritoneal versus hemodialysis, with options for hemodialysis including in-center or home hemodialysis. In a later section on Person-Centered Care, we describe how a person-centered approach may help support treatment decisions among older adults.

Challenges to Managing CKD in Older Adults

Along with challenges in evaluating CKD in older adults, there are also challenges applying guideline-recommended care to all older adults with CKD.[26] Clinical practice guidelines were developed to standardize care, provide consistent recommendations, and reduce variations in routine care. However, clinical practice guidelines are limited by existing evidence from available clinical trials, the focus on only one condition at a time, and the lack of consideration for heterogeneity in treatment preferences and life expectancy seen in older adults.

Limited evidence base. Evidence-based clinical practice guidelines are informed by the results of published clinical trials.[27] Often older adults are excluded from clinical trials with specific upper-age cutoffs or by excluding those with other chronic conditions, cognitive impairment, or limited life expectancy. Additionally, clinical trials often do not measure outcomes or investigate research questions that are important to older adults, such as how a given treatment affects function and quality of life. Therefore, findings from clinical trials, and accordingly the recommendations in clinical practice guidelines, are often not relevant to older adults. When compared to real-world patient populations, clinical trials in CKD often include younger patients, with lower mortality and lower comorbidity, who are more likely to benefit from the given interventions. For many older adults, these interventions may have limited to no benefit.

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High burden of multimorbidity and geriatric conditions. The vast majority of older adults with CKD have other chronic conditions.[28] Multimorbidity can limit the management of CKD in multiple ways. Applying multiple single-disease practice guidelines results in care that is burdensome and potentially harmful. The number of medications, health behaviors, and specialists' visits that patients have can be overwhelming. Older adults with CKD have also described specific situations where they have received conflicting (i.e., discordant) treatment advice.[22] For example, patients may be told to limit water intake to address heart failure by one specialist and told to increase water intake to prevent AKI by another. Specialists, focused on one organ system at a time, may not anticipate these discordant situations, leaving patients to reconcile opposing self-management requirements.[29]

In addition to the high prevalence of co-occurring medical conditions, older adults with CKD often experience geriatric conditions such as cognitive impairment and falls.[4] Cognitive impairment is underrecognized in this population and can lead to difficulty understanding CKD self-management tasks and fully participating in complex decision-making. Falls are also common in CKD, with rates of serious fall injuries increasing in the months preceding and following dialysis initiation.[30] While geriatric conditions impact a patient's ability to function and live independently, these are not addressed by clinical practice guidelines.

Heterogeneity in life expectancy and health goals. Applying a standardized approach to management recommended in the clinical practice guidelines may not be appropriate because of varying life expectancy and health goals among older adults. While the average number of years remaining in life decreases at older ages, within each age group there is marked heterogeneity.[31] For patients who may live 5 to 10 more years, many preventive measures may be appropriate. Many patients, however, will not live long enough to benefit from treatments with a long time horizon. While many guidelines now recognize the need to individualize care, few provide guidance on how to do so.

In addition to variability in life expectancy, health goals and treatment preferences also vary among older adults. Often this results in differences in how individual patients weigh tradeoffs related to treatments decisions. In CKD, there are many small and large tradeoffs that older adults must consider. For example, avoiding nonsteroidal anti-inflammatory drugs (NSAIDs) to prevent CKD progression may come at the cost of worse arthritis pain. For some this means occasional pains, but for others pain may limit mobility and social participation. For those with ESRD, a tradeoff example is the prolongation of life with dialysis, but at the cost of a higher percentage of days spent in the hospital at the end of life. Because guidelines most often prioritize preventing disease-based outcomes, these recommendations do not take into account the ways in which individual patients weigh tradeoffs when making treatment decisions.

Person-Centered Care

Care for patients with CKD has historically been grounded in a disease-based approach dictated by medical evidence and knowledge, meaning that care is focused on management of an individual disease without full consideration for the unique individual being treated.[26,27] Conversely, person-centered care focuses on the whole person or individual and their personal circumstances to guide clinical decisions and care strategies. This represents a shift from the traditional paternalistic model of health care, in which patients are passive recipients, to an equal, collaborative relationship between those who use and those who provide nephrology services. Providing person-centered care is appropriate for all older adults, but this is especially true in CKD because of the uncertainty in lab-based diagnosis, the high burden of multimorbidity and geriatric conditions, and the known heterogeneity in life expectancy and treatment goals (Table 26.2).

Four principles guide the provision of person-centered care: (1) care is delivered with dignity, compassion, and respect; (2) care is well coordinated; (3) care accounts for the evolving preferences, needs, values, goals, and family circumstances of the individual; and (4) care involves the individual as an active participant in their own care.[32] Following these principles should increase shared decision-making, help individuals gain a better understanding of both their disease and their treatment, and improve care experiences, disease management, and health outcomes.[32] Although there is still much to learn about the effects of person-centered kidney care, fewer hospitalizations and improved BP control have been identified as benefits.[33] Performing geriatric assessment, engaging and supporting family caregivers, and incorporating palliative care into care of older adults

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Table 26.2 Examples of CKD clinical practice guideline recommendations, challenges applying these in older populations, and the potential role of person-centered care

Recommendation	Challenge	Role of person-centered care
Use lab-based biomarkers including eGFR	Uncertainty about accuracy of eGFR in older adults	• Consider co-occurring chronic conditions, overall health, and function when making diagnoses
Monitor eGFR to assess for progression	Low understanding of purpose of monitoring visits	• Explain purpose of visits in the context of patients' health-care goals • Involve family caregivers in the process early in the course of CKD
Treat underlying causes of CKD	Risk for treatment-related complications including hypotension and hypoglycemia	• Identify vulnerable patients using geriatric assessment including falls risk, gait and balance abnormalities, or mobility limitations
Adhere to self-management tasks to prevent progression	Barriers to self-management including conflicting treatment advice or functional and cognitive limitations	• Reconcile treatment conflicts (i.e., discordant recommendations) • Recognize that family caregivers contribute to self-management and include them in CKD education
Identify and treat CKD-related complications	Multifactorial etiology of conditions such as anemia	• Consider whole person, not just one organ system at a time
Start dietary restrictions as first-line treatment for some CKD complications	Functional and cognitive limitations interfere with patients' ability to adhere	• Identify functional and cognitive limitations through geriatric assessment • Engage family caregivers
Refer high-risk patients to nephrology for preparation for renal replacement therapy	Heterogeneity in life expectancy, health goals, and treatment preferences	• Identify health goals and preferences • Use geriatric assessment to guide estimation of prognosis • Incorporate palliative care in routine CKD care

CKD = chronic kidney disease, eGFR = estimated glomerular filtration rate

with CKD are all practical approaches to move toward person-centered care.

Geriatric Assessment

In order to provide person-centered care for older adults with CKD, it is necessary to recognize the physical, cognitive, social, and environmental context in which the person experiences CKD. A practical and evidence-based way to identify this context is through geriatric assessment.[34] As described in Chapter 3, geriatric assessment is a multidomain, often interdisciplinary, evaluation of a patient's function, cognition, mental health, medications, social support, living situation, and health-care goals. Geriatric assessment provides two types of information necessary for providing person-centered CKD care: functional capacity and prognosis. First, geriatric assessment identifies functional capacity, defined here as one's ability to participate in CKD self-management and decision-making. For example, a patient who needs help grocery shopping because of mobility limitations and mild cognitive impairment will be limited in their ability to independently implement CKD-related dietary restrictions. Identification of both strengths and weaknesses related to capacity allows medical providers to tailor treatment plans to the patient's individual needs. Obtaining the long-term perspective of the patient's primary care physician about functional capacity is often helpful. Geriatric assessment also provides important information about prognosis. While it is known that patients with functional limitations or cognitive impairment have higher mortality and worse prognosis after dialysis initiation, these problems are underrecognized among older adults with CKD.[35]

While there are multiple tools available, the most relevant measures for patients with CKD include basic and instrumental activities of daily living, a history of falls, gait and balance abnormalities, and brief screening for cognitive impairment and depression. Self-reported measures of limitations with walking mobility (difficulty walking one quarter of a mile) and life-space mobility (how far, how often, and how much help someone needs with movement in their community) have also been

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shown to be important predictors of adverse outcomes and can be used to identify those who may need additional evaluation by geriatrics or physical therapy.[36] In routine CKD care, a review of medications is often limited to those that affect kidney function or that need to be dosed based on reduced kidney function. A geriatric assessment of polypharmacy should include a review of all prescription and OTC medications to identify potentially inappropriate medications such as anticholinergics and sedatives. Social and environmental contextual factors that should be assessed include social support, availability of caregivers, and living situation, including independent, assisted living, or nursing home residency. Models of care that include risk stratification (e.g., 70 years and older, eGFR <30 ml/min/1.73 m^2), followed by brief assessments to guide referral to geriatrics, physical therapy, pharmacy, or other care, are feasible in clinical practice and can provide information to support person-centered care.

The Importance of Family in Person-Centered Care

Family-centered care. Family-centered care acknowledges, meaningfully involves, and addresses the needs, values, preferences, and goals of family members who care for older adults with CKD by integrating them into the patient's care team as care partners, or full partners in patient care and decision-making. A family-centered care approach entails understanding and improving care partners' care experiences; lessening care strain and burden in care partners' daily lives; providing care partners with access to information and relevant tools for making informed, shared decisions and developing a care plan; and promoting communication and trust among patients, care partners, and health-care providers.

The vital role of family care partners in CKD. As non-health-care professionals, care partners voluntarily dedicate their time and energy to informally support the daily and instrumental needs of CKD patients. Care partners engage in numerous essential activities while caring for older adults with CKD, such as scheduling and attending patients' medical visits; transporting patients to dialysis clinics; operating home dialysis machinery; making frequent trips to the hospital; helping patients transition to and cope with negative feelings about dialysis; dealing with patients' maladaptive behaviors (e.g., treatment nonadherence); monitoring patients' symptoms, renal-friendly diet, and medication compliance; participating in decisions about dialysis, transplantation, or conservative care; advocating for patients' receipt of high-quality care; and establishing a partnership with patients' kidney team.[37]

Lack of recognition and preparation. Despite acknowledgment and support for the family within the paradigm of person-centered care, family care partners of older adults with CKD often receive little attention and support in practice. They have been referred to as a shadow workforce or invisible members of patients' health-care team. Unsurprisingly, care partners of CKD patients describe being overlooked and excluded from care processes by health-care providers, encounter difficulties accessing the health-care system, lack disease- and treatment-related knowledge, and indicate unmet practical, educational, and psychosocial support needs.[38,39] Related to these suboptimal experiences, care partners feel unprepared to provide complex CKD care and are uncertain about their role in patient care.[39]

Care partner health and well-being. Family involvement in the care of patients with CKD has been associated with a host of positive patient outcomes, including improved self-management behaviors and treatment adherence, enhanced quality of life, decreased mortality risk, reduced anxiety and depressive symptoms, fewer treatment complications, and lower odds of hospital readmission.[40] While their involvement in CKD patients' care can be rewarding (e.g., experiencing personal growth), family members commonly report poor quality of life, negative changes in psychosocial well-being, and difficulties juggling non-caregiving responsibilities.[37] Further, studies have observed worry, role strain, physical and emotional exhaustion, restlessness, social isolation, health problems, limited time for self-care, and low levels of caregiving satisfaction among care partners of older CKD patients.[41]

The importance of supporting family care partners. Person- and family-centered care recognize that supporting family care partners is central to providing high-quality care for CKD patients. Care partners who are not adequately supported may experience negative physical and mental health consequences that hinder their ability to provide care; these consequences, in turn, can adversely affect the physical and mental health of patients with CKD. To provide better and more meaningful support for care partners, the delivery of person- and family-centered care involves treating care partners as

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individuals with their own information, training, and support needs that require empathy and attention. Person- and family-centered care therefore has the potential to help care partners remain in their caring role as well as to benefit patients and care partners alike.

Palliative Care

Palliative care is a form of person- and family-centered care that addresses the stress, pain, and burden of serious illnesses through symptom management, support provision, shared decision-making, identification of goals for end-of-life care, and facilitation of advance care planning to optimize quality of life and alleviate suffering for patients and families.[42] When incorporated alongside standard care, palliative care can lead to better symptom management, disease adjustment, and end-of-life preparedness. Palliative care is relevant for all patients, regardless of their age, disease stage, or life-prolonging treatment, but should be engaged early in and throughout the disease course to increase the likelihood that patients receive goal-concordant care. Treatment decision-making, conservative management of CKD, advance care planning, and setting goals for end-of-life care are distinctive features of palliative kidney care.

Treatment Decision-Making and Conservative Management

Patients who transition from CKD to ESRD typically choose a dialysis modality as their form of treatment. For older patients, however, the complications and side effects of dialysis, coupled with physiological changes associated with aging, comorbid conditions, and geriatric conditions, can mean that the burdens of dialysis (e.g., fatigue) outweigh potential benefits with respect to survival advantage, functional status, symptom burden, quality of life, and care intensity at the end of life. Consequently, conservative management is a viable treatment alternative for older patients who are on or must decide whether to initiate dialysis. Comprehensive conservative care aims to delay the progression of kidney failure, preserve function, minimize the risk of adverse events or complications, optimize quality of life and health, prioritize comfort, facilitate advance care planning and shared decision-making, coordinate end-of-life care, and provide personalized, non-dialytic, and active management of kidney failure symptoms with full medical treatment. Compared to patients on dialysis, patients who choose conservative care management are able to maintain their quality of life, experience less hospitalization, and more often die at home or in hospice.

Advance Care Planning and Goals for End-of-Life Care

Advance care planning is an ongoing process involving patients' consideration and discussion of preferences, goals, and values for current and future transitions in the stage and treatment of CKD, including care at the end of life, with family members and health-care providers before they become incapacitated or decisions are triggered by urgent health events. Informal consideration and discussion of advance care planning may result in the designation of a surrogate decision maker and documentation of care goals through an advance directive (e.g., living will). In the context of CKD, advance care planning may address complex decisions related to commencing, continuing, withholding, or withdrawing dialysis.[43] These decisions need to be made early in the disease course, given the high prevalence of cognitive impairment and unpredictable, heterogeneous disease trajectories in dialysis patents. Advance care planning among patients with CKD is associated with reduced emotional burden on surrogate decision makers, decreased uncertainty about the future, decreased decisional conflict, improved satisfaction and psychological outcomes in bereaved families, patient–care partner congruence in decision-making, surrogate decision-making confidence, person-centered care delivery, and quality of care at the end of life. Despite these benefits and patient and care partner interest in advance care planning, low rates of completed advance directives and infrequent discussion of goals for end-of-life care with health-care providers are characteristic of the CKD population, even when death is expected.

Summary

Disorders of the kidney, both acute and chronic, are common at older age. Diagnosis, evaluation, and staging of kidney disease is based primarily on blood and urine lab tests. Guideline-recommended care for CKD includes identification and management of underlying causes to slow the progression, recognition and treatment of CKD-related complications, and preparation for renal replacement therapy prior to ESRD. Despite the high burden of CKD in older adults, the evidence underlying existing guidelines comes primarily from young and middle-aged adults and often does not take into account

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multimorbidity, geriatric conditions, and heterogeneity in life expectancy and health goals common at older age. A person-centered approach that includes geriatric assessment, engages and supports family caregivers, and incorporates palliative care principles across the spectrum of kidney disease is appropriate for older adults. Ongoing research focuses on developing and implementing person-centered models of care for older adults with CKD and will lead to advances in care for this population. Included in this is the need to identify ways to make geriatric assessment a part of routine care, to organize care to be more inclusive of family caregivers, and to increase access to high-quality conservative management.

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Chapter 27

Urological Conditions

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Introduction

Urological problems are extremely common in older adults. The incidence and prevalence of most urological conditions increase with advancing age in both men and women. Estimates indicate that approximately 20% of all primary care visits include some type of urological complaint. Indeed, urology consistently ranks third, behind ophthalmology and cardiology, in terms of the total annual number of outpatient clinical visits by older Medicare recipients in the United States. As the population ages, there is a growing need for more clinicians across all specialties focused on care of older adults.[1] This chapter addresses evaluation and management of many common urological conditions seen in older adults. Several topics relevant to urology are also covered in more detail in other chapters including urinary incontinence (Chapter 28), gynecologic issues (Chapter 30), sexuality (Chapter 48), and cancer (Chapter 41).

Urinary Tract Bleeding

Hematuria

Hematuria is one of the most common clinical conditions seen in urology. The common causes of hematuria in older adults are summarized in Table 27.1. Hematuria can be either gross or microscopic, and it may be episodic or persistent. Any episode of gross hematuria should be considered abnormal. On microscopic urinalysis, the generally accepted upper limit of normal is zero to three red blood cells per high-powered field. Because it is a common presenting sign for many forms of genitourinary pathology, older patients with gross or persistent microhematuria should undergo a thorough evaluation including upper urinary tract imaging and cystourethroscopy. Guidelines for the evaluation of asymptomatic microhematuria have been developed and recently updated by the American Urological Association.[2] Depending on the degree of hematuria and inherent risk factors, the evaluation may be tailored for each specific patient. However, age is a strong risk factor, and all adults ≥60 years of age are considered to be high risk for genitourinary malignancies. Other factors that place patients in the high-risk category include >30-pack-year smoking history, >25 RBC/HPF on microscopic urinalysis, or any history of gross hematuria.

Imaging with CT urography (computed tomography obtained with and without intravenous contrast and including delayed imaging) is the preferred radiographic evaluation.[3] Administration of IV contrast does require adequate baseline renal function, which should be assessed prior to imaging in older adults. Stones tend to be seen best on non-contrast images. Immediate and delayed contrast phases are useful for identifying renal perfusion and function, the course and caliber of the ureters, the characteristic of renal masses, cysts, and other lesions, and hydronephrosis or ureteral obstruction.

Although larger lesions in the bladder may be identifiable on CT imaging or ultrasound, smaller mucosal lesions may not be evident on these studies. Therefore, cystourethroscopy is an essential part of the complete urological evaluation for hematuria. Bladder cancers, particularly transitional cell carcinomas, typically start in the urothelium and are often visible as papillary lesions on cystoscopy. Carcinoma in situ is a particularly aggressive form of bladder cancer that may initially present with either microscopic or gross hematuria. On cystoscopy, this typically appears as a red, velvety patch of tissue. Histological examination of bladder biopsies and cytological examination of either voided urine or bladder washing specimens may be useful to help diagnose bladder malignancies. In the United States, transitional cell carcinoma is the most common form of bladder cancer in older adults, and cigarette smoking is one of the most common risk factors. Worldwide, squamous cell carcinoma is also a common form of bladder cancer and is often associated with prior *Schistosomiasis* infection.[4]

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Table 27.1 Causes of hematuria in older adults

Benign conditions
Stones
Urinary tract infection (UTI)
Pyelonephritis
Glomerular diseases of the kidney
Inflammatory conditions
Prostatitis
Cystitis
Urethritis
Malignant conditions
Bladder cancer
Transitional cell carcinoma
Squamous cell carcinoma
Carcinoma in situ
Ureteral cancer
Renal cancer
Renal cell carcinoma
Prostate cancer
Urethral cancer

Although an episode of gross hematuria can be quite distressing for patients, emergency evaluation is not usually necessary. Exceptions include patients experiencing clot retention with difficulty passing urine or patients requiring blood transfusions for anemia secondary to the hematuria. Cystourethroscopy with clot evacuation may be required in these cases. Electrocoagulation is used if a specific bleeding site or vessel is identified. However, in many cases, a specific source cannot be identified. Chemical coagulation with bladder infusions of dilute alum or formalin may be required in cases of persistent gross hematuria.

In older patients with renal insufficiency, a plain X-ray of the kidneys, ureters, and bladder (KUB) and renal ultrasound can be performed as the initial imaging evaluation. This should be supplemented with cystourethroscopy and retrograde ureteropyelography to look for abnormalities in the ureters or renal collecting systems. Filling defects may indicate some type of space-occupying lesion such as a stone, tumor, polyp, fungus ball, blood clot, or stricture.

Acute conditions including urinary tract infection (UTI), prostatitis, or recent passage of stones may be associated with hematuria. Urinalysis should be repeated after these conditions have been treated to document resolution. If hematuria persists, then urological consultation for further evaluation should be obtained, including imaging and cystoscopy. Hematuria is also frequently seen in elderly patients receiving chronic anticoagulation therapy; however, these individuals still require a complete urological evaluation, because 15–20% will be found to have significant underlying genitourinary pathology.

If an older adult patient has persistent microhematuria despite a negative urological evaluation with upper tract imaging, cystourethroscopy, and cytology, then referral to a nephrologist to evaluate for possible glomerular bleeding would be warranted. This is particularly true in patients with a history of either proteinuria or hypertension.[2] Repeat hematuria evaluation including imaging and cystoscopy may also be considered if microhematuria remains persistent over time.

Urinary Tract Infections (UTIs)

Bacteriuria and UTIs are among the most common urological diagnoses in older adults. The estimated lifetime risk for development of a UTI in women is greater than 50%, and associated costs are staggering. In the year 2000, the estimated overall annual expenditures for UTI care in the United States were $2.47 billion for women and $1.03 billion for men.[5,6] Epidemiological trends indicate the incidence and prevalence of UTIs increases with advancing age. Although seen in both sexes, a higher proportion of women are affected, with a ratio of 3:1. Various age-related physiological changes may predispose older adults to UTIs.[7] These include hormonal and vaginal changes associated with menopause, alterations in cognitive function, prostate disease, and changes in bladder physiology.

Asymptomatic bacteriuria should be differentiated from symptomatic UTI.[8] Symptomatic UTIs should be treated in patients of any age. Diagnosis should be confirmed with urinalysis and urine cultures. Determination of drug sensitivity on urine culture is important to ensure appropriate antibiotic therapy has been administered. This is particularly important given increasing rates of drug resistance seen with many common bacteria. Typical symptoms of acute UTI include fever, dysuria, urinary urgency and frequency, burning with urination, and suprapubic pain or discomfort.[9] Elderly patients may not develop these symptoms because of normal alterations in overall immune status associated with aging. Older adults may exhibit other symptoms resulting from UTI including lethargy, anorexia, or

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confusion. However, diagnostic accuracy of these nonspecific symptoms is often poor.[10] In elderly patients with new onset of delirium, a urinalysis and urine culture should be checked to determine if a UTI is present. Transient urinary incontinence may also be caused by UTI.

Oral antibiotic therapy is most commonly used for acute UTI if possible. Uncomplicated UTIs can usually be treated with simple, low-cost antibiotics such as amoxicillin or ampicillin, nitrofurantoin, fosfomycin, or trimethoprim-sulfamethoxazole. In patients who are allergic to these compounds, cephalosporins or doxycycline may be used as second-line therapy. Fluoroquinolones are usually reserved for complicated UTIs including those associated with concomitant pyelonephritis, urosepsis, or stone disease. The choice of appropriate antibiotic therapy should be guided by the patient's overall medical condition and renal function status, and the results of the antibiotic sensitivity profile for the specific organism. The duration of therapy generally ranges from 3 to 7 days, and is dependent on a variety of factors including overall complexity of the infection and response to therapy. Intravenous antibiotic therapy may be required in cases of severe infection, pyelonephritis, or urosepsis, or in cases where the causative bacterial organism is resistant to all available oral medication options.

The most common organisms seen in elderly patients with UTI include gram-negative bacteria such as *Escherichia coli*, *Pseudomonas*, *Klebsiella*, and *Proteus*. The most common gram-positive organisms seen in older adults with UTI include *Staphylococcus aureus* and *Enterococcus*. In patients with recurrent, culture-documented UTI, a clinical investigation to search for a nidus of infection is warranted. Common causes of recurrent infection include urolithiasis or other foreign body, chronic urinary retention, vesicoureteral reflux, and atrophic vaginitis. Treatment of the underlying condition may lead to resolution or a decrease in the frequency of UTIs.

Asymptomatic bacteriuria is quite common, particularly in both community-dwelling and elderly women living in institutional settings. In a community-based, cross-sectional analysis of 432 people aged 80 years or older, 19.0% of women and 5.8% of men were found to have asymptomatic bacteriuria.[11] In this study, urinary incontinence, reduced mobility, and systemic estrogen replacement therapy were identified as independent risk factors for asymptomatic bacteriuria in women. There is general evidence-based consensus that asymptomatic bacteriuria need not be treated with antibiotic therapy.[12]

UTIs associated with systemic bacteremia in elderly patients in a geriatric hospital carry a high risk of morbidity and mortality. In a retrospective study of 191 patients aged 75–105 years with concomitant positive urine and blood cultures, the in-hospital mortality rate was 33%.[13] A variety of factors associated with impaired physical and cognitive function were associated with increased mortality; however, in this study, advanced age itself was not identified as a significant risk factor. Mental status changes and a history of frequent UTIs may be associated with increased mortality in elderly patients with UTI.

Indwelling catheterization is clearly associated with an increased risk of UTI in older adults. If possible, clean intermittent catheterization is preferred in patients with urinary retention. Indwelling catheters should be used only if absolutely necessary. A number of treatments can be used to prevent development of UTIs in susceptible older adults. Increased hydration may decrease bacterial adherence to the urothelium of the bladder and urethra. Vaginal estrogen replacement may prevent development of UTI in postmenopausal women with atrophic vaginitis. Estrogen acts to acidify the vaginal fluid, which in turn facilitates growth of *Lactobacillus* species, which are part of the natural vaginal flora. *Lactobacillus* is an important component of this natural host-defense mechanism and helps prevent overgrowth of pathogenic bacteria associated with UTI. The estrogen is administered topically in the vagina to enhance absorption and direct action on the vaginal and periurethral tissues. Even in patients already on systemic estrogen replacement, additional vaginal administration is usually required to reach appropriate local tissue levels. Administration approximately three times weekly is usually sufficient. Exogenous estrogen administration is typically contraindicated in women with a personal history of uterine or breast cancer. However, oncologists may permit vaginal estrogen use in select women with a history of breast cancer that is in remission, particularly if the tumor was estrogen receptor negative.

Cranberries (*Vaccinium macrocarpon*) have long been considered a preventive agent for UTIs, and consumption of cranberry juice has been associated with decreased rates of UTI in elderly patients.[14] This is most likely due to urinary acidification and the azo-ring compounds found in the cranberries that prevents bacterial adherence to the urothelium. If patients intend to use cranberry for UTI prevention, they should be counseled to seek out products containing a high percentage of real juice rather than water. Cranberry tablets may be substituted for juice

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in diabetic patients or those on a reduced-calorie diet. Patients should avoid exceeding the recommended dosing because of the risk of developing calcium oxalate urinary stones at high concentrations. Overall data on cranberry supplementation has yielded mixed results. This option may be more effective in specific settings such as assisted living or long-term care facilities, or in older adults with a known propensity for recurrent UTIs. Other supplements including D-mannose have also shown promise as an option to prevent UTIs.[15]

Urinary Catheters

Chronic use of indwelling urinary catheters should generally be avoided.[16] Indwelling catheters can be associated with significant potential complications including UTIs, urosepsis, stone formation, and tissue erosion. Care should be taken to remove the catheter as soon as feasible, and to monitor the patient for signs and symptoms of UTI. With extended time, chronic catheter irritation may lead to squamous metaplasia of the bladder epithelium and squamous cell carcinoma of the bladder. Surveillance cystoscopy is recommended on a regular basis to monitor for this potential complication.

Measures to avoid catheter-associated urinary tract infections (CAUTIs) have become important targets to enhance quality of care, particularly in acute hospital and residential care facilities.[17] Rates of CAUTI in hospitals are reportable and important quality measures, particularly in the care of older adults. The Centers for Disease Control and Prevention (CDC) provide a guideline for the prevention of CAUTIs that includes priority recommendations related to the appropriate use, aseptic insertion, and maintenance of urinary catheters. Further, the guideline notes administrative infrastructure and support (including staff education) is necessary, as is a quality improvement program.[18]

If chronic indwelling catheter use is required, suprapubic catheter drainage is usually preferred over urethral catheterization. A suprapubic catheter may be easier for caregivers to change and is often more comfortable for patients. In patients who are sexually active, a suprapubic catheter is useful because it moves the catheter away from the genitals. Chronic urethral catheterization can also lead to urethral or bladder neck erosion and subsequent urinary incontinence, particularly if the catheter is frequently on tension. Urinary leakage around an indwelling catheter is usually caused by either catheter blockage or bladder spasms. Gentle irrigation of the catheter with sterile saline can be used to relieve obstruction from urinary sediment. Placement of larger catheters should be avoided because this will only serve to dilate the urethra or suprapubic tract and will not correct the underlying problem. With time, use of larger catheters can lead to urethral or bladder neck erosion and worsening urinary incontinence. Treatment of urinary incontinence in patients with this type of urethral erosion can be very difficult and often involves major surgery such as a cystectomy with urinary diversion or augmentation enterocystoplasty with closure of the bladder neck.

Antimuscarinics or other medications for treatment of detrusor overactivity may be useful to reduce bladder spasms and improve catheter-associated incontinence. The most common medications used in the treatment of overactive bladder, including dosages and potential side effects, are listed in Table 27.2. Care should be taken when prescribing these agents in older adults, and the patient and family or caregivers should be instructed to watch closely for any side effects. The most common adverse effects associated with the antimuscarinic agents include dry mouth, constipation, and confusion or other cognitive changes. Recent epidemiological data suggests a potential risk of development or progression of dementia with ongoing use of anticholinergic medications.[19] Chemodenervation with intradetrusor injection of onabotulinum toxin A is also a clinical option for treating refractory bladder overactivity.

Stone Disease

Approximately 20% of all adults will develop urinary stone disease at some point in their lives. Overall rates of stone formation and passage do not differ in older adults compared with the general population.[20] Those older adults with a prior history of stone disease are at significantly increased risk for development of recurrent stone episodes. Inadequate hydration is one of the primary risk factors for stone disease. This is a common problem in older adults who may reduce fluid intake in an effort to avoid urinary incontinence, or because of changes in smell, taste, and sense of thirst. Epidemiological and health-care utilization analyses have revealed that Medicare beneficiaries with a diagnosis of stone disease have a 2.5 to 3-fold higher rate of inpatient hospitalization for the condition compared with younger patients. However, in the absence of associated urosepsis or other complex comorbidities, older adults with stone disease can often be treated effectively as an outpatient. Small stones (<5 mm) can often be treated conservatively with increased hydration and oral

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Table 27.2 Medications for overactive bladder

Medications	Typical dosages
Antimuscarinics	
Oxybutynin	5 mg twice daily to four times daily (maximum dose 30 mg total per day)
Oxybutynin (time released)	5, 10, or 15 mg once daily
Oxybutynin (transdermal patch) Oxybutynin (transdermal gel)	3.9 mg/day, patch changed twice weekly 1 packet topically daily
Tolterodine	1 or 2 mg twice daily
Tolterodine (time released)	4 mg once daily
Darifenacin	7.5 or 15 mg once daily
Solifenacin	5 or 10 mg once daily
β-3 agonists	
Mirabegron	25 or 50 mg once daily
Potential side effects of anticholinergic medications	
Dry mouth	
Constipation	
Confusion	
Blurry vision	
Headache	
Tachycardia	
Prolongation of QT interval on electrocardiogram	
Potential side effects of β-3 agonists	
Hypertension	

analgesics. Stones may pass spontaneously in many cases. Oral alpha-blocker medications such as tamsulosin can help with relaxation of ureteral smooth muscle, which can aid in spontaneous stone passage with natural peristalsis. Patients are encouraged to collect and strain their urine to capture the stones for chemical analysis. This can help identify stone composition, which may guide dietary recommendations to assist with future stone prevention.

Large stones often require surgical intervention. Cystoscopy and ureteral stent placement can be used to bypass an obstructing stone and help relieve renal colic. Indications for stent insertion include upper tract obstruction, particularly with significant urinary infection or bacteriuria, a solitary functioning kidney, underlying renal insufficiency, or intractable nausea, vomiting, or pain. Subsequent surgical treatment may include ureteroscopy with stone fragmentation and removal or extracorporeal shock wave lithotripsy. Percutaneous nephrostolithotomy may be required for large stones in the renal pelvis or calyces. Endoscopy is performed through a tract from the back directly into the renal collecting system. Laser lithotripsy is used to fragment the stones and extract the pieces. Surgical outcomes are quite favorable for older adults, and overall results are comparable with data from younger patients.

Stone composition may also change with advancing age.[21] Alterations in stone chemistry in older adults may be related to associated changes in vitamin D and calcium metabolism, which can be affected by age-related physiological changes. The overall proportion of uric acid stones also appears to increase with advancing age. This may be related to a progressive defect in urine ammoniagenesis, which is observed with aging, and which leads to a low urinary pH observed in patients who form uric acid stones. In addition, adults with diabetes tend to have a higher incidence of uric acid stone production compared with those who do not have diabetes.[22] This may explain higher rates of uric acid stone production observed in some older adults, who also have a greater tendency to have diabetes mellitus or a history of gout.

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However, overall rates of stone formation are similar in older and younger adults.

Urological Malignancies

The incidence and prevalence of most urological malignancies increase with advancing age. In some cases, there may be differences in the type or progression of cancer compared with younger patients. For a more detailed discussion regarding evaluation and management of cancers in older adults, please refer to Chapter 41.

Bladder Cancer

Bladder cancer frequently presents initially with either gross painless hematuria or persistent microhematuria. In the United States, transitional cell carcinoma is the most common type of bladder cancer.[23] On cystoscopic examination, this usually appears as either a papillary or sessile tumor of the bladder mucosa. Carcinoma in situ is a particularly aggressive form of bladder cancer. Cystoscopically, this typically appears as a velvety red patch in the bladder mucosa. Bladder wash cytology and biopsies are used to establish the diagnosis. Various biomarkers have been developed to assist with diagnosis and management. Blue-light cystoscopy is a recent advance that can be used to enhance endoscopic surgical management and monitor for recurrence.[24]

Choice of treatment for bladder cancer is dependent on grade and stage of the tumor. Low-grade tumors that do not invade into the muscular layer of the bladder are usually treated with endoscopic resection. Tumor recurrence occurs in up to 70% of patients, and careful postoperative monitoring is essential. Management consists of repeated surveillance cystoscopy and cytology every 3 months for 2 years, every 4 months for 2 years, every 6 months for 2 years, and then annually. Adjuvant therapy with intravesical administration of Bacillus-Calmette-Guérin (BCG) or mitomycin-C may be used to help decrease tumor recurrence. High-grade noninvasive tumors may also be treated with a combination of surgical resection and intravesical chemotherapy.

Invasion of tumor into the muscularis propria is an ominous pathologic finding and is associated with a high risk of disease progression. Radical cystectomy with urinary diversion has long been considered the gold-standard therapy. This is a major surgical procedure that can be associated with significant risk of morbidity and mortality. Recent studies indicate elderly patients can safely undergo this type of surgery, although risks and potential benefits need to be carefully considered for each individual patient.[25,26]

Benign Disorders of the Prostate

Benign prostatic hyperplasia (BPH) and prostatitis are the most common nonmalignant conditions of the prostate.

Benign Prostatic Hyperplasia (BPH)

The prostate gland secretes fluid that forms the ejaculate and provides nutrient factors required for function and survival of sperm. Benign enlargement of the prostate gland typically begins at approximately 40–50 years of age.[27] Enlargement is driven in part by the presence of serum testosterone. Proliferation of both stromal and epithelial components of the prostate gland can contribute to BPH. The effect of prostatic enlargement is variable. Some men experience few symptoms; however, many men develop obstructive voiding symptoms including urinary frequency, hesitancy, nocturia, and a slow urinary stream. Nocturia can be particularly bothersome for some men.[28] Getting up once per night is considered normal, and usually does not require therapy. However, data has shown that experiencing two or more episodes of nocturia each night leads to substantial impairment in overall and health-related quality of life.[29] Some men experience acute or chronic urinary retention with incomplete emptying of the bladder. Irritative voiding symptoms, including urinary urgency or urgency urinary incontinence, are also common and bothersome. Pain is uncommon unless men have acute urinary retention or need to strain to urinate. Prostate size does not necessarily correlate with symptoms. In fact, some men with relatively small prostate glands may have severe symptoms, particularly if the median lobe of the prostate gland is enlarged. Voiding symptoms associated with BPH can have a significantly negative impact on both overall and health-related quality of life.[30] Fortunately, there are a wide variety of both surgical and nonsurgical therapies for BPH that can be quite effective in relieving these bothersome symptoms. These are outlined in Table 27.3.

Medical Therapies

Currently, there are three main categories of drugs used for the pharmacological treatment of BPH.[31] These include α-adrenergic antagonists, 5-α-reductase inhibitors, and nutritional supplements and phytotherapies. The α-adrenergic antagonists include terazosin (Hytrin), doxazosin (Cardura), tamsulosin (Flomax), and alfuzosin

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Table 27.3 Treatment options for benign prostatic hyperplasia

Medical therapies	
Alpha-adrenergic antagonists	
Nonselective	
Terazosin (Hytrin)	1–10 mg PO at bedtime (must titrate dose)
Doxazosin (Cardura)	1–8 mg PO at bedtime (must titrate dose)
Selective	
Tamsulosin (Flomax)	0.4–0.8 mg PO at bedtime
Alfuzosin (Uroxatral)	10 mg PO once daily
5-alpha-reductase inhibitors	
Finasteride (Proscar)	5 mg PO once daily
Dutasteride (Avodart)	0.5 mg PO once daily
Surgical therapies	
Transurethral resection of the prostate (TURP)	
Transurethral incision of the prostate (TUIP)	
Open suprapubic prostatectomy	
Open retropubic (nonradical) prostatectomy	
Minimally invasive therapies	
Transurethral photovaporization of the prostate (PVP)	
Transurethral microwave thermotherapy (TMT) Holmium laser enucleation of the prostate (HoLEP)	
Lasers (various)	
Prostatic stents (not commonly used in clinical practice) Prostatic urethral lift (PUL)	

(Uroxatral). These drugs act by blocking α-adrenergic receptors in the tissue of the prostatic urethra and bladder neck. This leads to relaxation of smooth muscle in these tissues, which causes a decrease in bladder outlet resistance. These drugs have been shown to work well, particularly in men with smaller prostate glands. The medications are typically prescribed once daily at bedtime. This helps reduce some potential side effects including orthostatic hypotension. The more selective medications tamsulosin and alfuzosin appear to have fewer overall side effects compared to the older, less selective medications in this class.[32] These more selective agents are more commonly used in clinical practice. Men should be warned to rise slowly and make sure they have their balance before getting up from bed. This is particularly true for men who have nocturia and get up to go to the toilet during the night. Retrograde ejaculation, a condition where semen goes backwards into the bladder with orgasm due to relaxation of the bladder neck, and erectile dysfunction are also potential adverse effects of the alpha-blocker medications. Another potential side effect, particularly with use of tamsulosin, is the floppy-iris syndrome.[33] This causes relaxation of the smooth muscle in the iris of the eyes and can be a problem if the medication is not discontinued prior to cataract surgery. The condition causes intraoperative billowing of the iris musculature with risk of prolapse and progressive miosis. Men taking these medications must advise their ophthalmologist prior to any surgical interventions.

The 5-α-reductase inhibitors act by blocking the enzyme that helps catalyze the conversion of testosterone into dihydrotestosterone (DHT). Limiting the amount of circulating DHT leads to shrinking of the prostate gland, although the full effect may not be seen for several months after starting the medication. The two main drugs in this category include finasteride (Proscar) and dutasteride (Avodart). These medications generally work better in men with larger prostate glands. Potential side effects include decreased libido and development of gynecomastia or breast discomfort. These drugs also cause an

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approximate 50% reduction in circulating serum prostate-specific antigen (PSA). It is recommended that a PSA level be checked prior to initiating these medications. After starting a 5-α-reductase inhibitor, observed PSA levels should be doubled to determine the corrected PSA for a given patient.

Some studies have suggested that using a combination of an α-adrenergic antagonist and a 5-α-reductase inhibitor may have better overall efficacy compared with monotherapy, particularly for men with larger prostates or more severe voiding symptoms.[32] Although this dual therapy may be beneficial for some patients, potential side effects and additive costs of these medications must also be considered.

A number of natural remedies and plant extracts have gained popularity for the treatment of the symptoms of BPH. The most widely used preparation is saw palmetto (*Serenoa repens*).[34] The exact mechanism of action is unknown, but theories suggest it may be similar to either 5-α-reductase inhibitors or other hormonally active agents. To date, there have been relatively few studies examining the efficacy of these compounds, particularly in elderly men. These agents are available in health-food stores without a prescription. It is difficult to counsel patients about the safety and efficacy of these types of treatments because of the overall paucity of data. These phytotherapeutic agents are also not subject to regulation by the US Food and Drug Administration (FDA), and there may be significant variations between products and even between batches of the same product.

The roles of micronutrients and other nutritional components have recently attracted attention as a potential option for treatment and perhaps prevention of BPH. Research data is limited, particularly for elderly men; however, this is a rapidly growing area of both basic science and clinical research. Zinc has long been advocated as a mineral important for prostate health. Other agents being examined for their potential influence on prostate physiology include lycopenes, bioflavonoids such as soy, and selenium.[35] If these types of nutritional agents show a clinically significant effect in either treating BPH or preventing clinically significant symptoms, it may alter the ways in which this disorder is managed in the future.

Surgery

Some older men with symptomatic BPH may require surgical treatment, particularly if medical therapy has not been clinically successful.[36] Traditional surgical options include open prostatectomy using either a suprapubic or retropubic approach or transurethral resection. Open surgery is still used in some men with very large prostate glands (>100 g), although this has been supplanted by newer laser enucleation procedures (see detailed descriptions below).[37] For most men, transurethral surgery has replaced open surgery as the technique of choice, because of lower overall morbidity and mortality and improved recovery. Transurethral resection of the prostate (TURP) remains the gold standard to which all other forms of therapy are compared. The main risks of TURP include bleeding and infection and development of hyponatremia from absorption of hypotonic irrigation solution during surgery. Recent technical advances have led to development of bipolar surgical systems that can utilize isotonic saline for intraoperative irrigation. This has reduced the incidence of post-TURP hyponatremia syndrome and the associated morbidity of the procedure. Another potential adverse effect is development of retrograde ejaculation following TURP. In men with a smaller prostate or with an elevated bladder neck, a transurethral incision of the prostate (TUIP) may help to avoid this potential complication.

Minimally Invasive Therapies

A number of minimally invasive surgical therapies have been developed to treat symptomatic BPH.[38] Examples include transurethral photovaporization of the prostate (PVP) and transurethral microwave thermotherapy (TMT), which heats the prostate, leading to subsequent sloughing of the treated tissue. Several different techniques using various laser-energy methods have also been developed. Holmium laser enucleation of the prostate (HoLEP) is a newer technique that can be used particularly for men with large prostates (>100 grams).[39] In these cases, the prostate is shelled out from its capsule endoscopically using laser energy, and the pieces are divided or morcellated for extraction from the bladder. Another newer procedure is the prostate urethral lift (UroLift®) in which thin suspensory implants are deployed into the tissue of the prostate to pull open the urethra and improve voiding symptoms.[40]

Intraurethral prostatic stents have also been developed for treatment of mild-to-moderate BPH.[41,42] These devices are placed across the prostatic urethra and function to push open the urethral lumen with a radial spring-like configuration. The overall popularity of urethral stents for routine management of BPH has declined recently because of associated complications including erosion, migration, or stricture. Removal can be difficult as the components of the stent become incorporated in the urethral epithelium. Nonetheless, these

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devices could be useful in highly select patients, such as frail elderly men with diminished life expectancy who may be too ill to undergo a more involved procedure.

Many of these current minimally invasive options do offer potential advantages for elderly patients. Some can be performed using local anesthetic in the outpatient office setting. This obviates the need for general or regional anesthesia, which may be advantageous for older men with multiple comorbidities. Some may reduce potential perioperative bleeding, which can be associated with traditional TURP. This could be beneficial for elderly men who may be on anticoagulation therapy for treatment of cardiovascular disease. Another potential benefit of minimally invasive procedures is improved preservation of sexual function following surgery.[43] Although all minimally invasive treatments have shown promise in clinical trials with short-term follow-up, the long-term efficacy of each has yet to be fully determined.

Prostatitis

Several types of infection and inflammation can affect the prostate. These include acute prostatitis, chronic prostatitis, and prostatodynia. Although the precise prevalence of prostatitis is unclear and varies among studies, epidemiological data suggests an overall prevalence of 2–10% in adult men.[44,45] Prostatitis can have substantial clinical effects in elderly men. Medicare beneficiaries are 2 to 2.5 times more likely to require inpatient hospitalization for treatment compared with younger men.[6]

Acute bacterial prostatitis is typically associated with a rapid onset of symptoms including fever, chills, irritative voiding symptoms with frequency and urgency, dysuria, and pelvic or perineal pain. Elderly men may not mount a full symptomatic response, and clinical findings may be more subtle because of natural changes in immune response with aging. The most common cause is ascending infection with bacteria from the distal urethra. This may be exacerbated by sexual activity or urethral instrumentation such as cystoscopy or catheter insertion. Rectal examination will usually reveal a swollen and tender prostate. Prostate massage should *not* be performed if acute bacterial prostatitis is suspected, because this can lead to systemic dissemination of bacteria and subsequent urosepsis. Urine cultures should be obtained to identify the involved organism(s) and to guide antibiotic therapy. If the patient is acutely ill, hospitalization with intravenous antibiotic administration may be necessary. Acute urinary retention may require suprapubic tube insertion. Urethral catheterization should be avoided to prevent bacterial dissemination and development of urosepsis. Oral antibiotic therapy is usually continued for 4 weeks. The most common antibiotics used for this are doxycycline or the fluoroquinolones because they can achieve adequate tissue concentrations in the prostate. CT examination may be useful to identify prostatic abscesses that would necessitate surgical drainage.

Chronic prostatitis occurs more commonly than acute prostatitis in elderly men.[45] Typical symptoms include urinary urgency and frequency, dysuria, nocturia, low back pain, scrotal or perineal discomfort, or suprapubic pain. Findings on rectal examination can be variable. Some men will have significant prostate swelling or tenderness but others will not. Secretions obtained from prostatic massage can be helpful to establish the diagnosis. Microscopic examination may reveal bacteria or white blood cells. A urine culture should be obtained to help guide antibiotic selection. Initial therapy with approximately 2 weeks of antibiotics is indicated, although longer courses of antibiotics may be necessary. Avoiding dietary irritants such as caffeine, alcohol, or carbonated beverages may also be helpful.

Prostatodynia refers to a syndrome in which patients have symptoms suggestive of acute or chronic prostatitis without objective clinical findings. Pain is one of the hallmarks of this condition. Antibiotics often do not help to alleviate symptoms in these patients. Treatment may be difficult and needs to be individualized for each patient.

Penile Disorders

In addition to penile cancers, a wide variety of benign conditions can affect the male genitalia.[46] Many of these are inflammatory conditions that are influenced by both anatomical factors and other comorbid conditions such as diabetes mellitus.

Phimosis

Phimosis is a condition characterized by narrowing of the prepuce, which makes retraction of the foreskin difficult or impossible. This can lead to pain and inflammation and may be associated with difficult voiding for some patients. Some dermatological conditions such as lichen sclerosus and balanitis xerotica obliterans (BXO) increase the incidence of developing phimosis and potentially penile cancer.[47] Surgical treatment with either a dorsal slit to release constriction of the foreskin or circumcision may be needed to treat the condition.

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Paraphimosis

In paraphimosis, the foreskin is retracted and becomes trapped behind the coronal sulcus. Tissue edema and swelling occurs that prevents the foreskin from reducing back over the glans penis. This can be very painful, and, if left untreated, may lead to tissue necrosis or significant infection.[48] One of the most common causes of paraphimosis is retraction of the foreskin for placement of a urinary catheter or other manipulation of the penis, with failure to properly reduce the foreskin back to anatomical position after the procedure. It can also occur following sexual intercourse if the foreskin is not returned to anatomical position covering the glans penis. Manual reduction of a paraphimosis should be attempted and may require use of a penile block with injectable local anesthetic. Once an adequate anesthetic level is obtained, gentle pressure can be applied circumferentially to the penis to reduce tissue edema in the glans. The clinician's thumbs are then placed on either side of the glans penis, and the first two forefingers of each hand are used to pull the foreskin back down past the coronal sulcus and over the glans. If this is unsuccessful, surgical reduction with a dorsal slit or circumcision may be required. Treatment of paraphimosis should be considered a urological emergency.

Peyronie's Disease

One of the more common benign conditions of the penis is Peyronie's disease.[49] This condition is characterized by development of a painful lump within the tissue of the penis associated with curvature of the penile shaft toward the lesion with erection. This is caused by a fibrous plaque in the tunica albuginea, the dense connective tissue that forms the outer layers of the corpus cavernosum of the penis.

Treatment of Peyronie's disease can be difficult, and a variety of different therapies have been tried with variable success.[50] Oral anti-inflammatory agents, including vitamin E, colchicine, and p-aminobenzoate (Potaba), have been useful in some men and may reduce pain associated with both erection and the plaques themselves. In 2013, the FDA approved use of injectable collagenase clostridium histolyticum (Xiaflex) for treatment of Peyronie's disease.[51] This acts to break down collagen plaque through an enzymatic reaction. Several treatment sessions may be necessary to yield clinical improvement, and penile stretching or modeling have been used as adjuvant measures in combination with injection.[52]

Scrotal Disorders

In addition to testis cancer, several benign conditions of the scrotum can occur in elderly men. These can be quite bothersome for patients and often require additional clinical evaluation and treatment.

Epididymitis

Acute epididymitis is a common condition in older men.[6] This is usually caused by bacterial infection and may be associated with recent urinary tract instrumentation or chronic urethral catheter use. It often occurs in conjunction with other genitourinary infections including acute cystitis or prostatitis. Symptoms include swelling of the affected hemiscrotum with pain and swelling of the involved epididymis. Patients can also experience systemic symptoms of fever, chills, and malaise. Scrotal ultrasound can be useful to establish the diagnosis. Epididymitis is typically associated with increased arterial blood flow to the epididymis in response to acute inflammation. A urine culture should be obtained to guide antibiotic therapy, although empiric treatment should be administered prior to obtaining final culture results. Scrotal support, bed rest, and topical application of ice packs can provide symptomatic relief.

Scrotal Edema

Benign scrotal edema is a very common condition seen in older men, particularly in the acute hospital setting. Patients with vascular disease, hypertension, severe ascites, congestive heart failure, and pulmonary edema are at increased risk for developing scrotal edema. Excess fluid accumulates in the most dependent areas, including the legs and feet, the presacral tissues, and the scrotum. The condition is usually painless. In some cases of severe scrotal swelling, compression of the urethra and penis can cause voiding difficulty and urinary retention. This may require placement of a urinary catheter. Treatment typically involves conservative therapy with scrotal elevation and support, and ice packs if the patient has pain. Diuretics may be indicated to treat underlying conditions such as congestive heart failure (CHF) or pulmonary edema. Scrotal swelling will typically resolve with time once the underlying cause has been adequately addressed.

Urethral Strictures

Urethral strictures are scars that develop in the urethra and may lead to narrowing or even obliteration of the

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urethral lumen. Historically, sexually transmitted infections (STIs) such as gonorrhea were a major cause of urethral stricture disease. They are now most often associated with a history of urethral or pelvic trauma. Traumatic urethral instrumentation increases risk for development of a urethral stricture. In men who have undergone radical prostatectomy for prostate cancer or TURP for BPH, scarring may develop at the junction between the urethra and bladder, leading to a bladder neck contracture.

Urethral dilation may be adequate in some cases of mild strictures, although this usually requires ongoing dilation by intermittent self-catheterization. Longer or more complex urethral strictures in men are typically treated surgically.[53]

Urethral strictures are uncommon in women and are most typically caused by prior urethral trauma due to obstetrical issues or straddle injuries. Urethral dilation is not typically indicated unless an instrument cannot be passed easily during cystoscopy or surgery. There is no data to support the routine use of urethral dilation in women for the treatment of voiding dysfunction. Women with clinically significant urethral strictures typically require open surgical reconstruction for successful management.

Benign Disorders of the Lower Female Urinary Tract

Urethral Caruncle

Urethral caruncles typically present as benign, polypoid lesions of the distal urethra and are most often seen in postmenopausal women. These are usually small lesions, although in some cases they can reach up to 1–2 cm in diameter.[54] Etiology is unknown but may be related to urethral prolapse and chronic irritation associated with estrogen deficiency. In some patients the lesion may be painful or may bleed to the touch. Care must be taken to differentiate urethral caruncles, which are typically soft and mobile, from urethral carcinomas, which are typically firm or hard and more fixed in position. Excisional biopsy may be performed if there is a question about the histological composition. In most cases, urethral caruncles can be treated nonsurgically with warm Sitz baths and anti-inflammatory medications. Topical estrogen can be quite useful and helps to shrink the lesions. Complete resolution may be observed with continued estrogen application. Excision is indicated only if more conservative therapies are ineffective or if the patient experiences clinically significant voiding dysfunction from urinary obstruction.[55]

Urethral Diverticulum

A urethral diverticulum is an outpouching of the anterior urethral wall in women that may be associated with significant lower urinary tract symptoms. The classic clinical triad includes dysuria, dyspareunia, and postvoid urinary dribbling. Although the exact etiology is unknown, theories include obstruction of paraurethral ducts with development of an inclusion cyst in the urethral wall. Subsequent spontaneous drainage of the cyst cavity into the urethral lumen leads to development of an epithelialized tract between the diverticulum and the urethra. Congenital abnormalities can also lead to a thinning of the anterior urethral wall, which in turn could cause development of an opening between the diverticular space and the urethra. Physical examination usually reveals a soft tissue bulge in the suburethral area in the vagina. These are typically tender to palpation because of the trapping of infected urine or sediment in the diverticular sac. Radiographic examination with voiding cystourethrography or pelvic magnetic resonance imaging (MRI) can be helpful to establish the correct diagnosis. Excision including removal of the entire diverticular sac is indicated for symptomatic diverticula.[56] In some cases, the diverticular sac may be quite close to the external urethral sphincter, and care must be taken not to injure this structure during surgery, which could lead to urinary incontinence. If the opening to the diverticulum is small, the urethra may be repaired primarily; however, a vascularized pedicle flap may be necessary to repair larger defects in the anterior urethra. Successful excision and repair of the urethral diverticulum can help improve sexual function in affected women.[57]

Genitourinary Fistulae

A fistula is defined as a connection between two hollow organs or between a hollow organ and the skin. Fistulae can occur in the genitourinary tract in older adults, often resulting from other underlying conditions or after treatment for these conditions. The most common types of fistulae involving the urinary system in older women include vesicovaginal fistulae between the bladder and vagina, and vesicoenteric fistulae between the bladder and bowel.[58] Men can also develop fistulae, most commonly between the bowel and bladder. In developed countries, most vesicovaginal fistulae are iatrogenic and related to prior pelvic surgery such as

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hysterectomy. Colonic diverticular disease leads to an increased risk of vesicocolonic fistulae. Inflammatory bowel disorders such as Crohn's disease increase the risk of fistulae between the bladder and small intestine. Other risk factors include prior pelvic surgery or radiation for a history of pelvic malignancy. With a vesicovaginal fistula, women typically experience continuous urinary leakage from the vaginal vault. Chronic UTI with enteric bacteria is the most common problem associated with vesicoenteric or vesicocolonic fistulae. These patients may also experience pneumaturia with passage of air during urination. These associated symptoms often have a strong negative influence on quality of life. Evaluation includes identification of the type and location of the fistula tract through physical examination, endoscopy, and imaging. Treatment is most often surgical with excision of the fistula tract and repair of the affected organs.

Atrophic Vaginitis and Lichen Sclerosus

In postmenopausal women, decreased vaginal estrogen levels can lead to tissue changes in the vaginal mucosa. The most common condition is atrophic vaginitis, which may involve variable degrees of tissue inflammation. On clinical examination, the tissue appears either pale and thin with loss of natural rugations, or may be inflamed and tender. Patients may experience pelvic pain or dryness. Women who are sexually active may complain of dyspareunia. Unless contraindicated by a personal history of uterine cancer or estrogen receptor positive (ER+) breast cancer, topical estrogen replacement is indicated to help reduce symptoms. Vaginal estrogen therapy for atrophic vaginitis can also help to reduce the risk of recurrent urinary tract infections in affected women by helping to promote growth of *Lactobacillus*, the natural vaginal flora.[59]

Lichen sclerosus of the female genitalia can be quite bothersome for affected patients.[60] The tissue tends to develop white patches that can be thickened and crinkly, and patients often experience itching or burning sensations. If untreated, it can cause permanent scarring. Topical steroids are often used to treat symptoms and prevent progression. Over time, lesions may resolve with treatment.

Pelvic Organ Prolapse

Various forms of pelvic organ prolapse are common in elderly women. Loss of anterior pelvic floor support results in a protrusion of the bladder into the vaginal vault, which is referred to as a cystocele. A rectocele occurs if the posterior aspect of the vaginal wall is involved. In an enterocele, the apex of the vaginal vault is prolapsed. These entities may occur alone or in combination. Uterine prolapse can also occur in women with an intact uterus but dysfunction or laxity of the supporting tissues. Pessaries can be used to reduce prolapse and provide an effective, nonsurgical form of therapy for many patients. In some cases, surgery with either repair of the vaginal vault or closure of the vaginal introitus (colpocleisis) may be indicated. Additional information on pelvic organ prolapse is provided in Chapter 30 on gynecological disorders in elderly women.

Sexual Health

Several comorbid conditions that are common in older adults can have a significant negative influence on sexual health, including diabetes, hypertension, heart disease, and vascular insufficiency. Erectile dysfunction is often an early warning sign of underlying cardiovascular disease. Urinary incontinence can negatively influence sexual activity in both men and women.[61,62] As social views have evolved over time, there has been increased recognition of some of the challenges that LGBT people face with aging. A recent national summit on addressing the needs of older LGBT people dealing with cancer, including various forms of urological cancers, examined many of these issues.[63] In addition to needs for patients themselves, spouses, partners, and other family members or loved ones may face unique issues related to health needs. These may include family and elder law issues, need for social services, and other ancillary care areas.

Urology in Palliative and End-of-Life Care

Older adults receiving palliative care or near the end of life may have unique urological clinical needs.[64] Some urologic conditions, particularly locally advanced or metastatic genitourinary cancers, may necessitate palliative care. This may lead to multiple other clinical needs that are often best addressed by a multidisciplinary care team. Adequate pain management, nutritional support, assistance with basic and instrumental activities of daily living, and support of mental and spiritual health needs are among the central aspects of quality palliative care. Some urologic conditions may also become more prominent near the end of life, including voiding dysfunction, urinary incontinence or retention, and UTI. Supporting individual patient and family goals of care for these conditions becomes paramount and can help optimize quality of life.

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Conclusion

A wide variety of urological conditions commonly occur in older adults. Associated urologic symptoms such as urinary incontinence, UTI, pain, sexual dysfunction, and others can have substantial negative effects on overall and health-related quality of life. Appropriate evaluation and treatment are important for effective management. Successful treatment may lead to reduction or elimination of symptoms and significant improvements in quality of life for older patients.[65,66] Successful diagnosis and treatment of urological conditions frequently have important implications related to patient autonomy, shared decision-making, relief of caregiver burden, and other aspects of geriatric care.

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Chapter 28

Urinary Incontinence

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Urinary incontinence (UI), the involuntary loss of urine, has a prevalence of up to 50% in older women and about 22% in older men, and these rates are even higher among nursing home residents.[1–3] UI is often referred to as a silent health condition and despite high prevalence among women, only 25% report symptoms and seek care associated with UI.[4]

Only approximately one third of incontinent women have UI to such a degree that they viewed it as a significant bother.[5] According to data from the National Health and Nutrition Examination Survey 2007–10, 12% of older women reported severe or very severe UI. Among noninstitutionalized women aged 65 or older, more than 50% reported a urinary leakage, compared to 25% of noninstitutionalized men in the same age range.[1] Frequent or severe UI can have a devastating impact on people's lives. This can lead to social withdrawal, depression, sexual distress, sleep quality, increased economic burden, and can contribute to the decision to go into a nursing home.[6–8] Leaking small amounts of urine and/or bowel can often be managed by wearing absorbent products such as pads and all-in-one undergarments, and has only a modest impact on quality of life.

UI prevalence increases with age in older women and men compared to younger age groups (<65 years).[1] In US adults aged 80 and older, the prevalence of UI in women is 26.7% and in men is 13.0%. The prevalence of combined UI and fecal incontinence in this age group is 10.5% in women and 3.3% in men.[1] Older men may have increases in incontinence prevalence with age similar to older women.[1] Fecal incontinence is covered in more depth in Chapter 29.

The loss of urinary continence will not always occur with aging. Many specific age-related changes, such as functional impairments in mobility, dexterity, cognition, and reduction in bladder capacity and sensation, contribute to UI. Other established risk factors that are not age-related include obesity, parity, and delivery mode in women for UI. The strongest single risk factor for UI in men other than age is prostatectomy or transurethral resection.[2,5,9]

With regard to care-seeking, an estimated 60% of people with UI who are identified through surveys have not reported their incontinence to a health-care provider,[4] perhaps because they are embarrassed or believe nothing can be done to help.[4,9–12] This is unfortunate because continence problems are reversible in many and can be managed in most cases. Health-care providers, therefore, should specifically screen for incontinence.[2,8,10–12,13]

Physiological Mechanism for Continence, Micturition, and Defecation

Innervation of the lower urinary tract system is under cholinergic, adrenergic, and somatic control. The early phase of bladder accommodation is mediated by β-adrenergic receptors in the bladder dome. Bladder contraction is mediated by cholinergic (parasympathetic) activity, whereas relaxation of the urethral and anal internal and external sphincters is mediated by adrenergic (sympathetic) pathways in the pudendal nerve via a spinal reflex mediated by the S2–S4 sacral nerve roots. Normal bladder capacity is 300–600 ml.

Central nervous system control of bladder function is mostly inhibitory; that is, reflex bladder contractions are actively inhibited until a socially appropriate time and place to urinate is found. This inhibition occurs through neural linkages from the sensorimotor cortex of the frontal lobes to the brainstem, cerebellum, thalamus, and spinal cord. Micturition normally involves a conscious disinhibition of bladder and/or bowel contractions. Thus, stroke and other neurological processes can result in UI because of loss of central cortical inhibition. Excessive bladder-filling may overcome higher cortical inhibitory inputs, resulting in the involuntary contraction of the bladder via the reflex arc (referred to as uninhibited bladder contractions). The urethra and anus are

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composed of internal (smooth muscle) and external (striated muscle) sphincters. Somatic innervation through the pudendal nerve allows voluntary contraction of the external sphincter and pelvic floor musculature that protects against urine loss from sudden increases in abdominal pressure. Voluntary contraction of the external urethral muscle also reflexively inhibits bladder contraction and can interrupt voiding.

Continence depends on voluntary inhibition of reflex bladder contraction and intermittent, as needed, voluntary contraction of the striated pelvic floor muscles to counter increases in intraabdominal pressure. Micturition requires voluntary disinhibition of bladder contractions, which reflexively leads to relaxation of both the internal and external urethral sphincters.

Classification of Urinary Incontinence and Lower Urinary Tract Symptoms

Transient incontinence is defined as new leaking of sudden onset that is generally associated with an acute medical or surgical illness or drug therapy, and it is usually reversible with resolution of the underlying problem. Functional incontinence is another term that has been used for this condition. Causes of transient UI are varied (Table 28.1). Drug side effects contribute greatly to this problem; therefore, a review of prescription and over-the-counter medications is extremely important.

Established UI is usually chronic, requires investigation, and is amenable to treatment in many cases. There are four types of established UI: stress, urgency, mixed incontinence, and overflow (also termed chronic urinary retention and incontinence with a high postvoid residual).[13]

Urgency Urinary Incontinence

Urgency UI occurs more commonly in older adult women and men compared to their younger counterparts and results from unsuppressed bladder contractions (detrusor instability) (Table 28.2). These uninhibited contractions are associated with an irresistible urge to void and usually result in loss of a large volume (>100 ml). Patients with urgency urinary incontinence may also have symptoms of urgency, frequent urination, and nocturia, which is called overactive bladder-wet (OAB-wet) in contrast to those who have OAB symptoms without urgency UI (OAB-dry). By definition, to have OAB, one must have urinary urgency without a urinary tract infection (UTI). Detrusor hyperreflexia is a term describing unsuppressed bladder contractions associated with a neurological disorder.

Table 28.1 Identification of reversible conditions that may cause or contribute to urinary incontinence (UI)

Conditions affecting the lower urinary tract
• Urinary tract infection (symptomatic with frequency, urgency, dysuria)
• Atrophic vaginitis or urethritis
• Prostatectomy
• Stool impaction
• Radiation treatments for cancer: prostate, colon, cervical
Drug side effects that may contribute to UI
• Diuretics: polyuria, frequency, urgency
• Caffeine: aggravation or precipitation of UI
• Anticholinergic agents: urinary retention, overflow incontinence, constipation
• Psychotropic medications: sedation, delirium
• Antidepressants: anticholinergic actions, sedation, diarrhea
• Antipsychotics: anticholinergic actions, sedation, immobility, rigidity, constipation
• Sedatives, hypnotics, central nervous system depressants: sedation, delirium, immobility, muscle relaxation
• Opioid analgesics: urinary retention, constipation, fecal impaction, sedation, delirium
• α-Adrenergic blockers: urethral relaxation
• α-Adrenergic agonists: urinary retention (found in many cold and diet over-the-counter preparations)
• β3-Adrenergic agonists: urinary retention
• Calcium channel blockers: urinary retention, constipation
• Alcohol: polyuria, frequency, urgency, diarrhea, sedation, delirium, immobility
• Nonsteroidal anti-inflammatory medications: constipation
Increased urine production
• Metabolic disorders (hyperglycemia, hypercalcemia)
• Excess fluid intake
• Volume overload
• Venous insufficiency with edema
• Congestive heart failure
Impaired ability or willingness to reach the toilet
• Delirium
• Chronic illness, injury; restraint that interferes with mobility
• Psychological disorders

Modified from Fantl et al.[62]

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Table 28.2 Types of urinary incontinence (UI) and characteristics

Urinary incontinence type	Characteristics
Urgency UI	• Often occurs with a strong urgency sensation and uninhibited detrusor (bladder) contraction • Large-volume leakage
Stress UI	• Hypermobile urethra • Internal sphincter insufficiency • Reduced pelvic floor musculature • May also be secondary to trauma (e.g., obstetrical) or surgery (e.g., prostatectomy) • Small or large amounts of leakage may occur
Overflow UI	• More common in older adults with impaired mobility and functional impairments (e.g., long-term care residents) • Usually involves prostatic enlargement in men and prolapse in women • Worsened with medications with anticholinergic side effects • May worsen with constipation symptoms

Urgency UI is more common in older women, those with diabetes, and those reporting UTIs.

All central and peripheral neurological disorders have a high risk of resultant dysfunction in the urinary tract.[14] Any damage to the structural integrity of the cholinergic inhibitory center of the central nervous system, or the afferent innervation from the lower spinal cord where the reflex arc is located, can cause detrusor hyperreflexia. Processes such as Alzheimer's disease, cerebrovascular atherosclerosis, multiple sclerosis, Parkinson's disease, spinal cord tumors or transection, and cervical spondylosis (among others) may result in UI by this mechanism.[15]

Stress Urinary Incontinence

Stress UI occurs much more often in women than men. Stress UI results from a hypermobile urethra, internal sphincter insufficiency, or reduced support by the pelvic floor musculature in the bladder outlet. Gynecological surgery, weight gain, and decreased effects of estrogen on pelvic tissues, vasculature, and urethral mucosa are possible causes. Multiple childbirths and obstetric injury are notable risk factors for UI[16] in the immediate postpartum period,[17] as well as in women aged 40–64 years, but it is unclear that these remain risk factors for older women. Sphincter weakness may also be the result of urethral inflammation, neurological disease, radiation therapy, or α-blocker drugs. Patients are likely to complain of losing small amounts of urine with coughing, sneezing, straining, lifting, or changing posture. Multiple and vaginal childbirth is an important risk factor for stress UI in younger women but is less important in older women. In older women, stress incontinence is commonly associated with higher body mass index (BMI) and education level.[18]

In men, stress incontinence is nearly always associated with surgical or radiation therapy treatment of prostate cancer, though it may also occur following surgical or procedural interventions for benign prostatic hyperplasia. Men with post-prostatectomy urinary incontinence may have complaints ranging from small amounts of urine lost coincident with coughing, sneezing, laughing, or lifting to nearly constant urinary dribbling.

Overflow Urinary Incontinence (Bladder Outlet Obstruction and Neurogenic Bladder)

Bladder outlet obstruction is more common in men than women, occurring primarily because of cellular benign prostatic hyperplasia (BPH), resulting in benign prostatic enlargement. Less common causes of bladder outlet obstruction include urethral stricture or, in rare cases, prostatic neoplasm. BPH may result in lower urinary tract symptoms (LUTS) such as frequency, urgency, nocturia, straining, hesitancy, weak urinary stream, postvoid dribbling, or incomplete emptying. In women, urethral stricture or severe bladder prolapse may also impede urine flow. In both men and women, partial obstruction may become complete obstruction with the use of anticholinergic or α agonist pharmacological agents, or with severe constipation. Atonic and neurogenic bladder are terms

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describing impaired bladder contractions resulting from low spinal cord lesions, diabetic or alcoholic neuropathy, and/or intake of muscle relaxants, opioids, or antidepressants. Acute urinary retention is painful. The clinical presentation of chronic, progressive bladder outlet obstruction or neurogenic bladder is constant dribbling or leaking associated with an enlarged, palpable bladder. The physical examination finding of a grossly enlarged bladder is very specific, but poorly sensitive for establishing the diagnosis of outlet obstruction. Patients generally strain to urinate, experience hesitancy, and voluntarily and involuntarily voided urine volumes are frequently small.

Urinary Incontinence Evaluation

History

Evaluation should begin with a detailed history of the nature, severity, and burden of incontinence and identifying the most easily remedied contributing causes. UI in older adults compared to younger adults is much more often multifactorial with contributions from concomitant medical conditions, functional impairment, cognitive dysfunction, and contributing medications. Given this, a broader, more comprehensive assessment including attention to comorbidity, medications, frailty, and function is warranted. While UI is a common condition in older adults, UI may also be the sentinel symptom of cancer or neurologic disease. An abrupt onset of UI, gross or microscopic hematuria, pelvic pain, or coincident or worsening new neurologic symptoms should prompt an expedient and thorough search for the underlying cause.

An incontinence diary for urination filled out before the patient's visit is helpful. A history of leakage occurring with specific activities or before/after toileting can also help determine the type of incontinence. Questionnaires may also be used to help determine the predominant symptoms associated with urinary leakage.[19]

Important items of the medical history include data about pelvic surgery, cancer, neurological disease, diabetes mellitus, congestive heart failure, pelvic floor radiation, and previous treatment of UI or FI. While parity and mode of childbirth is extremely important in younger and middle-aged women, it is less clearly a significant risk factor for UI in the older woman. Specific questions should be asked about prescription and over-the-counter medication use, alcohol and tobacco use, and quantity and type of fluid intake, along with food sensitivities (lactose intolerance). Inquiries should be made about the physical layout of the patient's residence and whether impaired mobility limits access to toilet facilities.

The patient should be encouraged to bring a bag containing all prescription and nonprescription drugs to the clinic for review. Medications that may contribute to UI or LUTS can include alpha-adrenergic agonists or antagonists, calcium channel blockers, and diuretics. Sedatives and opioids may alter awareness to the need to void and may precipitate functional UI or nocturnal UI (enuresis).

Men being evaluated for LUTS potentially attributable to BPH should complete a symptom inventory such as the American Urological Symptom Index (equivalent to the International Prostate Symptom Score). Given that women with LUTS may also score high on this inventory,[20] the index is not diagnostic of prostatic enlargement. The AUA-SI gives a grade of severity (mild 1–8 points, moderate 9–17 points, severe 18–35 points).[21]

Physical Examination and Diagnostic Testing

A careful, directed physical exam should be performed.[22] The physical examination should focus on the abdomen and urogenital area, perineal skin integrity, mobility, and the central and peripheral nervous systems. The abdominal examination is insensitive for a high postvoid residual (PVR) or chronic urinary retention, but gross bladder distention (for example, ≥500 ml) can usually be detected. In acute urinary retention, the distended bladder is a firm, midline mass that originates from the pelvis and is dull, and perhaps uncomfortable, to percussion. The rectal examination may reveal fecal impaction, a pelvic mass, external hemorrhoids, enlarged prostate gland, or a prostate nodule. It is very important to assess perianal sensation and the patient's ability to contract and relax the anal sphincter voluntarily. An abnormal clinical sign can suggest serious lumbosacral disease, possibly requiring emergency treatment. Men should be examined for phimosis and balanitis. Assessing for penile retraction is useful for informing the selection of continence draining products, if needed. In women, a pelvic examination is indicated to assess urethral, uterine, or bladder prolapse and to evaluate the patient for any pelvic mass. A gray, dry vaginal mucosa is suggestive of atrophic vaginitis.

The most important diagnostic distinction is between overflow UI and the other types of UI. Most studies show a poor correlation between the underlying cause and the patient's symptoms. UI that results from several multiple

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causes (mixed UI with functional incontinence) means drawing on recommendations across multiple algorithms.

Diagnostic Tests for Incontinence

Selected tests are recommended for the evaluation of patients with urinary leakage. On initial evaluation, a urinalysis and/or urine culture, if indicated, should be done. Properly collected clean catch urine is adequate for culture even for nursing home residents,[23] although some persons will require an in-and-out catheterization to obtain an appropriate specimen. Further evaluation is indicated for older patients with transient gross hematuria or asymptomatic microscopic hematuria as this may signal an occult genitourinary malignancy.[24]

Tests for blood urea nitrogen (BUN), creatinine, glucose, and calcium are recommended if compromised renal function or polyuria is suspected in patients not taking diuretics.[22] The PVR urine volume may be measured in patients with symptoms of UI, particularly those with risk factors for urinary retention such as BPH, urethral strictures from past surgeries, meatal stenosis, phimosis, prostate cancer, hematuria-related clot formation, prostatitis, balanitis, stones, fecal impactions, and medication side effects.[25] The PVR can be measured less invasively by ultrasonography, but can also be checked by in-and-out catheterization.[26,27] A bladder ultrasound scan for PVR can be obtained 5–10 minutes after the patient has voided. The portable ultrasound scan has been shown to be highly reliable, especially at low and very high bladder volumes, offering a clinically relevant result.[26,27] Catheterization can be done by inserting, in sterile fashion, a No. 14 French straight catheter into the bladder. Caution is indicated for patients with outflow obstruction, as a single catheterization may cause infection. Although the definition of a high PVR is controversial, a volume of 200 ml or more suggests either outlet obstruction or neurogenic bladder and is an indication for further urological evaluation.[25] Many clinical trials do not initiate pharmacological treatment of urgency UI or OAB if the PVR is ≥150 mL.

Clinical tests for stress UI may be useful. A cough stress test can be performed with the patient having a comfortably full (200–300 mL) bladder where the patient is instructed to cough vigorously once (followed by three additional coughs, if negative). Cough stress testing can be done with the female patient in the lithotomy position while the examiner is positioned to observe any leakage, or men or women can perform this test while standing. Sensitivity is highest when the patient is standing. Cough stress testing is insensitive if the patient cannot cooperate, is inhibited, or the bladder volume is low.[28]

Formal Urodynamic Testing

After the basic evaluation, treatment for the presumed type of incontinence should be initiated, unless there is a need for further evaluation. Further evaluation may be indicated for the following: failure of initial treatment, a history of surgery or radiotherapy, neurological conditions[14] where noninvasive testing has proved insufficient, marked prolapse on physical examination, PVR greater than 200 ml, inability to pass a catheter, or in patients considering more invasive treatment options who desire further evaluation.[29] Common urodynamic tests that provide more detailed diagnostic information include urine flowmetry, voiding cystourethrography, multichannel cystometrogram, pressure-flow study, urethral pressure profile measurement, and sphincter electromyography.[29]

Treatments for Urinary Incontinence

An accurate diagnosis of UI is essential for appropriate treatment. Any cause of transient incontinence identified during evaluation should be addressed specifically. Behavioral, pharmacological, and surgical therapies are all effective in older people (Table 28.3). It is generally advisable to begin a treatment regimen with the least risk and burden to the patient and caregiver. In all types of UI, except those characterized by urinary retention or poor bladder contractility, behavioral techniques should be considered as first-line therapy unless the patient has a specific preference for another type of therapy.[30] In cases of obesity, weight loss can be of benefit in improving symptoms associated with UI.[31]

Management of UI in long-term care settings differs from management in the ambulatory care setting for two principal reasons. First, comorbidities such as dementia and mobility limitations are more frequent and more severe in the long-term care setting, and these complicate management. Second, in the outpatient setting, behavioral interventions can be implemented by the patients themselves or by highly motivated caregivers who are usually family members. In long-term care, these interventions are implemented by nursing assistants whose capacity may be compromised by high patient-to-staff ratios. For these reasons, treatment of UI in the long-term care setting is discussed separately.

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Table 28.3 Therapeutic modalities in urinary incontinence

Type of incontinence	Recommended therapies
Urgency urinary incontinence	Diet and fluid management
	Behavioral therapy: Timed/scheduled voiding
	Pelvic muscle exercises with or without biofeedback
	Drug therapy:
	Anticholinergic agents
	Oxybutynin (IR 2.5–5 mg qd to tid; ER 5–30 mg qd; 3.8-mg TD patch changed 2×/wk)
	Tolterodine (IR 1–2 mg bid; 2–4 mg qd ER)
	Fesoterodine (4–8 mg qd)
	Trospium (20 mg qd to bid)
	Solifenacin (5–10 mg qd)
	Darifenacin (7.5–15 mg qd)
	Beta-3 agonist agent
	Mirabegron (25–50 mg qd)
Stress urinary incontinence	Pelvic muscle exercises with or without biofeedback
	Behavioral therapy: Timed voiding
	Surgery: Referral for consultation for minimally invasive surgeries
Neurogenic urinary incontinence	**No drug therapy**
	Management: Intermittent catheterization
Functional urinary incontinence	Correct underlying cause

Modified from Fantl et al.[62]
Treatments recommended for management of urinary incontinence are adapted from the Agency for Health Care Policy and Research (AHCPR) 1996 clinical practice guideline Urinary Incontinence in Adults with updated drug recommendations.

Ambulatory Care Treatment of Urinary Incontinence

Successful treatment of functional incontinence relies on the recognition that physical, pharmacological, psychological, and environmental problems coexist that can cause or worsen UI. Providing the patient with assistive devices such as a urinal or bedside commode; reassessing drug indications, doses, and schedules; treating depression; providing supportive prompts and cues and toileting assistance; and eliminating barriers in the path to the toilet may improve incontinence dramatically. A visit to the patient's home by a health-care provider and discussion with family members can be very helpful in identifying barriers to continence and implementing simple changes that improve continence. Treatments that are specific to each type of UI are discussed below.

Urgency Urinary Incontinence

The treatment of urge incontinence entails designing interventions to decrease or block uninhibited bladder contractions, improve bladder capacity, and prolong the time from symptoms of urgency to voiding. Effective strategies include: (1) dietary and lifestyle changes, (2) behavioral training (timed voiding), and (3) drugs to reduce bladder contractions.

Dietary Management

Self-monitoring techniques such as reducing caffeine[32] and alcohol intake, management of constipation, and drinking an adequate intake of fluids throughout the day have been shown to be helpful in reducing UI in women.[33]

Behavioral Treatment

Behavioral treatments are efficacious for treatment of urgency UI in both men and women. Approaches do not eliminate urgency but allow the individual to maintain bladder control with occurrences. Behavioral strategies are effective and can be used without risk of adverse effects. Bladder control can be enhanced through patient behavior changes through the use of specific techniques such as pelvic floor muscle training used along with urge suppression strategies. Urge suppression techniques are effective in improving bladder symptoms through suppression of detrusor contractions. Patients learn how to respond adaptively to urgency by voluntarily inhibiting detrusor contraction, and thus postpone urination and urine loss.[34]

One behavioral treatment for UI or OAB is timed voiding. Timed voiding may be combined with pelvic floor muscle exercises to strengthen the pelvic floor muscles, and patients may be taught to contract pelvic floor muscles when they experience an urge to void to reflexively inhibit bladder contractions.[35] The rationale for this treatment is that patients with urge incontinence may void too frequently and may gradually develop an intolerance for bladder-filling. The treatment is to instruct the patient to void at fixed, short intervals such as every 30

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minutes and to increase gradually the duration of the intervals to 2–3 hours. Patients are encouraged to resist the urge to void between these intervals. A typical regimen for pelvic muscle exercises is described later under treatments for stress UI.

Pharmacological Therapy

Anticholinergic agents have long been the mainstay of pharmacologic therapy for patients with urgency and mixed incontinence, along with OAB. More recently, beta-3 adrenergic agonists have been approved for this indication as well. While there are six anticholinergic medications approved for the treatment of urgency UI (also OAB) in the United States, there are only two beta-3 agonists.

Common side effects of anticholinergic medications may include dry mouth, dry eyes, constipation, and urinary retention.[36] In general, these anticholinergic medications are roughly equivalent in their effectiveness.[22] Systematic reviews have found that extended-release (ER) formulations of the anticholinergic agents have fewer dry-mouth and constipation side effects than the immediate release formulations for the treatment of UI and OAB.[22] More attention to the use of anticholinergic medications has come lately with a growing literature of epidemiological studies showing the association of medication usage with the development of cognitive impairment.[37] It is possible that these effects are cumulative and irreversible, so caution is warranted. Oxybutynin, an older medication in this category with reported impact on cognition, was the most widely used bladder anticholinergic agent among US older adults receiving Medicare Part D benefits.[38] It has both anticholinergic and smooth muscle relaxant properties. Studies with the immediate release (IR) formulation have shown a 15–58% greater reduction in urge UI compared with placebo. The ER and transdermal (TD) formulations have similar efficacy with fewer side effects. Up to 50% of patients in clinical trials of the IR medication had side effects such as dry mouth and constipation that limited therapy. The ER and TD formulations are associated with a lower rate of dry mouth. Given the availability of generic ER oral formulations, this is more commonly used than IR formulations. The initial dosage for the ER formulation is 5 mg daily. While it can be titrated up to 30 mg daily, the highest dosage is more commonly 20 mg. The initial dosage for IR demonstrated to be effective in clinical trials is 2.5–5 mg three times a day, although some elderly patients benefit from only 2.5 mg daily. The 3.8-mg TD patch, used to reduce dry mouth, is applied twice weekly and is now available over the counter (while marketed over the counter for use in women, men can use it as well). Narrow-angle glaucoma and urinary retention are contraindications for treatment with oxybutynin.

Solifenacin is the second most widely used bladder anticholinergic, and darifenacin is the least often used.[38] Both are more selective muscarinic receptor (M3) antagonists. Solifenacin is not currently available as a generic formulation (as of 2021), but darifenacin is. These drugs were also approved for treatment of OAB symptoms. Initial dosage of solifenacin is 5 mg, which may be titrated to 10 mg, and the dose of darifenacin is 7.5–15 mg daily. Studies have not shown these drugs to be associated with a clear increase in efficacy or decrease in side effects compared with other anticholinergic drugs such as tolterodine.

Tolterodine, a muscarinic receptor antagonist, was found to be as effective as oxybutynin in double-blind studies, and it had a lower incidence and decreased severity of the side effect of dry mouth. As with oxybutynin, tolterodine should not be used in patients with narrow-angle glaucoma or urinary retention. Both extended-release and immediately released formulations are available as generic.

Trospium is a nonselective muscarinic receptor antagonist metabolized in the kidneys for treatment of the OAB symptom complex of urgency, frequency, and incontinence. The dose for older persons and those with renal impairment is 20 mg daily. Trospium must be taken on an empty stomach.

Fesoterodine, an isomer of tolterodine, is approved for the treatment of OAB and UI. As of 2021, there is not currently a generic version of this medication. Data suggests that even frail, older adults may have some benefit from this medication from a randomized, controlled trial done specifically in this population.[39]

Mirabegron, a nonselective beta-3 adrenoreceptor agonist (beta-3-AR), was approved by the Food and Drug Administration (FDA) in 2012 as the first agent in this category. It is indicated for pharmaceutical treatment for OAB and UI in men and women. The starting dosage is 25 mg and can be titrated to 50 mg; the recommended highest dosage for patients with liver and renal impairment is 25 mg daily. Mirabegron is not indicated for patients with severe liver impairment or uncontrolled hypertension. Mirabegron is a moderate inhibitor of CYP2D6, and monitoring should be done if the patient is taking another medication (e.g., metoprolol, desipramine) with a similar metabolic pathway. Mirabegron has been studied as an add-on therapy to solifenacin therapy and showed statistical improvement in urgency UI and

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OAB (urinary frequency).[40] The most common side effects were hypertension (mean increase of blood pressure 0.5 to 1 mmHg), nasopharyngitis, UTI, and headache.

There is an additional, recently approved selective beta-3 agonist (vibegron) that was approved for use at the 75 mg per day dose. There is still limited clinical prescribing experience with vibegbron. Neither mirabegron nor vibegron have supplemental warnings in the package insert with use in the geriatric population.

For all older patients taking anticholinergic drugs, the lowest dose possible that is efficacious should be prescribed. PVRs should be monitored on a regular schedule to identify urinary retention that causes worsening of incontinence. In these cases, the drug dose should be reduced, and in severe cases, discontinued.

Sacral Nerve Stimulation with Percutaneous Tibial Nerve Stimulation (PTNS)

PTNS has emerged as a viable, minimally invasive, low-risk, and relatively low-cost option for management of OAB and UI. This treatment has comparable efficacy to bladder drug treatments with fewer side effects. PTNS is an office-based procedure to indirectly stimulate the sacral nerves through the posterior tibial nerve and appears to facilitate neuromodulation via the sacral S3 nerves. A small, acupuncture-sized needle is inserted into the medial aspect of the lower extremity and attached to an electrical stimulation device. Often, 12 weeks of treatment are needed to see improvement in symptoms.

The posterior tibial nerve contains mixed sensory motor nerve fibers that originate from L4 through S3 nerve roots involving nerves that provide sensory and motor control of the pelvic floor and viscera. The site of stimulation in PTNS, posterior and superior to the medial malleolus (inner ankle), is an acupuncture point ("sanyinjiao" or "spleen-6") in traditional Chinese acupuncture. PTNS uses a surgically implanted neuromodulation device, Interstim™, that has been approved in Europe since 1994. This device has shown improvement in symptoms compared to conservative measures.[41,42]

New approaches such as neural sacral modulation and installation of botulinum toxin have been developed for urgency incontinence that remains unresponsive to medical and behavioral treatment. Studies including older participants have shown that improvement rates with sacral neuromodulation range from 60% to 90%.[43] If failure does occur, this treatment is reversible and the device can be removed.

Instillation of Botulinum Toxin

Injection of botulinum toxin into the detrusor muscle is also a treatment option for women with urgency or mixed incontinence who are unable to tolerate pharmacotherapy or unresponsive to conservative therapy. However, caution must be taken to avoid use of this therapy in patients with a history of urinary retention or recurrent UTIs. While botulinum has been studied in men, including older men, there have been a few reports of some improvement in overactive bladder symptoms, but issues of limited compliance rates for these techniques in older populations require further study.[44,45]

Stress Incontinence (Sphincter Insufficiency/ Pelvic Floor Muscle Weakness)

Behavioral Treatment

The rationale behind behavioral treatments for stress incontinence is that this type of incontinence results from transient increases of bladder pressure above urethral pressure, which can be corrected by strengthening the external urethral sphincter muscle so that urethral sphincter pressure is higher or by elevating the bladder neck. The simplest and least costly behavioral technique is referred to as pelvic floor muscle training (PFMT), which may be known to patients as Kegel exercises.[46] These are often taught verbally by instructing patients to squeeze the pelvic floor muscles as if they are holding back urine or holding back a bowel movement but to keep their abdominal wall muscles relaxed (i.e., to avoid inappropriate increases in intraabdominal pressure that may increase the likelihood of stress incontinence). These instructions may include the suggestion that patients place a hand on the abdomen to detect abdominal wall contraction or that they continue to breathe while squeezing pelvic floor muscles. Patients are instructed to squeeze pelvic floor muscles and hold a maximum squeeze for 3–10 seconds, with 10-second rest periods in between squeezes. They are directed to perform 15 squeezes three times a day for at least 8–12 weeks.[47,48]

The literature on PFMT and exercise has demonstrated that it is effective for reducing stress, urge, and mixed urinary incontinence in most outpatients who cooperate with training. Cochrane Database systematic reviews concluded that there is Grade A evidence for PFMT and that it should be offered as first-line treatment to women with stress, urge, or mixed incontinence[48,49] (or even for men with postprostatectomy UI).[47]

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While less research has been conducted with men, a growing number of studies have shown the utility of this approach in men. However, a recent randomized trial of an exercise-based behavioral training program for men with persistent postprostatectomy incontinence demonstrated a 51% reduction in incontinent episodes, significantly greater than the 24% reduction in the control group.[34,47,50]

The initial instruction in pelvic floor muscle exercises should be done during a digital rectal or pelvic examination: With the examining finger in the anal canal or vagina, the therapist instructs the patient to squeeze the pelvic floor muscles and provides verbal feedback on whether she is squeezing correctly. The examiner can also place a hand on the rectus abdominis muscles to detect inappropriate contractions. Biofeedback may be useful to teach pelvic floor muscle exercises in the treatment of stress UI,[51] particularly with patients who experience difficulty isolating the pelvic floor muscle or are unable to correctly contract the pelvic floor, or who lack motivation. The use of biofeedback is also influenced by availability of a specialist, access, and the patient's preference. Although this may be helpful to many patients, studies have generally shown no significant difference in efficacy between Kegel exercise training alone and with use of biofeedback.

An alternative treatment for stress UI in women, particularly where there is also uterine prolapse, is to be fitted for use of a vaginal pessary. This can help elevate the bladder neck and may be especially useful for women who do not desire surgery or are poor surgical candidates.[52] Pessaries come in various sizes and shapes and should be selected based on effectiveness and patient preference/comfort following a therapeutic trial. Referral to a specialist experienced in the use of pessaries should be considered.

Drug Treatment

Estrogen has direct effects on urethral mucosa and periurethral tissues and increases the number and responsiveness of α-receptors in women. Vaginally applied estrogen cream, ring, or tablets may improve stress and mixed UI. The duration of topical estrogen application has not yet been established. Several large studies have shown that oral estrogen or estrogen plus progesterone worsens stress, urge, or mixed UI.[53,54] Based on this data, oral estrogen is not recommended as a treatment for UI.

Surgical Management

When conservative therapy has failed, surgery may be appropriate. In women with urethral hypermobility of the bladder neck and stress UI, minimally invasive surgeries with synthetic slings[55,56] are outpatient procedures with a low complication rate.

Overflow Incontinence (Neurogenic Bladder or Bladder Outlet Obstruction)

Overflow urinary incontinence is of two primary types: neurogenic bladder (detrusor underactivity) and bladder outlet obstruction.

Neurogenic bladder may occur either in older men or women and is a potentially life-threatening condition because it increases the risk of reflux of bacteria to the kidneys. In patients with severe neurological deficits, intermittent clean catheterization every 2–4 hours by the patient or caregiver is often the best management. If this is not possible or practical, an indwelling catheter may be necessary. The use of chronic indwelling catheters is generally not encouraged because of the frequency of complications, including urolithiasis, symptomatic bacteriuria, periurethral abscess, and acute pyelonephritis. Appropriate management of an indwelling catheter depends on proper insertion using sterile technique and maintaining a closed sterile system. Urethral cleansing, routine bladder irrigation, and prophylactic antibiotic therapy should be avoided,[57] as these procedures do not prevent bladder colonization and are likely to result in the selection of resistant organisms.

For patients with mild overflow incontinence, a prompted voiding schedule (reminding the patient to void every 2–3 hours) may be beneficial. Overflow incontinence resulting from a hypocontractile bladder is generally poorly responsive to behavioral or pharmacological therapy. Surgery is not indicated.

Severe bladder outlet obstruction is much more common in men but can occur in older women. Women may present with bladder outlet obstruction secondary to pelvic organ prolapse, specifically anterior compartment prolapse, or cystocele. This may require surgical repair.

In older adults, the bladder can be shown to be simultaneously overactive and underactive. In this situation, the bladder contracts inappropriately during bladder-filling but may not contract sufficiently during bladder emptying. This bladder dysfunction is termed detrusor (bladder) overactivity with detrusor underactivity (DO-DU) and used to be called detrusor hyperactivity with impaired contractility (DHIC).

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Treatment of Male Lower Urinary Symptoms Suggestive of Benign Prostatic Hyperplasia

Use of a questionnaire, such as the American Urological Association Symptom Inventory,[21] is useful in deciding on initial treatment for men with BPH. Men with milder symptoms can be managed by watchful waiting. For men with moderate to severe symptoms, medications are often the treatment of choice. Current medication approaches for BPH include α-antagonists (nonselective alpha-blockers such as doxazosin and terazosin, or selective agents like tamsulosin, silodosin, and alfuzosin) that reduce the dynamic component of prostatic obstruction; 5-α reductase inhibitors (such as finasteride and dutasteride), which reduce the size of the prostate gland; and more recent use of phosphodiesterase inhibitors (sildenafil, tadalafil, vardenafil), which may regulate smooth muscle tone in the prostate gland. These can be used in combination to treat moderate to severe symptoms. There is a growing use of minimally invasive surgical techniques that have proved efficacious for BPH other than transurethral resection of the prostate (TURP). In these patients, full evaluation, including urodynamic testing prior to surgery, may be indicated to rule out coexisting causes of incontinence.

Managing Urinary Incontinence with Single-Use Absorbent and Incontinence Products

Single-use absorbent pads, undergarments, bed sheets and chair covers, and other continence products are designed for management rather than treatment of UI with or without concomitant fecal incontinence (Table 28.4). Although evaluation and treatment are recommended, many patients will either depend on continence materials exclusively or will use absorbent products for security in circumstances where they are fearful of incontinence.

Single-use absorbent pads typically consist of a superabsorbent layer surrounded by an outer barrier and an inner membrane that conducts moisture away from the skin.[58] They are supplied both as small pads to be worn in undergarments by patients who typically experience small-volume leakage or as all-in-ones (wraparound pads, adult briefs), worn in place of undergarments by patients who at least occasionally have large-volume leakage of urine. For men with light incontinence, male pads may be constructed as pouches or in the shape of a shield or leaf that surrounds the penis. Pads are also available specifically for accidental bowel leakage.

Table 28.4 Single-use absorbent products for incontinence

Type	Description
Pads	Waterproof-backed absorbent products held in place using separate, close-fitting underwear
Unbacked pads	Absorbent products without a waterproof backing
Male pads	Waterproof-backed absorbent products specifically designed for men that cover the penis and scrotum, and are held in place using separate, close-fitting underwear
Male pouches	Waterproof-backed absorbent products specifically for men, designed as a pocket that the penis – and sometimes the scrotum, too – is placed within
Pull-on pads	Single products with an absorbent core, waterproof backing, without fasteners, that resemble regular underwear
All-in-ones (wraparound pads)	One-piece products with an absorbent core secured using adjustable adhesive tabs or a hook-and-loop fastening system at the sides
Belted pads	One-piece products with an absorbent core, waterproof backing secured using an adjustable belt with adhesive tabs or a hook-and-loop fastening system

Adapted from the Fader et al.[58] International Continence Society (ICS) Standardization Steering Committee (https://onlinelibrary.wiley.com/doi/10.1002/nau.24488)

For men, sheaths made of latex or plastic, as a condom, that can be attached around the penis and that connect via tubing to a collection bag are more commonly used than absorbent pads. Sheaths are associated with a higher incidence of UTIs than pads, although this risk can be minimized by ensuring that the connection between the sheath and collection device does not kink or become obstructed. Other risks associated with sheaths are allergic reactions to latex, irritation, and compression from sheath binding straps.

Treatment in Long-Term Care Facilities

Prevalence estimates of UI from Minimum Data Set (MDS) data in both men and women aged 65 years and older residing in long-term care facilities range from

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30–77%, with up to 60% also having FI.[1] The prevalence of UI reported in a population-based study involving 95,911 older nursing home residents from eight southeastern states was 65% on admission, suggesting that the majority of nursing home residents are incontinent.[59] It is possible to decrease the frequency and severity of UI in at least half of incontinent long-term care patients through prompted voiding, but this approach may be challenging in long-term care facilities, because the financial and staff resources needed to implement this program are seen as prohibitive. Prompted voiding may also have some benefit for adults with constipation and FI in long-term care settings.[60]

Behavioral Treatment for Urinary Incontinence

Prompted voiding is the behavioral program that has been most thoroughly evaluated.[60,61] A staff member approaches incontinent residents every 2 hours (or at intervals individualized to each patient) and asks them if they would like assistance to get to the toilet. Assistance is provided if needed, and staff are instructed to encourage patients to void/defecate and to praise them for success at remaining continent between prompts. Increasing fluid intake and exercise may also be included. In a large number of studies, this approach reduced the severity of UI (the proportion of times checked that the patient was found to be wet) by approximately 50% overall, although relatively few patients become fully continent. When prompts were no longer provided, the rate of UI quickly returned to its former level. Moreover, approximately half of nursing home residents do not benefit because of the severity of cognitive impairment or other functional limitations. A trial period may be indicated to focus prompted voiding techniques on those who respond.

Management studies have shown that the average time required to assist a patient to the toilet to urinate greatly exceeds the time required to change absorbent undergarments or bed pads and linens.

Summary

UI remains a common, underreported, and vexing problem in older patients that can lead to social isolation. New therapeutic options using behavioral, pharmacological, and surgical approaches can lead to symptomatic improvements or cure for this important clinical problem and increased quality of life for the older patient.

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Chapter

29 Fecal Incontinence

Alayne D. Markland, Jan Busby-Whitehead, and William E. Whitehead

Fecal incontinence (FI), the involuntary loss of solid or liquid feces, has a lower prevalence than urinary incontinence (UI), but is equally prevalent in older men and women. In a systematic review of 38 studies across several countries, the median prevalence among older men was 8.1% and among older women was 8.9% (7.7% across all ages, range 2–21%).[1] FI may be more stigmatizing than UI for the individual because of the inability to contain the odor, the lack of absorbent products to contain large-volume leakage, and the incontinence-associated dermatitis that results because of the fecal material in close contact with skin. Frequent or severe FI can have a devastating impact on people's lives, leading to social withdrawal and depression and contributing to the decision to go into a nursing home.[2]

In addition to older age, other risk factors for FI include diarrhea, fecal urgency, UI, diabetes mellitus, and hormone therapy (current and prior therapy) in postmenopausal women.[1,3–5] Many specific age-related changes, such as functional impairments in mobility, dexterity, cognition, and reduction in bowel capacity and sensation, contribute to FI.[6] Other strong risk factors for FI in men, other than age, are prior anorectal surgery and radiation exposure.[7]

An estimated 60% of people with FI who are identified through surveys have not reported FI to a health-care provider, perhaps because they are embarrassed or believe nothing can be done to help.[8] Health-care providers, therefore, should specifically ask about bowel symptoms, including FI and known risk factors. From a patient perspective, the term "accidental bowel leakage" is preferred when discussing FI.[9] Like UI, FI is not a normal part of aging despite physiologic changes in the aging gut and anorectal anatomy.

Physiological Mechanism for Defecation

The processes involved in healthy defecation are complex (Figure 29.1). Defecation begins with the entry of stool in the rectal vault. Progressive rectal distention leads to reflex relaxation of the internal anal sphincter. The urge to defecation increases as fecal material continues to enter the rectal vault from the sigmoid colon. When defecation is desired, the anorectal angle is straightened under voluntary pelvic muscle control, and increased abdominal pressure results in pelvic floor descent, contraction of the rectum occurs, and inhibition of the external anal sphincter results in evacuation of the rectal contents.

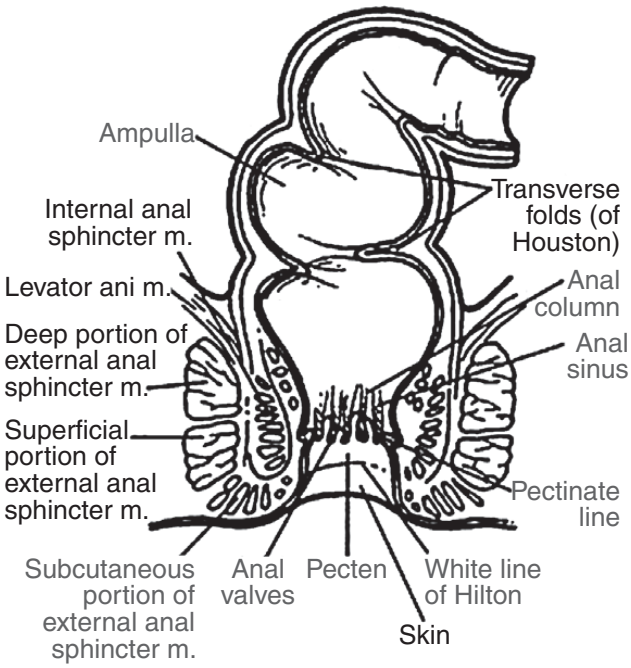

Figure 29.1 Recto-anal anatomy.

Innervation of the defecation process is under cholinergic, adrenergic, and somatic control. Lower rectal contraction is mediated by cholinergic (parasympathetic) activity, whereas relaxation of the internal and external sphincters is mediated by adrenergic (sympathetic) pathways in the pudendal nerve via a spinal reflex mediated by the S2–S4 sacral nerve roots. Central nervous system

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control of bowel sphincter function is mostly inhibitory; that is, reflex bowel contractions are actively inhibited until a socially appropriate time and place to defecate is found. This inhibition occurs through neural linkages from the sensorimotor cortex of the frontal lobes to the brainstem, cerebellum, thalamus, and spinal cord. Defecation normally involves a conscious disinhibition of bowel sphincter muscle relaxation. Thus, stroke and other neurological processes can result in FI because of loss of central cortical inhibition.

The anal sphincter complex is composed of internal (smooth muscle) and external (striated muscle) sphincters. Somatic innervation through the pudendal nerve allows voluntary contraction of the external sphincter that protects against bowel loss from sudden increases in abdominal pressure. Voluntary contraction of the external anal muscles can also inhibit bowel wall contraction and delay defecation. The puborectalis muscle is also striated muscle and is under voluntary control. The puborectalis is innervated through branches of S3 and S4 nerve roots. Despite having independent innervation, the external anal sphincter and the puborectalis function as a unit in maintaining continence. The external anal sphincter can be contracted to increase rectal pressure further. The puborectalis forms a sling around the anorectal and separates the anal canal from the rectum by 80 to 110 degrees at rest. Voluntary contraction of the puborectalis may decrease the angle further to improve continence.

Loss of continence can result from dysfunction of the anal sphincters, abnormal rectal compliance, decreased rectal sensation, altered stool volume and consistency, cognitive function, or a combination of these mechanisms. Similar to UI, FI is multifactorial. With normal aging, mild impairments can occur in several of the defecation mechanisms. Mild impairments in any one mechanism will not cause FI. More commonly, moderate to severe impairments in one or two mechanisms lead to FI because of the inability of other defecation mechanisms to compensate.

Classification for Fecal Incontinence

Identification of the reversible types of fecal incontinence is an important first step in classification and treatment. Reversible or transient incontinence is defined as new leaking of sudden onset that is generally associated with an acute change in one of the defecation mechanisms, and usually improves with resolution of the underlying problem. "Functional incontinence" is another term that encompasses a decrease in physical ability or mobility that may or may not be transient. Causes of transient or reversible FI are diverse (Table 29.1). Drug side effects contribute greatly to this problem, usually causing alteration of stool consistency; therefore, a review of prescription and over-the-counter medications is extremely important.

Table 29.1 Identification of reversible conditions that may cause or contribute to fecal incontinence (FI)

Conditions affecting the anorectal area
Bowel infection (symptomatic with frequency, urgency, diarrhea)
Stool impaction
Radiation treatments for cancer – prostate, colon, cervical
Hemorrhoidectomy
Drug side effects that may contribute to FI
Caffeine: aggravation or precipitation FI (bowel stimulation)
Anticholinergic agents: constipation
Psychotropic medications: decreased rectal sensation
Antidepressants: anticholinergic actions, sedation, diarrhea
Antipsychotics: anticholinergic actions include sedation, immobility, rigidity, constipation
Sedatives, hypnotics, CNS depressants: sedation, delirium, immobility, muscle relaxation
Opioid analgesics: constipation, fecal impaction, sedation, delirium
Calcium channel blockers: constipation
Alcohol: diarrhea, sedation, delirium, immobility
Digoxin: diarrhea if serum levels are elevated
Metformin: diarrhea
Nonsteroidal anti-inflammatory medications: constipation
Colchicine: diarrhea
Increased stool production and loose stool
Malabsorption syndromes, including lactose intolerance and bile acid malabsorption
Irritable bowel syndrome
Inflammatory bowel diseases
Impaired ability to reach the toilet
Delirium
Chronic illness, injury; restraint that interferes with mobility
Psychological disorders

CNS – central nervous system

Source: Modified from Fantl JA, Newman DK, Colling J, et al. Urinary incontinence in adults: Acute and chronic management. Clinical practice guideline 2 (1996 update). AHCPR 96–0682. 3–1–1996. Rockville, MD: US Department of Health and Human Services, Public Health Service, Agency for Health Care Policy and Research, 1996.

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Table 29.2 Types of fecal incontinence (FI) and characteristics

FI type	Characteristics
Urgency FI	Often occurs with a strong urgency sensation to have a bowel movement Liquid stool or diarrhea often associated with the inability to hold stool in the rectal vault
Passive FI	Accidental bowel leakage without the sensation of the need to defecate
	May not be able to differentiate between passing gas and having a bowel movement
	May also involve seepage after a bowel movement Staining may be a type of passive accidental bowel leakage
Overflow FI	More common in older adults with impaired mobility and functional impairments (e.g., long-term care residents)
	May also involve seepage or smaller amounts of stool loss around an impaction
	Associated with symptoms of constipation
	May need to treat constipation symptoms to improve FI

Urge, Passive, and Mixed Fecal Incontinence

Types of FI are similar to UI and include urge, passive, mixed (urge and passive), and overflow. Seepage and staining can be types of passive FI. Seepage can also occur with fecal impaction and severe constipation. Accidental bowel leakage is the preferred terminology to use when discussing FI with patients[9] (see Table 29.2).

Fecal Incontinence and Fecal Impaction

FI can result from constipation with stool impaction and may be more common in certain frail, older populations. In a recent study, 81% of residents in long-term care settings had symptoms of constipation and FI.[10] However, the true prevalence of impaction and FI in nursing home residents and home-care settings has not been clearly identified. Because constipation with FI is difficult to diagnose, treatments normally target constipation.

Fecal Incontinence Evaluation

History

Evaluation should begin with a detailed history of the nature, severity, and burden of FI, and identify the most easily remedied contributing causes. A bowel diary for defecation patterns, including stool consistency, filled out before the patient's visit is helpful. A bowel diary can be done on paper or through phone and mobile health applications.[11] Providing a history of leakage that occurs with specific activities or before/after toileting can also help determine the type of FI. Questionnaires may also help to determine the predominant symptoms, severity, volume/stool consistency, and impact on quality of life. Caregivers can also provide important information about FI symptoms.

Important items of the medical history include data about childbirth, pelvic surgery, cancer, neurological disease, diabetes mellitus, congestive heart failure, pelvic floor radiation, prior hemorrhoid surgery, and previous treatment of FI or other anorectal disorders. Specific questions should be asked about prescription and over-the-counter medication use, alcohol use, and fluid intake, along with food sensitivities (e.g., lactose intolerance and gluten sensitivity). Inquiries should be made about the physical layout of the patient's residence and whether impaired mobility limits access to toilet facilities. The patient should bring a bag containing all prescription and nonprescription drugs, including supplements, to the clinic so that medications that may contribute can be identified.

Physical Examination and Diagnostic Testing

The physical examination should focus on the abdomen, urogenital area, and anorectal area, including assessment of the central and peripheral nervous systems. The abdominal examination may be important for recognition of prior surgeries (scarring), areas of tenderness, or masses. The digital rectal examination may reveal fecal impaction, rectal prolapse, external hemorrhoids, or an enlarged prostate gland. The digital rectal examination is required to assess resting anal tone and squeeze tone with the patient voluntarily squeezing the puborectalis and the external anal sphincter. It is very

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important to assess perianal sensation for the anocutaneous reflex (anal wink sign) and the patient's ability to contract and relax the anal sphincter voluntarily. A cotton swab can be used to evaluate the anocutaneous reflex by gently stroking the perianal skin toward the anus and observing reflexive contraction of the external anal sphincter (should be done bilaterally). An abnormal sensation suggests serious lumbosacral disease, or more nerve root involvement. In women, a pelvic examination is indicated to assess urethral, uterine, bladder, or recto-vaginal prolapse and to evaluate the patient for any pelvic mass. A gray, dry vaginal mucosa is suggestive of atrophic vaginitis.

Diagnostic Tests for Fecal Incontinence

Selected tests are recommended for the evaluation of patients with FI. Symptoms, severity, chronicity, and response to prior treatments can indicate if further diagnostic testing is needed.

Laboratory tests for causes of loose stool may include stool studies for the evaluation for infectious causes of diarrhea and malabsorption syndromes (including *Clostridium difficile* evaluation, fat malabsorption, lactose intolerance, and the presence of leukocytes). Other testing could involve serum tests to evaluate for celiac disease if chronic diarrhea is suspected.

Specialized Tests for Fecal Incontinence

Specialized testing for FI with endoscopy may be indicated if other warning signs are present, such as hematochezia, a family history of colon cancer/inflammatory bowel disease, anemia, positive fecal occult blood test, unexplained weight loss ≥10 pounds, constipation that is refractory to treatment, and new-onset constipation or diarrhea without evidence of potential primary cause. If these symptoms are present, colonoscopy may be needed to evaluate for colonic lesions, mass or obstruction, volvulus, megacolon, strictures, or mucosal biopsy. Abdominal radiographs may indicate significant stool retention in the colon and suggest the diagnosis of megacolon, a volvulus, or a mass lesion. Abdominal ultrasound could be ordered if acute or chronic cholecystitis symptoms are suspected as a potential cause for the change in bowel symptoms.

Evaluation may be needed to identify anatomic abnormalities or factors such as external or internal anal sphincter tears or scarring, rectal sphincter muscle weakness, and impaired rectal sensation.[13] Three types of specialized testing may help with the diagnosis and etiology of FI symptoms.[14] Although findings from these tests may identify specific treatments, few have been evaluated for cost-effectiveness.

1. Anorectal manometry measures internal and external anal sphincter pressure at rest and during contraction. High-resolution manometry and three-dimensional high-definition anorectal manometry provide greater anatomic detail than air- or water-perfused manometry for deficits along the anorectal sphincter complex.[15] Sensation and rectal capacity can also be evaluated with a rectal balloon. Balloon expulsion tests can be used with anorectal manometry to evaluate pelvic floor dyssynergia and other defecation disorders.
2. Two- or three-dimensional endorectal ultrasound and magnetic resonance imaging (MRI) evaluate structural defects in the external or internal anal sphincters, the rectal wall, and the puborectalis muscle.[16] Often, scarring or thinning of the muscle layers can also be detected. Endorectal ultrasound is more sensitive for detection of internal sphincter anatomy, whereas MRI is superior for discriminating between external anal sphincter tear and scarring when external anal sphincter atrophy is present.
3. Defecography evaluates the defecatory process after a barium paste is inserted rectally and the patient defecates under fluoroscopy. Defecography can assess rectal emptying or structural abnormalities in the pelvic floor, like obstruction or the presence of a rectocele. MRI defecography (dynamic MRI imaging) can also be used with specialized protocols to evaluate the soft tissue surrounding the anorectal area, including the anal sphincter and other pelvic floor muscles.[17]

Treatments for Fecal Incontinence

Accurate diagnosis of FI is essential for appropriate treatment.[18,19] Any cause of transient incontinence identified during evaluation should be addressed specifically. Behavioral, pharmacological, and surgical therapies are all effective in older people (see Table 29.3).[18] It is generally advisable to begin a treatment regimen with the least risk and burden for the patient and caregiver. In all types of FI, except those characterized by an obstructive process, behavioral techniques should be considered as first-line therapy unless the patient or caregiver has a specific preference for another type of therapy.[19]

Management of FI in long-term care settings differs from management in the ambulatory care setting for two principal reasons. First, comorbidities such as dementia

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Table 29.3 Therapeutic modalities in fecal incontinence

Type of incontinence	Recommended therapies
Urgency and passive fecal incontinence	*Behavioral therapy:* Pelvic floor muscle therapy with or without biofeedback
	Timed or scheduled toileting to improve bowel habits
	Drug therapy (to manage loose stool consistency):
	Psyllium fiber (1–3 teaspoons daily), may use more or less if needed Loperamide (2–4 mg daily), may use less if needed
	Cholestyramine powder (1 packet or 3 gm daily) – use 10–20 minutes prior to meals
	Injectable therapy: Dextranomer and sodium hyaluronate
Overflow fecal incontinence	*Office-based neuromodulation or surgery for neuromodulation:* Referral for consultation if indicated
	Management: Correct underlying causes for constipation

and mobility impairment are more frequent and more severe in the long-term care setting, and these complicate management. Second, in the outpatient setting, behavioral interventions can be implemented by the patients themselves or by highly motivated caregivers, who are usually family members. In long-term care, these interventions are implemented by nursing assistants whose motivation may be compromised by high patient-to-staff ratios. For these reasons, treatment of FI in the long-term care setting is discussed separately.

Initial treatments for FI include conservative measures (dietary modifications, bowel habit training, and pelvic floor muscle training with or without biofeedback and electrical stimulation) and pharmacologic treatments (constipating and/or stool bulking agents).[19,20] Conservative therapies are often combined and improve mild FI by 50–95%, depending on the modality used.[19] When FI is not responding to initial treatments, perianal injectable bulking agents and sacral neuromodulation (surgical and nonsurgical) may be considered.[21]

Dietary and Behavioral Interventions for Fecal Incontinence

Dietary modifications should focus on avoiding triggers for loose stool such as incompletely digested sugars (e.g., fructose and lactose-containing products) and/or gluten products. Increasing dietary or supplementary fiber may improve loose stool and decrease diarrhea by bulking the stool. If used with adequate fluid intake, fiber may also improve stool consistency. In general, solid stool may be easier to retain in the rectum. Recent data suggests that fiber treatment may be beneficial for FI and that constipating agents may increase other bowel symptoms. [18,22] Bowel habit training and scheduled toileting with or without laxatives to empty the rectum may help those with cognitive or mobility problems.

Pharmacologic Treatment

The goal of pharmacologic therapy is to reduce stool frequency and improve stool consistency. The most commonly used pharmacologic treatments involve antidiarrheal drugs for diarrhea-associated FI, such as loperamide (Imodium) and diphenoxylate plus atropine (Lomotil).[18] No antidiarrheal drugs have specific approval for the treatment of FI in the United States. In clinical studies of these drugs for FI treatment, people on an antidiarrheal drug as compared to placebo reported improvements in fecal urgency, FI episodes, unformed stools, constipation, abdominal pain, diarrhea, headache, and nausea. They also reported decreased use of pads.[23] However, given the anticholinergic properties of diphenoxylate plus atropine, this treatment should be avoided in older adults.

Occasionally, when antidiarrheal drugs are not effective for improving diarrhea-associated FI, a trial with bile acid binders (e.g., cholestyramine) is warranted. Cholestyramine is a bile salt binding medication used to lower cholesterol and can reduce diarrhea associated with the production of excess bile acid. It may be more effective in patients with a history of prior cholecystectomy or ileocolonic resection. Limited data exists on the use of this medication specifically for FI.[24]

Using rectal suppositories or enemas may reduce FI episodes by further evacuation of rectal contents in older patients. This method is also used to help with neurogenic bowel dysfunction due to spinal cord injury.[25]

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Biofeedback

If initial management with dietary changes, fiber, and pharmacologic therapy fails, biofeedback may be considered. Biofeedback involves a trained provider who utilizes an instrument with visual or auditory feedback on the proper control of voluntary muscle contraction and relaxation of the external anal sphincter and recognition of anal sphincter sensation.[26] Strength training, sensory training, and coordinated training (strength and sensory) occur in most biofeedback protocols for FI.[27] The goal is improved external anal sphincter muscle contraction in response to improved rectal sensation or distention. For success, patients who undergo biofeedback treatment need to be aware of their defecation symptoms and be able to actively participate during the office-based treatments and a home exercise program.

In an early study of biofeedback for FI, 108 patients with incontinence at least once a month were recruited and treated with education about the effects of diarrhea and constipation on FI, drugs to minimize diarrhea or constipation, and pelvic floor exercises to improve sphincter contraction for 1 month. The patients reporting adequate relief (21%) were dismissed from the trial, and the remainder were randomized to biofeedback or pelvic floor exercises. After 3 months, the patients treated with biofeedback showed greater reductions in the Fecal Incontinence Severity Score than those on pelvic floor exercises, and these results were sustained for the 1-year trial. Thus, biofeedback had a greater impact than pelvic floor exercises after excluding those who responded to conservative management.[38]

Data comparing efficacy of pharmacologic treatments and anal sphincter exercises alone to biofeedback for FI is limited.[26] In a trial of 300 women with greater than one episode of FI per month and normal stool consistency, titrated dose of loperamide, six sessions of biofeedback, and a combination of both treatments were compared to a control education and oral placebo in a 24-week clinical trial.[28] No significant differences were noted in the interventions compared to the control group. In further subgroup analysis, overweight women and women with higher symptom severity at baseline had higher clinical response rates to the interventions.[29]

Injectable Agents

Injectable agents are most often given for passive FI to improve resting sphincter tone. An office-based procedure involves the perianal injection of Dextranoner microspheres stabilized in hyaluronic acid. Data from clinical trials reported reduced FI episodes in the short term. However, in longer-term follow-up (up to 2 years) of patients who received anal injections compared to anal sphincter training, some of the initial improvements in FI episodes were not sustained.[30] Studies involving other injectable agents into the perianal area have not shown significant improvements in FI episodes. Other injectable materials, including the use of adipose-derived human stem cells, have been used to augment the internal anal sphincter, but more data is needed on safety and efficacy.[31,32]

Sacral Neuromodulation

Percutaneous tibial nerve stimulation (PTNS) and surgically implanted nerve stimulation devices involve the treatment of FI by stimulating the sacral nerves that help control defecation. The exact mechanism of action of sacral nerve neuromodulation is not known. PTNS is an office-based procedure to indirectly stimulate the sacral nerves through the posterior tibial nerve and is the same treatment mentioned for UI (see Chapter 28 on UI). A small, acupuncture-sized needle is inserted into the medial aspect of the lower extremity and attached to a stimulation device. Often, 12 weeks of treatment are needed to see improvement in symptoms. Data from adequately powered randomized controlled trials for PTNS treatment for FI failed to show overall improvement in reducing FI episodes in 227 patients compared to a sham PTNS treatment.[33]

Surgically implanted neuromodulation devices exist for refractory FI. These surgically implanted devices stimulate the sacral nerves (at S3), and their use has led to improvement in symptoms compared to conservative measures.[21] Limited data exists to guide treatment response in older adults or more frail adults with FI.

Other Surgical Approaches

Other surgical therapies include anal sphincter repairs, artificial bowel sphincter implant, and colostomy.[21] Overlapping sphincteroplasty can be used to repair a torn anal sphincter, which most commonly results from an obstetrical injury and can be an occult finding in older women. Although short-term benefits may exist, longer-term data on outcomes is needed. Use of an artificial bowel sphincter has significant associated morbidity, and colostomy is effective but it is used essentially as a salvage procedure.

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Managing Fecal Incontinence with Absorbent Pads and Other Continence Products

Absorbent pads and other continence products are designed for management rather than treatment of FI. Although evaluation and treatment are recommended, many patients will either depend on continence products exclusively or will use absorbent pads for security in circumstances where they are fearful of incontinence.

Disposable absorbent pads typically consist of a superabsorbent layer surrounded by an outer barrier and an inner membrane that conducts moisture away from the skin. They are supplied both as small pads to be worn in undergarments by patients who typically experience small-volume leakage or as briefs worn in place of undergarments by patients who at least occasionally have large-volume leakage of urine. While these products are more appropriate for urine leakage, they are also often used for FI with mixed results. Light pads that are folded and inserted between the buttocks are available specifically for fecal seepage.

A vaginal insert with a pressure-regulated pump to temporarily occlude the rectum has been shown to reduce FI episodes by 50% in about 80% of women at 1 month in a cohort study.[34] The use of a self-inserted silicone rectal plug may also be effective for some women with FI.[35] Additional studies are needed to compare efficacy of treatment with these devices to other treatments, as well as longer-term follow-up.

Treatment in Long-Term Care Facilities

Prevalence estimates of FI in long-term care facilities are up to 60% from Minimum Data Set data in both men and women aged 65 and older. Prompted voiding that is often used to manage UI may also have some benefit for adults with constipation and FI in long-term care settings.[36] For the most part, in nursing homes UI and FI are managed rather than treated or prevented, usually by placing absorbent pads on beds or chairs or by keeping residents in absorbent undergarments and changing these two to three times per day.

Behavioral Treatment for Fecal Incontinence in Long-Term Care Settings

Prompted voiding is the behavioral program that has been most thoroughly evaluated. A staff member approaches each incontinent resident every 2 hours (or at intervals individualized to each patient) and asks the resident if he or she would like assistance to get to the toilet. Assistance is provided if needed, and staff are instructed to encourage patients to void/defecate and to praise them for success at remaining continent between prompts. Increasing fluid intake and exercise may also be included. In a few studies, this approach increased the number of bowel movements in the toilet but did not decrease the number of FI episodes. Moreover, approximately half of nursing home residents do not benefit because of the severity of cognitive impairment or other functional limitations. A trial period may be indicated to focus prompted voiding techniques on those who respond to an initial 3-day evaluation period.

Management studies have shown that the average time required to assist a patient to the toilet greatly exceeds the time required to change absorbent undergarments or bed pads and linens.[37]

Summary

FI remains a fairly common, often underreported, and vexing problem in older patients that can lead to social isolation and limit caregiver ability to manage. Therapeutic options using behavioral, pharmacological, and surgical approaches can lead to symptomatic improvements or cure for this important clinical problem and increased quality of life for the older patient.

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Chapter 30

Gynecologic Issues

Rebecca Jackson and Ashley R. Tran

Introduction

Routine gynecologic care contributes to the health and wellness of women well beyond the reproductive years, and is an important part of preventive care. The Women's Preventive Service Initiative recommends that women receive at least one preventive care visit per year beginning in adolescence and continuing across the lifespan. Whether or not a routine pelvic exam should be done during that visit is controversial. The American College of Obstetricians and Gynecologists (ACOG) recommends that pelvic exams should be performed when indicated by medical history such as cervical dysplasia, gynecologic malignancy, or in utero diethylstilbestrol exposure or symptoms such as abnormal bleeding, dyspareunia, pelvic pain, sexual dysfunction, vaginal dryness, vaginal bulge, urinary issues, or inability to insert a tampon. For asymptomatic women, the decision about a routine pelvic exam should involve shared decision-making between the patient and her gynecologic care provider.[1]

If a pelvic exam is going to be performed, adjustments may need to be made. Lower and/or adjustable tables can help with pain and decreased range of motion in hips, knees, and spine. Pillows can help with cervical disc disease or kyphosis and congestive heart failure. Using speculums with smaller width can help with atrophic changes.[2] If a pelvic exam is deferred, the annual exam serves as an excellent opportunity to discuss overall health, urinary and bowel function, potential gynecologic and breast issues, sexual dysfunction, and cancer screening. It is important to keep in mind that elderly patients are less likely to report symptoms than younger patients, especially when pertaining to gynecologic concerns and sexual dysfunction. Sensitive history-taking with careful attention to risk factors and sexual history is an important part of caring for older women.

This chapter focuses on the most common gynecologic issues of the elderly: vulvovaginitis and other vulvar conditions, menopausal symptoms, pelvic floor prolapse, sexual dysfunction, and gynecologic malignancies. Urologic conditions, breast cancer, and osteoporosis are covered in Chapters 27, 41, and 34, respectively.

Vulvovaginitis and Other Conditions

Vulvovaginitis is a common complaint in the postmenopausal woman. The decrease in estrogen that accompanies menopause leads to increased irritation of the vaginal tissue, along with increased susceptibility for infection and trauma. The three most common causes of vulvovaginitis complaints are atrophic vaginitis, candidiasis, and lichen sclerosus.

Atrophic changes of the vulvovaginal area are very common following menopause, when the decreased presence of estrogen leads to thinning of the vaginal tissue. Symptoms of vaginal atrophy include vaginal dryness, irritation, itching, discharge, and dyspareunia. Physical findings include atrophic vaginal tissue that may be pale, smooth, shiny, erythematous, and friable with petechiae, loss of natural ruggae, and introital stenosis.[3] The approach to treatment of vulvovaginal atrophy is based upon the woman's level of discomfort. Intravaginal lubricants and long-term use of moisturizers can be helpful in treating mild to moderate symptoms.[4,5,6] Vaginal estrogens, available in cream, ring, and tablet formulations, are the most effective at treating severe postmenopausal atrophic vaginitis.[4,5,6] It should be noted that unopposed systemic estrogen has been shown to increase risk of endometrial and breast cancer, but local estrogen therapy is not associated with an increased risk of cancer and does not require concurrent treatment with progesterone.[4,5]

Candidiasis. Vulvovaginitis caused by candida infection presents similarly in patients of all ages, with complaints of pruritus, irritation, and discharge. Physical findings include inflammation and erythema, along with a discharge demonstrating buds and hyphae on microscopy. Treatment is similar for all age groups, including

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several different antifungals in topical or suppository formulations, or with a single dose of fluconazole in oral form.[3] It is important to note that candidiasis occurs less frequently in postmenopausal women unless vaginal estrogen is being used. Vulvovaginitis symptoms refractory to treatment with antifungals should be evaluated for other etiologies.

Lichen sclerosus. This is a chronic and progressive condition that causes thinning of the vulvar epithelium. Women often report pain, pruritis, and dyspareunia. It may present with thin, white "cigarette-paper" patches and can be treated with topical steroids.[7] Untreated lichen sclerosus can lead to permanent changes to the vulvar anatomy and is also associated with squamous cell carcinoma. Women should be examined annually with this in mind. Diagnosis is based upon the presence of characteristic clinical findings, and treatment can be initiated without a biopsy.[8] Histologic confirmation is indicated in any of the following findings: asymmetry, border irregularity, color variation, sudden change, bleeding, nonhealing ulceration, persistence, and lack of firm diagnosis.[8]

Pruritus. The differential for pruritus without discharge is large and includes primary dermatologic conditions, as well as manifestations of systemic illnesses (see Table 30.1 for a full differential diagnosis). A full history should be obtained, including use of topical creams, lotions, detergents, and bath products. Any ulcerations or lesions may warrant biopsy to rule out vulvar intraepithelial neoplasia or squamous cell carcinoma.

Table 30.1 Differential diagnosis of pruritis without discharge[2]

Dermatitis
Vulvar intraepithelial neoplasia (VIN)
Cancer
Lichen sclerosis
Squamous cell hyperplasia
Hypoestrogenic atrophic vaginitis
Mechanical irritation and wetness from urinary incontinence
Dermatoses (lichen sclerosus, lichen planus, psoriasis, contact and seborrheic dermatitis)
STIs
Systemic causes

- Uremia
- Hepatic disease
- Diabetes
- Thyroid disease
- Lymphomas/leukemias
- Graft versus host disease

Vaginal discharge. Any vaginal discharge should be evaluated by pelvic exam, vaginal pH, and wet prep, with screening and culture for sexually transmitted infections (STIs) when indicated. The three most common causes of vaginal discharge are bacterial vaginosis, trichomoniasis, and candidiasis. Bacterial vaginosis occurs when various anaerobes replace normal vaginal flora. It frequently causes an increase in vaginal discharge with a distinct fishy odor. Treatment for bacterial vaginosis is the same in elderly as in younger patients, with either oral metronidazole or clindamycin, or vaginal metronidazole.[9] Trichomonas may cause foul-smelling discharge and irritation, and may be treated with a single 2-gram dose of metronidazole. As trichomonas is sexually transmitted, both sexual partners should be treated. Candidiasis presents with irritation, pruritus, and/or discharge and may be treated as discussed above.[9] The differential diagnosis for chronic vaginal discharge includes: desquamative inflammatory vaginitis, chronic endometritis with malodorous purulent discharge, vesicovaginal fistula, or enterovaginal fistula following bowel inflammation and injury as in cases of diverticulitis.[10,11]

Vulvodynia. This is pain or discomfort of the vulva. Typically women describe a burning, stinging, irritation, or rawness. Vulvodynia without evidence of infection, dermatologic conditions, or vagina atrophy can be treated with topical lidocaine and low-dose antidepressants.

Bleeding. All postmenopausal bleeding requires a workup to rule out endometrial cancers. Benign causes of vaginal bleeding include: atrophic endometrium, endometrial hyperplasia, cervicitis, cervical or endometrial polyps, hormone therapy (HT), endometriosis, leiomyomas, vaginal atrophy, and friability.[12] Workup should include a full history, medications including hormonal therapies (i.e., tamoxifen), complete exam to evaluate for causes of bleeding other than vaginal (i.e., hematuria or rectal bleeding), ultrasound to evaluate endometrial lining, and an in-office endometrial biopsy if the endometrial lining is thickened. Hysteroscopy with dilation and curettage (D&C) may be indicated in refractory or inconclusive cases and would warrant referral to a gynecology specialist.[12]

STIs. The elderly are at increased risk of contracting STIs. Nearly half of all Americans living with HIV are age 50 and older.[13] Though new HIV diagnoses are declining, one in six new diagnoses in 2017 were in this age group.[14] There has been an overall increased incidence across all age groups of chlamydia, gonorrhea, and

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syphilis since 2011.[9] The CDC reports 7.4/100,000 new cases of chlamydia in men over age 65, and 2.3/100,000 new cases in women over age 65. New gonorrhea cases account for 9/100,000 men over 65 years and 1/100,000 women over 65 years. The incidence of syphilis increased by 28.6% during 2017–18, with 1.9/100,000 new cases in patients over 65 years.[15]

Age-related vaginal atrophy creates micro-tears in the vaginal mucosa, allowing bacteria more access, while a waning immune response makes them more susceptible to infection. Clinicians should encourage open communication between sexual partners and educate the patient about safer-sex techniques, including use of latex condoms and limiting the number of partners, to reduce the risk of infection and illness. Any presence of rash, blisters, or discharge should be evaluated right away.[16]

Menopausal Symptoms

Vasomotor Symptoms

Vasomotor symptoms (VMS) are the most common symptoms of menopause, occurring in 75–80% of women.[17] VMS are sometimes called hot flashes or hot flushes. When they occur at night, they are called night sweats. Women typically describe a sudden sensation of extreme heat in the face, neck, and chest, associated with sweating.[5] The pathophysiology is not completely understood, but is thought to be due to narrowing of the thermoregulatory zone induced by estrogen withdrawal.[18] VMS become common during the menopausal transition and peak approximately 1 year after the final menstrual period. It used to be thought that VMS would stop after a few years, but there is increasing evidence that for many women symptoms continue for much longer.[19,20]

Women with mild VMS can manage these with lifestyle modifications, such as reducing core body temperature, regular exercise, weight management, smoking cessation, and avoiding triggers like hot drinks and alcohol.[5]

Systemic HT is the most effective treatment for VMS. In women who have not had a hysterectomy, HT includes estrogen and a progestin (EPT), whereas women who have had a hysterectomy can take estrogen alone (ET). HT prescriptions decreased dramatically after the Women's Health Initiative (WHI). The WHI was a large randomized controlled trial of healthy menopausal women aged 50–77 years designed to look at the primary outcome of coronary heart disease prevention, which demonstrated an increased risk of breast cancer, coronary heart disease, stroke, and venous thromboembolic events with use of EPT.[21] Newer data suggests that these risks may be different in younger women. In 2017 the North American Menopause Society (NAMS) released their updated position statement on hormone therapy which states, "For women aged younger than 60 years or are within 10 years of menopause onset and have no contraindications, the benefit-risk ratio is most favorable for treatment of bothersome VMS."[6] Both NAMS and ACOG support extended use of HT, even beyond age 65, when patient and clinician agree that the benefit of symptom control outweighs the risks.[5,22] Contraindications to HT are personal history of breast cancer, coronary heart disease, previous history of venous thrombotic event, stroke or transient ischemic attack, active liver disease, or unexplained vaginal bleeding. The route of administration is important as well. Transdermal estrogen has been shown to have lower risk of thromboembolic events and should be used for women who are at moderate risk for cardiovascular disease, with a 10-year risk of 5–10%.[23]

For women with a contraindication to HT or who prefer to avoid HT, there are nonhormonal options for treatment as well. Selective serotonin reuptake inhibitors (SSRIs), selective serotonin-norepinephrine reuptake inhibitors (SSNRIs), clonidine, and gabapentin have been shown to be effective treatment for VMS. Each has been shown to be more effective than placebo. Paroxetine 7.5 mg is the only nonhormonal medicine that is approved by the Food and Drug Administration (FDA) for treatment of VMS[5] (see Table 30.2 for a summary of available options for treatment of VMS).

Genitourinary Syndrome of Menopause

The term genitourinary syndrome of menopause (GSM) was created in 2014 by the International Society for the Study of Women's Sexual Health and the North American Menopause Society to encompass the symptoms in the vulvovaginal area as well as the urethra and bladder that are caused by a lack of estrogen. GSM includes symptoms of dryness, burning, and irritation of vulva and vagina; decreased sexual function with lack of lubrication and/or pain with intercourse; and urinary urgency, dysuria, and recurrent urinary tract infections. Women can present with any or all of these symptoms.[24] Up to 45% of women report experiencing these symptoms.[25] First-line treatment is nonhormonal lubricants with intercourse and regular use of long-acting vaginal moisturizers. For

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Table 30.2 Food and Drug Administration-approved treatments for vasomotor symptoms of menopause

Treatment category	Preparation	Doses available	Notes
Oral estrogen	Conjugated equine estrogen (CEE)	0.3, 0.45, 0.625, 0.9, 1.25 mg	*For women with an intact uterus a progestin should be added for endometrial protection*
	Micronized estradiol*	0.5, 1, 2 mg	
	Esterified estrogen	0.3, 0.625. 1.25 mg	
	Estropropipate	0.75, 1.5, 3 mg	
	Conjugated synthetic estrogen	0.3, 0.45, 0.625, 0.9 mg	
Oral estrogen-progestin combinations	CEE/medroxyprogesterone	0.3/1.5, 0.45/1.5, 0.625/2.5, 0.625 mg/5 mg	
	Estradiol/norethindrone acetate*	0.5/0.1, 1 mg/5 mg	
	Estradiol/norgestimate	1 mg/0.09 mg	
	Ethinyl estradiol/ norethindrone acetate	2.5 mcg/0.5 mg, 5 mcg/1 mg	
	Estradiol/drosperinone	0.5/0.25, 1 mg/0.5 mg	
Oral estrogen-	CEE/bazedoxifene	0.45 mg/20 mg	
SERM combination			
Estrogen patches	Estradiol (twice weekly)*	0.025, 0.0375, 0.05, 0.075, 0.1 mg/day	*For women with an intact uterus a progestin should be added for endometrial protection*
	Estradiol (weekly)*	0.025, 0.0375, 0.05, 0.06, 0.075, 0.1 mg/day	
Estrogen-progestin combination patches	Estradiol/norethindrone (twice weekly)	0.05/0.14, 0.05 mg/0.25 mg per day	
	Estradiol/levonorgestrel (weekly)	0.045 mg/0.015 mg per day	
Other topical estrogens	Estradiol gel	0.25, 0.5, 0.52, 0.75, 1 mg per pump	*For women with an intact uterus a progestin should be added for endometrial protection*
	Estradiol spray	1.53 mg/spray	
Nonhormonal	Paroxetine (selective serotonin reuptake inhibitor)	7.5 mg	

* **available as a generic**

women with moderate to severe symptoms, or milder symptoms not relieved with nonhormonal topical options, low-dose vaginal estrogen should be used. Vaginal estrogen can be administered as a cream, tablet, capsule, or ring. In usual doses of vaginal estrogen to treat GSM, the addition of a progestin is not required for endometrial protection.[5,26] In women with a personal history of breast cancer, nonhormonal treatments should be the first-line approach. If these are unsuccessful in treating symptoms, vaginal estrogen may be used after discussion of risks and benefits and with consultation with an oncologist.[27]

Other treatments for GSM include oral ospemifene and vaginal dehydroepiandrosterone (DHEA). Ospemifene is a selective estrogen receptor modulator (SERM) that has been shown to improve vaginal dryness and dyspareunia in postmenopausal women.[28] Both ospemifene and vaginal DHEA have been shown to be more effective than placebo, but neither has been compared to vaginal estrogen.[28,29]

Pelvic Organ Prolapse

Pelvic organ prolapse is a common yet underdiagnosed condition for older women. Symptoms can greatly affect quality of life by altering a woman's function and activity. Women living with prolapse reported feeling self-conscious and isolated, having loss of interest in activities, and avoiding intimacy because of embarrassment or discomfort.[30] Pelvic organ prolapse is the descent of one or more of the anterior vaginal wall, posterior vaginal wall, uterus, or vaginal vault (after hysterectomy) into or beyond the vagina. While most women with prolapse are asymptomatic, pelvic organ prolapse can cause a sensation of pressure or bulge in the vagina and is associated with difficulty voiding (urinary incontinence or retention) or difficulty having bowel movements (fecal incontinence or constipation).[31] The cause for pelvic organ prolapse is multifactorial and poorly understood. Common risk factors include pregnancy, aging and decreased estrogen levels, constipation, chronic cough, obesity, and repeated heavy lifting.[31] Treatment for pelvic organ prolapse is based on the severity of symptoms. The Pelvic Organ Prolapse Quantitation (POPQ) system is commonly used to grade the degree of prolapse.[32] Asymptomatic women may require only observation. Conservative management begins with pelvic floor muscle strengthening with Kegel exercises, or pelvic floor physical therapy.[32] Another nonsurgical option is the vaginal pessary, which supports the pelvic organs and can provide immediate improvement in symptoms.[32] In general, the pessary can be left in place and removed every 3 months in the clinician's office for cleaning and inspection of the vagina. However, if symptoms are severe and conservative management is not effective, surgical repair may be warranted.[33]

Sexuality and Sexual Dysfunction

Although menopause may trigger changes in sexual function, aging does not signal the end of a woman's sex life. Sexuality and a healthy sex life remain an important part of the postmenopausal woman's overall quality of life. One national study revealed older adults reported having sex an average of two to four times per month. Another study looking at men over the age of 80 years reported 29% of the study participants were having at least weekly sex, while 38% were not sexually active.[16] Clinicians should be comfortable asking about sexuality and any concerns that their patients have in a sensitive manner. Elderly patients may be less likely to bring up their concerns, so integrating screening into regular interview questions and normalizing the conversation can help. The clinician should discuss normal age-related physiologic changes, address how medications and chronic medical conditions can affect sexual function, and teach about safer-sex practices as well as protection from STIs and abuse.[34] When asking patients about sexual activity, keep in mind that 2.5–3% of older adults report having two or more sexual partners in the last year, so screening for STIs is still indicated.[16] In addition, screening for sexual abuse is indicated in older adults, both those living independently and those residing in long-term care facilities. Patients with cognitive impairment may not be able to consent to sexual activity and are at risk for abuse.[16,34]

Most sexual dysfunction and dyspareunia in the postmenopausal woman is caused by lack of natural lubrication associated with atrophic changes that happen in the absence of estrogen.[11] Most female concerns are related to normal, age-related atrophic changes, which can be treated with lubricants or low-dose vaginal estrogen creams.[11] Other causes of sexual dysfunction in postmenopausal women include skin conditions such as psoriasis and lichen sclerosus (discussed above), urinary tract conditions such as chronic inflammation and incontinence, pelvic adhesions, hot flashes, and other medical conditions such as diabetes, heart disease, and cancer. It should be noted that depression, and depression medications, namely SSRIs, can contribute to sexual dysfunction. Stress and body image issues can be major factors

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as well.[35] A biopsychosocial assessment of both partners is recommended prior to treatment of woman's sexual dysfunction if not lubrication-related.[5]

Screening and Diagnosis of Gynecologic Cancers

There are conflicting recommendations from different organizations, including the American Cancer Society, the US Preventive Task Force, and ACOG, as to the upper age limit for screening for gynecologic cancers. A woman's life expectancy, risk of cancer death, and unique values and preferences must be considered. In general, cancer screening is not recommended if life expectancy is less than 5 years.[36]

Endometrial Cancer

Endometrial cancer is the most common gynecologic cancer in the United States (and developed countries). Women in the USA have a 2.8% lifetime risk of developing uterine cancer. The incidence peaks between ages 60 and 70.[37]

There is no current screening for endometrial cancer. Most women with endometrial cancer will present with abnormal uterine bleeding or postmenopausal bleeding. All women who complain of postmenopausal bleeding require evaluation. The initial evaluation can be either with transvaginal ultrasound or endometrial biopsy.[38,39] An endometrial thickness of less than or equal to 4 mm in a woman with postmenopausal bleeding is associated with a low risk of endometrial cancer or premalignant conditions.

Endometrial cancer can be divided into two types. Type 1 cancers are most common, are histologically endometrioid adenocarcinomas, are FIGO (International Federation of Gynecology and Obstetrics) grades 1 and 2, and typically have a good prognosis. Type 2 neoplasms include FIGO grade 3 endometrioid endometrial cancers and other histologies including serous, clear cell, mucinous, squamous, transitional cell, mesonephric, and undifferentiated. Type 2 endometrial cancers have a worse prognosis.[40,41]

The greatest risk factor for type 1 endometrial cancer is excess estrogen, either exogenous or endogenous. Exogenous sources include unopposed estrogen – systemic estrogen without concurrent progestin[42] – and use of tamoxifen, which is a selective estrogen receptor modulator.[43] Endogenous sources include chronic anovulation, obesity,[44] early menarche/late menopause,[45] and estrogen-secreting tumors, such as granulosa cell tumors. Women with Lynch syndrome have a significant risk of developing type 1 endometrial cancer.[46] Age is a risk factor for both type 1 and type 2 endometrial cancers. Smoking actually decreases risk for type 1 endometrial cancer,[47] but increases risk for type 2.[48] Use of combination oral contraceptive pills,[49] depot medroxyprogesterone acetate,[50] and the levonorgestrel IUD[51] decrease the risk of endometrial cancer.

The prognosis for endometrial cancer depends on the disease stage, grade, and histology. Most women are diagnosed with early-stage disease and endometrioid histology, and therefore have a good prognosis. The 5-year survival rate for stage 1 disease is approximately 90%.[52]

Treatment usually involves a total hysterectomy, bilateral salpingo-oophorectomy, lymph node dissection, and evaluation for extrauterine disease. Postoperative radiation and chemotherapy is used in women with high-risk disease.[41]

Ovarian Cancer

In the United States, ovarian cancer is the second most common gynecologic malignancy, but it has the highest death rate of all gynecologic cancers. In 2021, there were an estimated 21,410 new cases and 13,770 ovarian-cancer-related deaths in the USA.[37] While fallopian tube cancer and peritoneal cancer have been thought to be uncommon, it is now believed that many serous ovarian cancers actually originate in the distal fallopian tube.[53]

Unfortunately, there is no screening that has been shown to be effective at reducing mortality for asymptomatic average-risk patients.[54,55,56] Women with a strong family history of breast, ovarian, or colon cancer should be referred for genetic testing.[55,56] Increasing age is associated with an increased risk of ovarian cancer, with a median age of 66 at diagnosis. Factors that increase the number of ovulatory cycles such as early menarche/late menopause and nulliparity increase the risk for ovarian cancer.[57] Factors that are protective for ovarian cancer include bilateral salpingo-oophorectomy, bilateral salpingectomy, tubal ligation, hysterectomy, oral contraceptive use, breastfeeding, and multiparity.[58,59]

Women with early-stage disease may complain of abdominal bloating, urinary frequency or urgency, feeling full quickly, or pelvic and abdominal pain. While these symptoms are vague and common, when they are persistent and represent a change from baseline, they should prompt investigation.[60] Some women will have an adnexal mass found on pelvic exam and should be referred for transvaginal ultrasound. Adnexal cyst size

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greater than 10 cm, papillary or solid components, irregularity, presence of ascites, and high-color Doppler flow should increase the clinician's level of suspicion for malignancy.[61] In postmenopausal women with an adnexal mass, CA-125 levels can be useful to evaluate the likeliness of malignancy and need for referral to a gynecologic oncologist.[61] Women with advanced disease may present with more acute symptoms. They may present with a malignant pleural effusion or bowel obstruction.

Postmenopausal women with elevated CA 125 level, ultrasound findings suggestive of malignancy, ascites, a nodular or fixed pelvic mass, or evidence of abdominal or distant metastasis should be referred to a gynecologic oncologist.[61] Ovarian cancer is staged surgically. Treatment includes cytoreductive surgery and chemotherapy. Older women should undergo a geriatric assessment looking at functional status, comorbid medical conditions, cognition, psychological status, social functioning, and nutritional status to determine appropriateness of surgery and/or chemotherapy.[62]

Cervical Cancer

In the United States, cervical cancer is the third most common gynecologic malignancy.[37] Cervical cancer is related to infection with human papillomavirus (HPV), which is detected in over 99% of cases.[63] Risk factors for HPV infection are also risk factors for cervical cancer, including early onset of sexual activity, multiple sexual partners, a history of STIs, immunosuppression, and smoking.[64] Low socioeconomic status and nonwhite race also increase both incidence and mortality from cervical cancer. Most cases of cervical cancer occur in women who have been inadequately screened.[65]

Preferred current screening recommendation for women aged 30 to 64 is cervical cytology with cotesting for HPV every 5 years. Cervical cytology alone every 3 years is also acceptable. For women over age 65 with adequate negative screening results and no history of CIN 2 or higher, screening should be discontinued. Adequate negative prior screening test results are defined as three consecutive negative cytology results or two consecutive negative cotest results within the previous 10 years, with the most recent test performed within the past 5 years.[66] This recommendation is supported by ACOG, the American Cancer Society and the United States Preventive Services Task Force. Women treated in the past for CIN 2 or higher remain at risk of persistent or recurrent disease for at least 20 years after treatment. Because of this increased risk, women with a history of CIN 2 or higher should continue to undergo routine age-based screening for 20 years after treatment, even if it requires that screening continue past age 65 years.[67] For women with HIV or other immunocompromised women, screening either with cytology alone or cotesting should continue every 3 years throughout their lifetime.[68] Women who were exposed to diethylstilbestrol in utero also require surveillance beyond the age of 65.

Early cervical cancer is often asymptomatic, highlighting the importance of screening. For those women who are symptomatic, the most common symptoms are abnormal uterine bleeding or postcoital bleeding.[69] Depending on the extent of disease at diagnosis, treatment for invasive cervical cancer can involve radical hysterectomy, radiation, and/or chemotherapy.[69] Treatment for high-grade precancerous lesions involves cervical conization procedures.

Vulvar Cancer

Vulvar cancer is less common than endometrial, ovarian, and cervical cancer. It accounts for approximately 5% of gynecologic malignancies.[37] Vulvar cancer is most common in women aged 65 and older.[37] Squamous cell carcinoma is the most common histology, comprising at least 75% of cases. Other histologies include basal cell carcinoma (8%) and melanoma (6%).[70] Risk factors for vulvar cancer include HPV infection, vulvar lichen sclerosus, cigarette smoking, and immunodeficiency.[71]

Vulvar cancer usually presents with vulvar pruritus or a vulvar lesion, noticed by the patient or clinician. Diagnosis is made with vulvar biopsy. Vulvar cancer is usually treated with surgery, though chemoradiation can be used for advanced or metastatic disease.[71]

Vaginal Cancer

Vaginal cancer is relatively uncommon and has a similar incidence to vulvar cancer.[37] Most vaginal cancers are squamous cell cancers, though melanoma, sarcoma, adenocarcinoma, and other histologic types can occur. Metastatic disease or local extension from adjacent gynecologic malignancies is more common than primary vaginal cancer.[72] Vaginal cancer is related to HPV infection and shares many of the same risk factors as cervical cancer, such as multiple lifetime partners, early age at first intercourse, and smoking.[73]

Vaginal bleeding or vaginal lesion are the most common presenting symptoms. The lesion can be a mass, plaque, or ulcer. Diagnosis is made by vaginal biopsy.

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It is considered to be primary vaginal cancer in a woman with no history of gynecologic malignancy that could better define the disease as recurrent cancer as opposed to a new primary. Treatment for vaginal cancer depends on the extent of the disease and may involve surgery or radiation.[74]

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Chapter 31

Endocrine Disorders

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Introduction

Dysfunction of the endocrine glands can occur at any point in the life cycle. Although many endocrine diseases will present with classic signs and symptoms, atypical presentation of hormonal dysregulation can make diagnosis in the elderly particularly challenging. Normal physiologic changes associated with aging, as well as medical comorbidities and medications, may all cloud the identification of endocrine dysfunction in this complicated population. As such, the diagnosis of endocrinopathies in the elderly population requires a careful medical history, detailed physical exam, rational biochemical workup, and, if necessary, directed imaging. Management of endocrine disorders can be equally complex. Many endocrine disorders are treated with medications that may complicate an already-lengthy list, causing unwanted side effects or even drug–drug interactions. If therapy includes possible surgical referral, careful assessment of the risk–benefit ratio and candidacy of the elderly patient is imperative. Endocrine guidelines have been designed to assist the clinician with accurate diagnosis and rational therapeutic decision-making; however, guidelines cannot supplant the need for patient-centered care in this vulnerable population in whom disorders of the endocrine glands fail to adhere to "textbook" scenarios.

Parathyroid Disease and Other Disease of Calcium Metabolism

Primary Hyperparathyroidism

Parathyroid hormone (PTH)-mediated hypercalcemia is often an incidental finding on routine laboratory assessment. Primary hyperparathyroidism (PHPT) is the most common cause of hypercalcemia and occurs in 1 in 1,000 persons in the United States. The incidence of PHPT peaks in the seventh decade of life. More than 80% of patients with PHPT are considered asymptomatic at the time of diagnosis.[1] Classic symptoms of the disease include nephrolithiasis and bone disease (osteoporosis, osteitis fibrosa cystica). Other symptoms of PHPT are often vague, nonspecific, and may overlap with common complaints among elderly patients. These include easy fatigability, myalgias, bone pain, depressed affect, abdominal cramping or constipation, weakness, headaches, irritability, forgetfulness, and difficulty rising from a chair. Laboratory findings in PHPT universally include hypercalcemia as well as inappropriately normal or high serum PTH levels. Additional studies may show low serum phosphate, hypercalciuria, high levels of renal cyclic adenosine monophosphate (cAMP), and enhanced markers of bone resorption.

Patients with confirmed PHPT who are symptomatic should undergo parathyroidectomy. Unfortunately, the distinction between symptomatic and asymptomatic patients is rarely well defined. A careful history for onset of symptoms compared to trajectory of serum calcium may be helpful in challenging cases. If a patient is not clearly symptomatic, guidelines should be followed to determine management.[2] Specifically, parathyroidectomy is indicated in asymptomatic patients with PHPT who meet any one of the following conditions: (1) serum calcium concentration of 1.0 mg/dL or more above the upper limit of normal; (2) bone density at the hip, lumbar spine, femoral neck, or distal 1/3 radius that is more than 2.5 standard deviations below peak bone mass (T score ≤ -2.5), or vertebral fracture by X-ray, computed tomography (CT), magnetic resonance imaging (MRI), or vertebral fracture assessment (VFA); (3) creatinine clearance that is reduced to <60 mL/min; (4) 24-hour urine calcium >400 mg/24 hr and increased risk on urinary biochemical stone risk profile or presence of nephrolithiasis or nephrocalcinosis by X-ray, ultrasound, or CT; (5) age less than 50 years.

The type of surgical intervention is dictated by the number of parathyroid glands involved. PHPT is most often caused by a single adenoma (80–85%) or four-gland

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hyperplasia (10–15%).[1] It is rarely part of hereditary syndromes such as multiple endocrine neoplasia types 1 and 2A. Neck imaging is not necessary for diagnosis of PHPT, but it should be ordered if surgery is indicated. Localization of the overactive gland(s) can decrease the invasiveness and morbidity of surgery. Both sestamibi scan and neck ultrasound are useful for localization.[3]

Parathyroidectomy should be performed by an experienced surgeon who uses intraoperative PTH monitoring. Surgery is associated with a cure rate of 95–98%. There is a low rate (1–3%) of complications, including laryngeal-nerve palsy and postoperative hypocalcemia, particularly when vitamin D is not replete prior to surgery. Successful parathyroidectomy is followed by a prompt normalization of the PTH level, serum and urinary calcium levels, and gradual increases in bone mineral density (up to 10% over the course of several years).

In elderly patients who are asymptomatic and have serum calcium concentrations <1.0 mg/dL above the upper limit of normal, parathyroidectomy can be deferred. Surveillance should include monitoring with yearly measurement of serum creatinine, calcium, and bone mineral density. Although counterintuitive, calcium intake through dietary sources or supplements should reach 1,000 mg daily for skeletal protection. Vitamin D deficiency often coexists with PHPT and should be actively managed to prevent additional bone loss and superimposed secondary hyperparathyroidism. Vitamin D should be repleted to levels ≥20 ng/mL both prior to surgery and with conservative management.[2] In elderly patients who meet criteria for parathyroidectomy but are medically unfit for surgery or do not wish to have surgery, medical management with cinacalcet or bisphosphonates may be effective. While cinacalcet may lower calcium and PTH levels, it unfortunately does not improve bone mineral density, calciuria, or nephrolithiasis risk.[4] In patients with PHPT who are medically unfit for surgery and have low bone mineral density, bisphosphonates may have a dual benefit of improving bone density and lowering calcium levels.[5]

Vitamin D Deficiency

Vitamin D is predictive of a wide variety of clinical outcomes related to mortality, cardiovascular health, cognitive function, muscle and bone health, and multiple sclerosis. The prevalence of vitamin D deficiency in persons over the age of 65 has been estimated at 50%, although this is highly variable and dependent on geographic location and socioeconomic, clinical, and other factors.[6] Further complicating this statistic is the lack of consensus regarding the definition of "deficiency." Although the Institute of Medicine defines sufficient as serum 25-hydroxy vitamin D above 20 ng/mL (50 nmol/L), many organizations – including the Endocrine Society, National Osteoporosis Foundation, and American Geriatric Society – recommend that the elderly maintain a minimum serum level of 30 ng/mL (75 nmol/L) to reduce the risk of falls and fracture.[7–9] Elderly and institutionalized patients are at risk for vitamin D deficiency because of age-related decline in renal function and skin structure modifications that affect vitamin D production. Low sunlight exposure, female sex, dark skin pigmentation, malnutrition, and obesity are also risk factors for vitamin D deficiency.[10]

A 2005 Cochrane review concluded that calcium and vitamin D treatment in frail elderly people who are confined to institutions reduces hip and vertebral fractures.[11] Further, a 2014 Cochrane review suggested vitamin D3 supplementation may reduce mortality in elderly people living independently or in institutional care.[12] A 2019 systematic review and meta-analysis with a mean age of 68.6 years showed that combined supplementation of vitamin D (400–800 IU daily) and calcium (1,000–1,200 mg) was associated with a reduced rate of any fracture and hip fracture.[13] For individuals with vitamin D deficiency on lab testing, high-dose repletion in the form of weekly ergocalciferol 50,000 IU may be necessary for several weeks. In some, ergocalciferol every other week standing has been shown to effectively maintain serum levels. Vitamin D deficiency has been implicated as a cause of bone pain, weakness, impaired cognition, low mood, and fatigue. Repletion may improve these symptoms, although data has not been definitive.

Secondary and Tertiary Hyperparathyroidism

Secondary hyperparathyroidism is diagnosed in the setting of elevated PTH with concurrent normo- or hypocalcemia. The most common causes of secondary hyperparathyroidism in the elderly are vitamin D deficiency and chronic kidney disease (CKD). PTH increases in CKD as an adaptive response to declining glomerular filtration rate (GFR). Produced in the kidney, circulating 1,25-dihydroxyvitamin D levels begin to decrease in stage 2 CKD and continue to fall with ongoing renal insufficiency. As GFR decreases below 60 mL/min/1.73 m^2, phosphate is retained in the kidney and stimulates synthesis and secretion of PTH. Hypocalcemia

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develops as the GFR decreases below 50 mL/min/1.73 m^2, further stimulating release of PTH.

Chronic secondary hyperparathyroidism of any cause can result in bone loss and fractures, cardiovascular disease, and increased mortality. Elderly patients with secondary hyperparathyroidism due to CKD should be followed by a nephrologist and/or endocrinologist for implementation of calcium, vitamin D, and cinacalcet and/or calcitriol therapy to lower PTH levels.[14] If left untreated, secondary hyperparathyroidism may evolve into tertiary disease in which parathyroid glands begin to function autonomously. This phenomenon is most commonly observed in those with longstanding renal disease or hemodialysis patients. Laboratory studies in tertiary hyperparathyroidism mimic those of primary disease with elevations in serum calcium and PTH levels. If pharmacotherapy is not effective in controlling serum calcium and PTH levels, subtotal parathyroidectomy may be necessary.

Hypercalcemia of Malignancy

In the elderly population, hypercalcemia in the presence of a low or suppressed PTH level should always prompt screening for occult malignancy. Parathyroid hormone-related protein (PTHrP) has been identified as the most common cause of hypercalcemia in patients with nonmetastatic solid tumors. PTHrP elevations are seen most commonly in squamous cell carcinoma (lung, head, neck), breast, prostate, renal, and bladder cancers.[15] Patients with non-Hodgkin's lymphoma, chronic myeloid leukemia, and adult T-cell leukemia may also have elevated PTHrP levels. The two other major mechanisms of hypercalcemia of malignancy are (1) osteolytic metastases with local release of cytokines (including osteoclast-activating factors), and (2) tumor production of 1,25-dihydroxyvitamin D (calcitriol). Osteolytic metastases account for approximately one fifth of cases of hypercalcemia of malignancy due to bone destruction by osteoclasts and occur in solid tumors with bone metastases and multiple myeloma. In lymphoma, hypercalcemia may be due to increased and PTH-independent production of 1,25-dihydroxyvitamin D (calcitriol), increased intestinal calcium absorption, and bone resorption. In most cases, treatment of the underlying malignancy improves serum calcium levels; occasionally, antiresorptive medications or steroids are also needed.

Hypoparathyroidism

Congenital hypoparathyroidism is usually part of a genetic syndrome detected early in life. In elderly patients, new-onset hypoparathyroidism is more likely to be iatrogenic. Acquired hypoparathyroidism is often due to inadvertent removal of the parathyroid glands or irreversible damage to their blood supply during thyroidectomy, parathyroidectomy, or neck dissection surgery. Patients may present with perioral numbness and tingling, tetany, hypotension, and arrhythmias, depending on the acuity of serum calcium declines. In the outpatient setting, these patients are treated with high-dose calcium and/or calcitriol, with the goal albumin-corrected calcium level at the lower end of normal range (8.0 mg/dL–8.5 mg/dL) to prevent nephrolithiasis.

Disorders of the Thyroid Gland

Hypothyroidism

The most common form of hypothyroidism is due to autoimmune (Hashimoto) thyroiditis in which circulating thyroid antibodies damage the thyroid gland over time, impairing its ability to make thyroid hormone. Hypothyroidism may also result from surgical removal of the thyroid or radiation therapy. The symptoms of hypothyroidism can be nebulous and similar to those occurring in natural aging. These include fatigue, cold intolerance, constipation, dry skin and hair, weight gain, and impaired concentration. It is important to consider depression and anemia in these patients as well, since all three conditions share the same symptoms and are commonly diagnosed in the elderly.[16] Measurement of serum thyrotropin (TSH) should be used to screen for an underactive thyroid gland. The anterior pituitary releases TSH in response to decreased circulating levels of free thyroxine (FT4) and free tri-iodothyronine (FT3) inadequately produced by the thyroid gland. Overt hypothyroidism is defined by elevated TSH with frankly low FT4 levels. There has been some debate as to whether the reference range for normal TSH should be adjusted for age. It is well known that TSH increases with each decade of life despite maintenance of circulating thyroid hormone. Some studies have shown that elderly individuals with high TSH and lower FT4 have a prolonged survival, suggestive that changes in thyroid function are adaptive mechanisms to conserve energy expenditure with age.[17] Given these findings, the latest American Thyroid Association (ATA) guidelines suggest raising the target serum TSH to 4–6 uIU/L in persons over 70–80 years old.[18]

Overt hypothyroidism requires treatment with thyroid hormone to alleviate symptoms and prevent progression to myxedema, an endocrine emergency characterized by

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undetectable thyroid hormone levels, obtundation, hypothermia, and decreased cardiac output that can progress to heart failure.[19] Patients with established hypothyroidism should be treated with synthetic levothyroxine (T4), which is peripherally converted in peripheral tissues to T3, the active form of thyroid hormone. Levothyroxine is typically administered as a once-daily dose of 1.6 ug/kg. Elderly persons without known heart disease can be initiated at the full weight-based dose of levothyroxine, though some experts prefer initiating a lower dose and increasing it slowly in this population.[18] Careful titration to avoid iatrogenic thyrotoxicosis is imperative because elderly are more likely to experience adverse effects of thyroid hormone excess such as atrial fibrillation, myocardial ischemia, and osteoporotic fractures.[20] TSH levels should be monitored 6 weeks after initiation of levothyroxine and at 6-week intervals after any dose adjustments. After dose stabilization, TSH levels can be monitored annually.

Subclinical Hypothyroidism

Subclinical hypothyroidism (SCH) is defined by elevated TSH with normal FT4 levels. This condition can occur with the progression of autoimmune disease or temporarily in the setting of thyroiditis. Possible reasons to consider treatment of subclinical hypothyroidism include alleviating hypothyroid symptoms, preventing progression to overt hypothyroidism if antibodies are present, and prevention of adverse events.[21] The TRUST trial, which randomized over 700 men and women 65 years and older with SCH to levothyroxine or placebo, did not show an improvement in symptoms of hypothyroidism with levothyroxine treatment.[22] Further, despite evidence that subclinical hypothyroidism has been associated with increased risk of heart failure, coronary heart disease events, and mortality from coronary heart disease, there is insufficient evidence to suggest that treatment of SCH reduces ischemic heart disease events or all-cause mortality in individuals older than 70 years.[21,23]

Given limited data supporting the treatment of subclinical disease, current guidelines recommend levothyroxine therapy (LT4) only when serum TSH concentrations are ≥10 uIU/mL in the setting of normal FT4.[21] Treatment should be considered in patients with serum TSH concentrations 7.0–9.9 uIU/L on the basis of individual factors, including symptoms, thyroid antibody positivity, or evidence of atherosclerotic cardiovascular disease. Treatment is not recommended when TSH is 4.5–6.9 uIU/L.[21] Treatment of subclinical hypothyroidism usually requires only 25 ug–75 ug of levothyroxine daily; TSH levels should be monitored 6 weeks after initiating therapy. Within an elderly population, the benefits of levothyroxine therapy in SCH are minimal, and there is a higher risk of complications of iatrogenic thyrotoxicosis; for this reason, treatment decisions should be made on an individual basis.

Hyperthyroidism

Hyperthyroidism is defined by low TSH levels and frankly elevated FT4 and/or T3 levels. Elderly patients with hyperthyroidism are more likely to present with cardiovascular symptoms (tachycardia, atrial fibrillation), dyspnea, edema, weakness, and weight loss and are less likely to present with tremor or nervousness. Hyperthyroidism in the elderly is often termed "apathetic," as the classic hyperactive symptoms are frequently absent. Overt hyperthyroidism is associated with increased cardiovascular risk, osteoporosis, and mortality in the elderly. Compared to younger patients, a higher proportion of elderly patients with hyperthyroidism have toxic multinodular goiter. Thyrotoxicosis can also be due to autoimmune stimulation of the thyroid gland (Graves' disease), thyroiditis, or iodine-containing medications (namely amiodarone).[24] Given the broad differential of hyperthyroidism, neck examination is a key component of the physical exam. Multiple thyroid nodules may suggest toxic multinodular goiter, whereas a single thyroid nodule could be an autonomous toxic nodule. Presence of a diffuse goiter may suggest Graves' disease, although many elderly patients with Graves' disease may have a nonpalpable thyroid gland. A tender thyroid gland suggests subacute (granulomatous) thyroiditis.

Neck ultrasound and radioactive iodine (RAI) uptake and scan should be performed in patients with suppressed TSH and nodule(s) on examination. Toxic nodules that appear "hot" on nuclear medicine testing can be definitively treated with radioactive iodine, though thionamide treatment can be temporizing. Nodules seen on neck ultrasound but "cold" on scan should be biopsied to rule out thyroid cancer. If RAI scanning shows diminished radiotracer uptake and poor gland visibility, subacute or subclinical thyroiditis should be suspected. Subacute thyroiditis can occur transiently following an upper respiratory illness and is associated with neck pain. The condition can be treated with NSAIDs and/or a short course of prednisone. Subclinical, painless thyroiditis is thought to be autoimmune mediated and does not typically require therapy. Both subacute and subclinical thyroiditis can be associated with a hypothyroid phase that

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follows hyperthyroidism. In some cases, the hypothyroidism does not resolve and requires lifelong LT4 therapy. As such, TSH and free T4 should be monitored closely every 6 weeks.

Graves' disease will show diffuse RAI uptake and increased vascularity on neck ultrasound. Graves' disease can be initially treated with thionamides for 12–18 months (methimazole, propylthiouracil), and lasting remission can occur in 20–30% of patients. Unless contraindicated, methimazole is preferred over propylthiouracil (PTU), given its once-daily dosing. Starting dose depends on the severity of thyrotoxicosis, but is usually 5–15 mg daily for mild Graves' disease. Beta-blockers are also recommended in Graves' disease, if appropriate. Thyroid function tests with TSH, free T4, and total T3 should be monitored every 6 weeks in Graves' patients, with adjustment of methimazole dose to achieve normal free T4 and total T3 levels and a TSH in the detectable range. If Graves does not go into remission with methimazole treatment for 12–18 months or becomes difficult to control, RAI treatment may be pursued. In cases where RAI is required to manage hyperthyroidism, resultant hypothyroidism should be anticipated and managed accordingly.[24]

Amiodarone is an iodine-containing anti-arrhythmic medication that can cause thyrotoxicosis in one of two ways: amiodarone-induced thyrotoxicosis (AIT) type 1 is iodine-induced and occurs in individuals with autoimmune thyroid disease or thyroid nodules; AIT type 2 is destructive thyroiditis caused by toxic effects of amiodarone on follicular thyroid cells. Thyroid ultrasound with doppler sonography may assist in distinguishing type 1 (increased vascularity) from type 2 (absent vascularity). The treatment is different for the two types. AIT type 1 is treated with thionamides, radioactive iodine, or surgery, if necessary. AIT type 2 is treated with steroids. A baseline TSH should be checked in all patients prior to initiating amiodarone and monitored at least yearly.

Subclinical Hyperthyroidism

Subclinical hyperthyroidism is defined by low TSH levels and normal FT4 and total T3 levels. It is important to distinguish this condition from nonthyroidal illness or "euthyroid sick syndrome," which can be due to chronic or critical illness. Euthyroid sick syndrome is characterized by low or normal TSH, low or normal FT4, and low total T3 and FT3. This condition is common in the elderly and affects up to one third of critically ill hospitalized patients.[25,26] The failure of TSH to rise in response to low thyroid hormone levels in critical illness is due in part to central hypothyroidism from alterations in the hypothalamic-pituitary-thyroid axis. Euthyroid sick syndrome is usually transient and does not require treatment, as the laboratory findings normalize when the underlying illness has resolved.

Many studies have shown that subclinical hyperthyroidism has adverse outcomes including cardiovascular disease (atrial fibrillation, heart failure, coronary heart disease), bone loss, fractures, and dementia, especially in patients 65 years and older.[27] Patients older than 65 years with subclinical hyperthyroidism should be treated if TSH levels are persistently <0.1 uIU/L. If TSH level is between 0.1 and 0.4 uIU/L, treatment should be considered in this age group, especially if there are coexisting conditions such as osteopenia, osteoporosis, or cardiovascular disease.[24]

Thyroid Nodules

Almost 50% of patients >65 years of age have thyroid nodules on ultrasound examination, the prevalence confirmed by autopsy studies. More than 95% of thyroid nodules are benign. Elderly patients with palpable thyroid nodules should be evaluated with thyroid ultrasound, as the incidence of thyroid cancer increases with age. Those with thyroid nodule(s) and suppressed TSH should have 24-hour radioactive iodine uptake and scan (as detailed earlier), to identify hyperfunctioning, "hot" nodule(s). Hyperfunctioning nodules rarely harbor malignancy, and cytologic evaluation is rarely necessary.

Elderly patients with thyroid nodules should be evaluated by an endocrinologist to determine if biopsy with fine-needle aspiration (FNA) is indicated. In general, nodules measuring 1.0 cm or larger with suspicious features (hypoechoic, microcalcifications, increased vascularity, infiltrating margins, etc.) should undergo biopsy. All patients with abnormal cervical lymph nodes detected on exam or ultrasound and thyroid nodule(s) should also undergo FNA. Very low suspicion patterns such as mixed cystic-solid nodules do not require FNA biopsy unless they are 2.0 cm or larger in size; benign patterns such as spongiform do not need to be biopsied. If biopsy reveals a benign nodule or nodules, patients can be followed every 1–2 years with thyroid ultrasound to monitor for changes in size or character, in which case repeat biopsy or surgical removal may be warranted.[28]

Thyroid Cancer

Differentiated thyroid carcinoma has a good prognosis if detected early and treated with thyroidectomy

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(and radioactive iodine, if indicated). However, patients older than 65 often have more aggressive disease with multiple, larger tumors and more advanced-stage disease, nonpapillary histology, and extrathyroidal extension. Anaplastic thyroid carcinoma, which carries the worst prognosis among thyroid cancers, presents almost exclusively after the age of 60 years. If biopsy of a thyroid nodule reveals thyroid cancer cells or findings suspicious for thyroid cancer, lobectomy or total thyroidectomy should be performed. Thyroid lobectomy is preferred for a solitary, cytologically indeterminate nodule and for unilateral intrathyroidal differentiated thyroid cancer measuring <1 cm. Total thyroidectomy is generally recommended if the primary thyroid carcinoma is >4 cm, there are contralateral thyroid nodules present, regional or distant metastases are present, the patient has a personal history of radiation therapy to the head and neck, or the patient has first-degree family history of differentiated thyroid cancer. If the thyroid cancer is >1 cm and <4 cm without extrathyroidal extension or evidence of lymph node metastases, the initial surgery can be either a total thyroidectomy or lobectomy depending on patient preference. Older patients (age >45 years) are recommended to undergo total thyroidectomy given higher recurrence rates in this age group.[28] Age alone is not a contraindication to thyroidectomy. Studies have shown no difference in complication rates (permanent hypoparathyroidism, recurrent laryngeal-nerve palsy, or postoperative bleeding) between older and younger patients who undergo thyroidectomy for thyroid cancer.[29] Treatment decisions including extent of thyroidectomy/neck dissection, radioactive iodine remnant ablation, TSH suppression, and ongoing follow-up should be made by an experienced endocrinologist and thyroid surgeon.

Disorders of the Pituitary Gland

Hypopituitarism

The incidence of hypopituitarism in the adult general population (mean age of diagnosis 50 years; range 18–79 years) has been estimated at 4.2 cases per 100,000.[30] Pituitary adenomas, particularly nonfunctional pituitary adenomas, are the most common cause of hypopituitarism in the elderly, with an incidence of 7–9.9% in this population.[31–33] The incidence of pituitary adenomas in the elderly is increasing, likely related to the increasing frequency of neuroimaging in this population. Pituitary adenomas are nonfunctional in 65–84% of cases, with the most common functional pituitary adenomas secreting growth hormone (GH, 17%), prolactin (4.5–10%), and adrenocorticotropic hormone (ACTH, 0–6%). Gonadotrophic adenomas, which are usually included in the nonfunctional pituitary adenoma group, tend to increase with age, particularly over the age of 50.[34] Other causes of hypopituitarism include nonadenomatous tumors such as craniopharyngiomas, meningiomas, gliomas, and metastases, as well as infiltrative lesions such as hemochromatosis, granulomatous diseases, histiocytosis, and autoimmune lymphocytic hypophysitis. Pituitary apoplexy, surgery, and radiation can result in hypopituitarism up to 10 years following therapy.[35]

Clinical manifestations of either partial or complete hypopituitarism may be nonspecific and attributed to the natural aging process or related comorbidities. Pituitary tumors are incidentally discovered on imaging in approximately 5–15% of elderly patients. Because of the predominance of nonfunctioning pituitary adenomas, clinical symptoms are often associated with local mass effect on the optic chiasm resulting in visual impairment. Because of age-related decline in visual acuity, cataracts, and macular degeneration, the link between visual symptoms and pituitary pathology may be misdiagnosed in up to 20% of cases. Other manifestations of mass effect – including headaches, cranial nerve palsy due to cavernous sinus invasion or pituitary apoplexy, and ophthalmoplegia – are less common.[36]

Symptoms of hormonal deficits are less commonly recognized as a presenting feature of pituitary tumors, largely because of their nonspecific nature and overlap with those related to aging and medical comorbidities. However, with thorough preoperative evaluations, rates of symptoms at presentation can be identified in up to 50% of individuals.[37] Hyponatremia, whether due to age-related changes in vasopressin secretion or adrenal insufficiency, can be a presenting feature of hypopituitarism in up to 9.5% of cases in the elderly.[38] Conversely, diabetes insipidus is unlikely to be due to a pituitary adenoma and is more commonly seen in cases of craniopharyngioma, lymphocytic hypophysitis, infiltrative disease, or pituitary metastases.[37]

Biochemical hormonal assessment with cortisol, IGF-1, free T4, and prolactin levels, as well as neuroradiological imaging with a pituitary-protocol MRI with baseline visual field testing, are important components of the diagnosis of hypopituitarism at any age. However, the diagnosis of pituitary dysfunction in the elderly individual poses specific challenges. There are morphologic changes in the hypothalamus and pituitary in addition

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to age-related declines in GH, androgens, and estrogen that must be considered. Additional illnesses and medications may further confound assessment. Classically, acquired hypopituitarism progresses such that GH deficiency appears first, followed by hypogonadotrophic hypogonadism, and subsequently thyroid and/or adrenal insufficiency.[37]

GH deficiency is diagnosed on the basis of subnormal serum insulin-like growth factor 1 (IGF-1) levels measured against gender- and age-specific norms. GH deficiency is then confirmed with one of a few different provocative tests. In the United States, glucagon stimulation tests are still common; however, in other countries, Arginine-GHRH testing is used. More recently, macimorelin, an agonist of the ghrelin receptor, was developed. It is given orally and results in dose-dependent increases in GH. Eliminating some of the side effects seen with glucagon stimulation testing, the macimorelin test is now preferred; however, cost limits its use in some US centers.[39] In the setting of acromegaly, documentation of elevated IGF-1 levels should be followed by confirmatory oral glucose tolerance testing with measurement of GH levels.[37] Assessment of central hypogonadism in the elderly man is made on the basis of 8 a.m. testosterone levels in the setting of low or inappropriately normal luteinizing hormone (LH) and follicle-stimulating hormone (FSH) levels. Prolactinomas typically present as macroadenomas (>2 cm) in the elderly. There may be associated hypogonadism in men as well as galactorrhea. Prolactin levels may be elevated as a side effect of several medications used commonly in the elderly, including antipsychotic agents. Nonfunctional pituitary adenomas may also result in modest elevations in prolactin levels due to stalk compression. The diagnosis of central hypothyroidism can be made on the basis of low plasma-free T4 levels in the setting of TSH levels in the low or inappropriate-normal range. Adrenal insufficiency is diagnosed based on low morning serum cortisol levels with confirmatory subnormal response to the 250 mcg ACTH stimulation test (<18 mcg/dL).

When selecting the appropriate treatment for pituitary tumors, whether surgical or medical, it is important to consider the impact of mass effect, hormonal deficits, and hormonal excess. Transsphenoidal surgery is regarded as both safe and successful in the elderly in the hands of an experienced neurosurgical team. Studies show no increase in perioperative morality or severe anesthesiologic complications in individuals aged 65 and older. The most frequent postoperative complications include diabetes insipidus and cerebrospinal fluid leak.[33,40] Transsphenoidal surgery is also the treatment of choice for elderly patients with visual disturbances due to nonfunctioning pituitary adenomas. More than 70% of individuals will demonstrate postoperative visual improvement, although a small percentage may develop visual deterioration due to hemorrhagic or ischemic damage.[33] The indication for surgical intervention in nonfunctional sellar masses without visual impairment is less clear. Conservative interval monitoring with laboratory studies and imaging may be a reasonable option and should be considered in those at high surgical risk.[37]

Hormonal replacement in elderly patients with hypopituitarism can be complicated and requires addressing each of the impaired axes. For ACTH deficiency, cortisol replacement regimens must be tailored to achieve hemodynamic stability while avoiding overtreatment that could adversely impact bone health and metabolism. It is of paramount importance to ensure that individuals with ACTH deficiency wear a medical alert tag and are aware of the need to contact their physician for dose adjustments in acute illness or prior to invasive procedures. In the elderly, thyroid hormone should be started at a low dose and uptitrated gradually to a goal of 1.3 +/– 0.2 mcg/kg/day.[41] In hypopituitarism, dosing is titrated to free T4 levels and not to TSH levels. Like over-replacement of glucocorticoid, care needs to be taken to avoid over-replacement of thyroid hormone to avoid impairing bone health and increasing the risk of atrial fibrillation. Further, prior to initiating thyroid hormone replacement, central adrenal insufficiency should be identified and treated to avoid precipitating an adrenal crisis.

Indications for replacement of testosterone and growth hormone in cases of hypopituitarism are less well established, particularly in the elderly population. Testosterone replacement therapy has important implications for maintaining lean body mass, bone mineralization, erythropoiesis, and sense of well-being. However, there have been retrospective studies that have revealed a possible link between testosterone replacement and increased cardiovascular risk in older men. While in totality studies have been equivocal, the Endocrine Society has issued statements regarding possible concerns about testosterone therapy and cardiovascular outcomes.[42] Testosterone initiations should take place after carefully assessing the risk–benefit ratio for the individual in question. Deficiency in growth hormone is associated with visceral obesity, insulin resistance with impaired glucose tolerance, and dyslipidemia. In the absence of contraindications such as malignancy or proliferative diabetic retinopathy, GH replacement may be beneficial in select cases. Lower-dose therapy is typically

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required in the elderly, and IGF-1 levels should be closely monitored.[36,38]

Ectopic ACTH Production

Clinical assessment, physical exam, and laboratory findings suggestive of Cushing's syndrome in the absence of a pituitary or adrenal adenoma are rare in the elderly. Similar to Cushing's syndrome of other etiologies, the most common clinical manifestations of ectopic ACTH production include muscle weakness, change in body weight, hypertension, hirsutism, low bone mineral density, and hypokalemia.[43] Distinguishing ectopic ACTH secretion from Cushing's disease can be quite difficult. Typically, the high-dose dexamethasone suppression test is the first-line test, followed by intrapetrosal sphenoid sinus sampling. Identifying the source of ACTH secretion can be equally challenging. Bronchial carcinoid tumors, small cell lung carcinoma, and gastroenteric pancreatic tumors may all secrete ACTH. Because pulmonary tumors are often a source of ectopic ACTH production, CT chest is the most useful form of imaging, with either CT or MRI of the abdomen/pelvis as the next step. Treatment includes surgical removal of the tumor and/or medical therapy.[44]

Disorders of the Adrenal Glands

Adrenal Nodules

Among one of the more common "incidentalomas," adrenal nodules are often detected in imaging tests ordered for the workup of other conditions. The prevalence of adrenal nodules increases with age, ranging from 0.2% among individuals 20–39 years of age to 7% in individuals over the age of 70.[45] Although often benign, adrenal nodules may be functional and/or malignant. Thus, further evaluation of the incidentaloma is often warranted. In the general population, nonfunctional adenomas are the most common (80%), followed by cortisol-producing tumors (Cushing's syndrome), pheochromocytomas, aldosteronomas, adrenocortical carcinoma, and metastatic disease.[46–48]

Important diagnostic information may be gleaned from adrenal protocol CT. Features suggestive, but not diagnostic, of malignancy include size >4 cm, irregular borders, lack of homogeneity, calcifications, and washout of contrast after 10 minutes less than 50%. Unless CT findings are clearly characteristics of benign findings such as a myelolipoma or cyst, lesions ≥4 cm should be resected to rule out adrenal cortical carcinoma.[46,49,50] In general, FNA of adrenal nodules is recommended only if there is high suspicion for metastatic disease or infection. Biochemical assessment must always be completed first, as FNA of a pheochromocytoma can result in hemorrhage and hypertensive crises.[50] For those lesions less than 4 cm, guidelines from the American Association of Clinical Endocrinologists and the American Association of Endocrine Surgeons recommend that patients with an adrenal incidentaloma undergo clinical and biochemical evaluation for hyperaldosteronism (if hypertensive), as well as for the presence of a pheochromocytoma and hypercortisolism.[46] The evaluation of each of these entities will be described in further detail.

Primary Aldosteronism

All patients with hypertension and adrenal nodules should be screened for primary aldosteronism (PA). PA encompasses a group of disorders characterized by inappropriately high and relatively autonomous aldosterone production that is nonsuppressible by sodium loading. PA is most commonly caused by an adrenal adenoma, unilateral or bilateral adrenal hyperplasia, or, less commonly, glucocorticoid-remediable aldosteronism.[51] Diagnosis of PA has important implications for patients, as evidence suggests that these individuals have higher cardiovascular morbidity and mortality than age- and sex-matched patients with essential hypertension and the same degree of blood pressure elevation.[51–53] Although a minority of patients present with hypokalemia, normokalemic hypertension is the most common presentation of hyperaldosteronism.[54] Screening for PA includes measurement of plasma renin activity (PRA) and aldosterone levels. An aldosterone level of 15 ng/dL or greater in conjunction with a plasma aldosterone to PRA ratio of 20 or greater is suggestive of aldosterone excess. Unfortunately, there are many medications that can confound laboratory studies. Patients should be taken off of spironolactone, epleronone, amiloride, triamterene, potassium-wasting diuretics, and products derived from licorice root for at least 4 weeks prior to testing. Additional antihypertensives such as angiotensin-converting enzyme inhibitors and angiotensin receptor blockers may make the diagnosis more difficult and require careful clinical judgment.[51,52] Confirmatory testing is recommended with aldosterone-suppression testing using oral sodium loading, saline infusion, fludrocortisone suppression, or captopril challenge.

Given the high incidence of adrenal nodules in the elderly, all individuals older than 40 years of age should undergo adrenal vein sampling if surgical intervention is

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considered to confirm laterality. In individuals with evidence of unilateral PA, whether it is due to an aldosterone-producing adenoma or unilateral adrenal hyperplasia, laparoscopic adrenalectomy should be offered to all patients after a careful consideration of surgical risks. Nonsurgical candidates are best served with a mineralocorticoid receptor antagonist such as spironolactone or eplerenone. After surgery, approximately 50% of individuals maintain blood pressure <140/90 mmHg without antihypertensive drugs. However, among the most common reasons for persistent hypertension after adrenalectomy are older age and longer duration of disease.[51]

Pheochromocytoma

In the general population, pheochromocytomas constitute approximately 4–7% of adrenal incidentalomas.[49] Despite textbook symptomatology, only 10% of patients 12–85 years of age will present with the classic triad of headaches, sweating, and palpitations; further, 12.5% patients are normotensive at the time of diagnosis.[55] Other less common signs and symptoms of pheochromocytomas include tremors, pallor, dyspnea, generalized weakness, anxiety attacks, orthostatic hypotension, visual blurring, papilledema, weight loss, polyuria, polydipsia, constipation, and psychiatric disorders. However, older patients with pheochromocytoma may be less likely to experience classic symptoms of sympathetic overactivity and catecholamine excess.[56] This may be attributed to decreased baroreceptor function and slowed responsiveness to catecholamines with aging, as well as concurrent comorbidities and medications. Catecholamine excess leading to myocardial injury and cardiomyopathy are of particular concern in this vulnerable age group.[56,57]

Appropriate screening includes measurement of plasma-free metanephrines as well as 24-hour urinary metanephrines and normetanephrines. Management is surgical, but should be postponed until liberalization of fluid and salt intake and initiation of alpha-adrenergic blockade for at least 1–2 weeks preoperatively to minimize the risk of intraoperative hemodynamic instability. Either phenoxybenzamine or doxazosin is appropriate, with titration to the goal of normotensive blood pressure readings and mild orthostasis. Beta-blockade may be initiated only after adequate alpha-blockade if the patient demonstrates persistent tachycardia or arrhythmias. Patients require close hemodynamic monitoring intra- and postoperatively.[58]

Cushing's Syndrome

Patients with cortisol-producing adrenal adenomas, Cushing's disease, or ectopic ACTH secretion may present with either subclinical or overt Cushing's syndrome. The scope of this section will be limited to cortisol-producing adrenal adenomas. Of the functional adrenal incidentalomas, cortisol production is the most common at approximately 5.3%.[47] Symptoms of Cushing's syndrome may overlap with those seen in normal aging to include truncal weight gain, sarcopenia, hypertension, glucose intolerance, fatigue, depression, sleep disturbances, easy bruisability, or osteoporosis. The Endocrine Society guidelines suggest that the diagnosis of Cushing's syndrome is likely if two out of the three following tests are positive: 24-hour urine-free cortisol, late night salivary cortisol, and dexamethasone suppression (1 mg overnight or 2 mg two-day test).[59] An important consideration in the elderly population when selecting appropriate testing is how concomitant medications will affect cortisol levels and rate of clearance of dexamethasone. For example, medications such as phenobarbital, phenytoin, carbamazepine, primidone, rifampin, and pioglitazone may accelerate the metabolism of dexamethasone. In contrast, aprepitant/fosaprepitant, fluoxetine, and dilitazem may decrease dexamethasone metabolism. Similarly, carbamazepine and fenofibrate are examples of medications that may affect 24-hour urinary cortisol results.[59]

In the appropriate candidate, surgical management of a cortisol-secreting adenoma involves laparoscopic adrenalectomy. Postoperatively, all patients require initiation of steroid replacement therapy until the hypothalamus-pituitary-adrenal (HPA) axis recovers. For those with multiple comorbidities, the benefit of surgery may not outweigh the associated risks. In these cases, medical therapy for Cushing's syndrome will require treating the end-organ damage associated with cortisol excess (i.e., increased bone resorption, hypertension, etc.). The degree of cortisol secretion from the adenoma may also be tempered with medications and should be instituted in conjunction with an experienced endocrinologist.

Adrenal Insufficiency

Adrenal insufficiency (AI) may be broadly categorized as primary or secondary, depending on whether disease originates in the adrenal glands (primary) versus the hypothalamus or pituitary (secondary); the diagnosis of primary adrenal insufficiency peaks in the fourth decade of life, and secondary adrenal insufficiency in the sixth

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decade.[60] AI, irrespective of etiology, is up to six times higher in the elderly, possibly related to decreased responsiveness of the HPA axis.[61] The most common cause of secondary adrenal insufficiency is iatrogenic, with longstanding steroid use (systemic, injections, topical) the most frequently encountered. Additionally, megestrol acetate, ketorolac, and opiates, as well as certain antipsychotics and antidepressants, can cause secondary AI. Though less commonly observed in the elderly, primary disease may occur as a result of anticoagulants precipitating bilateral adrenal hemorrhage. Other broad, but important, etiologic categories of primary AI include infection (HIV-1, CMV, tuberculosis, candidiasis, and histoplasmosis), metastatic disease, infiltrative disorders (amyloidosis, hemochromatosis, and sarcoidosis), infarct due to coagulation disorders, and autoimmune disease.[62]

AI may be a difficult clinical diagnosis to make in the elderly given that symptoms of chronic adrenal insufficiency may be nonspecific, classically including weakness, fatigue, anorexia, abdominal pain, nausea, vomiting, constipation or diarrhea, postural dizziness, myalgias, and arthralgias. Altered sensorium, abdominal pain, and fever together with severe hypotension may be seen in the setting of adrenal crisis, a medical emergency.[60–63] In the absence of exogenous steroid administration, basal cortisol levels of ≥15 mcg/dL–19 mcg/dL rule out AI, whereas 8 a.m. cortisol levels ≤3 mcg/dL are virtually diagnostic and are made using ACTH testing. As noted briefly earlier, for this stimulation test, serum cortisol is measured prior to and 60 minutes following an intramuscular injection of cosyntropin 250 mcg. A serum cortisol peak greater than 18 mcg/dL indicates an appropriate response.[62] However, mild adrenal insufficiency may be missed by the 250-mcg test; if suspicion for AI is high, a 1-mcg cosyntropin test may be a more sensitive measure to establish the diagnosis of AI.[62–64]

Treatment for AI includes glucocorticoid replacement, generally in the form of hydrocortisone. To mimic physiologic secretion of cortisol, hydrocortisone is taken twice daily with half to two thirds of the total dose (generally 15 mg–25 mg) given in the morning and the remaining dose mid-afternoon. Mineralocorticoid replacement, necessary only in primary adrenal insufficiency, includes fludrocortisone 0.05 mg–0.2 mg in the morning with optional dehydroepiandrosterone replacement (25 mg–50 mg).[62,63] In contrast, adrenal crisis should be managed with IV fluid resuscitation and immediate IV hydrocortisone at a dose of 100 mg followed by 100 mg–200 mg every 24 hours with close cardiac monitoring.[62] If there is concern for coexisting hypothyroidism, glucocorticoid replacement should precede levothyroxine therapy.

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Chapter 32

Diabetes Mellitus

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Introduction

Diabetes mellitus is a dominant chronic disease in the older adult population in the United States and around the world. The prevalence of diabetes is expected to grow with the increase in the population of adults aged 65 and over, the prevalence of obesity, and physical inactivity. Clinicians are faced with many unique challenges when caring for this older diabetic population. The clinician's major challenges are (1) to avoid symptoms and complications of hyper- and hypoglycemia, (2) to minimize or delay micro- and macrovascular complications, if possible, and (3) to maximize daily functioning. Underlying these challenges is the realization that the geriatric population is a heterogeneous one. Goals of care and treatment decisions may vary, depending more on the patient's functional abilities and on other comorbidities or coexisting geriatric syndromes, and less on the age of the patient. This chapter will focus on specific aspects of diabetes care in the older adult.

Epidemiology

An estimated 14.3 million people or 26.8% of those 65 years of age or older in the USA are afflicted with diabetes (combined diagnosed and undiagnosed), the majority of whom have type 2 disease.[1] Almost 42% of adult Americans with diabetes are 65 years of age or older, with an approximately even split between men and women. Diabetes is more prevalent in minority groups. After adjusting for population age differences, Native Americans are twice as likely, non-Hispanic Blacks 1.6, Hispanics 1.7, and Asian Americans 1.2 times as likely to have diabetes as non-Hispanic Whites. The direct costs of medical care are great with almost $237 billion being spent in 2017, about 61% of it on older adults with diabetes.[2] The majority of this expense is for medications ($49.5 billion) ahead of inpatient costs ($44.9 billion). Even after adjusting for inflation, compared to costs in 2012, costs for insulin and other antihyperglycemics increased by 45%. The absolute number of older persons with diabetes is expected to rise for at least three reasons: (1) older adults are more likely to develop diabetes, (2) there will be an increased percentage of the population of older adults in minority groups, and (3) older adults with diabetes are living longer.[3] The most rapid rate of rise of the diabetes burden is expected to be in the population of adults 75 years and older. If detection improves, the number of older adults with clinically recognized diabetes may rise even further, since 21% of patients of all ages with diabetes were estimated to be undiagnosed in 2018.[1]

Diagnosis

Although the "poly" symptoms (polydipsia, polyuria, and polyphagia) are considered to be pathognomonic of diabetes, this is often not true in older persons for several reasons. First, these symptoms are nonspecific and may be due to other conditions, such as urinary difficulties or diuretic use. Second, they may not be present because of age-related or disease-related changes in organ function. For example, thirst mechanisms often become impaired with age. Third, they may be masked by other conditions. Thus, relying on them will result in both false positives and false negatives. The challenge to the clinician is to maintain a high level of suspicion yet be prudent with glucose testing.

Because of consistent correlation between hemoglobin A1c (A1c) and complications related to diabetes and improved standardization of the A1c assay, the 2009 International Expert Committee recommended that the A1c assay supplant glucose testing as the first-line diagnostic test for diabetes. The current American Diabetes Association (ADA) criteria for the diagnosis of diabetes are: (1) A1c ≥6.5% *or* (2) a fasting plasma glucose level of >126 mg/dL *or* (3) an elevation in plasma glucose to ≥200 mg/dL 2 hours following a 75-gram oral glucose tolerance test *or* (4) symptoms of diabetes and a random glucose level of ≥200 mg/dL.[4] The diagnosis should be

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confirmed by a second test on a different day unless the patient has obvious hyperglycemia.

Most older diabetics are classified as either type 1 or type 2. Type 1 diabetics require insulin and are ketosis prone. Type 2 diabetics are insulin resistant and ketosis resistant. Many type 1 diabetics are living to older ages. In addition, insulin-deficient diabetes can develop from viral or autoimmune causes or pancreatic trauma, therefore when caring for a diabetic patient, it is important to establish when and how the diagnosis of diabetes was made in order to institute proper therapy and to anticipate the types of complications that are likely to develop.

Management

To the older adult, receiving the diagnosis of diabetes may evoke multiple emotions including dread, fear, and sadness. Not only are complications devastating, but following complex dietary, medication, and monitoring regimens can be overwhelming. Daily functional, nutritional, and medical assistance from professional and lay caregivers is often necessary when patients are physically and/or cognitively impaired. When developing a treatment plan with the older diabetic patient, it is important to customize the plan to the individual and involve the patient in his or her own self-care, to the extent that this is possible, and to be sensitive to the patient's perception of his or her quality of life, as it is affected by various therapeutic interventions.

Collaborative management is a conceptual model to better care for patients with chronic diseases such as diabetes. Collaborative management consists of: (1) defining problems from the perspective of the patient and physician and the health-care team, (2) targeting key problems, goal setting, and planning methods to achieve goals, (3) creating patient education and support services, and (4) evaluating patient progress in a frequent and regular follow-up plan.[5] It is important for all the members of the health-care team to provide as much education to the patient as possible in order to engage the patient as an active participant in his or her own management. Evidence suggests that interventions targeted at improving the diabetes care delivery system and promoting diabetes self-management lead to improved patient outcomes and metabolic control.

In 2003, the first guidelines for improving the care of the older adult with diabetes were created by the California Health Care Foundation (CHCF) and the American Geriatrics Society (AGS). These published guidelines stressed the importance of setting individualized goals of care using the best evidence available for this very heterogeneous group. In 2012, the ADA convened a Consensus Development Conference on Diabetes and Older Adults to address questions about diabetes care in those adults ≥65 years old for which evidence from clinical trials is often lacking.[6] The guidelines also included recommendations for individualizing management of diabetes in the older adult taking into account eight geriatric syndromes, or conditions, for which there is evidence or strong consensus opinion that persons with diabetes are at greater risk. The conditions include cognitive dysfunction, functional impairment, falls and fractures, polypharmacy, depression, vision and hearing impairment, pain, and urinary incontinence. Most recently in 2013, the American Geriatrics Society Expert Panel on the Care of Older Adults with Diabetes Mellitus updated the previous CHCF/AGS guidelines from 2003.[7]

The remaining sections of this chapter will elaborate on diet and exercise, blood sugar targets and glycemic control, pharmacologic therapy, monitoring, managing cardiovascular risk factors, eye care, lower-extremity complications, nephropathy screening, cognitive function, and family considerations. More specific information on the screening for and management of the above related geriatric syndromes can be found in other chapters of this book.

Diet and Exercise

In general, diabetes is closely related to being overweight or obese, although a subset of older patients is either of normal weight or underweight. What constitutes an optimal diabetic diet for older persons has not yet been determined, but weight loss, even if modest, in obese older persons can improve metabolic control, thereby reducing symptoms of hyperglycemia.

Weight loss in obese diabetics should be attempted to improve insulin sensitivity and reduce the need for medications. A registered dietitian should be an active member of the diabetes management team to assist the patient and/or caregiver in creating an individualized diet plan. While a recent systematic review was unable to find strong evidence for an ideal percentage of carbohydrates, fats, and proteins in an individual's diet with diabetes,[8] calorie consumption should be consistent with weight management goals. When weight loss is not possible, the best strategy for many patients should focus on achieving a balanced diet of all three macronutrients that includes high-fiber unprocessed carbohydrates, more unsaturated fats than saturated or trans fats, and

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leaner meats and meat alternatives for protein. Adequate fluid intake of non-sucrose-containing beverages is also important. This alone may help to reduce glucose levels and will correct mild volume contraction related to osmotic diuresis. Older diabetics with frailty or living in long-term care facilities must have diets appropriate to prevent or correct malnutrition, necessitating less restrictive diets. It is important for patients to enjoy mealtimes to satisfy nutritional needs as well as positively contributing to their quality of life.

Weight maintenance and glycemic control may be added benefits of regular exercise, specifically with resistance training. Physical activity has been found to increase insulin sensitivity of muscle and other tissues that have insulin receptors. Other cardiovascular risk factors, e.g., hyperlipidemia and hypertension, may be reduced by regular exercise as well. Self-esteem, risk of falls, and quality of life may also improve. However, exercise in the older diabetic may not be without risk. Exercise can exacerbate angina or ischemia in a patient with underlying cardiovascular disease (CVD). The presence of peripheral neuropathy may result in soft-tissue or musculoskeletal injuries. Symptomatic hypoglycemia can occur, especially in patients taking oral hypoglycemic drugs. Despite these possible associations, there is no evidence supporting prescreening older adults receiving diabetic care prior to beginning a low- to moderate-intensity physical activity program "not exceeding the demands of brisk walking or everyday living."[9]

Blood Sugar Targets and Glycemic Control

Establishing individualized goals of therapy are of great importance when treating older adults with diabetes. In certain patients, such as the frail, demented nursing home resident with sporadic eating habits, controlling symptoms of hypoglycemia or hyperglycemia is more important than preventing macrovascular and microvascular complications of diabetes. Other older, more active patients with longer life expectancies may benefit from tighter glucose control. The benefits of improved glycemic control in reducing microvascular complications of diabetes, such as retinopathy and nephropathy, are seen at approximately 8 years, according to the United Kingdom Prospective Diabetes Study (UKPDS), which excluded participants ≥65 years.[10]

To date, there is limited evidence from clinical trials that establishes recommendations for glycemic targets in adults ≥75 years. While no independent effect of intensive glycemic control on cardiovascular events has been shown in randomized controlled trials, several cohort studies have examined the association between A1c values, mortality, and cardiac events in adults with type 2 diabetes ≥50 years.[11,12] It appears that there is a U-shaped curve with lower mortality between 6.0% and 9.0% compared to ≤6.0% and ≥11%. Therefore, setting glycemic targets for individual patients remains prudent. Glucose target setting must take into account avoiding hypoglycemia, which is associated with both acute and chronic complications such as falls, poor cognitive function, and increased risk of death due to cardiovascular causes. Poor glycemic control with glucose levels persistently above 200 mg/dL can lead to dehydration, nocturia, poor wound healing, fatigue, urinary tract infections, dizziness, and falls. Patients may require admission to the hospital for hyperglycemic crisis, a diagnosis that carries a higher mortality in older adults than it does for younger individuals.

Several societies have provided guidance in establishing targets in the elderly. The 2013 update of the AGS Expert Panel on the Care of Older Adults with Diabetes Mellitus recommends A1c values between 7.5% and 8.0%. For older adults with "few comorbidities and good functional status," A1c values of 7.0–7.5% may be appropriate, while for those with "multiple comorbidities, poor health, and limited life expectancy," A1c values of 8.0–9.0% are acceptable.[7] The ADA recommends for healthy elderly persons a goal A1c of <7.5%, for those with multiple comorbid illness and mild to moderate cognitive impairment a goal of <8%, and for those very poor health, moderate to severe cognitive impairment, and end-stage chronic illness a goal A1c of <8.5%. In 2019 the Endocrine Society also released guidelines that recommended tailoring glycemic control to a patient's health and functional status (Table 32.1).[13]

Patients receiving palliative or end-of-life care will often require less medication. In discussion with the patient and caregivers, goal setting around symptom management can focus on avoiding hypoglycemia and severe hyperglycemia and simplifying treatment regimens to preserve comfort. This often involves de-escalation of medications and limiting insulin usage, though basal insulin may be appropriate.

Pharmacologic Therapy

Drug therapy is warranted if the combination of diet, exercise, and weight loss is not successful in reaching glycemic control and if benefits outweigh potential risks of treatment. Multiple oral agents are currently available

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Table 32.1 Conceptual framework for determining blood glucose targets in adults aged 65 years and older

Overall Health Category		Group 1: Good Health	Group 2: Intermediate Health	Group 3: Poor Health
Patient characteristics		No comorbidities **or** 1–2 non-diabetes chronic illnesses* **and** No ADL€ impairments and ≤1 IADL impairment	3 or more non-diabetes chronic illnesses* **and/or** Any one of the following: mild cognitive impairment or early dementia; ≥2 IADL impairments	Any **one** of the following: End-stage medical condition(s)**; Moderate to severe dementia; ≥2 ADL impairments; Residence in a long-term nursing facility
		Reasonable glucose target ranges and HbA1c by group		
		Shared decision-making: individualized goal may be lower or higher		
Use of drugs that may cause hypoglycemia (e.g., insulin, sulfonylurea, glinides)	No	Fasting: 90–130 mg/dL Bedtime: 90–150 mg/dL; <7.5%	Fasting: 90–150 mg/dL Bedtime: 100–180 mg/dL; <8%	Fasting: 100–180 mg/dL Bedtime: 110–200 mg/dL; <8.5%¥
	Yes£	Fasting: 90–150 mg/dL Bedtime: 100–180 mg/dL; ≥7.0 and <7.5%	Fasting: 100–150 mg/dL Bedtime: 150–180 mg/dL; ≥7.5 and <8.0%	Fasting: 100–180 mg/dL Bedtime: 150–250 mg/dL; ≥8.0 and <8.5%¥

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Note: While glucose targets are highlighted for each group in this framework, overall health categories can also be considered for other treatment goals such as blood pressure and dyslipidemia.

* Coexisting chronic illnesses may include osteoarthritis, hypertension, chronic kidney disease stages 1–3, or stroke, among others.

** One or more chronic illnesses with limited treatments and reduced life expectancy, These include metastatic cancer, oxygen-dependent lung disease, end-stage kidney disease requiring dialysis, and advanced heart failure.

£ As long as achievable without clinically significant hypoglycemia; otherwise, higher glucose targets may be appropriate. Note also that the lower HbA1c boundary was included, as data suggesting increased hypoglycemia and mortality risk at lower HbA1c levels are strongest in the setting of insulin use. However, the lower boundary should not reduce vigilance for detailed hypoglycemia assessment.

¥ HbA1c of 8.5% correlates with an average glucose level of approximately 200 mg/dL. Higher targets than this may result in glycosuria, dehydration, hyperglycemic crisis, and poor wound healing,

€ ADLs include bathing, dressing, eating, toileting, and transferring, and IADLs include preparing meals, shopping, managing money, using the telephone, and managing medications.

Includes data from Cigolle CT, Kabeto MU, Lee PG, Blaum CS. Clinical complexity and mortality in middle-aged and older adults with diabetes. *J Gerontol A Biol Sci Med Sci*. 2012; 67(12):1313–1320 (39); and from Kirkman MS, Jones Briscoe V, Clark N, et aJ. Diabetes in older adults. *Diabetes Care*. 2012; 35(12):2650–2664 (40).

Abbreviations: IADL, instrumental activity of daily living; ADL, activity of daily living; SU, sulfonylurea.

and will be discussed below. Oral agents are easy to use and are frequently preferred by patients but vary significantly in cost and side-effect profiles (Table 32.2). For reasons of low tolerability and limited effects on A1c, alpha-glucosidase inhibitors are not often used in older adults. Pramlintide, an amylin mimetic, is administered subcutaneously before meals, but will also not be discussed below given its limited use.

Biguanides. The only biguanide available in the United States is metformin, which is considered initial therapy in type 2 diabetes. Metformin is a unique treatment for

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Table 32.2 Selected oral hypoglycemic agents for use in older adults

Class	Medication	Dosage schedule	Mechanism of action	Side effects
Biguanide	Metformin (Glucophage®)	Two or three times daily	Decreases glucose release from the liver	Bloating, gas, dyspepsia, loss of appetite in first few weeks. Not for use if significant liver or kidney problems. Will not cause hypoglycemia. May help with weight loss.
Sulfonylurea	Glipizide (Glucotrol®), Glyburide (Micronase®, Diabeta®), Glimepiride (Amaryl®)	Once or twice daily	Stimulates pancreas to secrete more insulin	Hypoglycemia, sometimes rash or dyspepsia, weight gain.
Meglitinide	Nateglinide (Starlix®), Repaglinide (Prandin®)	Rapid onset of action and short-acting, so must be taken before each meal two, three, or four times daily	Stimulates pancreas to secrete insulin after a meal	Hypoglycemia, but less likely than sulfonylureas.
Thiazolidinediones	Pioglitizone (Actos®), Rosiglitazone (Avandia®)	Once or twice daily	Sensitizes tissues to insulin	Fluid retention, so increased risk of heart failure, macular edema, bone loss in women, weight gain. Avoid use if severe heart or liver failure.
Dipeptidyl peptidase-4 (DPP-4) inhibitors	Sitagliptin (Januvia®), Saxagliptin (Onglyza®), Linagliptin (Tradjenta®), Alogliptin (Nesina®)	Once daily	Increases insulin secretion after a meal	Dyspepsia, diarrhea, pharyngitis, nasal congestion. Will not cause hypoglycemia.
GLP-1 Receptor Agonists	Exenatide (Byetta®), Exenatide LAR (Bydureon®), Liraglutide (Victoza®), Dulaglutide (Trulicity®), Lixisenatide (Adlyxin®), Semaglutide injectable (Ozempic®), Semaglutide oral (Rybelsus®)	Varied injectable forms from twice daily, daily, or weekly	Glucose-dependent action to stimulate insulin secretion. Slows gastric emptying, suppresses glucagon	Nausea, vomiting, diarrhea. Will not cause hypoglycemia. Limited study with very low glomerular filtration rate. Contraindicated for gastroparesis, increased risk of medullary thyroid cancer.
Sodium-glucose cotransporter-2 (SGLT2) inhibitors	Canagliflozin (Invokana®), Dapagliflozin (Farxiga®), Empagliflozin (Jardinance®) Ertugliflozin (Steglatro®)	Once daily	Blocks reuptake of glucose by kidneys, thereby increasing loss of glucose in the urine	Potential dehydration, vaginal and penile yeast infections, urinary tract infections.
Glucosidase inhibitors	Acarbose (Precose®), Miglitol (Glyset®)	Short-acting, so must be taken with each meal	Slows absorption of carbohydrates	Bloating, gas, diarrhea, abdominal pain in first few weeks of use, may cause elevations of transaminases.
Pramlitide	Symlin®	Injection before each meal	Decreases postprandial glucagon	Hypoglycemia, dizziness, nausea, vomiting.

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diabetes in that it suppresses hepatic glucose production and improves insulin sensitivity to promote cellular glucose uptake. Therefore, the drug alone does not cause hypoglycemia. It also has a positive effect on lipids by lowering triglycerides and low-density lipoprotein (LDL) cholesterol, does not contribute to weight gain in the obese patient, and may even assist with weight loss. Data from the UKPDS showed a decrease in macrovascular complications of diabetes and in overall mortality in obese patients with newly diagnosed diabetes taking metformin independent of glucose control compared with patients on only dietary changes and with patients on a sulfonylurea after 10 years of follow-up.[14]

Given the cardiovascular benefits and side-effect profile that does not include hypoglycemia, metformin is an excellent first choice for the management of diabetes in the older adult. While age should not be a contraindication to the use of metformin, renal function should be monitored closely during treatment. It should probably be avoided in patients with conditions associated with renal insufficiency (e.g., hepatic or cardiac failure) and in patients with renal failure who are more susceptible to lactic acidosis. Current United States Food and Drug Administration (FDA) guidelines do not recommend initiation of metformin in patients whose estimated glomerular filtration rate (GFR) (eGFR) is less than 45 mL/min/1.73 m^2. For patients who are taking metformin and whose GFR falls below 45 mL/min/1.73 m^2, the risks and benefits of continuing the drug should be considered. Metformin is contraindicated in patients with an eGFR <30 mL/min/1.73 m^2. Drug clearance decreases with increase in age independent of renal function, so low to moderate doses, 500–2,000 mg/day, should be used in older adults. Side effects include nausea, vomiting, anorexia, and diarrhea, most of which resolve within a few weeks and can be avoided with slowly titrating up the dose.

Contraindications to the use of metformin are drug hypersensitivity, metabolic acidosis, and renal impairment as above. The drug should be stopped 2 days prior to radiological procedures involving contrast dyes. The most serious potential side effect of metformin is lactic acidosis, but this is a much less common problem than with the biguanide phenformin, which is no longer available.

Sulfonylureas. The sulfonylurea drugs are the most frequently prescribed. They are generally efficacious, especially in patients who are not obese and within the first 2–5 years after diagnosis. Given that their mechanism of action is to stimulate the beta cells to produce more insulin, their loss of efficacy with time is probably due to a progressive diminution in pancreatic beta cell function. The sulfonylureas may contribute to modest weight gain probably due to the effect of increased circulating insulin. All sulfonylureas are now available in generic form and are reasonably priced. Use of the long-acting sulfonylureas, glyburide and chlorpropamide, should be avoided in older adults because of the risk of prolonged hypoglycemia.[15] Glipizide is preferable because it does not have any active metabolites and has a half-life of 2–5 hours. It must be taken 30 minutes before meals so as not to delay absorption.

Melitinides. Nateglinide and repaglinide act similarly to the sulfonylureas in stimulating the pancreatic insulin release, but are very short-acting, with half-lives of 1–1.5 hours, and for this reason are less likely than sulfonylureas to cause hypoglycemia. These agents can be used when patients have erratic eating habits, such as those with dementia. The medication can be administered with a meal or held if the meal is not eaten. However, dosing schedules of two or three times daily may be burdensome.

Thiazolidinediones (TZDs). Rosiglitizone and pioglitizone are peroxisome proliferator-activated receptor-gamma (PPAR-γ) agonists. Activation of PPAR-γ receptors regulates gene transcription involved in lipid metabolism and in glucose production, transport, and utilization, thus decreasing peripheral insulin resistance. The pharmacokinetics of these drugs do not appear to be altered by age. They have been shown to significantly improve glucose control in older patients with diabetes.[16] These drugs do increase the risk for fluid retention and should not be used in patients with New York Heart Association Class III or IV heart failure. Serum transaminase levels should be checked at the start of therapy, and periodically thereafter. Therapy should be stopped if the patient exhibits signs or symptoms of liver disease or if the transaminases are elevated to three times normal.

There has been some controversy as to the safety of the TZDs. The TZDs have been associated with macular edema, bone loss, and increased fracture risk in women. Concern was raised about rosiglitazone potentially increasing the risk of myocardial infarction and death from cardiovascular causes in a meta-analysis of 42 randomized controlled trials.[17] For several years the FDA restricted prescribing this medication, but in 2015, after further data review, the agency concluded that it did not increase risk, and the restrictions were lifted. Pioglitazone, on the other hand, may have a protective effect in reducing the risk of myocardial infarctions in patients with diabetes and existing macrovascular disease.[18]

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Dipeptidyl peptidase-4 (DPP-4) inhibitors. These oral antihyperglycemic agents increase levels of incretin, an intestinal hormone that inhibits glucagon release and stimulates insulin, thus lowering blood glucose levels. While expensive, they have an intermediate effect on A1c levels but low risk of hypoglycemia and are well tolerated, giving them advantages for use in the frail elderly. Furthermore, they are one of the few oral agents that is safe to use even in advanced renal disease. In 2013, there were reports of pancreatitis and pancreatic cancer in patients taking DPP-4 inhibitors and GLP-1 receptor agonists, raising the question of a causal association. Data from large, randomized, controlled studies has not demonstrated an increased risk of these problems, however. The FDA and the European Medicines Agency (EMA) have stated that evidence of a causal association was "inconsistent with current data" but have implemented strategies for continued monitoring of the safety of these drugs.[19]

GLP-1 receptor agonists (GLP-1RAs). These therapies also leverage the incretin pathway, and work by suppressing glucagon, enhancing glucose-dependent insulin secretion, and slowing gastric emptying. GLP-1RAs are more potent than DPP-4 inhibitors, with an average A1c lowering of 0.6–1.2 percentage points. Most are administered via a pen injector device that differs between medications, which should be considered if patients fear needles or have decreased dexterity or vision. Injection frequency ranges from twice per day to once per week. Semaglutide was recently released in an oral form that can be taken daily. In addition to other effects, GLP-1RAs also suppress appetite, and in combination with slowed gastric emptying typically lead to a weight loss of 2–3 kg. This can be of great benefit for overweight older adults who have a limited ability to increase physical activity and may have challenges following reduced-calorie diets. This effect should be considered carefully in patients at risk for malnutrition or frailty. Because these medications promote insulin release only when glucose is elevated, the risk of hypoglycemia is very low.

All GLP-1RAs are undergoing or have completed large cardiovascular safety trials. These trials generally enrolled patients at very high risk of CVD events. Trials to date have shown that patients at high risk for CVD events taking liraglutide, dulaglutide or injectable semaglutide had lower rates of adverse cardiovascular outcomes.[20,21,22] As a result, the ADA now recommends that for patients with type 2 diabetes uncontrolled on metformin that a GLP-1RA be considered as a second-line preferred agent in patients with established CVD. These studies also showed that several GLP-1RAs may have an added benefit of slowing the progression of diabetic renal disease, with reduced rates of nephropathy progression, mainly driven by lower rates of the development of macroabuminuria.[23,24]

There are several other considerations for safety and tolerability. While a history of pancreatitis is no longer considered an absolute contraindication to use, an informed discussion should be had with the patient about the lack of data in this setting, and patients who develop pancreatitis should have the drug discontinued. Among the most frequent common side effects are nausea, vomiting, and diarrhea. For most patients, the side effects do not require stopping the medication, and can be minimized by appropriate dose titration. Patients with a history of gastroparesis should not use GLP-1RAs. Because findings in rodent studies showed an increase in a rare type of thyroid cancer, patients with an increased risk for medullary thyroid cancer should not use these medications. Finally, because of an increase in reports of acute kidney injury, it is not recommended to use exenatide in patients with a creatinine clearance below 30 mL/min, and patients with a reduced creatinine clearance below 50 mL/min require ongoing monitoring. There is insufficient data at this time to assess the safety of other GLP-1RAs in advanced renal disease, but limited study to date has not shown any adverse effects.[25]

SGLT-2 inhibitors. SGLT-2 inhibitors work at the kidney to lower the threshold of glucose excretion, promoting glucosuria and thereby lowering serum glucose. In the form of a daily pill, they generally do not cause hypoglycemia, and result in modest weight loss and blood pressure lowering. A1c lowering is modest, with an average decrease of 0.5–0.7 percentage points. Recent cardiovascular trials in patients with coexisting CVD and type 2 diabetes have shown a decrease in cardiovascular morbidity and mortality for patients using empagliflozin and canagliflozin.[26,27] In addition, the diuretic action of these medications has shown them to be effective in reducing admissions for heart failure. Large trials have also shown a slowed progression of nephropathy.[28,29] Based on this data, the ADA recommends adding an SGLT-2 inhibitor or GLP-1RA with CVD benefit to metformin in patients with or at high risk for CVD, heart failure, or chronic kidney disease (CKD). Currently there is limited data regarding the safety and tolerability of these agents in very elderly patients, who may be at

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increased risk for the most common side effects of these medications, which include for women genital yeast infections (up to 10%) and balanitis in men. Urinary tract infections, though a less common complication, occur in about 2.5% of patients, thus a history of prior frequent infections is a contraindication to use. In addition, by lowering blood pressure, patients may experience dizziness. Patients already taking diuretic medication may need to have the dose adjusted. These agents are not approved for use in patients with reduced renal function (GFR <60 mL/min/1.73 m^2: ertugliflozin; GFR <45 mL/min/1.73 m^2: other agents). Some studies have raised a concern about an increase in amputation risk, primarily with canagliflozin, thus this agent should not be used in patients at increased risk, such as those with peripheral vascular diseases and neuropathy.

Insulins. Insulin therapy may be needed to achieve metabolic control and may be preferable to oral agents in some patients. The decision to treat with insulin must include an assessment of patient beliefs about insulin and the potential for its safe use. For example, since most older patients have type 2 disease, they are likely to have had experiences with family members or friends with diabetes. Insulin therapy is frequently instituted after several years of diagnosed disease duration, at a time when disease complications may be manifest. Thus, the development of worsening complications may be falsely attributed to the insulin itself, and fears should be explored with patients. In addition, visual and cognitive function and manual dexterity require careful evaluation if patients will be administering the insulin themselves. If not, the adequacy of informal or formal supports to manage insulin therapy consistently and safely must be reviewed.

When exogenous insulin is used as sole therapy for older diabetics, its dosage and injection schedule should be individualized to each patient's needs. In addition, many insulin formulations are available as pens, which can be quite useful for older adults with vision or manual dexterity challenges. The ideal insulin regimen should have a low risk of hypoglycemia while still controlling symptoms of hyperglycemia. Newer basal insulins, such as glargine and degludec, have no peak and an action duration of 24 hours or more. When these are combined with rapid-acting insulin, such as lispro or aspart, prior to meals, glycemic goals for fasting and postprandial states can be achieved. However, older adults with physical and cognitive impairments may not be able to monitor plasma glucose and/or self-administer different types and doses of insulin reliably. A starting dose of 10 units or 0.2 units/kg/day of a basal or intermediate-acting insulin (neutral protamine Hagedorn [NPH]) minimizes the risk of hypoglycemia. Adding a dose before dinner or at bedtime will help to lower fasting blood glucose values. Additional doses of short-acting insulin can be given with or between the NPH to control hyperglycemia. Insulin regimens should be tailored according to the individual patient's response as well as to his or her acceptance of the regimen. Per the 2019 Beers Criteria, sliding scale insulin is associated with hypoglycemia without managing hyperglycemia across all sites of care for older adults and should be avoided.[15]

Home Blood Glucose Monitoring

Although self-monitoring of blood glucose is safe and relatively easy for most patients, its use has not been studied systematically in older persons. The main reasons to consider glucose monitoring in older patients are: (1) to recognize hypoglycemia in patients treated with hypoglycemic medications, particularly during times of illness or when medication changes are planned; and (2) to guide adjustments of therapy in conjunction with A1c levels. The A1c reflects glucose levels over the previous 8–12 weeks and is therefore useful in monitoring glycemic control over time. However, the A1c must be interpreted with caution in conditions that affect red blood cell turnover, such as advanced kidney disease, erythropoietin therapy, and those patients with bleeding, myelodysplastic syndrome, and valvular heart disease. While monitoring blood glucose at home can be important in identifying and treating hypoglycemia, and valuable to assess the effect of medication adjustments, many patients find performing frequent fingerstick checks challenging and uncomfortable. With the advent of continuous glucose monitors (CGMs), patients are able to continually monitor blood sugar via subcutaneous sensors. Such technology is used more often by patients with type 1 diabetes and these patients are now living longer. While data in older adults is limited, trials have shown the use of CGM devices in older adults with type 1 and type 2 diabetes can improve A1c while decreasing glycemic variability without an increase in hypoglycemia.[30,31] Considerations in prescribing CGM devices include affordability, ability of patients and caregivers to utilize the devices, provider knowledge in interpreting CGM data, and whether monitoring the patient's blood glucose at home is in line with the patient's overall goals of care.

Cardiovascular Risk Factors

While diabetes was ranked as the seventh leading cause of death in American adults in 2017,[32] about two thirds of deaths in adults with diabetes are due to heart disease or

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stroke. Mutable risk factors for CVD include hypertension, hyperlipidemia, smoking, and obesity. There is strong evidence to support achieving moderate blood pressure targets to lower the risk of heart disease and stroke in older adults with diabetes, although exact targets remain controversial. Society recommendations from the ADA, the Eighth Joint National Committee (JNC8), and the American College of Physicians (ACP) range from <130/80 mmHg in high-risk groups with progressive renal disease or a prior stroke to <150/90 mmHg for those in poor health or with end-stage chronic illness. Most trials, however, have enrolled participants who are middle-aged to young-old-aged (65–75 years old) such that benefits to the older age groups have been inferred. In the ACCORD-BP study, lowering systolic blood pressure below 120 mmHg was not associated with a lower risk of major adverse cardiovascular events but was associated with a decreased risk of stroke.[33] As a group, older adults with diabetes may be most likely to benefit in terms of decreased CVD morbidity and mortality given their decreased life expectancy compared with persons of the same age without diabetes. Reductions in macrovascular endpoints from treating hypertension in controlled studies are seen at approximately 2–5 years.[34,35]

Updated guidelines from the American College of Cardiology and the American Heart Association continue to recommend that all adults aged 40–75 years with diabetes be considered for at least moderate-intensity treatment with a statin for primary prevention of CVD (Class 1 evidence).[36] Moderate intensity is defined as an LDL-C reduction of 30–49%. The 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors (statins) are the preferred pharmacological therapy. Statins have some gastrointestinal side effects, but minimal drug–drug interactions. Liver transaminases and creatinine kinase levels should be checked prior to starting therapy, but if normal, no further testing is required unless the patient experiences muscle symptoms. Given that there is limited evidence supporting the benefit of statin therapy in older diabetics ≥75 years for primary prevention of CVD, statins should be discontinued in this age group. For secondary prevention, there is evidence that moderate-intensity statin therapy can be beneficial in CVD risk reduction in adults ≥75 years. Decisions for treatment should be based on life expectancy, goals of care, and potential for harm over benefit.

Aspirin therapy should be used in patients with diabetes and known CVD in doses of 75–162 mg daily to decrease the risk of another cardiovascular event. However, the odds of gastrointestinal bleeding in older adults taking aspirin are 1–10 in 1,000 annually and probably increase with age and dose.[37] Therefore, given the risk for harm of bleeding from gastrointestinal or other sources (i.e., intracranial) and limited evidence for efficacy in primary prevention for reducing the risk for cardiovascular events, current recommendations are to not prescribe aspirin therapy for these individuals unless ≤70 years old, a 10-year risk of CVD ≥10%, and not at increased risk of bleeding.

Eye Care

Given the importance of vision to overall functional independence, eye disease in older diabetics deserves critical attention by clinicians. Data from the 2013 Behavioral Risk Factor Surveillance System indicates that among diabetics aged 65 years and older, over 10% have serious trouble seeing, even with glasses.[38] This is not only because of diabetic retinopathy, but also because older diabetics are likely to have comorbid eye conditions, namely cataracts, glaucoma, and macular degeneration. The risk of developing retinopathy increases with duration of disease and with poor glucose control. Nearly all of type 1 diabetics and >60% of type 2 diabetics have retinopathy after 20 years. Because patients with type 2 diabetes frequently have had their disease for some time prior to diagnosis, over one third may already have retinopathy at the time of diagnosis.[39]

Because of this, all newly diagnosed patients with diabetes should be referred for ophthalmologic evaluation. This evaluation should include a comprehensive and dilated-eye examination by an ophthalmologist or an optometrist, since even in the best hands, dilated ophthalmoscopy only has a sensitivity of about 80% for detecting proliferative retinopathy.[40] In addition, there is strong evidence that diagnosis and treatment of diabetic retinopathy reduces its progression and visual loss. Current ADA guidelines recommend at least yearly eye exams if any retinopathy is present, but if the initial dilated-eye exam is normal, the frequency of eye exams should be every 1–2 years. More conservative recommendations from the Endocrine Society of America support annual eye exams even for those older diabetics without retinal disease.

Lower-Extremity Complications

Older adults with diabetes are at increased risk of developing peripheral arterial disease, sensory neuropathy, joint malformations, and foot ulcers, all of which can contribute to functional disability. Resulting gait abnormalities can lead to traumatic falls, and ulcerations

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can lead to lower-extremity amputations. Prevention of diabetic foot ulcers would lead to reductions in amputation rates. It is well known that patients do not engage in preventive care of their feet and that physicians infrequently examine feet. Improved self-care and physician attention to foot abnormalities, however, can be achieved relatively easily and inexpensively, resulting in reduction of foot lesions in one randomized controlled trial.[41] More recently, a Cochrane Collaboration review found mixed results on the effect of patient education for the prevention of diabetic foot ulceration.[42] Even without better evidence, patients should be instructed in self-examination methods, nail and callus care, washing techniques, and what constitutes appropriate footwear. Since many older persons have fungal infections of their nails and may not be able to safely cut them, referral to a podiatrist is a prudent strategy.

For their part, health-care providers should perform a comprehensive foot exam at least annually, although there is no evidence to support this interval of screening. The foot examination can be used to reinforce important patient foot care behaviors. The exam should include (1) an assessment of vascular perfusion by palpation of the lower-extremity pulses, (2) a neurological exam to assess sensorimotor deficits using the Semmes-Weinstein 5.07 (10-g) monofilament, (3) an assessment of skin integrity, and (4) a musculoskeletal exam to evaluate range of motion of the foot and ankle as well as bony abnormalities.

The rate of amputation for people with diabetes is at least twice that for people without diabetes. Black people and males are more likely to undergo a lower-extremity amputation.[43] In addition to the monetary costs of amputations, they obviously have a profound effect on patients' mobility and may precipitate institutionalization and death.

Nephropathy Screening

Diabetes is the leading cause of end-stage renal disease. Evidence from the UKPDS has shown that glycemic and blood pressure control provide protection from advancing renal disease. Albuminuria (≥30 mg/24 hrs) is an early sign of diabetic nephropathy and is associated with an increase in cardiovascular mortality. The simplest test for albuminuria is a spot urine albumin-to-creatinine ratio where normal is accepted as <30 μgm albumin/mg creatinine. Since it may be helpful for risk assessment, testing for albuminuria should be performed at diagnosis of diabetes, then yearly in its absence. Ongoing testing may not be necessary in patients with expected limited lifespan.[13] Once a patient has albuminuria, there is little evidence to support continued monitoring. The ADA recommends ongoing annual monitoring as some therapeutic interventions, such as treatment of uncontrolled hypertension and institution of angiotensin-converting enzyme (ACE) or angiotensin receptor blocker (ARB) therapy, may reduce albuminuria and be associated with reduced cardiovascular and renal complications.

Cognitive Function

Compared with age-matched controls, older adults with diabetes are more likely than their nondiabetic counterparts to perform poorly on cognitive tests,[44,45] and have a 73% increased risk of developing dementia.[46] One systematic review found consistent deficits in verbal memory in diabetics compared to nondiabetics despite significant heterogeneity among the methods of the studies reviewed.[47] These changes are similar to those associated with normal aging, but whether they are manifestations of an accelerated aging process or occur via other mechanisms is not clear. There may be adverse effects on cognition from hyper- and hypoglycemia and hyper- and hypo-osmolar states, furthering the importance of maintaining metabolic stability. Older diabetics are also more likely to have strokes, which predisposes patients to cognitive deficits and further necessitates treatment of other modifiable risk factors such as hypertension. Because of diabetes-related changes in cognitive function and the increased likelihood of dementia with age, periodic assessment of cognitive function in older diabetics is essential. This can serve to reassure the "worried well" who may have concerns about memory problems, and to identify those beginning to experience subtle difficulties. Careful attention to these issues may uncover adverse drug effects, other metabolic derangements (e.g., hypothyroidism), or depression, and will identify those who may need additional help from clinicians and caregivers in adhering to complex treatment regimens.

Family Considerations

There is growing evidence that families play significant roles in the management of older persons with chronic disease in addition to the well-known role they play in the general daily care of frail older persons.[48] A study of 357 family members of diabetic patients 70 years of age or older demonstrated that over half (71% were spouses) participated in the patients' diabetes care.[49]

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If patients are having difficulty adhering to treatment regimens, if they rely on family members for certain activities (e.g., food preparation, managing medications), or if they have functional disabilities, their family members or other caregivers need to be educated about diabetes and receive instruction and support in methods of management.

Summary and Conclusions

Diabetes is a common condition in older persons and is associated with considerable economic and personal costs. Attention to the prevention and management of cardiovascular, eye, and foot disease is therefore critical. The risks and benefits of tight glycemic control will vary for a given individual depending on cognitive status, functional status, and life expectancy. Glycemic targets should be individualized. In addition to the focus on glucose management, cardiovascular risk factors should be addressed, and attention should be given to preventing eye and lower-extremity complications, where screening and treatment interventions also have known efficacy.

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Chapter 33

Lipid Management

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Overview

Since the last edition of this book chapter, recommendations for the management of lipid disorders to reduce cardiovascular risk have evolved. At present, the emphasis of therapies directed at low-density lipoprotein cholesterol (LDL-C) remain the mainstay of the current treatment paradigm. Lipid metabolism does not vary greatly between younger and older adults. Age and exposure to elevated serum cholesterol over time are relevant in the manner in which we approach lipoprotein-mediated disease processes in older adults. Age, a powerful predictor of risk for vascular disease, will be highlighted as outlined in the 2018 American Heart Association (AHA)/American College of Cardiology (ACC) cholesterol treatment guidelines.[1]

We anticipate lipid management recommendations to evolve even more rapidly in the near term. These recommendations will likely be based on data from the REDUCE-IT study, data on the role of PCSK-9 inhibitors in older patients, and emerging science on residual risk – all directed at improving the care of vascular disease patients, regardless of age.

Age and Cardiovascular Risk

Clinicians typically focus on patients' age as a "nonmodifiable risk factor." Age contributes considerably to absolute 10-year cardiovascular risk in the Framingham risk equation. This contribution may be related to lipid exposure over time, modest elevations of cholesterol, and increased exposure to traditional cardiovascular risk factors with increasing age. Multiple observations have concluded that atherosclerosis is a process that begins in early life.[2,3] Compelling work by Hobbs and others focused on identifying the variant of the PCSK-9 gene has supported the concept that even a moderate reduction of LDL-C over a lifetime can result in a lowering of coronary events.[4] Their hypothesis that LDL-C might be a sufficient and necessary cause of atherosclerosis suggests a lower threshold of benefit, one that was presaged by Brown and Goldstein.[5] The concept of lifetime exposure was further supported by the analysis by Ferrence et al. in which a delay of statin therapy until later in life results in a nearly threefold increase in cardiovascular events.[6] More recently, Robinson and others made the case for early aggressive treatment of LDL-C in atherosclerotic disease. This fact is also reinforced by the data describing similar numbers of cardiovascular events in individuals ≤65 years as those ≥65 years of age.[7] Thus, advanced age reflects an increased duration of exposure to various risk factors and an accumulation of coronary disease burden.[8]

The current treatment recommendations from 2018 endorse aggressive treatment in individuals with known disease using statins with reductions greater than or equal to 50%, with an option to act more aggressively with the addition of ezetimibe or a PCSK-9 inhibitor to reduce LDL-C below 70 mg/dL.[1] These risk-based guidelines include robust discussions with patients at risk for disease, i.e., primary prevention, in association with considerations like strong family history, chronic kidney disease, metabolic syndrome, and chronic inflammatory disease. It should also be noted that the current treatment recommendations address evidence from clinical trials for patients between 45 and 75 years of age. For the elderly, >75 years of age, evidence for statin therapy is not as strong, therefore clinical assessment of risk status in a clinician–patient risk discussion is paramount for deciding whether to initiate statin therapy.[1] However, in a patient already on statin therapy, it is reasonable to continue therapy. Also, as stated in 2013, "Guidelines are intended to define practices meeting the needs of patients in most, but not all, circumstances, and should not replace clinical judgment." This is particularly relevant to older patient populations.[1]

Recognizing that age is such a powerful predictor for risk of heart disease, clinicians should address common modifiable risk factors in older patients, such as LDL-C, in order to slow the development of subclinical disease. It is

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quite likely that older individuals will become increasingly eligible for lipid-lowering therapy, given the accumulation of age-related conditions such as diabetes, hypertension, and chronic kidney disease. In a well-considered review of treatment of high cholesterol in older patients, the authors suggest a more patient-centered approach (as opposed to a 10-year risk paradigm), given the wide heterogeneity in physical and functional health in older individuals. Issues such as frailty and safety serve as an important platform for a conversation between the clinician and the patient on treatment options. As outlined in the 2018 AHA/ACC guidelines:

- In the presence of cardiovascular disease (CVD), 70–75 years: treat in the same way as younger adults; >75 years: it is reasonable to initiate moderate-/high-intensity statins. Weigh potential CVD risk reduction against adverse effects, drug–drug interactions, frailty, and patient preferences before initiating therapy. Continue high-intensity statins if well tolerated.
- In primary prevention in individuals between 70 and 75 years: treat in the same way as younger adults; >75 years: it is reasonable to initiate moderate-/high-intensity statins. Weigh potential CVD risk reduction against adverse effects, drug–drug interactions, frailty, and patient preferences before initiating therapy. Continue high-intensity statins if well tolerated.[1]

The cutoff point of ≥75 years of age is subjective yet consistent with the majority of available evidence. In these patients with CVD and a reasonable life expectancy (more than 1–2 years), lifetime risk of CVD is considered high enough to benefit from initiating or continuing lipid-lowering therapy. However, in case of (severe) negative effects (e.g., side effects) or if life expectancy is limited (less than 1–2 years), the benefits of lipid-lowering therapy is unlikely to outweigh potential adverse effects of treatment. For frail, older patients without pre-existing CVD, we propose to actively deprescribe and avoid initiation of lipid-lowering treatment. Their lifetime risk of CVD is not sufficient to benefit from lipid-lowering therapy in terms of preventing a cardiovascular event in a given life expectancy. Also, frailty may exacerbate adverse events associated with lipid-lowering therapies (particularly statins).[9]

Clinical Trials and Duration of Therapy

Numerous clinical trials demonstrate the benefits of lowering LDL-C in older persons with established coronary heart disease (CHD). Increased levels of LDL-C carry predictive power for the development of CHD in older persons as well as younger individuals.

As reported in the Cardiovascular Health Study in 2002, the use of statin therapy in study participants at baseline who were 65 years or older and free of CVD resulted in a 56% lower risk of CVD events and 44% lower all-cause mortality.[10] Although the value of lipid lowering in older patients with known coronary disease is evident, the decision to manage risk has been modified in the current guidelines.

We are often asked, "How long will I be on this medication?" In more medical terms, do the benefits we see with the clinical trials last a lifetime? The answer, of course, is not known but it would seem to lie in what we know and believe about the role of LDL-C in causing CVD and the probability that lowering LDL-C with statins stabilizes or prevents atherosclerosis progression. We have strong indications that the benefit of long-term statin therapy occurs and persists in older as well as in younger and middle-aged individuals. In the 10-year follow-up of the Simvastatin Survival Study, a reduction in mortality from coronary disease, with no increase in death from cancer or other causes, was observed in the simvastatin-treated group.[11] Follow-up from the Heart Protection Study further supports the observation that ongoing lipid-lowering treatment with a statin confers benefits over time without an increased risk of non-cardiovascular mortality.[12] Furthermore, the Cholesterol Treatment Trialist Collaboration meta-analysis of 27 statin therapy trials demonstrated even a substantial risk reduction among low-risk patients with a 5-year risk of a coronary event of less than 10% – a reduction comparable to the event reductions seen in higher-risk groups. Again, there was no compromise in safety related to cancer or other non-cardiovascular mortality over the long term. Although the most common concern for patients is muscle-related side effects, as referenced in this meta-analysis, statin therapy is associated with a small increased risk of myopathy (excess incidence of about 0.05% over 5 years) and, more rarely, of rhabdomyolysis (excess incidence of about 0.01% over 5 years).[13] The risks of myopathy are typically dose-related but, with the exception of simvastatin 80 mg daily (or lower doses in Asian populations), intensive statin regimens have not been shown to result in substantial myopathy risks.[14] A recent report from Thompson et al. concluded that the long-term adverse events related to statin

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therapy – including myopathy, central nervous system effects, and the occurrence of diabetes – appear to be low but that the cumulative risk needs further study.[15]

Primary Prevention in Older Patients

Recent guidelines strongly support the use of low-, moderate-, or high-dose statins for risk reduction in patients based on their LDL-C levels and 10-year atherosclerotic cardiovascular disease (ASCVD) risk.[1] Based on this construct, the prospects for reducing clinical CHD in the older patients by LDL-C lowering are good. The first specific prevention trial to evaluate the role of lipid lowering with statins in older patients (aged 70–82) was the Prospective Study of Pravastatin in the Elderly (PROSPER). Older men (n = 2,804) and women (n = 3,000) at high risk of developing CVD and stroke were randomized to placebo or 40 mg pravastatin. Patients were evaluated over an average of 3.2 years and assessed based on a composite endpoint of major coronary events, including nonfatal myocardial infarction and CHD death. Each endpoint was reduced with treatment by 19% and 24%, respectively. Although no reduction in stroke occurred in the treatment group, there was a 25% reduction in transient ischemic events. These results support the notion that the benefits of statin therapy could be safely extended to older persons.[16]

Another trial in evaluating the possible role of lipid lowering in at-risk patients was completed in a large hypertensive population. The Anglo-Scandinavian Cardiac Outcomes Trial – Lipid-Lowering Arm (ASCOT-LLA) program evaluated more than 10,000 patients aged 40–79 (average 63 years of age) with at least three risk factors in addition to high blood pressure. Patients were randomly assigned to either atorvastatin 10 mg or placebo. The original study design was to provide follow-up for an average of 5 years, but the trial was stopped at a median follow-up of 3.3 years because of a marked reduction of events in the treatment group. Treatment with atorvastatin resulted in a reduction in the incidence of fatal and nonfatal stroke by 27% (P = 0.024), total cardiovascular events by 21% (P = 0.0005), and total coronary events by 29% (P = 0.0005). A practical observation from this study underscores the value of modest doses of statin resulting in improved disease outcomes.[17]

Another recent randomized trial evaluated the role of rosuvastatin as a lipid-lowering agent in primary prevention in elderly persons with high C-reactive protein and low LDL-C levels. This double-blinded trial, based on a secondary analysis of the Justification for the Use of statin in Prevention: An Intervention Trial Evaluating Rosuvastatin (JUPITER) trial, randomized patients to rosuvastatin 20 mg or placebo. Participant profiles differed from the original JUPITER trial participants in that among more than 17,000 patients, 32% were aged 70 years or older. These patients were predominantly female and more likely to be hypertensive as compared to younger participants enrolled in the JUPITER trial.[18] The primary endpoint of this study was occurrence of first major cardiovascular event or death from components of primary endpoints. Inclusion criteria included LDL-C level at screening of 130 mg/dL and C-reactive protein level of 2.0 mg/L at the beginning of the study. LDL-C was reduced to half (54 mg/dL) and C-reactive protein was reduced by 36–37% in the treatment group as compared to placebo at 12 months. An absolute reduction of 48% or more was seen in the primary endpoint in the participant group treated with 20 mg rosuvastatin. Although the trial was stopped early, assessment of cumulative risks in the treatment group continued for up to 4 years. Statistically, the number needed to treat (NNT) to prevent one cardiovascular event or death in adults over 70 years was 24 (95% CI: 15–57) as compared to 36 (95% CI: 23–77) in younger individuals, thus indicating the relative benefit in treating older patients based on age.[18]

The 2018 ACC/AHA guidelines on lipid management reflect the results from the above studies while also acknowledging the overall limitation in prospective, randomized research data for primary prevention in adults >75 years of age. The guideline committee emphasizes that it remains reasonable to continue to follow the primary prevention recommendations even above age 75, but an individualized assessment of a patient's risk of adverse event as well as frailty becomes paramount in the care of older adults. For many adults older than 75 who have an LDL-C value of 70–189 mg/dL, a moderate-intensity statin is a reasonable approach to primary prevention (LOE IIb/B-R). In older age groups, however, it is even more important to consider competing factors that may affect their medication adherence, adverse effects, and cardiovascular risk. The presence of functional or cognitive impairment, multimorbidity, and frailty all lead to limited life expectancy in older patients. Given the fact that it can take up to 5 years to see a statistical benefit from statin in regard to stroke prevention, it may be reasonable to deprescribe or decide not to initiate a statin for primary prevention in those whose aggregate risk outweighs the potential benefit (LOE IIb/B-R). Unfortunately, the decision and qualitative calculation

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of an individual's aggregate risk and potential benefit is not intuitive. For example, the most clinically frail patients are most likely to have a limited life expectancy, but these patients are often of advanced age and have multiple comorbidities that may put them at the highest risk for cardiovascular events. Furthermore, those with impairment in cognition and functional status are often unable to participate in various nonpharmacologic moderations of cardiovascular risk such as an exercise regimen. For this reason, the ACC/AHA committee recommends a model of shared decision-making to come to an individualized treatment plan for each patient. This should also include reassessment over time to ensure that previously prescribed treatments remain within the ongoing goals of care for individuals. Patients who tolerate the medications well and without adverse events may prefer to stay on lipid-lowering medications even after the point at which the potential benefit continues to outweigh the risk. The guideline committee also supports this type of individualized decision-making.[1]

An evolving area that can be helpful to assess individual risk and to guide the decision on primary prevention in individual patients is the use of coronary artery calcium (CAC) scoring. The 2018 AHA/ACC guidelines recommend that up to age 80, CAC scoring can be considered in asymptomatic individuals with LDL-C values between 70 and 189 mg/dL. In these individuals, if CAC is zero, then statin should be avoided (LOE IIb/B-R).[1]

Atherosclerotic Cardiovascular Disease and Secondary Prevention

Beginning with the 2013 guidelines, the definition of heart disease was expanded to include all ASCVD. This includes CHD, stroke, and peripheral arterial disease (PAD), all of which now are considered "CAD equivalents" and linked to atherosclerosis.[1] Given the increased risk for all-cause and cardiovascular mortality, individuals with PAD – regardless of age – should be aggressively managed with respect to coexisting risk factors.[19] Historically, the effect of cholesterol on development of stroke has been less clear. However, more recently high serum cholesterol was identified as a predictor of risk in patients with ischemic stroke.[20] Given that almost 30% of the 700,000 strokes that occur each year are recurrent events, it is critical that we identify the risk and determinants of recurrent stroke and review the evidence base to support improved outcomes in this patient population.

The first trial to demonstrate a reduction in stroke risk in noncoronary disease patients with a history of cerebrovascular events (stroke or transient ischemic attack [TIA]) was the Stroke Prevention by Aggressive Reduction in Cholesterol Levels (SPARCL) trial. This study included more than 4,700 patients with a stroke or TIA within 6 months before study entry. These individuals had no known history of CHD and baseline LDL between 100 mg/dL and 190 mg/dL. After treatment assignment of either placebo or atorvastatin 80 mg, patients were followed for a median period of 4.9 years. In patients with a recent stroke or TIA, treatment with 80 mg of atorvastatin per day decreased the risk of stroke, major coronary events, and revascularization procedures. These results strongly support the initiation of atorvastatin treatment soon after a stroke or TIA.[21]

The 2018 guidelines for management of blood cholesterol continue to strongly advocate for the use of high-intensity statin therapy for nearly all patients under 75 years of age (LOE I/A) with clinical evidence of ASCVD. These most recent guidelines include a new treatment algorithm for identifying those at "very high risk" for which more intensive risk modification is recommended. The "very high-risk ASCVD" population has been defined as multiple major ASCVD events (multiple myocardial infarctions, ischemic stroke, symptomatic PAD) or one major ASCVD event with multiple high-risk conditions (e.g., age >65, diabetes, hypertension, chronic kidney disease, or congestive heart failure).[1]

For those not in the "very high-risk" cohort, the group continues to advocate for the initiation of moderate- or high-intensity statin for those over 75 years of age, with the added recommendation of individualized consideration of the risk-to-benefit ratio and shared decision-making (LOE IIa/B-R). This recommendation is consistent with prior treatment recommendations for treatment of ASCVD in older age groups. The benefit of lipid-lowering therapy for secondary prevention in older adults is clearer than when used for primary prevention in the same age group. The inclusion of moderate-intensity statin in the recommendation for this age group (as opposed to specifying high-intensity statin for those <75 years) is reflective of moderate-intensity statins being better tolerated in the older age groups. The multi-society expert panel also advises that if a patient older than 75 with clinical ASCVD is already tolerating a high-intensity statin, there is no need to de-escalate therapy to a lower-intensity drug provided that intensive secondary prevention remains within the patient's goals of care and benefit continues to outweigh risk (IIa/C-LD).[1]

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There is no specific recommendation for use of non-statin medication such as ezetimibe or PCSK-9 inhibitor in older adults, but it is reasonable to consider these treatment options if an older patient is considered to be very high risk for future ASCVD events as described above. As with the other treatment recommendations in older adults, a rigorous assessment of patient risk, including drug interactions, adverse events, life expectancy, and frailty, is needed along with shared decision-making prior to initiation of these medications. The expert panel recommends first adding ezetimibe for treatment intensification if LDL-C remains greater than 70 mg/dL on high-intensity (or maximally tolerated) statin therapy (IIa/B-R). In those patients who have LDL-C greater than 70 mg/dL even after addition of ezetimibe, PCSK-9 inhibitor can be considered (IIa/A). It is important to consider the low cost-effectiveness of PCSK-9 inhibitors in the shared decision-making process when considering these drugs for older adults.[1]

Therapeutic Lifestyle Changes in the Elderly

As in prior clinical recommendations, the 2018 AHA/ACC guidelines continue to emphasize the importance of lifestyle modification as a critical component of health promotion and ASCVD risk reduction prior to and along with the use of cholesterol-lowering drug therapies. Healthy diet or lifestyle modifications were recommended as background therapy for nearly all randomized controlled trials of cholesterol-lowering drug therapy.[1] The 2013 Lifestyle Management Work Group Guideline for lifestyle recommendations identifies patterns of nutrition rather than specific diets such as the DASH (Dietary Approach to Stop Hypertension) or Mediterranean diets. These "patterns" include an emphasis on intake of fruits, vegetables, and whole grains. Sources of proteins should include low-fat dairy products, poultry, fish, and legumes. Individuals should also limit intake of sweets, sugar-sweetened beverages, red meats, and overall calorie intake from saturated fat.[22] Plant sterols (2 gm/day) and up to 25 mg of soluble fiber can aid in lowering LDL-C, which could be used alone or in conjunction with appropriate pharmacotherapy.[8]

Appropriate levels of physical activity can also improve cardiovascular outcomes across all age groups. Older patients are often a heterogeneous group, many of whom have multiple comorbid conditions or physical limitations that make broadly applicable recommendations regarding physical activity problematic. The 2008 Physical Activity Guidelines for Americans endorse regular physical activity as key to healthy aging. Although the 2013 AHA/ACC Lifestyle Management Guideline suggests 2.5 hours per week of moderate-intensity exercise, an individual approach should be outlined to support as much physical activity as patients' abilities and conditions allow.[23]

Ideally, the components of an exercise regimen would be multifaceted for older adults. Aerobic exercise remains the mainstay of cardiovascular risk reduction from a standpoint of physical activity, but a well-rounded exercise program in older adults should also include components of muscle-strengthening, flexibility, and balance training. While these other modalities of training may not directly address a patient's cardiovascular risk, the overall health and maintenance of these domains helps to limit falls, osteoarthritis, and sarcopenia, all of which contribute to frailty, which does have significant indirect implications in CVD.

It is paramount for clinicians to screen older adults' level of physical activity in order to help advise them as to appropriate activity levels and activity prescription. The American College of Sports Medicine (ACSM) along with the AHA recommends the development of an activity plan for all older adults. This should be created in collaboration with primary care providers, subspecialists, physical therapists, occupational therapists, and personal trainers when appropriate. It is common for patients with functional limitations to perceive exercise as impossible, unhelpful, or even a risk to future injuries. Studies such as the Lifestyle Interventions and Independence of Elders (LIFE) trial proved that regular exercise improves functional limitations, promotes health, and does not increase adverse events if conducted in a safe manner. The involvement of specialists such as physical therapists or specialized, multidisciplinary programs such as cardiopulmonary rehabilitation can provide extra benefit in these patients.[24]

Statin Therapy for Lipid Management in Older Patients

As emphasized in the 2018 AHA/ACC guidelines, the statin class has evidence derived from multiple randomized clinical trials as well as meta-analyses to inform clinical decision-making. Statin drugs remain the mainstay of lipid-lowering pharmacologic therapy for all age groups. Further emphasized in the 2018 ACC/AHA guidelines is the intensity of statin used in treating older adults. Statin therapy is divided into three classes: low-intensity, moderate-intensity, and high-intensity. While in general it is felt that high-intensity offers the best

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results in terms of overall reduction in ASCVD-related events, as discussed above, moderate-intensity statins appear to be better tolerated in older patients. For this reason, moderate-intensity statin is considered a reasonable approach to both primary and secondary prevention in older age groups.[1]

Statin-associated side effects (SASEs) range from very mild myalgias to severe and critical cases of rhabdomyolysis or hepatic failure. Although SASEs receive a lot of attention and concern from patients, randomized controlled trials and observational data suggest they are actually quite rare. Specifically, the serious side effects including myositis, rhabdomyolysis, statin-associated autoimmune myopathy, hepatitis, and liver failure are especially rare.[25]

Myalgias are the most common type of statin-associated muscle symptoms (SAMS), and nearly every prescriber will encounter a patient with muscle pain who is on a statin at some point in their career. Statin-associated myalgias with no evidence of muscle breakdown or myositis (normal creatine kinase level) occurred around 1–5% of the time in randomized controlled trials and in 5–10% of patients in observational cohorts.[25] Those most commonly affected by myalgias tended to be older, female, or had low body mass. Those with comorbid HIV, renal, liver, thyroid, or preexisting muscle disease also tended to be at higher risk of myalgias. Asian ancestry, excessive alcohol intake, extreme levels of physical activity, and high-risk medications also appear to increase the incidence of statin-associated myalgias. These symptoms, although often mild, can be quite bothersome to the patient and result in significant numbers of drug discontinuation, sometimes inappropriately. The guidelines regarding the assessment and management of these side effects will be discussed later in this chapter in the section Adherence: Making Medicines Work.

Other reported SASEs are often clinically relevant. There is a small increase in the development of new-onset diabetes mellitus in those taking statins, typically thought to occur at a rate of about 0.2% per year of statin treatment. This risk is most pronounced in those with other risk factors for diabetes (BMI >30, impaired fasting glucose), as well as those on high-intensity statin therapies.[25,26] Impairment in memory and cognition has been noted in case reports, but no randomized controlled trial has ever found an increase in the incidence of cognitive impairment among statin-treated patients. Additionally, no definite associations between statin treatment and cancer have been reported. Reports of multiple other potential side effects, including renal failure, cataracts, interstitial lung disease, and low testosterone, also appear to be unfounded in clinical data.[1,25,27]

The most comprehensive age-based analysis of safety for a statin was a pooled analysis of more than 50 clinical trials for atorvastatin that compared four different dosages of active drug to placebo in 5,437 patients older than 65 years (range of mean age 71–74), of which almost half of those studied were female (42%). This included a large number of individuals on either the 10-mg (n = 2,042) or 80-mg (n = 1,698) dose, along with a comparison group of almost 1,000 placebo-treated individuals. Serious adverse events (AEs) were rare (≤1%), and rates of discontinuation because of treatment AEs were low between treatment doses and placebo (2.1% vs. 1.7%). At the maximum dose, elevations of liver enzymes (AST/ALT) were higher than placebo (3.2% vs. ≤0.9%). Treatment-associated myalgia was low and no patient experienced persistent elevated creatine kinase of greater than 10 times upper limits of normal.[28]

The European Society of Cardiology conducted a large-scale systematic review of 14 primary prevention trials (n = 46,262) to assess the proportion of side effects directly attributable to statin therapy in an attempt to aid clinicians and patients in making decisions on a future course of therapy. Analysis showed an increase in type 2 diabetes by absolute risk of 0.5% ($P = 0.012$) while decreasing mortality by the same extent: 0.5% ($P = 0.003$). The study also looked at 15 randomized controlled trials (37,618 participants) for secondary prevention, and statins decreased mortality by 1.4% ($P < 0.001$). Asymptomatic increase in liver transaminase was seen 0.4% times more frequently in both primary and secondary prevention groups than the placebo group. Current guidelines highlight the very small increase in cases of new-onset diabetes (0.1 excess cases per 100 on moderate-dose statin treatment and 0.3 excess cases per 100 on high-dose statin treatment), myopathy, and hemorrhagic stroke (~0.01 excess cases per 100), making statins a relatively safe choice for the management of ASCVD risk.[29]

Non-Statins

A number of agents/classes have an impact on the lipid profile, but beyond the statins, there is limited data on their impact on cardiovascular events. Even in trials in which statins were combined with non-statins, there was little to no added benefit. Given the interpretation of the 2013 ACC/AHA guidelines, clinicians treating high-risk

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patients who have a less-than-anticipated response to statins, who are unable to tolerate a less-than-recommended intensity of a statin, or who are completely statin-intolerant may consider the addition of a non-statin cholesterol-lowering therapy. High-risk individuals include those individuals with known vascular disease including stroke or TIA, those with LDL-C ≥190 mg/dL, and those with diabetes.[1]

Limitations of Treatment in the Older Patient

The combination of multimorbidity, polypharmacy, decreased life expectancy, functional impairment, cognitive impairment, and frailty, among many other factors, leads to significant limitations in treating older patients with abnormal blood cholesterol. The current US guidelines emphasize the use of careful risk and benefit analysis as well as shared decision-making in treatment decisions, but a lack of substantial, dedicated research on treatment of these disorders in older patients makes estimating one's potential risk or benefit very difficult. Often the philosophy of the individual prescriber or a patient's previous experiences with lipid-lowering medications dictates the treatment decisions, which in some cases can become problematic.

In general, older patients do not receive evidence-based therapies for atherosclerosis-related disease on a par with their younger counterparts. The aforementioned shared decision-making is further complicated by the fact that those with advanced age and significant frailty are often simultaneously those with the shortest life expectancy, highest risk of adverse events, and highest risk for cardiovascular events. The traditional methods of risk assessment such as the pooled cohort equation have limited use above age 75. Furthermore, as age is a strong predictor of CVD, using traditional risk assessment models would indicate all patients over 75 for a statin for primary prevention, even in the absence of other risk factors. It is difficult to argue that *all* patients, especially those who have lived at least 75 years without clinically apparent ASCVD, should take a lipid-lowering medication. Boasting a strong negative predictive value, the CAC score offers promise at reassuring those without significant coronary calcium, but we have yet to determine a strategy for primary prevention in those with low or intermediate risk levels of coronary calcium.

In addition to limitations in treatment decisions, there are also significant limitations related to the pharmacotherapy in older patients. Statins, for example, are contraindicated in patients with active liver disease or unexplained persistent elevations of serum transaminases. Although cases of rhabdomyolysis are rare with statin treatment, patients should promptly report muscle pain, tenderness, or weakness to avoid severe cases. In those with preexisting functional or cognitive impairment, the early symptoms may be hard to recognize. The clinician should review these patients carefully for possible concomitant physiological compromise, including renal status, and the potential for drug–drug interactions as a contributor to risk for rhabdomyolysis.

Other pharmacological approaches also have specific limitations in this age group. Bile acid resins often interfere with absorption of other drugs and are difficult to dose. Fibrates carry warnings on their use in patients with compromised renal function, as well as distinct risk–benefit considerations when used in combination with statins. Sound judgment and a review of specific agents should be used in all patients, particularly older patients who are likely to be on multiple medications and may have age-related physiological compromise.

Beyond risk assessment, there may be other factors that come into play in older persons that affect the decision to employ LDL-lowering drugs. Ultimately, as with patients of any age, individualized therapy should match the needs of each specific patient.[30]

Insights on Risk Assessment and Residual Risk

We know that LDL-C lowering has demonstrated clear benefit in reducing cardiovascular disease, but even in patients treated to very low levels of LDL-C, cardiovascular events may still occur. Insights from the Cholesterol Treatment Trialist Collaborators and the Stroke Prevention by Aggressive Reduction in Cholesterol Levels (SPARCL) analyses demonstrate that treatment with statins alone results in a composite risk reduction of 30% with a "residual risk" of almost 70%.[13,26] Even in the studies with intensive LDL-C lowering (either utilizing ezetimibe or evolocumab), the risk of cardiovascular events remained. Results of the REDUCE-IT study revealed that treatment beyond LDL-C reduction may be of benefit in patients with established CVD with elevated triglycerides. This data suggests that the next frontier in lipid management may center on issues related to residual risk as identified by high triglycerides. We have appreciated for some time that elevated triglycerides provide a window of risk to reflect on an overproduction of atherogenic lipids. These are not traditionally assessed in a standardized lipid profile and are likely associated with increases in triglyceride-rich lipoproteins associated with post-hepatic transport by apolipoprotein

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B. While the causal role of LDL-C is clear, the presence of triglyceride-rich remnants represents abnormalities in lipid metabolism and a more pronounced proatherogenic state. Research in the future will add clarity to the precise role of these lipids in the contribution to CVD.[13,21,31–34]

Adherence: Making Medicines Work

Official guidelines offer a framework to address many specific patient care challenges, yet there are often issues up to and beyond the patient–physician encounter that impact the optimization of outcomes and quality of care. Adherence to treatment is one such issue. Poor adherence to therapies across all age groups represents a significant challenge to the health-care system, leading to worse outcomes and increased costs. Nonadherence to pharmacological therapies for chronic conditions such as hypertension, diabetes, and CVD in the elderly (which is frequently complex and includes on average five to eight different medications daily) is estimated at between 14% and 77%.[35] Older patients may require additional support for medication management and monitoring. Based on a study of older adults enrolled in two separate managed care plans, the primary challenges were related to the failure to prescribe appropriate medications, monitoring treatment, providing adequate education, and supporting continuity of treatment among providers.

Quality improvement needs to focus on errors of omission in which the underutilization of potentially beneficial medications manifests potential harm. These omissions may arise from the provider belief that there is insufficient evidence of clinical benefit associated with underrepresentation of older patients in clinical trials, concerns regarding polypharmacy, and substantial financial barriers with insufficient insurance coverage of outpatient prescription drugs.[36]

In an effort to increase adherence and widespread use of lipid-lowering medications where appropriate, the 2018 ACC/AHA guidelines recommend focused interventions to improve adherence in patients on lipid-lowering drugs (LOE I/A). Interventions such as telephone reminders, multidisciplinary education activities, and pharmacist-led interventions to streamline medication regimens have been shown to improve adherence as well as effectively help lower LDL-C levels.[1,37]

In the USA, subjective complaints of statin-associated side effects, typically myalgias, cause up to 10% of patients to discontinue the drug, often unnecessarily.[25] The 2018 ACC/AHA guidelines offer several recommendations to safely manage these side effects. Prior to treatment, it is helpful to identify risk factors for possible statin-associated side effects (LOE I/B-R) and to use this information to contribute to the shared decision process in regard to starting treatment. Patients can be reassured at the time of starting treatment that if side effects do occur, they are typically mild and can be managed successfully (LOE I/A). In patients who do develop SAMS, they recommend first an assessment of the symptoms as well as evaluation for non-statin causes and predisposing factors (LOE I/A). Myalgias are more likely to be statin-related if they are bilateral, involving the proximal muscle groups, and start within weeks or months of initiating a statin drug.[27] In most patients, statin-associated myalgias are mild and resolve after discontinuation of the therapy. In these patients it is recommended to reassess and restart statin either using a modified dose or alternative statin choice in order to determine maximal LDL-C lowering prior to considering a non-statin therapy (LOE I/B-R). If the SAMS are severe, it is recommended to measure serum creatinine kinase levels, observe for objective muscle weakness, and measure serum transaminase levels, in addition to other kidney and liver function tests, and to discontinue the medication (LOE I/C-LD). For patients who are at high ASCVD risk and have either severe SAMS or recurrent SAMS after an appropriate retrial of therapy, it is reasonable to consider other non-statin therapies such as ezetimibe or PCSK-9 inhibitors to achieve appropriate risk reduction (IIa/BR). Commonly, patients will inquire about routine laboratory monitoring of creatinine kinase or transaminase levels as well as the possibility of utilizing coenzyme Q10 supplementation to minimize statin side effects. At this time the ACC/AHA recommends against routine laboratory monitoring as well as coenzyme q10 supplementation for routine use (LOE III/B-R, C-LD).[1]

Other than the management of SAMS, the 2018 ACC/AHA guidelines also offer recommendations regarding those at high risk for diabetes or with a new diagnosis of diabetes. In these patients, it is recommended to continue statin therapy. The clinician should emphasize adherence, net clinical benefit, and lifestyle modifications to these patients (LOE I/B-R). Additionally, in patients who have elevated risk of ASCVD and chronic liver disease, treatment with statin therapy has previously been controversial. It is now thought to be reasonable to use statins after obtaining baseline laboratory measurements and a schedule of monitoring labs and safety checks has been established (LOE I/B-R).[1]

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Underutilization

The beneficial role of statins in secondary prevention is well documented for patients of all age groups. However, a recently published retrospective cohort study of more than 75,000 patients older than 65 showed the use of statin therapy declined with increasing age. This treatment paradox suggested that despite an annual 1% increase in mortality risk, there was a greater than 6% yearly decline in the use of preventive therapy. Other reviews as well have commented on this relative "clinical inertia" with respect to using statins in older patients.[38–40] Additional studies have further assessed these specific challenges. In a retrospective cohort analysis of more than 34,000 New Jersey Medicaid patients, Brenner and colleagues described a rapid decline in ongoing statin use in older patients. Unlike data reported in clinical trials, this analysis found that after 5 years, only 26% of patients were still taking their prescribed regimens. Several predictors of poor persistence were age (>75 years), socioeconomic factors, issues of ethnicity, and depression. These findings support the concept that advanced strategies for improved adherence require early intervention and specific targeting of high-risk groups.[41] A meta-analysis conducted by Mann and colleagues reviewed 22 cohort studies to find predictors of statin nonadherence. They found age >70 (odds of nonadherence 0.46; 95% CI 0.38–0.57), female gender (odds of nonadherence 1.07; 95% CI 1.04–1.11), and lower income (odds of nonadherence 1.18; 95% CI 1.10–1.28) to be associated with decreased adherence. CVD (odds of nonadherence 0.68; 95% CI 0.66–0.78) and other comorbidities, including hypertension and diabetes, led to increased adherence to therapy.[42]

Conclusion

Appropriate lipid management in older patients provides an important opportunity to address cardiovascular risk. Statins have proven to be the drug of choice because of their potent lipid-lowering effects, low cost, and relatively safe drug profile. Whereas the most recently published guidelines may have modified the clinical approach to risk assessment and management, the critical issue of a patient-centered focus remains at the forefront as we consider cardiovascular risk in older patients. As healthcare providers acknowledge the higher absolute risk of CVD in older patients, they should also acknowledge the benefits of currently appropriate diagnostic and treatment modalities. Although guidelines and randomized clinical trials help inform our approach to patient care, the clinician's clinical judgment, experience, and patient relationships will help truly improve outcomes for the individual.

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Chapter 34

Osteoporosis and Other Metabolic Bone Disorders

Michele F. Bellantoni

Aging and Bone Health

Peak bone mass achieved at about age 25 years is largely determined by genetic factors, although extreme malnutrition, impaired reproductive status, paralysis, and severe chronic illness may cause a significantly lower peak bone mass. Bone is an active organ with a normal physiology of removing older bone and replacing with new matrix to achieve tensile strength through a three-dimensional architecture of cross-linked and calcified proteins.

Aging is associated with reductions in the synthesis of new bone matrix proteins, while menopausal reductions in sex and adrenal hormones for women result in accelerated resorption of existing bone. Age-related male endocrine changes have less clinical impact, as men have greater peak bone mass than women and sex steroid synthesis decreases more gradually than menopausal endocrine changes. Reductions in sun exposure and inadequate vitamin D intake place some older adults at risk for impaired bone mineralization.

Osteoporosis is the most common metabolic bone disorder. The compromised bone architecture and reduced tensile strength result in bone fractures, most commonly in the setting of falls. One in two women and one in five men experience osteoporosis-related fracture.[1] Hip fractures are a significant consequence of osteoporosis and are associated with excess mortality, ranging from 8% to 36% at 1 year, with higher mortality in men than in women. Vertebral and pelvic fractures contribute to gait disorders and cause disabling pain. Severe kyphosis resulting from vertebral fractures in addition to age-related degenerative changes of the spine may contribute to restrictive lung disease, impaired bowel function, limited mobility, and physical frailty.

Osteoporosis Screening

The US Preventive Services Task Force (USPSTF) recommends bone mineral density testing for women >65 years of age, and for younger adult women who have fracture risks comparable to those of an otherwise healthy 65-year-old woman,[2] estimated by the World Health Organization (WHO) Fracture Assessment Tool as a 10-year risk of about 8.4% (forearm, shoulder, clinical spine, and hip fracture). However, the Osteoporosis Self-Assessment Tool calculated as 0.2 × (weight in kg – age in years) <2 is more predictive of low bone density for postmenopausal women aged 50–64 than the Fracture Risk Assessment >8.4%.[3]

The National Osteoporosis Foundation recommends bone mineral density testing in men >70 years of age,[4] although the USPSTF cites insufficient data to recommend screening bone density in men.[2] All agree that postmenopausal women and men over age 50 who experience a low-impact adult fracture of the hip, spine, pelvis, humerus, or forearm should undergo bone mineral density testing and consideration for osteoporosis drug therapies.

Medical conditions and certain drug therapies are associated with bone loss (Table 34.1). Fall risk is a consideration when determining need for bone mineral density testing. Fall risks are associated with neurologic conditions that impair gait and balance, cardiac conditions that predispose to orthostasis, and musculoskeletal conditions that impair gait such as degenerative and inflammatory arthritis and degenerative spine.

Dual energy X-ray absorptiometry is considered the most cost-effective bone mineral density technique,[5] although for older adults, degenerative changes of the spine may give falsely elevated results. The proximal radius and hip are two additional sites for clinical measurement. The WHO defines osteoporosis using only the mean lumbar spine (at least two vertebral bodies), total hip, proximal femur, and proximal radius bone sites. Osteoporosis is defined by bone density criteria as bone mineral density in at least one of these bone sites more than 2.5 standard deviations below normal gender-peak-specific bone mass (T score ≤ –2.5). Normal bone density

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Table 34.1 Medical conditions and therapies associated with bone loss

Endocrine	Gastrointestinal	Hematologic	Neurologic	Other
Diabetes mellitus	Celiac disease	Monoclonal gammopathy	Stroke	Familial hypercalciuria
Central obesity	Inflammatory bowel diseases	Systemic mastocytosis	Parkinson's disease	Chronic kidney disease
Hyperparathyroidism	Short gut syndrome	Chemotherapy	Antiseizure medications	Rheumatoid arthritis
Thyrotoxicosis	Bariatric surgery	Radiation therapy		Systemic steroid therapy
Cushing syndrome	Chronic anti-acid therapy			

is defined by the WHO as a bone density greater than one standard deviation below normal peak bone mass, (T score ≥ –1.0), with osteopenia T score between –1 and –2.5.

Although other technologies exist to measure bone density, dual energy X-ray absorptiometry is preferred based on access, radiation exposure, and cost. Low-dose computerized tomography used for lung cancer screening can also measure bone mineral density of the spine.

The clinical history and physical exam determine the need for additional diagnostic studies beyond bone densitometry. For example, a bone density that is significantly below the expected for age and gender in the setting of comorbid conditions of migraine syndrome and GI symptoms of bloating may prompt studies for celiac disease. Evaluation of hypercalcemia or renal calculi associated with osteoporosis includes serum-intact parathyroid hormone and 24-hour urine collection to assess for hypercalciuria. High serum levels of biomarkers of bone metabolism such as Type I collagen C telopeptide predict high bone turnover state and may guide the clinician in early diagnosis and treatments to prevent irreversible age-related bone loss.[6]

Without additional clinical findings of comorbid medical conditions, further diagnostic studies before offering drug therapies for postmenopausal or age-related osteoporosis include assessment of renal function, serum calcium, and serum 25-hydroxyvitamin D3.

Osteoporosis Treatments

Bone Health Nutrition

A heart-healthy diet may not meet the nutritional needs for bone health because fresh fruits, vegetables, and lean meats contain small amounts of calcium and vitamin D. The addition of low- and no-fat dairy products, plant milk (such as soy and almond milk), calcium-fortified beverages (such as calcium-fortified fruit juices), and foods such as breakfast cereals can achieve a bone-healthy diet without compromising cardiovascular health. Optimal calcium intake is 1,000 mg elemental calcium daily through dietary sources, although older adults taking loop diuretics may require 1,200 mg to account for calcium loss resulting from diuresis. In contrast, thiazide diuretics conserve calcium from renal loss.

For those who cannot obtain adequate calcium intake through diet alone, calcium supplements are available in various formulations – tablets, soft chews, gummies, and dissolvable powders – but may cause gas, bloating, and constipation to a greater extent than dietary sources. Anti-acid drugs, such as H2 blockers and proton pump inhibitors, impair digestion of calcium, although calcium citrate supplements do not require gastric acid for digestion. Clinical trials have demonstrated an association among calcium supplementation but not dietary intake of calcium and increased risk of cardiovascular disease.[7]

Vitamin D deficiency in the elderly can result in deep bone and muscle pain, muscle weakness, and hyperesthesia, as well as fractures from brittle bone that lacks calcium deposition, called osteomalacia.[8] Vitamin D is the hormone that facilitates calcium absorption from the gut. Changes in skin metabolism and decreased sun exposure are thought to contribute to vitamin D deficiency in older adults. Daily intake of 800 units of vitamin D is adequate to prevent vitamin D deficiency for older adults, with higher-dose supplements needed for those with gastrointestinal malabsorption, including those with acquired celiac disease (allergy to gluten, a protein found in wheat and other related grains). Vitamin D supplementation alone showed no significant association with risk of any fracture or of hip fracture, though vitamin D and calcium together have been associated with a 16% reduction of hip fracture.[9]

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Drug Therapies for Osteoporosis

Food and Drug Administration (FDA)-approved drug therapies are grouped into two classes: antiresorptive agents that prevent further bone loss, and anabolic therapies that stimulate new bone protein formation, though to a lesser degree they also stimulate osteoclastic bone resorption, with the exception of romosozumab that decreases bone resorption. The American Association of Clinical Endocrinologists guidelines on osteoporosis drug therapies include first-line agents alendronate, risedronate, zoledronic acid, and denosumab because all have been shown in clinical trials to reduce spine, nonvertebral, and hip fractures (Table 34.2).[10] Alendronate and risedronate are preferred over zoledronic acid and denosumab because of their lower cost and oral administration, though the latter was shown in meta-analysis to increase efficacy.[11]

Postmenopausal women with family history of breast cancer but without increased risk of thromboembolic disease may find that selective estrogen receptor modulator drug therapy addresses both medical conditions. Estrogen replacement, while effective, has long-term risks of breast cancer and increased risk of cardiovascular disease. Testosterone replacement for older men poses risks for cardiovascular health and prostate cancer.

Teriparitide and abaloparatide have greater fracture risk reduction than oral alendronate and risedronate, but with significantly higher cost, potential to cause hypercalcemia, and black box warning labels of osteosarcoma based on rodent toxicology studies. For these reasons, teriparitide and abaloparatide are indicated for high fracture risk or when recurrent fractures occur during treatment with antiresorptive drug therapies. Romosozumab, a monoclonal antibody targeting sclerostin, is highly effective in preventing fractures, but is associated with more serious cardiovascular adverse effects than oral bisphosphonate.[12] Calcitonin is a weak antiresorptive drug that has little clinical utility given the above alternatives.

Prolonged suppression of bone resorption may lead to suppressed bone formation, resulting in fragile bone called osteonecrosis, with clinical consequences of atypical femoral shaft fractures and jaw necrosis following invasive dental procedures.[13] However, the reduction in osteoporotic and hip fractures with antiresorptive medications far outweighs the increased risk in atypical fractures.[14] Oral bisphosphonate therapy is recommended for 5 years, and intravenous zoledronic acid recommended for 3–5 years.[10] Denosumab therapy is not time limited, as accelerated bone resorption can occur 1–2 years after denosumab therapy is stopped.

Over time, the withdrawal from oral bisphosphonates, often referred to as "drug holidays," can be associated with bone loss, as measured by serial bone densitometry. Resumption of oral bisphosphonates after several years of drug holiday is appropriate to stop further bone loss. Serial monitoring at intervals of 6 months to 1 year of serum markers of bone turnover such as Type 1 collagen C telopeptide may be useful in assessing response to drug therapy.[10]

Nonpharmacologic Management of Osteoporosis

Smoking cessation, avoidance of phosphate beverages that bind dietary calcium, and alcohol and caffeine intake limited to two servings per day are nonpharmacologic strategies to promote bone health. High-intensity resistance and impact training improves bone mineral density and physical function in postmenopausal women with osteopenia and osteoporosis.[15] Weight-bearing exercise has been estimated to reduce 20-year relative fracture risks of spine and femoral neck by about 10%. First-line drug therapies reduce fracture risks by 40–60%, though no trial has combined exercise and drug treatment. Physical activity recommendations for cardiovascular health serve bone health as well. Weight training with improper body mechanics risks nerve entrapment syndromes, soft-tissue injuries, and degenerative spine disease. However, weight-bearing exercise techniques are easy to perform safely in the home setting with proper patient education.

Monitoring of Bone Metabolism with Serial Bone Density

Serial bone density for monitoring osteoporosis treatment plans is recommended at 2-year intervals, although 1 year is appropriate in high-turnover states such as hyperparathyroidism or high-dose steroid use. Bone density monitoring for those with screening studies within osteopenia range and who do not require drug therapies should be no more frequent than every 2 years.[10] A normal bone density study need not be repeated within 5 years unless a medical condition associated with bone loss occurs, such as breast cancer requiring antiestrogen therapy.

Other Metabolic Bone Conditions of Older Adults

Four common metabolic bone conditions of older adults associated with increased fractures include osteomalacia

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Table 34.2 Osteoporosis drug therapies

Drug	Class	Route of administration	Fracture risk reduction		
			Vertebral	Nonvertebral	Hip
Alendronate	Bisphosphonate	Oral weekly	Yes	Yes	Yes
Risedronate	Bisphosphonate	Oral weekly or monthly	Yes	Yes	Yes
Zoledronic acid	Bisphosphonate	Intravenous once yearly	Yes	Yes	Yes
Denosumab	Monoclonal antibody against RANK-L receptor	Subcutaneous every 6 months	Yes	Yes	Yes
Ibandronate	Bisphosphonate	Oral monthly or intravenous every 3 months	Yes	*No effect demonstrated	*No effect demonstrated
Raloxifene	Selective estrogen receptor modulator	Oral daily	Yes	*No effect demonstrated	*No effect demonstrated
Teriparatide	Synthetic parathyroid hormone derivative	Subcutaneous daily	Yes	Yes	*No effect demonstrated
Abaloparitide	Synthetic parathyroid hormone-related protein derivative	Subcutaneous daily	Yes	Yes	No effect demonstrated
Romosozumab	Monoclonal antibody against Sclerostin	Subcutaneous monthly for 1 year	Yes	Yes	Yes
Calcitonin	Synthetic salmon calcitonin	Intranasal daily or subcutaneous daily	Yes	No effect demonstrated	No effect demonstrated

* The lack of demonstrable effect at these sites may be due to insufficient sample size for study.

Table 34.3 Metabolic bone conditions of older adults

	Bone pathology	Bone turnover state	Diagnostic criteria	Treatment
Osteoporosis	Excess bone resorption	High	Bone densitometry T score ≤ -2.5	Calcium, vitamin D, and drug therapies
Osteomalacia	Inadequately mineralized bone	Low	Low serum 25-hydroxyvitamin D	Vitamin D
Hyperparathyroidism				
Primary	Excess bone resorption	High	Hypercalcemia and high serum-intact PTH	Surgery; cinacalcet
Secondary	Excess bone resorption	High	Low serum 25 vitamin D	Vitamin D; cinacalcet
Tertiary	Excess bone resorption	High		Surgical resection of parathyroid(s)
Paget's disease	Disordered bone resorption and formation	High	Elevated serum markers of bone turnover X-ray	Bisphosphonates
Renal osteodystrophy	Low bone formation	Low	Chronic kidney disease Low serum markers of bone turnover	Vitamin D; phosphate-binding agents

(inadequately mineralized bone resulting from vitamin D deficiency), hyperparathyroidism (primary, secondary, and tertiary), Paget's disease, and renal osteodystrophy (Table 34.3).

Primary hyperparathyroidism is characterized by excessive production of parathyroid hormone, which leads to hypercalcemia via two mechanisms: (a) osteoclast-medicated bone resorption and (b) calcium conservation at the level of the glomerulus. The incidence in men >60 years of age is 1/1,000/year, twice the frequency for older women.[16] Head and neck irradiation in childhood and long-term lithium therapy are risk factors. More than 80% involve a single adenoma, although four-gland hyperplasia can result from either sporadic disease or as a hereditary multiple endocrine neoplasia Type 1 and 2A. The disease course can vary in severity from asymptomatic hypercalcemia to a syndrome of constipation, hypertension, osteoporosis, peptic ulcer disease, pancreatitis, kidney stones, mood disorders, and cognitive impairment. Additional laboratory findings include decreased or low normal serum phosphate, low serum 25-hydroxyvitamin D due to increased conversion to 1,25-dihydroxyvitamin D, possible elevation in serum alkaline phosphatase, urinary calcium excretion <250 mg/g Cr, and serum-intact parathyroid hormone >80 pg/mL. Radiographs may show diffuse osteopenia, subperiosteal resorption (a salt-and-pepper appearance of the phalanges, distal clavicles, or skull), and osteitis fibrosa cystica (deep areas of bone resorption filled by fibrous tissues). Osteopenia on bone density testing may be more severe at the proximal radius, a site with greater cortical bone than the lumbar spine and femoral neck. Definitive therapy for hyperparathyroidism is surgical resection of the adenoma, considered when either of the following are present: serum calcium >1 mg/dL above the upper limit of normal, bone mineral density T score <2.5, vertebral fracture, creatinine clearance <60 cc/min, nephrocalcinosis, renal calculi, or hypercalciuria >400 mg/dL.[17] Cinacalcet reduces serum calcium >1 mg/dL and bisphosphonates may reduce bone loss.

Secondary hyperparathyroidism results from prolonged and severe vitamin D deficiency, either from inadequate nutrient intake, chronic kidney disease, or gastrointestinal malabsorption syndromes including bariatric surgery and inflammatory bowel diseases. Multiple parathyroid gland hyperplasia may occur in chronic states, though early correction of vitamin D deficiency may prevent progression to clinically significant hyperparathyroidism. Tertiary hyperparathyroidism results from prolonged secondary hyperparathyroidism such as end-stage renal disease and persists even after renal failure is corrected with organ transplantation.

Otitis deformans, or Paget's disease, is a condition of focal areas of active bone turnover. The prevalence of

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Paget's disease ranges from 1–3% in areas of the United States to 10–15% of older Europeans, with a male-to-female ratio of 3 to 2 and 15% estimated to be familial autosomal dominant inheritance with identified genetic mutations involving osteoclast differentiation and function. The diseased bone is deformed, with thickened cortices and course trabeculations resulting in painful skeletal deformity and fractures. The most commonly affected sites include the femur, spine, pelvis, humerus, tibia, cranium, and sternum, causing hearing loss and nerve compression syndromes of the thoracic and lumbar spine. Osteosarcomas, fibrosarcomas, and benign giant cell tumors develop in 2–4% of patients with Paget's disease. The pathogenesis of Paget's disease is unknown, but it is hypothesized that the condition is a late manifestation of an earlier paramyxoviral infection such as measles, respiratory syncytial, or canine distemper viruses. Asymptomatic Paget's disease may present as an elevated serum alkaline phosphatase or as incidental radiographic findings.[18] Serum markers of bone turnover such as C telopeptide and osteocalcin are elevated, particularly when bone formation is markedly increased. Nuclear bone scans using technetium-labeled polyphosphonate or diphosphonate often reveal additional areas of disease not detected on X-ray. The radiographic changes of Paget's disease can be difficult to distinguish from those seen in metastatic prostate or breast cancer. Intravenous zoledronic acid is indicated to treat severe bone pain.[19] Oral bisphosphonate is effective for milder disease. Calcitonin is used when bisphosphonates are contraindicated because of renal insufficiency.

Osteitis fibrosa, or renal osteodystrophy, is inadequately mineralized bone that results from impaired vitamin D metabolism and inadequate calcium absorption due to chronic kidney disease.[20] Simultaneously, cortical bone may experience significant bone resorption due to secondary hyperparathyroidism. Chronic kidney disease is a stronger risk factor for fracture than conventional risk factors such as bone mineral density, sex, race, and age. Bone densitometry may underestimate fracture risk in the setting of chronic kidney disease. Renal clearance of serum biomarkers of bone turnover may be impaired in chronic kidney disease. Antiresorptive drug therapies are contraindicated when chronic kidney disease is associated with low bone formation. In addition, bisphosphonates and denosumab are contraindicated when creatinine clearance falls below 30 mL/min, and parathyroid hormone derivatives are not used when there is secondary hyperparathyroidism associated with vitamin D deficiency. Current treatment strategies are management of vitamin D deficiency, optimization of calcium through calcium phosphate binders, resistance exercise, and fall prevention.

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Chapter 35

Common Rheumatologic Disease

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As the population of the United States ages, rheumatologic disease is quickly becoming one of the more common diagnoses encountered in the geriatric population. According to the Centers for Disease Control and Prevention, approximately 54.4 million (22.7%) US adults had doctor-diagnosed arthritis, and 23.7 million (43.5% of those with arthritis) had arthritis-attributable activity limitations.[1] In addition to increasing prevalence, disability associated with musculoskeletal disorders is increasing. One study evaluating the burden of disease found that worldwide disability associated with musculoskeletal disorders had more than tripled since 2004.[2] With this increased burden of disease, there is a necessity for these diseases to be managed with an interdisciplinary approach. Through appropriate partnership of physical therapists, occupational therapists, physiatrists, orthopedic surgeons, and pain specialists, rheumatologic disease in the elderly can be effectively managed.

Osteoarthritis

Osteoarthritis is the most common type of arthritis. Over 30 million individuals in the United States have osteoarthritis, which has increased from 21 million in 1990 and 27 million in 2005.[3] Although osteoarthritis was once thought to be a normal consequence of aging, it is now felt to have more of a multifactorial etiology. It results from a complex interplay of a patient's age, genetics, bone mass, hormonal influences, occupation, physical activity, and injuries.[4]

Clinical Presentation

Patients with osteoarthritis classically report joint pain that increases at night and with use. They may also have increasing stiffness after prolonged immobility; however, this stiffness should last for 30 minutes or less. Patients may suffer from primary and/or secondary osteoarthritis. Primary osteoarthritis most commonly affects the hands, spine, hips, knees, and feet. Secondary osteoarthritis can affect any joint. Secondary osteoarthritis results from an underlying condition such as previous trauma, congenital disorders, endocrinology disorders (diabetes mellitus, acromegaly, hypothyroidism, hemochromatosis), or inflammatory arthritis (calcium pyrophosphate dehydrate deposition disease, rheumatoid arthritis, gout).

The physical examination of a patient with osteoarthritis may reveal tender joints. Patients may also have enlarged joints from bony osteophyte formation or from joint effusions; however, the joints are neither markedly warm nor erythematous. Warmth or erythema should lead to an evaluation for inflammatory causes of the patient's joint pain. Aspiration of joint fluid can sometimes be helpful in making the diagnosis. Synovial fluid from a joint with osteoarthritis will be noninflammatory, with less than 2,000 WBC/mm^3. There are no laboratory tests or biomarkers that are helpful in establishing the diagnosis of osteoarthritis. Although osteoarthritis is primarily a clinical diagnosis, radiographs can be helpful to confirm the diagnosis and rule out more serious causes of pain such as fracture or malignancy.

Hand Osteoarthritis

Elderly women are at higher risk for development of hand osteoarthritis. Patients with osteoarthritis of the hands commonly have bony enlargement of the joints. The most commonly affected joints are the proximal interphalangeal (PIP) joints, distal interphalangeal (DIP) joints, and first carpometacarpal joints. Patients often report that the symptoms are increased in their dominant hand. Pain may significantly interfere with performance of the activities of daily living. A significant proportion of patients with hand osteoarthritis may have erosive disease. These patients may have striking bony proliferation, erythema, or warmth on physical examination. This presentation may often be confused with an inflammatory arthritis but is degenerative in its pathophysiology. Radiographs can sometimes be helpful in differentiating between these diagnoses. Radiographs of patients with

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hand osteoarthritis classically show joint space narrowing with subchondral sclerosis and marginal osteophytes. Radiographs of erosive osteoarthritis may also show subchondral erosions or a "gull wing" appearance of the distal interphalangeal joints.

Spine and Hip Osteoarthritis

The cervical and lumbar spine are the most common sites of spine osteoarthritis. Patients may relate pain, stiffness, and difficulty with movement of these areas. They may also have radicular symptoms if nerve root impingement or spinal stenosis exists. Imaging of the spine may show osteophytes resulting in foraminal narrowing, spinal stenosis, or spondylolisthesis.

Hip osteoarthritis classically presents as referred pain to the groin and thigh, but the pain can also radiate to the buttock and knee. Patients usually report that the pain increases with walking but also with prolonged immobility. As the disease progresses, patients may demonstrate decreased range of motion. Radiographs of the hip in osteoarthritis will often show joint space narrowing with osteophyte formation.

Knee Osteoarthritis

The knee is the most common site of joint pain in the older adult. Risk factors for osteoarthritis of the knee include increased body mass index (BMI), previous knee injury, smoking, older age, intensive physical activity, female gender, and presence of hand osteoarthritis.[5] Patients with osteoarthritis of the knee usually report pain that is increased with ambulation, but they can also have stiffness after prolonged immobility. It may also lead to other joint strain secondary to an abnormal gait. Other sources of pain of the knee such as mechanical derangement, anserine bursitis, fracture, or referred pain from the hip or spine should be considered before making a diagnosis of osteoarthritis. Physical examination of a knee affected by osteoarthritis may show crepitus, bony proliferation, and valgus or varus deformity. It may also reveal joint swelling, but the synovial fluid will be noninflammatory with a white blood cell (WBC) count less than 2,000 WBC/mm^3. Radiographs may show joint space narrowing that is present more often in the medial joint compartment. They are also likely to show osteophytes.

Foot Osteoarthritis

While any joint of the feet can be affected by osteoarthritis, the first metatarsophalangeal (MTP) joint is the most commonly involved. Older age and female sex are independent risk factors for developing arthritis of this joint.[6] Patients classically have the physical finding of hallux valgus with lateral deviation at the first MTP joint. Hallux valgus may also result in formation of an inflamed bunion at the site, which may be confused with gout. Radiographs can help differentiate between the two, with osteoarthritis of the first MTP showing the classic joint space narrowing and osteophytes.

Treatment

Patients with osteoarthritis are treated with a combination of nonpharmacologic and pharmacologic modalities. Exercise should be encouraged, especially with knee osteoarthritis, as studies suggest that exercise, in combination with weight loss, is more effective in pain reduction than weight loss alone.[7] Involvement of occupational and physical therapists is encouraged. Splints, taping, orthotics, and wedged insoles can be helpful adjuncts in treatment.

Pharmacologic treatments for osteoarthritis do not change the natural history of the disease; rather, they focus on management of pain. Acetaminophen is the first-line treatment for osteoarthritis, with doses up to 4,000 mg daily showing significant reduction in osteoarthritis pain when compared to placebo.[8] More recent recommendations suggest a maximum of 3,250 mg of acetaminophen daily to decrease the likelihood of adverse effects.[9] If nonpharmacologic treatments and acetaminophen do not help with the pain, topical nonsteroidal anti-inflammatory medications (NSAIDs) are often preferred in the elderly over oral NSAIDs.[10] Oral NSAIDs can be used in the elderly with careful attention to the possible side effects such as gastrointestinal bleeding and hypercoagulability. Treatment with opioid analgesics may sometimes be necessary but should be used sparingly in the elderly because of increased falls and fractures.[11] Supplementation with glucosamine chondroitin sulfate remains controversial, as large-scale studies have shown conflicting results. In the 2019 American College of Rheumatology/Arthritis Foundation Guideline for the Management of Osteoarthritis of the Hand, Hip, and Knee, duloxetine is conditionally recommended, as evidence suggests this medication may have efficacy when used alone or in combination with NSAIDs.[12]

Intra-articular injections of corticosteroids or hyaluronic acid often provide relief for patients who have failed nonpharmacologic and pharmacologic treatment. Any joint can be injected with corticosteroid, but the knee

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is the most common site. Studies have demonstrated that a large majority of patients will have relief with intra-articular steroid injections to their knee.[12] Unfortunately, intra-articular steroid injections with steroids is not curative, and the pain is likely to return. Injection with hyaluronic acid is primarily performed in the knee joint. Results from trials regarding the efficacy of viscosupplementation to the knee with hyaluronic acid have been conflicting, with some trials suggesting benefit and others finding it no more effective than intra-articular placebo.[13]

Surgery is an option for some patients who have failed to respond to nonoperative treatments. Joint replacement of the knee and the hip are the most common surgical treatments of osteoarthritis. As these are complicated surgeries associated with significant perioperative morbidity, elderly patients must be fully medically evaluated before surgery is performed.

Rheumatoid Arthritis

Rheumatoid arthritis (RA) affects between 0.25% and 1% of the world's population[14] and, in patients older than 60, the overall prevalence may be as high as 2%.[15] Females are affected more often than males, and, although disease onset may occur at any age, approximately one third of all RA patients are >65 years of age.[16]

Clinical Presentation

RA is a symmetric inflammatory polyarticular arthritis that classically affects the small joints of the hands and feet. It may also affect medium- and large-size joints; however, this is less typical than the small joint pattern. Patients usually describe stiffness that is increased in the morning, joint pain, and swelling, and, often, constitutional symptoms. RA may present indolently over several months or may present acutely. The majority of patients with RA have a positive rheumatoid factor, but this is neither specific nor sensitive for the diagnosis of RA. Patients may also have a positive antibody to cyclic citrullinated peptide (CCP antibody). CCP antibody has a high specificity for the diagnosis of RA but has a lower sensitivity. Radiographs early in RA may be normal. As the disease progresses, one may see periarticular osteopenia and juxta-articular erosions.

As RA often presents in younger people, many elderly individuals with RA have been living with this disease for years. Patients without sustained access to disease-modifying antirheumatic drugs (DMARDs) may have serious morbidity from their RA. Patients with longstanding RA may have ulnar deviation of their hands, subluxation of numerous joints, swan neck and boutonniere deformities, joint contractures, and rheumatoid nodules. They may have undergone numerous orthopedic surgeries. With longstanding disease, patients may also suffer from extra-articular manifestations of RA such as uveitis, scleritis, pulmonary fibrosis, and cardiovascular disease.

Elderly-onset RA occurs in patients older than 60 years of age and occurs equally among males and females. Symptoms are more likely to appear abruptly as opposed to insidiously. Large joints such as the shoulder are also more likely to be involved. Patients with elderly-onset RA are less likely to have a positive CCP antibody.[17]

Treatment

The trend in the treatment of RA is to treat the disease early and aggressively. Tight control with DMARDs should be started as soon as possible.

Oral medications are sometimes preferred by the geriatric patient. Nonsteroidal and anti-inflammatory medications and corticosteroids may be used to help with pain; however, as these medications are not DMARDs, they should only be used as adjunctive therapy. Initial treatment with hydroxychloroquine or sulfasalazine may be considered for patients with very mild disease or for those with many comorbidities. The majority of patients with a new diagnosis of RA start with weekly oral methotrexate and daily folic acid supplementation. Methotrexate is generally well tolerated by the elderly; however, the weekly dosing schedule may sometimes be confusing to patients. If tight control is not achieved within the first 6–12 weeks, additional medications are added to the existing regimen. So-called triple therapy (hydroxychloroquine, sulfasalazine, and methotrexate taken together) has proved noninferior to methotrexate and etanercept together in one study.[18] Leflunomide is another oral medication that is often used by itself or in combination with other medications for RA.

Several oral Janus kinase (JAK) inhibitors – tofacitinib, baricitinib, and upadacitinib – have been approved by the Food and Drug Administration (FDA) for use in RA. However, among other potential side effects, thrombosis, including deep vein thrombosis, pulmonary embolism, and arterial thrombosis, has occurred in patients treated with JAK inhibitors, thus this may be a concern in elderly patients with risk factors for or prior history of thrombotic episodes.

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If oral medications do not control the disease or if they cannot be tolerated, biologic medications in subcutaneous and intravenous routes of administration are a potential option. The majority of biologic medications work best when used with oral DMARDs. Vaccinations such as pneumococcal, influenza, hepatitis B, and herpes zoster should be initiated whenever possible before the biologic medications are prescribed. Screening for tuberculosis should also occur before initiation of a biologic medication, as these medications are associated with reactivation of latent tuberculosis. Tumor necrosis factor (TNF) alpha inhibitors are the most commonly used biologic medications for RA. Adalimumab, certolizumab, etanercept, golimumab, and infliximab are FDA-approved for the treatment of RA. The choice between these medications is usually made based on patient preference and includes issues such as route of administration, frequency of administration, and cost. Although elderly patients may seem to be at higher risk for adverse events, studies have demonstrated that these medications are as safe in the elderly as they are in the general population.[19] Other options for patients who fail or cannot take TNF alpha inhibitors include rituximab, abatacept, tocilizumab, and sarilumab.

Crystal-Induced Arthritis

Deposition of monosodium urate crystals (gout) or calcium pyrophosphate crystals (pseudogout) are the most common causes of crystal-induced arthritis. Another cause of crystal-induced arthritis in the elderly, albeit less common, is that induced by calcium phosphate hydroxyapatite crystals.

Gout

Joint pain induced by monosodium urate crystals most commonly affects men and postmenopausal women older than 65 years of age.[20] There is an increasing risk for gout as one grows older because of increased hyperuricemia. Hyperuricemia is defined as a uric acid level greater than 7 mg/dL. Etiologies of hyperuricemia are likely to be multifactorial but may stem from older age, increased use of diuretics and aspirin, and a higher prevalence of comorbidities such as hypertension, congestive heart failure, and renal failure. Patients may remain asymptomatic with hyperuricemia for many years before their first attack of gout; however, the degree of the hyperuricemia is directly linked to their risk of gout.[21] As the uric acid rises greater than 7 mg/dL, the uric acid crystals can become insoluble and deposit into the joints or soft tissues, initiating a gout attack.

Clinical Presentation

The majority of first gout attacks present with involvement of the metatarsophalangeal joints, foot, or ankle. However, in the elderly, a first attack of gout may be polyarticular or involve the hands. Once the acute attack has resolved, patients enter into the intercritical period where they are asymptomatic from gout. A large majority of patients will eventually experience another acute attack of gout. Over time, if patients remain untreated, the intercritical period shortens and gout can become chronic. Elderly patients with chronic gout may have diffuse tophi, which are sometimes misinterpreted as osteoarthritis.

The definitive diagnosis of acute gout should be established by direct visualization of intracellular monosodium urate crystals under polarized microscopy. Polarized microscopy will demonstrate strongly negative birefringent crystals. In intercritical or chronic gout, the crystals may still be present but not intracellular. Imaging of early acute gout may only show soft-tissue swelling or the presence of a joint effusion. Classic radiographic features such as erosions with sclerotic margins and overhanging edges may not be present until many years after the first attack of gout. Visualization of tophi on imaging may also take several years.

Treatment

The treatment of gout in the elderly is essentially the same as that of the general population with the caveat that careful attention must be paid to medication side effects. Colchicine or NSAIDs are most commonly used for the acute episodes. Colchicine is usually prescribed according to renal function and provider discretion. NSAIDs may also help with the inflammation of gout; however, they must be used cautiously in the elderly population as geriatric patients are more likely to experience gastrointestinal, renal, and cardiovascular side effects from NSAIDs.[22] If colchicine or NSAIDs are not tolerated or do not control the symptoms, intra-articular or oral corticosteroids can be used.

Patients who have chronic frequent gout attacks, a severe polyarticular presentation of acute gout, or tophaceous deposits will benefit from medications to lower their uric acid levels. A serum uric acid level below 6 mg/dL is associated with less frequent gout attacks.[23] Allopurinol, febuxostat, probenecid, and pegloticase are FDA-approved treatments for lowering uric acid in gout. Allopurinol and febuxostat both inhibit xanthine oxidase. Although rare, allopurinol (and not

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febuxostat) is associated with a severe hypersensitivity syndrome; this allergic reaction is most common in patients with the allele human leukocyte antigen (HLA)-B*58:01 in Han Chinese, Japanese, Korean, Thai, and other Asian populations as well as European populations.[24] Febuxostat is an option for those who are allergic to allopurinol. It is also approved for use in patients with renal dysfunction as long as the creatinine clearance is greater than 30 mL/min. Recently, the FDA has added a black box warning for febuxostat stating that it is associated with an increased risk for cardiovascular death with the drug. The warning also states that febuxostat use should be limited to patients who do not respond to or cannot tolerate allopurinol.[25] Probenacid is an uricosuric agent; however, as it is associated with increased nephrolithiasis and cannot be used in patients with renal dysfunction, it is not often prescribed for the elderly. Pegloticase is effective in rapidly lowering serum uric acid levels, but its use has been limited because of high cost and the potential to develop antibodies to the medication, rendering it ineffective by that point.

Elderly patients with gout may also suffer from the effects of polypharmacy. As the use of diuretics may increase serum uric acid, efforts should be made by physicians to try to limit the use of these medications in gout patients. Aspirin can also increase serum uric acid levels; thus, it is important to ensure that patients with gout who are taking aspirin have a definite need for this medication.

Calcium Pyrophosphate Deposition Disease

According to one study, almost 50% of patients older than 84 years of age have evidence of calcium deposition in articular cartilage (i.e., chondrocalcinosis) on radiographs of knees, hands, wrists, and pelvis.[26] There are also metabolic and congenital conditions associated with chondrocalcinosis such as hypothyroidism, hypomagnesemia, hyperparathyroidism, and hemochromatosis. Whereas the majority of patients with radiographic evidence of chondrocalcinosis will remain asymptomatic, some patients may demonstrate an acute inflammatory arthritis from precipitation of calcium pyrophosphate crystals. This acute flare of disease is termed pseudogout.

Clinical Presentation

An attack of pseudogout may be monoarticular or polyarticular. It may affect any joint but is more common in medium and large joints. It may be severe enough to be confused with RA. Trauma, surgery, or severe medical illness can sometimes precipitate an attack. The diagnosis of calcium pyrophosphate deposition disease (CPPD) is made through assessment of synovial fluid under polarized microscopy. One should see weakly positive birefringent rhomboid-shaped crystals. An arthrocentesis should show joint fluid that is inflammatory with a WBC greater than 2,000 mm^3. Inflammatory markers are likely to be elevated. Radiographs may show chondrocalcinosis, but this is not specific for the diagnosis, as it can be found in asymptomatic individuals.

Crowned dens syndrome (CDS) is a clinical and radiological entity that manifests with neck pain and calcifications from calcium pyrophosphate dihydrate crystals superior to and surrounding the dens of the axis, which results in a "crown-like" appearance on coronal views. The presentation of CDS can be accompanied by fever and other symptoms that may result in the condition mistakenly being diagnosed as meningitis. These patients can be quite ill; prompt recognition of the condition is important and treatment with high-dose steroids is indicated.

Treatment

There are a few randomized controlled trials showing the benefit of pharmaceutical treatments for CPPD. NSAIDs are usually the first oral medication to be used. Colchicine is often prescribed in the acute setting and for prophylaxis, but the evidence behind this practice is limited.[27] Other options for treatment of pseudogout include intra-articular or oral steroids.

Basic Calcium Phosphate Hydroxyapatite Deposition Disease

Deposition of basic calcium phosphate hydroxyapatite crystals often causes a highly destructive arthritis of the shoulders bilaterally. This is seen most often in elderly women and named the Milwaukee shoulder. Synovial fluid may be serosanguinous but, under light microscopy, one may see basic calcium phosphate hydroxyapatite crystals. Radiographs of the shoulder may show severe glenohumeral degeneration with displacement of the humeral head and periarticular calcifications. These patients may be treated with NSAIDs and corticosteroids, but the disease may be difficult to control.

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Polymyalgia Rheumatica/Giant Cell Arteritis

Polymyalgia Rheumatica

Polymyalgia rheumatica (PMR) is a disease that is found only in older adults. As one ages, the incidence of PMR also rises, with a peak incidence in the eighth decade of life. Women are affected more often than men.[28] People of northern European descent have a significantly higher risk of being affected, but PMR has been seen in all racial and ethnic groups.[29]

Clinical Presentation

Patients with PMR classically report pain of the shoulders and pelvic girdle. These symptoms usually occur along with fatigue and morning stiffness. Physical examination usually reveals a patient in severe discomfort at rest; however, besides this finding, the physical examination may be normal.

Although PMR is quite common in the elderly population, as there are no specific diagnostic tests for this condition, there is considerable variation in diagnosis and treatment. The majority of patients with PMR have an erythrocyte sedimentation rate (ESR) greater than 40 mm/hr and an elevated C-reactive protein (CRP), but some patients may also have normal inflammatory markers.[30] In cases in which imaging is used for diagnostic testing, magnetic resonance imaging (MRI), ultrasound, and positron emission tomography (PET) may show subacromial bursitis or interspinous bursitis, but these findings are somewhat nonspecific.[31]

Treatment

Responsiveness to treatment with low-dose prednisone has been one of the hallmarks of a diagnosis of PMR. Patients with PMR are usually slowly tapered off the prednisone within 1 year. Relapses are common and may necessitate increasing the dose and duration of prednisone. The 2015 Recommendations for the Management of Polymyalgia Rheumatica from the European League Against Rheumatism/American College of Rheumatology Collaborative Initiative recommend considering low-dose methotrexate (7.5–10 mg weekly) in addition to glucocorticoids in patients at high risk for relapse or on prolonged steroid therapy.[32]

Giant Cell Arteritis

Giant cell arteritis (GCA) is also found exclusively in older adults. Although it may be diagnosed in patients as young as 50 years of age, recent studies have suggested that the mean age of GCA diagnosis is approximately 76 years of age.[33] Similar to PMR, GCA is diagnosed more often in patients of northern European descent.

Clinical Presentation

Patients with GCA present with symptoms related to arterial insufficiency. As the temporal artery can often be affected, in the past GCA was also referred to as temporal arteritis. As it is now clear that GCA can affect numerous other vessels, the disease is more appropriately referred to as GCA. Patients classically present with a combination of headache, jaw claudication, and, possibly, vision loss. Vision loss may occur as a result of ischemia of the retina, choroid, or optic nerve. Patients are also likely to have constitutional symptoms such as fevers and weight loss. They may also have symptoms of PMR. As the aorta is often involved in GCA, patients may have symptoms of lower-extremity claudication. Physical examination may be normal or may show artery abnormalities such as enlargement, tenderness, or an absent pulse.

The majority of patients with GCA will have an elevated ESR. It may be as high as 100 mm/hr or higher, though in a small minority of patients, the ESR may be normal.[34] MRI, ultrasound, CT, and PET have also been used in the diagnosis of GCA, but temporal artery biopsy remains the gold standard for diagnosis.

Treatment

As vision loss can occur rapidly and irreversibly, treatment with 60 mg of prednisone should be initiated quickly if one has a high clinical suspicion. If vision is threatened, high-dose pulse intravenous methylprednisolone should be considered. Steroids are typically tapered slowly to avoid relapse, with patients often requiring steroid treatment for 1–2 years.

In terms of additional treatment options, tocilizumab is a recombinant humanized monoclonal antibody that acts as an interleukin-6 (IL-6) receptor antagonist that has been approved by the FDA for treatment of GCA. A landmark 2017 study demonstrated higher rates of sustained steroid-free remission at 52 weeks in GCA patients treated with tocilizumab versus placebo, and

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this study has supported the use of the medication as a steroid-sparing agent early in the course of disease.[35]

Remitting Seronegative Symmetric Synovitis with Pitting Edema

Remitting seronegative symmetric synovitis with pitting edema (RS3PE) is a disease of people older than 50. Patients typically present with acute pain and subcutaneous swelling of the dorsum of the hands. They may also have a polyarthritis that classically affects the small joints of the hands but may affect any joint. Laboratory tests may reveal elevated inflammatory markers. Imaging may show subcutaneous edema and tenosynovitis, as well as synovitis.[36] Some feel that this syndrome may be a subset of RA or PMR. Low-dose prednisone is very effective in treating this condition.

Sjogren's Syndrome

Sjogren's syndrome is the most common medical cause of xerostomia in the elderly. The incidence of Sjogren's syndrome in the elderly is not known; however, estimates of the prevalence range between 1% and 14%.[37]

Clinical Presentation

Patients with Sjogren's syndrome usually report dry eyes and dry mouth. There can also be systemic manifestations with neurologic, pulmonary, musculoskeletal, gastrointestinal, hematologic, and renal involvement. Diagnosis can be difficult but usually involves a combination of autoantibodies (anti-SSA and SSB), ocular staining, and, possibly, labial salivary gland biopsy. A Schirmer test may also be used. This involves placing a piece of paper in the lower lid of the eye. After a specified time, the paper is removed and evaluated for wetness. This test is not specific for Sjogren's syndrome. Other diagnostic tests often used to evaluate patients with sicca include salivary and parotid gland scintigraphy and sialometry. The elderly with Sjogren's syndrome must also be carefully evaluated for the development of lymphoma – marginal zone lymphoma or mucosa-associated lymphoid tissue (MALT)-type lymphoma, which in all patients with Sjogren's syndrome is significantly higher than in the normal population.[38]

Treatment

Patients with Sjogren's syndrome should try to avoid using antidepressants, antihistamines, anticholinergics, and some neuroleptic medications, as these medications can exacerbate symptoms. Salivary and tear substitutes can help with sicca. Muscarinic agonists such as pilocarpine and cevimeline can also be used to stimulate secretions.

Inclusion Body Myositis

Inclusion body myositis (IBM) is an idiopathic inflammatory myopathy. It is rare in the general population, with an estimated prevalence of 0.49–1.07 per 100,000 in Olmstead County, Minnesota.[39] Although IBM is not frequently encountered in the general population, it is the most common muscle disease of aging. The majority of patients are older than 50 and are more likely to be male.[40] Patients usually present with an insidious onset of muscle weakness, often initially involving the proximal lower extremities first before becoming widespread. The pattern of muscle involvement can be symmetric or asymmetric. Muscle enzymes can be normal or only mildly elevated. Markers of systemic inflammation are normal. Autoantibodies to cytosolic 5'-nucleotidase 1A (cN1A) have recently been identified as a specific finding in IBM patients.[41] Electromyography and MRI can be helpful for diagnosis, but muscle biopsy remains the gold standard. A biopsy should show extensive endomysial inflammation with inflammatory cells surrounding myofibers. Rimmed vacuoles may also be visualized in the myofibers on Gomori trichrome stain. Electron microscopy should show filamentous inclusions and vacuoles. IBM is often resistant to treatment. High-dose prednisone is usually prescribed for several months; however, if the patient does not respond to treatment, it is discontinued. Methotrexate and azathioprine are also sometimes used, although data is limited regarding their success.[42]

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Chapter

36 Geriatric Emergency Medicine

Christine Binkley, Katherine Selman, and Kevin Biese

Case: *Mrs. Smith is an 86-year-old woman presenting to the emergency department by ambulance after a fall from standing in her kitchen. She has a forehead laceration and contusions to her head. The patient is pleasant, stating that she turned too quickly while making her evening meal when she fell. She explains, "These things just happen to me sometimes. I'm not sure why, but I think it's part of getting old." She appears alert and oriented x3 without any other complaints or injuries. In a traditional emergency department, she is evaluated with a head and neck CT. Her imaging studies result negative, and her wound is repaired. She lives with her daughter, who has been called to pick her up. Mrs. Smith is instructed to follow up with her primary care doctor in 3–5 days for wound recheck and to discuss the use of a walker.*

Introduction

The emergency department (ED) is a critical resource in the evaluation and treatment of acute medical conditions, with a growing proportion of visits from older adults aged 65 and over. In the USA, approximately 15% of all annual ED visits are made by patients older than 65, with more than one ED visit for every two adults over age 65 per year.[1,2] The geriatric population is forecast to surpass the pediatric population by the year 2034, with 20% of the population over the age of 65.[3] Because of this rapidly growing population, the need for all health services will increase, and the demand for emergency services will likely be disproportionately affected. The need for innovations specific to caring for older adults has become a growing focus within the practice of emergency medicine.

The ED is traditionally a fast-paced environment with a focus on rapid assessment of patients for acute resuscitative needs or time-critical therapies and to ultimately determine if a patient requires admission for their illness or injury. EDs were designed with the fundamental goal of treating a specific event, such as a heart attack or broken hip from a fall, while leaving the task of further investigation into the possible underlying syndromes that led to the event to their primary care doctors and geriatricians. Much of the work of the geriatric ED is to stabilize and treat the acute event while simultaneously identifying and addressing key elements of these underlying geriatric syndromes in a time-efficient way, and then transitioning the care to the outpatient setting. The purpose of this chapter is to highlight key topics in geriatric emergency medicine, focusing on unique considerations for the management of emergency care for the older adult, the development of geriatric emergency medicine, the different characteristics of a geriatric ED, and areas of current and future research.

Geriatric-Specific Issues in the ED

EDs are typically designed to maximize throughput for lowest cost, which translates into crowded departments fitted with bright fluorescent lights, slippery floors that are easy to sterilize, and thin mattresses on hallway beds to maximize capacity, with limited space for visitors. While younger patients are usually accepting of the sacrifices to their privacy and comfort in exchange for being seen as quickly as possible, these seemingly minor details in comfort and environment can lead to complications in older adults, including delirium, disorientation, falls, worsening pain, and possibly increasing frailty.

Older adults require more time for a thorough evaluation because of their often atypical and complex presentations. Instead of one acute medical complaint triggering the ED visit, they typically have a combination of functional and cognitive impairments, polypharmacy, comorbidities, and other unique age-specific multifactorial geriatric syndromes that complicate their clinical picture. Furthermore, social needs, such as the need for increasing caregiver support, are frequently driving factors resulting in an ED visit. As an important rule of thumb, it is vitally important to talk to a close family member or caregiver when possible to bridge gaps in the initial assessment of older adults.

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Geriatric patients are also more likely to present with vague, nonspecific complaints that are signs of serious pathology with higher rates of morbidity and mortality than younger patients. This leads to more extensive and lengthy workups in the ED, as well as higher rates of admission. In 2015, the percentage of ED visits that resulted in hospital admissions for patients aged 65 or older was 33.6% compared to an average overall admission rate of 15.2%.[2] With the current trajectory, admissions for this older population are expected to increase to over 50% by 2030.[4]

Upon discharge, older adult patients are at increased risk for readmission secondary to lack of access to necessary medications or follow-up visits. They are also at greater risk for medical complications and decline in both function and health-related quality of life after discharge from the ED.[5] With the complexity of care required and increased vulnerability in older adults, interventions focused on the specific needs of older adults are critical to providing optimal care across all healthcare settings and specialties, including the ED.

History of Geriatric Emergency Medicine

Given the projected growth in the older population in combination with the relative dearth of geriatricians, there has been significant interest in the development of geriatric expertise among other medical specialties. Studies have shown that not only is the older population increasing, but use of EDs by older adults is increasing at a rate greater than the increase of population.[6] Additionally, older adults are more likely to be admitted from the ED and more likely to require admission to the intensive care unit, indicating a greater resource use among this population.[6]

In 1992, the Society of Academic Emergency Medicine (SAEM) formed the Geriatric Emergency Medicine Task Force to examine care of older patients in EDs. This initial task force concluded that the needs of older adults, related both to medically complex care and psychosocial issues, were not being sufficiently researched or acknowledged at the time.[7] As a proposed solution, the task force recommended geriatric emergency care centers as a potential model to focus on the specific needs of and resources required by older adults.[7]

Similarly, a 1992 survey of American College of Emergency Physicians (ACEP) members demonstrated that physicians reported increased difficulty in management of older adults and inadequate training on geriatric-specific issues both during residency and as part of continued medical education programs.[8] A recreation of this survey in 2007 demonstrated similar results and showed persistent perceived difficulty in the treatment of older adults and unsatisfactory geriatric-specific training.[9] In response to the deficits in training, in 2009 the document Geriatric Competencies for EM Residents was published in order to specifically address skills for emergency medicine residents related to geriatric ED patients.[10] The competencies are divided into eight domains: atypical presentation of disease, trauma including falls, cognitive and behavioral disorders, emergent intervention modifications, medication management, transitions of care, pain management and palliative care, and effect of comorbid conditions.

While the first self-identified geriatric emergency department (GED) opened in 2008, in 2014 a multidisciplinary task force from ACEP, the American Geriatrics Society (AGS), the Emergency Nurses Association (ENA), and SAEM published the first formalized guidelines for GEDs. The GED guidelines outline key elements that GEDs should incorporate into practice to optimize care and to meet the needs of older patients. The guidelines are divided into six general categories: staffing; transitions of care; education; quality improvement; equipment and supplies; and policies, procedures, and protocols.[11] The team behind the GED guidelines, in partnership with the West Health Institute and John A. Hartford Foundation, then created the Geriatric Emergency Department Collaborative (GEDC: https://gedcollaborative.com) as a learning collaborative to discover, pilot, train, and disseminate best practices in geriatric emergency care.

Building on these guidelines, and in order to standardize care further, ACEP has developed a three-tiered accreditation process for GEDs that was first published in 2017, with the first sites becoming accredited in 2018. The accreditation provides additional structure and standardized criteria for defining GEDs, beginning with a "Bronze" Level III and progressing to a "Gold" Level I. A Level III center is expected to have at least one physician and one nursing champion, a geriatric-specific care initiative, and availability of mobility aids, as well as 24/7 access to food and drink. Level II centers require additional staff such as a case manager, physical therapist, and social worker. Level II centers also need at least 10 specific policies, guidelines, and procedures in place as well as quality improvement measures for these policies. Equipment and supplies specific to the care of older adults are expected to be incorporated into the GED (e.g., pressure-ulcer reducing mattresses). Finally, a Level

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I center includes an interdisciplinary geriatric assessment team and either a separate space for the GED or a plan for space preferentially caring for older adults, with physical environment adjustments such as enhanced lighting. Level I sites have also implemented 20 geriatric-specific care initiatives. As of May 21, 2020, there were over 150 accredited GEDs throughout the United States, the majority of which were Level III facilities. Current GED interventions have led to increased rates of discharge, reductions in hospital length of stay, and overall lower costs per hospital admission.[5,12,13,14]

Aspects of a Geriatric Emergency Department

Many people imagine a GED as a stand-alone entity that only cares for patients aged 65 or older, but it is often most useful to incorporate these initiatives into an existing department's workflow. There are four general aspects to making any ED friendlier for older adults: (1) structure, (2) training staff in the core principles, (3) processes and screenings for underlying syndromes, and (4) connections to the community. These key features help to build a cohesive model for the effective and efficient care of older adults in the ED.

Structure

The layout and design of a GED focuses on improving safety, mobility, comfort, memory cues, and sensorial perception for older adults while decreasing the risk of iatrogenic complications during their stay. Examples include the use of soft and natural lighting rather than harsh fluorescent lights, flooring that does not have contrasting patterns, and bright colors on signage to help with visual function, as well as extra-thick foam mattresses, sturdy chairs and recliners, and access to bedside commodes. Many of these changes also improve the quality of ED visits for patients of all ages and can be added to existing departments for relatively modest cost. A full list of possible recommendations is available on the GEDC website.

Training

Training staff in core principles of geriatric-focused emergency medicine is imperative in creating a cohesive geriatric-friendly ED. The Geriatric ED Collaborative describes eight principal domains for this education including atypical presentations of disease, trauma and falls, cognitive and behavioral disorders, emergency intervention modifications, medication management and polypharmacy, transitions of care, the effect of comorbid conditions, and end-of-life care. Beyond training existing staff, the creation of an interdisciplinary team by bringing in other key players offers a more integrated experience in the ED for older adults. These players include providers trained specifically in geriatric-focused emergency medicine as well as physical and occupational therapists, pharmacists, geriatricians, palliative care providers, social workers, case managers, and patient navigators.

Processes/Screening for Underlying Syndromes

EDs are generally focused on gathering the one reason for the visit: the "chief complaint." However, for older adults it is also imperative to identify background geriatric syndromes and social support needs that may put this patient at risk for poor outcomes after ED or hospital discharge. Screenings for syndromes such as dementia, delirium, fall risk, polypharmacy, and elder abuse in the ED provide an opportunity to address these underlying, and often underidentified, syndromes. While studies show mixed results on the use of these prognostic screening tools, it is believed that a simple, team-driven screening tool can be instrumental in preventing poor outcomes and improving the overall health-care experience for the elderly patient.[11]

Connection to the Community

A traditional ED is the front door of the hospital. A geriatric ED should be seen as the front porch where people can come when they have acute needs but, when possible, are referred back to the community for additional support. This can be accomplished through a variety of programs and interventions. Case managers, social workers, and/or patient care navigators can assist with arranging follow-up appointments, home health or physical therapy, and other resources that reduce the need for repeat ED visits. There are also local Area Agencies on Aging that can help connect patients to other essentials or key services they may be lacking such as food aid, like Meals on Wheels, or a home helper. These key interventions can help sustain older adults in their homes as an alternative to admission.

There is also a potential role for the use of paramedicine in the care of older adults. This refers to novel programs being developed and studied for the use of

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emergency medical services (EMS) in nontraditional ways to better integrate primary, home, and acute care. EMS can help "bridge" the acute phase of care back to the community by expanding the paramedic's role to include frailty assessments, determination of medical necessity for ED visit, and selection of appropriate alternative care pathways when applicable. While studies seem to indicate benefit to paramedicine, there are still controversies surrounding the increased workload for EMS and how to best integrate their new roles in the current health system.[15]

Alternative Model for GEDs

The delineated guidelines and accreditation are designed to allow for incorporation of best-practice geriatric emergency medicine principles into all EDs and allow for personalization of GEDs for individual health systems. There is no one uniform approach, and both the guidelines and accreditation process take into consideration that most hospitals are not capable of building and staffing separate EDs for older adults. Another key aspect is that GEDs generally only target 10–12% of older adults who come to the ED, and many have screening to identify which patients should be evaluated in the GED;[16] thus, GEDs can focus on older adults who are likely to benefit the most from a geriatric-specific approach to care.

A discrete physical GED has the benefits of a dedicated space with geriatric-specific environmental features such as enhanced lighting and often the ability to have dedicated staff, including geriatric-trained nurses, pharmacists, and physical therapists. However, a dedicated space may not be practical for most hospitals, depending on local patient populations, volumes, and flow. In fact, the vast majority of geriatric EDs do not have a separate space for older adults. Nevertheless, other GED models have been deployed successfully at other sites that do not require a separate ED space. In some EDs, a geriatric assessment team or consultation service works in conjunction with the primary ED team to provide recommendations and facilitate follow-up. Other systems have nursing and physician "champions" to create universal ED initiatives and close coordination with community resources to ensure that care is able to be delivered whether or not a geriatrician is present in the ED. Finally, some EDs may choose instead to create a GED within an ED observation unit to allow for full interdisciplinary team assessment in less than 48 hours.[16] There are many ways to develop a GED, most of which do not require space or significant resources. Over 90% of GEDA-accredited EDs are self-funded, needing no additional financial support.

The Institute for Healthcare Improvement (IHI) and John A. Hartford Foundation have established the Age-Friendly Health System initiative that coincides with GED core principles. This initiative is centered on incorporating the "4Ms" of high-quality care into standard practice. The 4Ms represent: what matters, medication, mentation, and mobility. "What matters" addresses the patient's care goals and ensures that treatment is in accordance with these goals. "Medication" promotes the safe and proper use of age-friendly medication. "Mentation" refers to the identification, treatment, and prevention of dementia, depression, and delirium. Finally, "mobility" centers on safe movement and function for older adults. A fifth "M" can also be added for mistreatment, which represents the identification of elder abuse. While the Age-Friendly Health System initiative is designed with entire health systems in mind, the focus on core issues emphasizes a shift in the mentality of treating older adults, which is equally pertinent to a GED.

Alternatively, there have been several programs incorporating ancillary staff to more dedicated care of older adults. For example, a program that incorporated physical therapy and pharmacy evaluation for older adults who fall showed increased patient and physician satisfaction with care delivery and did not significantly increase the length of stay.[17] Additionally, trained technicians or medical students can be utilized to screen older adults for geriatric syndromes such as cognitive dysfunction, which alleviates perceived time burden from nurses and physicians.[18] Many of these ancillary services already exist within the hospital system, and instead the GED finds novel ways to incorporate these employees into the care of older adults in the ED rather than the inpatient setting.

Current and Future Research

New studies are beginning to prove that the work being done in GEDs is beneficial in the cost, outcomes, and overall quality of care being provided to older adults. The use of intradisciplinary staff has shown a reduction in the overall cost of care after an ED visit by $2,000–$5,000.[19] One multisite GED study suggested a 16% reduction in risk of hospital admission from the ED with the use of senior-specific protocols.[20] In another study, providing seniors with targeted intervention in the ED with resources like physical therapy after a fall is associated with a 34% lower likelihood of another fall-related ED

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visit within 30 days.[21] These findings are promising, but more research is needed to more fully elucidate the impacts of specific interventions in GEDs. The Geriatric Emergency Department Collaborative and the NIH-funded Geriatric Emergency care Applied Research (GEAR) Network are two interdisciplinary teams of experts in the field of geriatrics and emergency medicine that are working together to study the impact of interventions designed to improve emergency care for older adults. Their main areas of focus are falls, medication safety, elder abuse, delirium, and transitions of care. As additional EDs become accredited as GEDs, further research opportunities include evaluating the implementation process in various health-care systems and assessing the quality improvement metrics.

Conclusion

Case: *In a GED, Mrs. Smith's visit takes a different course. While Mrs. Smith awaits the results of her CT scan, a physical therapist evaluates her and recommends use of a walker at home. He has one available to fit and to educate her on in the department. The ED pharmacist identifies two of her home medications that can contribute to dizziness and falls in older adults and recommends an alternative regimen to the treating physician. A transitional navigator arranges to have a home safety evaluation in the next 2 days. This information is all conveyed to Mrs. Smith's family physician via the electronic health record. The patient and her daughter are provided with a verbal and written copy of instructions that include recommendations on medication changes, follow-up appointments, and information on home safety and use of a walker. The next day, they receive a call from the transitional navigator to ensure the patient has not had any issues since leaving the hospital and has been able to arrange transportation to follow-up appointments.*

As the above case demonstrates, the GED addresses the needs of older adults in a more comprehensive approach aimed toward assessing overall risk and minimizing risk of future complications and return visits. Improving the care of older adults is attainable by any ED without significant cost to the hospital. The resources most utilized by GEDs already exist within hospitals, and a reorganization of staffing structure can make resources such as physical therapy and case managers more readily available to ED patients, thus providing more integrative care without requiring inpatient stay and without placing the responsibility to address an older patient's complex care requirements solely on one provider. Key aspects of geriatric emergency medicine include environmental changes within the ED itself; education for all members of staff; processes to screen for important underlying syndromes; and connections within the outpatient community. These principles of geriatric emergency medicine are universally applicable to all EDs who evaluate and treat older adults, and will only become more critical to appropriate care as the geriatric population increases.

Key considerations for proponents of geriatric emergency medicine in their health-care systems:

- A GED is an ED with enhanced (but not exclusive) focus on the special needs of older adults.
- A GED incorporates staff education, enhanced processes including screening, improved – often inexpensive – structures for older adults, and community connections.
- Initial data demonstrates decreased admissions and ED revisits and enhanced patient satisfaction with GED processes.
- A GED requires a physician and a nurse champion to drive forward improved care for older adults.
- GEDC (https://gedcollaborative.com) is a learning collaborative to share best practices in geriatric emergency medicine.
- Geriatric ED Accreditation (www.acep.org/geda) is a program standardizing tiered expectations for geriatric EDs, and granting recognition to EDs that achieve those standards.

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Chapter 37

Musculoskeletal Injuries

Joshua C. Rozell and Joseph D. Zuckerman

Introduction

Orthopedic trauma in the elderly patient presents both a medical and surgical challenge. This growing population requires injury management tailored to specific patient needs and particular attention to bone quality and soft tissues. Injury treatment is based on patient factors, injury factors, and other special considerations in order to optimize outcome. The goal of injury treatment in the elderly patient is allowing for return to preinjury functional status. This chapter will focus on the treatment of some of the more common musculoskeletal injuries encountered in the elderly, including proximal humerus fractures, wrist fractures, hip fractures, ankle fractures, and vertebral compression fractures.

Patient Factors

Preinjury Status

The goal of injury treatment in the elderly patient is a return to preinjury level of function. Therefore, a thorough history that includes preinjury activity level and independence is integral to guiding effective orthopedic care. For example, the treatment goals of an independent community ambulator who sustains a hip fracture are different than those of an institutionalized non-ambulator. The former requires early operative intervention combined with aggressive postoperative rehabilitation, while the latter generally requires a less aggressive approach that enables comfortable transfers and the ability to sit. For both, the goal is to return the patient to preinjury status; however, the approach to each differs significantly.

Systemic Disease

Elderly patients often have preexisting medical comorbidities that influence musculoskeletal injury treatment. Cardiopulmonary disease is common in this population and affects the patient's ability to tolerate anesthesia, undergo surgery, and participate in a postoperative rehabilitation program. Cardiopulmonary disease is a major determinant of the American Society of Anesthesiologists' preoperative risk assessment.[1]

The presence of neurologic disorders such as Parkinson's disease, Alzheimer's disease, and previous cerebrovascular accident affects injury management. Profound Parkinson's disease associated with severe contractures and significant functional incapacity limits the treatment options for both fractures and soft-tissue injuries. Patients who have suffered a stroke and resulting neurologic impairment are at an increased risk for fracture as a result of altered gait and balance, coupled with compromised bone quality due to impaired weightbearing, disuse osteopenia, and mechanics. Fractures occurring in patients who have suffered a stroke usually occur on the affected side.[2]

Endocrinopathies, such as diabetes and thyroid disorders, are common in the elderly. Diabetic patients are considered to be immunocompromised and have microvascular disease that increases the risk of wound complications and infection following surgery.[3,4] Independent of other known risk factors, diabetics also have greater risk of sustaining a fracture, require a longer time for fracture union, and have poorer expected outcomes of operative fracture fixation as compared to age-matched nondiabetic patients.[3–6]

Bone Quality

Strong bone should be both appropriately dense and appropriately mineralized. In the elderly, reduction of bone quality – osteopenia – is common and may be caused by either osteoporosis (decreased bone density with normal mineralization) or osteomalacia (decreased mineralization with or without a change in density). Osteopenia is most commonly caused by senile osteoporosis, but also may be due to other, treatable causes such as nutritional deficiencies, hyperparathyroidism, renal disease, tumors, and Cushing's disease.[7] A thorough

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medical evaluation should identify any of these modifiable causes.

Osteopenia affects fracture management because osteopenic bone is at higher risk for delayed union and nonunion. Additionally, osteopenic bone may affect the surgeon's ability to achieve a stable construct during operative fixation. Orthopedic implants do not have the same fixation in osteopenic bone as they have in robust, strong bone. Lower forces are required for screw pullout, leading to early failure of fixation. This problem is compounded by the osteopenia that develops, even in normal bone, during periods of immobilization. Several techniques may be employed to mitigate this problem: bone graft, bone graft substitutes, methylmethacrylate cement, and locked plating systems.[8]

Several prophylactic and therapeutic medical treatments for osteoporosis are currently available such as bisphosphonates, parathyroid hormone analogs, calcitonin, and selective estrogen receptor modulators, and should be considered for the elderly patient.[9–11] Osteomalacia in the elderly patient is often the result of nutritional deficiencies, whether due to malabsorption syndromes, aberrant metabolism of calcium, vitamin D, and phosphorus, or excessive use of phosphate-binding medications such as phenytoin or antacids. These conditions are generally treated medically by addressing the cause of the deficiency and providing increased dietary supplementation of the deficient metabolite.

Soft-Tissue Quality

The most characteristic age-related change in skeletal muscle tissue is a loss of muscle mass secondary to a decrease in the size and/or number of muscle fibers.[12,13] Functional changes associated with aging are alterations in reaction time, strength, reflex time, coordination, speed, and endurance.[14,15]

As a result of aging, skin becomes more fragile and requires extreme care in soft-tissue handling during surgical intervention. These changes may affect the treatment options considered. Age-related attritional changes can compromise soft-tissue repair; aggressive surgical management requiring lengthy rehabilitation may not be warranted in patients with preexisting soft-tissue compromise.

Injury Factors

Polytrauma

Although patients older than age 65 constitute a minority of the overall population, they represent over 28% of all fatal injuries in the United States.[16,17] For any operative injury, mortality and morbidity are greater in the geriatric patient. Traditional trauma rating systems used to triage patients and predict outcome are less reliable in the elderly.[18,19]

Recognition of skeletal injuries in the geriatric trauma patient requires vigilance. The possibility of fractures should be considered in any high-energy trauma. Visceral injuries uncommonly occur without skeletal injury in the elderly trauma patient.[20] Long bone injuries should be immobilized to decrease hemorrhage, mitigate muscle spasm and pain, and minimize the risk of fat embolization. The mortality of acute and delayed complications of pelvic fractures (hemorrhage or sepsis) is 17% in cases of closed pelvic ring fractures, and over 80% in cases of open fractures.[17] Early stabilization of pelvic ring fractures and long bone fractures facilitates patient mobilization and improves respiratory function, ultimately resulting in improved outcomes.[21]

Open Fractures

Fractures in which the bone is exposed to the outside environment through a defect in the soft-tissue cover are known as open fractures. Open fractures in the elderly, particularly those of the lower extremity, should be treated as limb-threatening injuries. One of the most common injuries is tearing of the medial soft tissue around the ankle after an ankle fracture (Figure 37.1). Preexisting conditions such as vascular insufficiency, diabetes mellitus, atherosclerosis, osteopenia, and immunocompromise adversely affect the outcome of these injuries and are more common in the geriatric population. The basic tenets of open fracture management must be followed to optimize outcome in this population: immediate antibiotic administration, timely and meticulous debridement of bone and soft tissue, fracture stabilization, bone grafting, and soft-tissue coverage when necessary.[22,23]

Comminution

Even low-energy fractures in the elderly patient may result in marked comminution, more commonly associated with high-energy trauma in the younger patient. This is often the result of relative weakness of osteopenic bone. Comminuted fractures in the geriatric population should be approached systematically by understanding the appropriate fixation construct necessary to produce reliable healing. Utilizing locked plating is advantageous in comminuted fractures to increase the overall construct strength and load to failure. Long bone fractures treated

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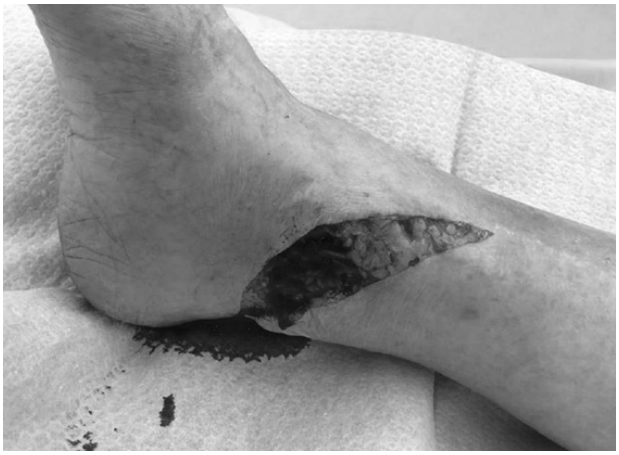

Figure 37.1 Clinical image of a 95-year-old female who sustained an open ankle fracture. The medial, oblique wound is a result of tensile failure of the skin as the displaced distal tibia tears through it. (A black-and-white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

with intramedullary devices or bridge plating should allow for robust secondary healing and callus formation in healthy hosts. Along with orthopedic management, patients with evidence of osteoporosis should also be treated medically to improve bone stock and optimize chances of fracture healing.[24]

Intraarticular Fractures

Intraarticular fractures require a stable, anatomic reduction to mitigate the risk of posttraumatic arthrosis, restore joint kinematics, and allow for early range of motion. Early joint motion promotes articular cartilage healing.[25] Intraarticular fractures in an arthritic joint are generally unique to the elderly patient and may require primary prosthetic replacement depending on the degree of comminution. Reviewing preoperative radiographs and asking if a patient had preexisting pain in the joint due to arthritis can help guide a treatment plan that will then serve as a long-term solution. This is applicable in displaced intraarticular fractures of the hip and proximal humerus.

Special Considerations

Periprosthetic Fractures

The treatment of fractures around a total joint replacement can be challenging. Risk factors include osteopenia and previous revision surgery. Treatment of displaced periprosthetic fractures must be individualized, with treatment goals of early patient mobilization, preservation of limb alignment, and stability of the bone-implant interface. Nonsurgical management may result in limb malalignment and prolonged recumbency with associated pulmonary, genitourinary, and thromboembolic complications. Surgical management is complicated by the presence of the implant, associated osteopenia, and potential implant instability. This often leads to difficulties with postoperative mobilization of the patient.

Periprosthetic fractures commonly occur around the junction of the implant and native bone because of stress shielding and the difference in modulus of elasticity between bone and metal. Once recognized, determination of implant stability (i.e., is the implant loose?) guides treatment. Treatment options may include open reduction, internal fixation for stable implants, or revision arthroplasty for loose implants. There is a high complication rate of treatment following periprosthetic fractures. Compared to fractures not near implants, there is a higher rate of nonunion, malunion, and infection.

Pathologic Fractures

Metastatic Disease

The skeleton is the third most common site of metastatic disease. The incidence of bony involvement in patients with a known malignancy is reported to be between 12% and 70%.[26–28] Common primary malignancies that metastasize to bone originate in the lung, breast, renal, thyroid, or brain. The proximal femur is the most common location of pathologic fracture and is involved in over 50% of cases. This is due to the significant mechanical stresses that occur across the hip joint and proximal femur during ambulation.[29]

Treatments are directed at early patient mobilization and protection of the long bones. Surgical treatment is the standard of care in treatment of neoplastic fractures, especially those of the femur. The goals are to restore function, alleviate pain, facilitate nursing care, decrease hospital stays, and improve patient quality of life. Patients may also present with pain associated with metastatic lesions in the long bones. In many cases, prophylactic fixation using an intramedullary device for impending fractures is performed to protect the bone from catastrophic failure (Figure 37.2). Surgical contraindications are few, but may include diminished mental status, inability to tolerate the operative procedure, and life expectancy less than 1 or 2 months.

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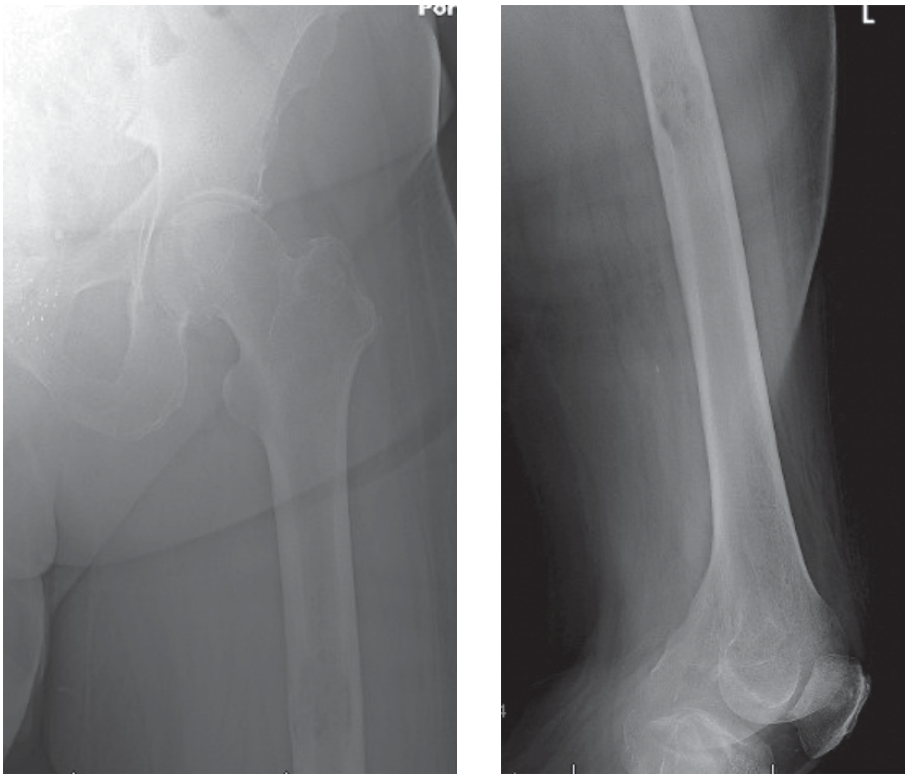

Figure 37.2 An anteroposterior (a) and lateral (b) radiograph of the left femur in a 72-year-old male with metastatic prostate cancer. There is a diaphyseal lytic metastatic lesion in the midshaft of the femur, resulting in thinned femoral cortices and impending fracture. The patient complained of severe thigh pain and difficulty ambulating. He was treated with a prophylactic femoral intramedullary nail placed to protect the entire femur, including the femoral neck.

Atypical Fractures of the Femur Associated with Bisphosphonate Use

Bisphosphonate treatment is steadily growing in the elderly population as patients live longer, and osteoporosis screening is widely promoted by primary care physicians and orthopedic surgeons. Atypical fractures in the femoral metadiaphysis associated with bisphosphonate use have been described.[30] Many patients report prodromal thigh pain. Occasionally, the thigh pain prompts radiographic evaluation before complete fracture occurs.

Investigation into the histology and biomarkers present in the deranged tissue has given rise to the theory that these fractures arise from stress fractures caused by suppression of bone turnover.[31,32] In patients on chronic bisphosphonate treatment, complaints of thigh pain should prompt a radiographic evaluation.

In the context of a low-energy fracture, the characteristic radiographic presentation of a bisphosphonate-associated fracture comprises the constellation of cortical thickening, a transverse pattern, minimal comminution, and a medial spike when the fracture extends through the medial cortex (Figure 37.3). If radiographs are normal but pain persists, a magnetic resonance imaging (MRI) should be obtained.[33] Operative treatment is strongly advised to avoid the complications associated with prolonged bed rest. Furthermore, surveillance of the contralateral limb should be performed in these patients with a low threshold for prophylactic treatment to prevent progression if findings of impending fracture are present.

Preoperative Considerations

Absolute indications for operative fracture treatment are open fractures, compartment syndrome, and neurovascular compromise. Relative surgical indications include displaced intraarticular fractures in which acceptable reduction and alignment cannot be maintained, and fractures that require stabilization in order to mobilize the patient. Fracture management in the elderly must take into consideration all of the injury, patient, and social factors, as well as the special considerations described above.

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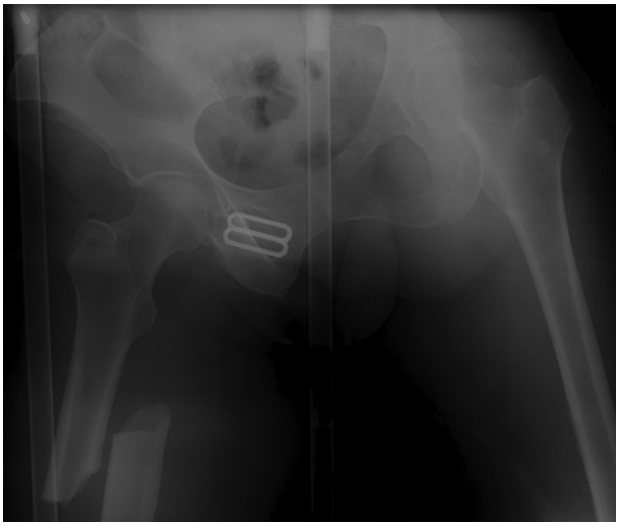

Figure 37.3 Anteroposterior radiograph of the pelvis of a patient on chronic bisphosphonate therapy, who sustained a fall. Note the hallmarks of atypical femur fractures: subtrochanteric location, transverse fracture line, lateral cortical thickening, and a medial cortical spike.

Timing of surgery in the elderly patient is controversial. Generally, surgery should be performed when comorbid medical conditions have been reduced. Retrospective analyses have offered equivocal assessments on the relationship between timing and morbidity.[34,35] A prospective study from our institution of 367 hip fracture patients demonstrated that surgical delay of greater than 2 days from hospital admission doubled the risk of patient death at 1 year when age, sex, and number of comorbidities were controlled.[36]

No significant difference has been demonstrated in survival rates in patients undergoing operative treatment of hip fractures with either regional or general anesthetics.[37,38] There is, however, a reduced incidence of thromboembolic events (deep venous thrombosis [DVT], pulmonary embolus) after use of regional anesthesia.[39] Because pulmonary embolism is a significant cause of morbidity and mortality in the geriatric patient, regional anesthesia is preferable when appropriate.

Intramedullary devices are the implant of choice for fractures in osteopenic bone when the location and fracture pattern are amenable. Intramedullary implants are closer to the mechanical axis of the bone and thus act as load-sharing devices. Plates, which are placed directly on the bone, are farther from the mechanical axis and act as load-bearing devices.

Hip Fractures

Principles

Hip fractures in the elderly can be a life-threatening injury because of the impact on the medical, functional, and psychological status of the patient. Over 250,000 hip fractures occur annually in the United States, which result in over $9 billion in health-care costs. Current age trends predict a more than doubling of the yearly incidence of hip fractures by the year 2050.[40–42]

The risk of a hip fracture increases with age, doubling every decade after age 50. Hip fractures occur most commonly in Caucasian women, followed by Caucasian men, African American women, and African American men. This may be due to differences in bone density between these groups. Institutionalized patients are also at an increased risk for hip fracture, with greater risk of mortality.[43,44]

Hip fractures in the elderly usually occur from low-energy trauma. They are categorized by anatomic location into two classes: intracapsular (femoral neck fractures) or extracapsular (intertrochanteric and subtrochanteric fractures). Fractures of the femoral neck are intracapsular, extraarticular fractures that occur in the region between the femoral head and the intertrochanteric line. The location of these fractures can compromise the primary blood supply to the femoral head, especially in displaced fractures.

The intertrochanteric region lies outside the hip capsule, between the greater and lesser trochanters. This area of metaphyseal bone benefits from a rich blood supply and, as a result, carries a lower risk of the healing complications associated with intracapsular fractures. The greater trochanter is a superolateral structure that serves as the insertion point for the hip abductors and short external rotators. The lesser trochanter is located distally at the posteromedial surface of the proximal femur and serves as the attachment site for the iliopsoas. The calcar femorale is a region of bone located along the posteromedial portion of the proximal femur that acts as a cortical strut to transmit the large forces across the intertrochanteric region.

Presentation and Initial Management

Patients who sustain a hip fracture present with hip and groin pain and an inability to bear weight on the affected extremity. The leg is usually positioned in external rotation and slight hip flexion. This position provides maximal capsular volume and the most comfort from the hematoma that develops in intracapsular fractures; in

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displaced extracapsular fractures, the displacement of the fracture results in a shortened and externally rotated position of the leg. There will often be a noticeable leg length discrepancy. Evaluation should include a thorough neurovascular exam of the affected extremity as well as examination of all other extremities to exclude concomitant injuries. Neurologic examination should assess mental status and any loss of consciousness associated with the fall.

Medical consultation should be obtained at the time of presentation to ensure the patient's condition is optimal for anticipated surgery. Injury radiographs, initial laboratory investigations, a chest radiograph, and an electrocardiogram should be performed. A baseline arterial blood gas may be warranted since hip fractures carry a risk for thromboembolic phenomena. A Foley catheter should be placed to eliminate the need for positioning on a bedpan or use of a urinal to minimize patient discomfort. Additionally, the Foley catheter allows accurate measurement of urine output, important for volume status assessment in these patients. Orthogonal films should include a true anteroposterior (AP) and cross-table lateral of the hip. A cross-table lateral projection is preferable to a frog-leg lateral, as the latter causes the bone fragments to rotate through the fracture site, further displacing fracture fragments and causing increased discomfort to the patient and the potential for further injury.

If the diagnosis of a hip fracture is suspected but not confirmed by routine radiographs, further imaging studies are indicated. An AP view with axial traction and internal rotation of the affected limb can improve the visualization of the entire femoral head and neck, while a computed tomography scan can better define the fracture characteristics and displacement. If doubt remains, a technetium bone scan or MRI should be obtained. A technetium bone scan requires 2–3 days after injury to minimize the risk of a false-negative result, while an MRI can accurately diagnose occult fracture within 24 hours after injury.[45]

The preferred treatment of hip fractures is operative because it allows early mobilization of the patient, thereby decreasing the risk of cardiopulmonary events, urinary tract infections, decubitus ulcers, and the rate of mortality in the first year. It also minimizes the period of non-weightbearing, decreases the risk of nonunion/malunion, and increases the ease of transfer.

As for any hospitalized patient, the potential for thromboembolic events (DVT or pulmonary embolus) is concerning. In addition to mechanoprophylaxis, chemoprophylaxis should be administered. However, the risk of bleeding in the injured or postoperative patient is an important consideration and must be balanced with the need for prophylaxis. Warfarin requires daily monitoring and carries a significant risk of bleeding. Low-molecular-weight heparins (LMWH) have been the contemporary choice, effective without the need for laboratory monitoring, although their subcutaneous administration raises compliance concerns posthospitalization.[46–48] Other promising alternatives have emerged. Rivaroxaban, for example, is one orally administered direct factor Xa inhibitor with predictable pharmacokinetics and similar efficacy to LWMH.[49] Discussion between the medical and surgical teams should decide upon the precise starting time postoperatively, but generally chemoprophylaxis should begin 12 hours postoperatively. An inferior vena cava filter may be used when anticoagulation is contraindicated or in patients at high risk for recurrent thromboembolism.

Outcomes

The goal of surgical treatment of hip fractures is to restore the patient's functional status to the preinjury level. At the 1-year follow-up, 41% of patients will regain their preinjury ambulatory status, 40% will require increased assistance, and 8% will become non-ambulatory.[50]

Mortality rates of hip fracture patients are twice that of age- and sex-matched controls.[34,51,52] The highest increase is seen in the first 6 months after injury, and progressively decreases to that of age- and sex-matched controls at 1 year. However, 1-year mortality can be as high as 30%.[52,53]

While the amount of time from injury to surgical treatment has been debated as a predictive factor, a prospective study from our institution in which age, sex, and other comorbidities were controlled demonstrated that a delay of surgical treatment for more than 2 days in patients who did not suffer from dementia and were ambulatory prior to injury doubled 1-year mortality.[36]

Postoperative rehabilitation should employ a weightbearing program as tolerated. Although some have recommended restricted weightbearing (or even non-weightbearing), in situations when fracture fixation is felt to be suboptimal, we feel that this has a very negative impact on the overall recovery and does not accomplish the intended goal of limiting forces across the hip (and therefore on the fracture fixation). Joint reaction forces across the hip are actually higher with non-weightbearing as opposed to toe-touch weightbearing. When elderly patients are allowed immediate

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postoperative weightbearing as tolerated, they tend to self-regulate the amount of weight on the injured extremity and will gradually increase the amount of weightbearing as their comfort allows. The approach encourages, rather than limits, their recovery of mobility and ambulation.

Femoral Neck Fractures

Femoral neck fractures, as previously discussed, are intracapsular fractures. They are generally classified as nondisplaced or displaced. Nondisplaced fractures may be treated nonoperatively for the sick or non-ambulatory patient, but persistent pain and nonunion may occur. For this reason and to allow for earlier mobilization, internal fixation with cannulated screws or a head screw with side plate (sliding hip screw) is preferable. The Fixation using Alternative Implants for Treatment of Hip fractures (FAITH) trial of 1,108 patients over the age of 50 who sustained low-energy femoral neck fractures randomized them to either sliding hip screw fixation or cancellous screw fixation and evaluated reoperation rate within 2 years of surgery. The study found similar rates of reoperations, treatment failures, and fracture healing between both groups. There were significantly higher rates of avascular necrosis and reoperation rates for avascular necrosis in patients who underwent the sliding hip screw fixation. Interestingly, subgroup analysis revealed that in patients who smoked or had basicervical or displaced fracture patterns, the reoperation rates were significantly lower in patients who had sliding hip screw fixation. Given certain biomechanical advantages using the sliding hip screw, this construct may lead to superior outcomes in certain populations such as smokers or those with poor bone quality.[54]

Displaced fractures in older patients are typically treated with arthroplasty. For younger, active patients, studies have shown improved outcomes and quality of life scores, and decreased rate of reoperation for total hip arthroplasty (Figure 37.4).[55] For patients who are older and low demand with poorer function status preoperatively, a hemiarthroplasty is performed (Figure 37.5). A recent trial demonstrated that patients in their 80s who were randomized to a total hip arthroplasty or hemiarthroplasty fared similarly at 2 years postoperatively in terms of hip function, complications and reoperations, pain, and activities of daily living.[56] In older patients with less cardiac reserve and more medical comorbidities, hemiarthroplasty is also a technically easier procedure and is associated with lower costs, less operative time, less blood loss, and lower risk of dislocation because of the use of larger femoral head components compared to total hip arthroplasty.[57]

Another surgical decision point is whether to cement the femoral stem. Cementation allows for immediate femoral stem fixation. A randomized controlled study comparing the use of cemented versus cementless hemiarthroplasty found that cementless fixation was associated with a significantly greater rate of intraoperative fractures and significantly inferior functional outcome scores at the 1-year follow-up.[58] A meta-analysis also found that cementless stems were associated with a significantly greater rate of overall complications as well as implant-related complications.[59] According to a study of over 347,000 patients in the Norwegian Hip Arthroplasty Registry, 10-year implant survival was lower in the uncemented group compared with the cemented group for patients over the age of 65.[60] Certain patient populations are better suited for cementation. For example, patients with pulmonary disease may have higher risks of complications associated with fat embolism as a result of cementation, while patients with chronic kidney disease and poor bone quality secondary to renal osteodystrophy are good candidates for cementation. The decision to fix the fracture versus replacing the joint has to be individualized, taking into account the patient's lifestyle, independence, physiologic age, bone quality, comorbidities, and radiographic findings.[61]

Osteonecrosis of the femoral head and nonunion following femoral neck fractures are seen more frequently in displaced fractures treated nonoperatively or with fixation rather than arthroplasty.[57] While nondisplaced fractures have rates of these complications between 5% and 10%, rates are 20–35% for displaced fractures.[62,63] This is due to disruption of the retinacular vessels and the medial femoral circumflex artery branches that supply the vascularity to the femoral head. Nonunion and osteonecrosis typically necessitate revision surgery.

Intertrochanteric Hip Fractures

Intertrochanteric hip fractures are classified as stable or unstable. Unstable fracture patterns are defined based on the presence of posteromedial calcar comminution, lateral femoral wall incompetence, a reverse obliquity pattern, or subtrochanteric extension of the fracture.[64]

Surgery is the treatment of choice for intertrochanteric hip fractures. Stable fractures can be treated with an

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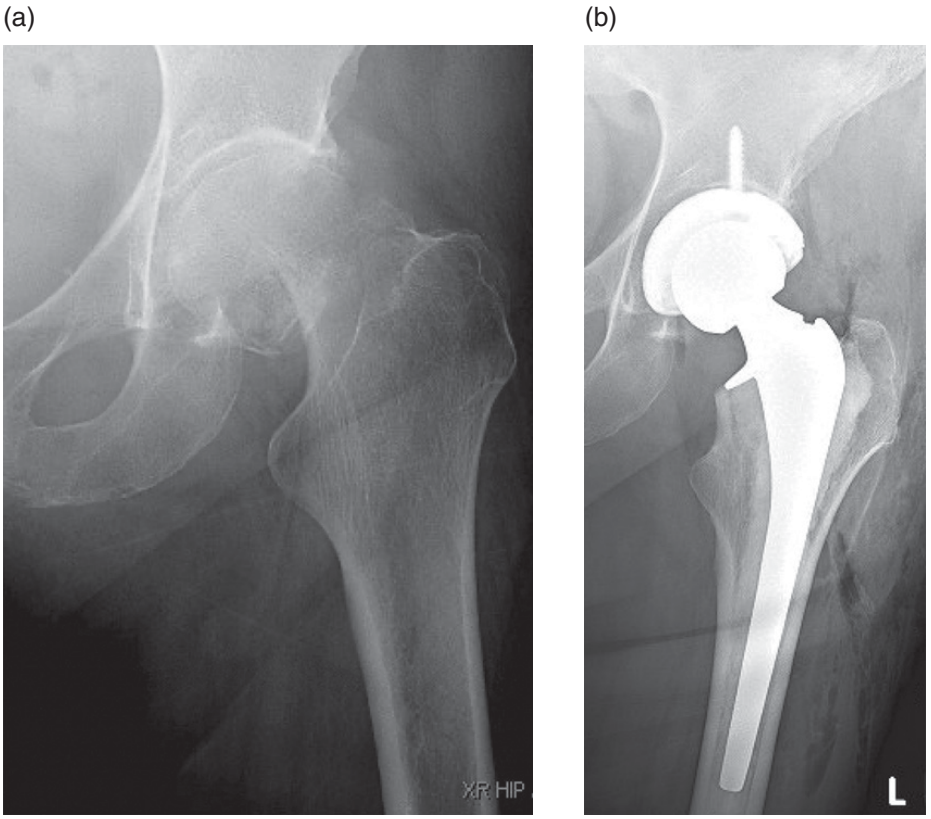

Figure 37.4 (a) An anteroposterior radiograph of an active, 72-year-old female with osteoporosis who sustained a left displaced femoral neck fracture after a fall. This patient required arthroplasty given the degree of displacement of the fracture. (b) Because of her functional status and activity level, the decision was made to perform a cemented total hip arthroplasty.

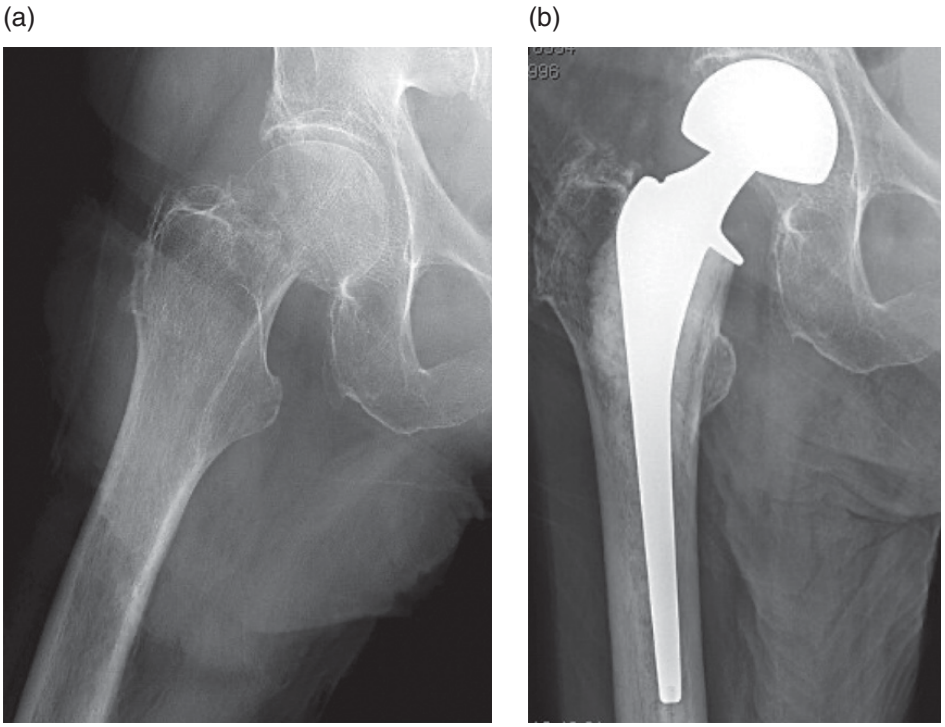

Figure 37.5 (a) An anteroposterior radiograph of a 94-year-old female with osteoporosis who sustained a right displaced femoral neck fracture after a fall. This patient was treated with a cemented hemiarthroplasty (b), given her limited functional status (ambulated only one block with a walker at baseline) and activity level.

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extramedullary device such as a screw and side plate. This fixed-angle device consists of placement of a lag screw parallel to the axis of the femoral neck, which is then inserted into a barrel attached to a lateral plate. The lag screw is allowed to slide within the barrel, thus allowing for micromotion and compression across the fracture site, leading to healing. To minimize lag screw fixation failure, the sum of the distance from the screw tip to the cortex of the femoral head on the AP and lateral radiographs should be 25 mm or less.[65,66] Treatment with this device requires a competent lateral femoral wall to act as a buttress for compression of the hip screw. For unstable fracture patterns, an intramedullary device is used with fixation into the femoral head (Figure 37.6). As the nail is closer to the center of rotation of the femoral canal, there is a shorter lever arm and therefore less of a moment arm, thereby increasing load to failure. The nail itself also acts as a lateral buttress in cases of lateral wall incompetence. Results from prospective randomized controlled trials have shown that for unstable fracture patterns, intramedullary nails were associated with superior radiographic outcomes and lower rates of malunion/nonunion compared to sliding hip screws.[67,68]

Ankle Fractures

The ankle joint is a modified hinge joint consisting of the lateral malleolus (tuberosity on the distal fibula), the medial malleolus (tuberosity on the distal tibia), the plafond (central articular surface of the distal tibia), and the talus. Binding these osseous structures are the lateral collateral ligament, the deltoid ligament, and the tibiofibular syndesmosis. The lateral collateral ligament is composed of three structures: the anterior talofibular ligament, the calcaneofibular ligament, and the posterior tibiofibular ligament. The deltoid ligament consists of an anterior, superficial portion that attaches to the navicular,

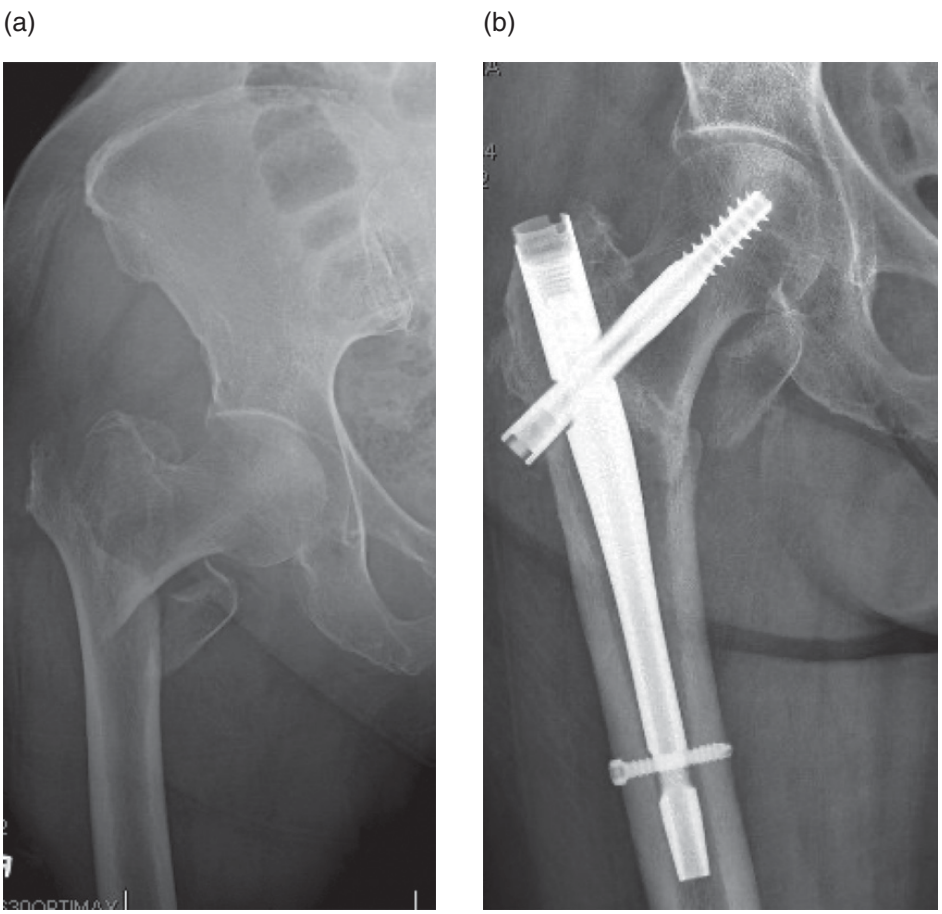

Figure 37.6 (a) An anteroposterior radiograph of a 94-year-old female who sustained an unstable intertrochanteric right hip fracture after a fall. The displacement of the lesser trochanter is an indicator of posteromedial calcar comminution and instability. She was treated with a short cephalomedullary nail (b).

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sustentaculum tali, and the talus, while the stronger, deeper, posterior portion originates on the posterior colliculus of the medial malleolus and inserts on the medial surface of the talus.

The clinical examination of a patient with a suspected ankle injury should include palpation of the above-mentioned osseous and ligamentous structures. Swelling and ecchymoses should be noted. Weightbearing ability must be determined (patients are rarely able to bear weight on an unstable fracture). A neurovascular exam should be performed, ensuring that ankle dorsi- and plantarflexion are intact, as is great toe flexion and extension. Sensation should be tested over the dorsum of the foot, first webbed space, medial and lateral edges of the foot, and the plantar surface.

The decision to evaluate an ankle radiographically after injury should be made using the Ottawa Ankle Rules. Only patients with tenderness over the inferior or posterior pole of either malleolus (defined as the distal 6 cm for these guidelines) and the inability to take four steps independently (even if limping) should have radiographs.[69] Radiographic examination should include three views of the ankle: an AP view, a mortise view (15- to 20-degree internal rotation oblique), and a lateral view. Ankle fractures can include isolated lateral malleolus/distal fibula fractures, isolated medial malleolus fractures, fractures of both malleoli (bimalleolar), or both malleoli and the posterior portion of the tibial plafond (trimalleolar). Isolated fractures of the lateral malleolus at the level of the joint line should be evaluated with external rotation stress views to exclude the presence of a medial soft-tissue injury that will lead to ankle instability and resultant ankle mortise widening.[70]

Ankle fractures are the third most common osseous injury in the elderly, behind hip and distal radius fracture.[71] Low-energy ankle fractures in the elderly are not usually the result of their weaker bones but rather their predisposition to falls, reaction time, and a worsening obesity epidemic.[72] The goal of treatment of ankle fractures in elderly patients is to restore the normal tibiotalar relationship and improve clinical outcomes, while also allowing for return to a functional level of activity and mitigating the risks of prolonged immobilization. Stable injuries, such as isolated lateral malleolus fractures without medial disruption, should be rested in the acute postinjury period with gradual return to weightbearing as tolerated with the use of a brace or fracture boot. Bimalleolar injuries (including an equivalent pattern of a lateral malleolus fracture with medial soft-tissue disruption and no medial malleolus fracture) and fractures associated with talar displacement and joint incongruity are unstable and require operative fixation (Figure 37.7). Even 1 mm of talar incongruity can lead to early posttraumatic arthritis.[73]

The timing of surgery depends largely on the condition of the soft-tissue envelope. Ankle fractures and fracture dislocations can be associated with significant swelling and the development of fracture blisters. When present, operative treatment should be delayed because of the risk of delayed wound healing or even an inability to close the wounds following the procedure. The return of skin wrinkles about the ankle herald adequate subsidence of swelling. If a reduction cannot be maintained in a plaster splint, an external fixator can be applied temporarily (usually 7–10 days) to allow the soft-tissue swelling to improve while also maintaining the joints in anatomic alignment. The ankle can then be fixed at a later date and the fixator removed at the same time. For open fractures, a systematic review by Hulsker et al. agreed that immediate fixation is safe following open ankle fractures, leading to a shorter hospital stay with reduced joint stiffness, and recommended that external fixation should only be used in cases where there is inadequate soft tissue to cover the implants.[74] Fixation options in the elderly take into account the bone quality and location of the fracture. For more distal fibular fractures, locking plate technology (Figure 37.7) can be used to offer increased construct pullout strength and in situations where bicortical fixation is not possible (i.e., at the lateral ankle joint). One study comparing nonlocked versus locked plating found that elderly patients treated with locked plating had a quicker return to weightbearing.[75] Patients with unstable injuries should remain non-weightbearing until bony union is achieved and soft-tissue supporting structures have had the chance to heal.

Proximal Humerus Fractures

Proximal humerus fractures are common in the elderly population. The proximal humerus is the third most common site of fracture[76] and often occurs as a result of a low-energy mechanism, such as a fall from standing position.

The current system used to classify fractures of the proximal humerus was first described by Neer.[77,78] This classification divides the proximal humerus into four anatomic segments: the head, the shaft, the lesser tuberosity, and the greater tuberosity. A segment is considered independent if it is displaced 1 cm or greater, or

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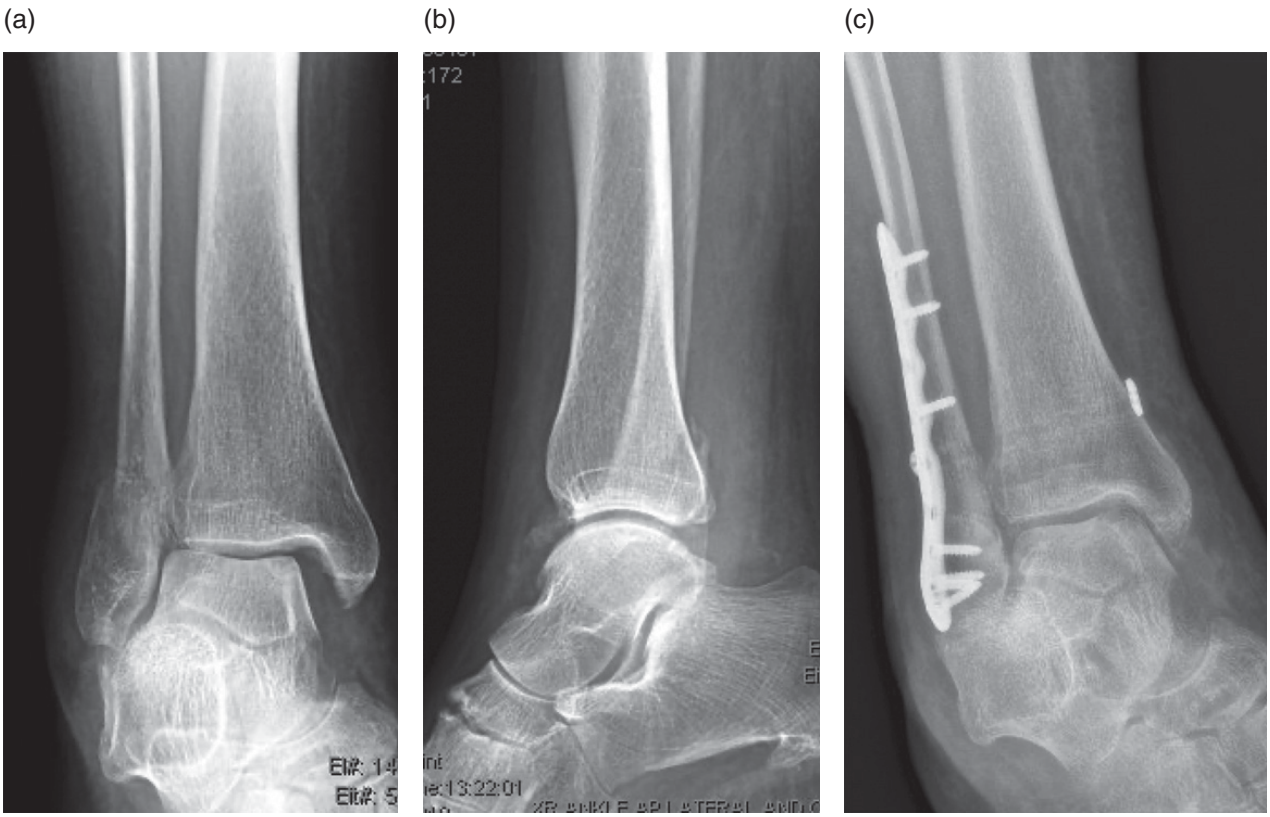

Figure 37.7 A mortise (a) and lateral (b) radiograph of a 67-year-old female who sustained an unstable ankle fracture after a twist and fall down the stairs. Medial clear space widening of the ankle mortise indicated medial ankle instability. She underwent an open reduction internal fixation of the distal fibula with a locking plate, as well as syndesmotic fixation with a suture-button device to stabilize the ankle mortise (c). The mortise is symmetrically reduced in the postoperative radiograph.

angulated 45 degrees or more from its anatomic position. This system provides treatment guidelines and predicts outcome based upon fracture type.

Minimally displaced fractures account for approximately 50–65% of all proximal humerus fractures. These fractures have an intact surrounding soft-tissue envelope, and all parts can be expected to move as a single unit. These injuries are treated with an initial period of immobilization in a sling, followed by range-of-motion exercises beginning about 1 week postinjury.[79] Isometric, pendulum, or passive range-of-motion exercises should be started within a few days of injury. The sling can be worn until healing is evident, which usually occurs by 4–6 weeks.[76]

Many patients with two-part fractures will respond acceptably to nonoperative management, which should be considered in osteoporotic patients with high physiological age, low demand, and minimal displacement. Surgery can be considered for fractures with significant displacement and patients with acceptable bone quality. Treatment may involve percutaneous pinning, open reduction and internal fixation, or intramedullary nailing. Fractures through the anatomic neck are rare and treated with reduction and internal fixation in younger patients and with arthroplasty in older patients. Greater tuberosity fractures are treated operatively if there is greater than 5-mm superior translation, to preserve rotator cuff function. Lesser tuberosity fractures may be treated nonoperatively unless there is a block to internal rotation.

Three- and four-part fractures are at increased risk for osteonecrosis of the humeral head and require operative management. Treatment preference – internal fixation versus prosthetic replacement – is determined by patient factors (age, activity level) and fracture factors (bone quality, comminution, and presence of dislocation). Currently, options for prosthetic replacement include

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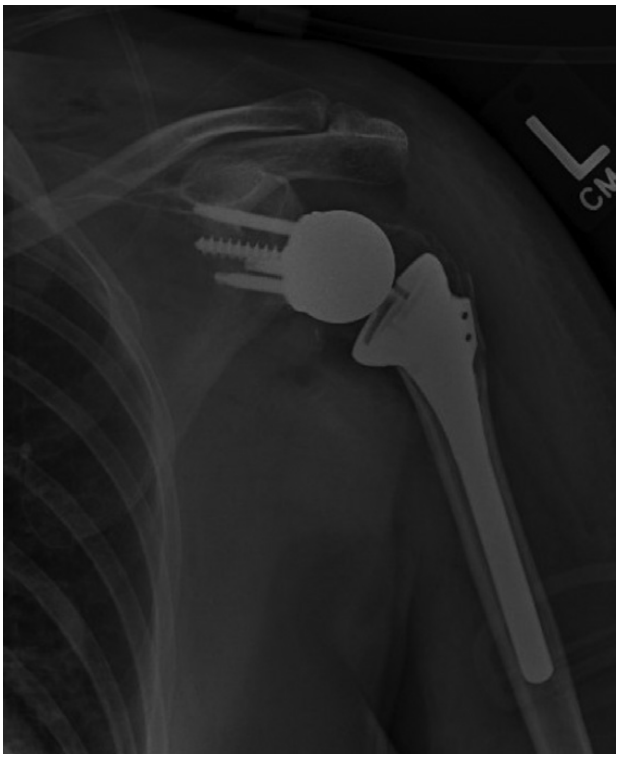

Figure 37.8 A 77-year-old female who sustained a left four-part proximal humerus fracture after a fall. Because of the difficulty in reconstructing the bone anatomically as well as her poor bone quality, she underwent a reverse total shoulder arthroplasty to maximize her chances of functioning and being able to perform activities of daily living.

hemiarthroplasty or reverse total shoulder arthroplasty (rTSA) (Figure 37.8).[76] Hemiarthroplasty was historically the treatment of choice for complex fractures, but results are mixed and depend on the position of the tuberosities and their healing. Recent studies pertaining to treatment with hemiarthroplasty have failed to show any benefit over nonsurgical management.[80] Results of rTSA have shown superior outcomes to hemiarthroplasty for fracture in terms of function and pain.[81,82]

Regardless of treatment approach, elderly patients with displaced proximal humerus fractures require a prolonged, supervised physiotherapy program to optimize functional outcomes. Minimally displaced one-part and adequately reduced two-part fractures can be expected to have good functional outcomes. Poor results may be related to compromise of the rotator cuff, malunion, nonunion, or osteonecrosis. Results of prosthetic replacement are predictable for pain relief, but less consistent for functional recovery.

Distal Radius Fractures

The distal radius is the second most commonly fractured bone in elderly persons and the most frequent upper-extremity fracture in women over 50 years of age.[83] The incidence increases dramatically with age, particularly for women,[84] and parallels trends seen for fractures of the proximal humerus and proximal femur. This increased incidence may be attributed to the presence of osteoporosis, poor eyesight, impaired coordination, and decreased muscular strength.

Unstable fractures are identified by marked comminution, greater than 1 cm of shortening, loss of palmar tilt, greater than 10 degrees of dorsal tilt, and intraarticular displacement. Unstable fractures account for 15–25% of distal radius injuries in the geriatric population and are generally associated with poorer outcome.

Closed reduction and splint/cast application is the treatment of choice for most distal radius fractures in elderly patients and should be attempted initially, even for unstable fractures. If closed reduction is successful, immobilization should continue for 6 weeks and radiographs should be performed weekly for 3–4 weeks to ensure that the reduction is maintained.

If closed reduction is not successful or cannot be maintained, operative intervention may be necessary, particularly when treating an active elderly patient with involvement of the dominant extremity. A variety of operative techniques have been described, such as external fixation, percutaneous pin fixation, and internal plate fixation. The choice is often surgeon-dependent, and each option has its own risk–benefit profile.

Functional outcome after distal radius fracture is often favorable, though dependent on patient factors, treatment method, and quality of reduction. Minimally displaced fractures treated in cast immobilization generally do well with minimal loss of preinjury function. Surgical fixation of displaced fractures demonstrates good to excellent results in 70–90% of patients with comminuted, unstable fractures.[85] However, in low-demand elderly patients, even closed treatment of displaced fractures may yield acceptable functional results despite a cosmetic deformity.[86] Patients treated surgically appear to have better grip strength but no difference in the ability to perform activities of daily living. In a randomized trial comparing volar locked plating with closed treatment in patients over 65 years old, the surgical group showed better wrist function in the early postoperative period. By 6 months, however, despite better grip strength in

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the operatively treated group, there was no significant difference in wrist function or pain between the groups.[87] Ultimately, the treatment of each patient should be individualized and should be based on fracture type, comorbidities, preinjury functional status, and hand dominance.

Vertebral Compression Fractures

In the setting of osteoporotic bone, vertebral compression fractures often result from low-energy trauma (i.e., a simple fall or even just sitting down in an awkward or forceful manner). Jensen et al. have estimated that 44% of women aged 70 or older have vertebral compression fractures.[88] Importantly, vertebral compression fractures in elderly patients are often a harbinger for other osteoporotic fractures, such as those of the distal radius, proximal femur, and proximal humerus.

Vertebral compression fractures most often occur in the midthoracic spine. When vertebral collapse occurs over several adjacent segments, kyphosis (humpback deformity) or scoliosis (lateral compression deformity) may develop.

Vertebral compression fractures may present as an incidental finding; however, most are associated with the acute onset of pain. The location of the pain is typically midline in the thoracolumbar spine but may be referred to the lumbosacral area. If a neurologic deficit is present, metastatic disease, infection, and Paget's disease must be included in the differential diagnosis.

Physical examination demonstrates decreased spinal range of motion, kyphotic deformity, and midline spinal tenderness to palpation. Radiographic evaluation should include standing anteroposterior and lateral views. If the patient is too uncomfortable to stand, then supine radiographs may be obtained. A bone scan can be helpful in differentiating old fractures from acute ones. A computed tomography scan can evaluate the integrity of the posterior elements and identify more severe injuries, such as burst fractures, that can compromise the spinal canal and cause compression of the neural elements.

Historically, a nonoperative approach has been the mainstay for the treatment of vertebral compression fractures. The protocol should involve appropriate analgesia and a short period of rest followed by mobilization.

Advances in operative techniques have changed the management of compression fractures. Vertebroplasty consists of percutaneous insertion of a large-gauge cannula into the vertebral body followed by infiltration of bone cement into the vertebra. Vertebral kyphoplasty is similar in principle: percutaneous insertion of a balloon tamp is used to create a cavity within the vertebral body, followed by the injection of cement to maintain vertebral height. While some studies have demonstrated that compared to traditional nonoperative treatment of vertebral compression fractures, operative intervention improves pain, restores some of vertebral height, lessens disability, shortens length of hospital stay, and returns patients to their preinjury level of function,[89] others looking at vertebroplasty have refuted this, concluding that these procedures are no better than sham surgery.[90,91] A more recent meta-analysis by Anderson et al., which included the studies by Kallmes et al. and Buchbinder et al., demonstrated that cement augmentation results in significantly greater pain relief, functional recovery, and improvement in quality of life scores than did nonsurgical or sham treatment. The results were significant for early and for late follow-up end-points between 6 and 12 months, favoring vertebroplasty.[92] Another more recent meta-analysis comparing vertebroplasty to kyphoplasty suggests that kyphoplasty may be superior to vertebroplasty for patients with pronounced kyphotic deformity or loss of vertebral height, and those with vertebral fissures or fractures compromising the posterior integrity of the vertebrae.[93]

Conclusion

Orthopedic injuries are responsible for significant morbidity and mortality in the geriatric population. As life expectancy continues to increase, the prevalence of these problems will increase as well. The ideal treatment should focus on prevention of predisposing risk factors to these injuries, such as osteoporosis and falls. The primary goals of treatment are to provide analgesia, allow for expedient patient mobilization, and return the patient to his/her preinjury level of function. Treatment should include patient education and rehabilitation to optimize outcome. Each treatment plan should be individualized based on patient factors, injury factors, and the other mitigating variables presented above that are unique to each patient, situation, and injury.

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Chapter 38

Dermatologic Conditions

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Introduction

Though the aging process affects every organ system, the physical signs of aging are most apparent on the skin. The skin is burdened with a disproportionate share of environmental exposures that occur through the course of our lives. Most notably, ultraviolet radiation (UVR) plays a major role in accelerating the natural aging process of the skin and induces a number of changes in structure and function.[1] As a result, elderly patients are more susceptible to cutaneous inflammatory, infectious, and neoplastic processes than their younger counterparts.[2] Elderly patients are also at risk of developing skin conditions that are related to common systemic diseases such as diabetes, vascular disease, and internal malignancy. It is important that clinicians understand these associations so that the skin conditions are recognized promptly and managed effectively as the geriatric population continues to grow. This chapter will explore the changes in structure and function of the skin that are a result of both natural and UVR-induced aging, and describe the appropriate evaluation and management of skin disease with special consideration for this population.

Structure and Function of the Skin

Overview of Skin Structure

The most obvious skin function is as a physical barrier to the environment. The thinnest and most superficial layer is the epidermis. It is composed primarily of keratinocytes, but melanocytes and Langerhans cells are also found in this compartment. The stratum corneum is the outermost layer made of lipids and proteins that are crucial for barrier function.

The thin basement membrane zone divides the epidermis from the dermis, which provides elasticity and strength. It is composed predominantly of collagen that undergoes constant remodeling and contains nerves, blood vessels, and the adnexal structures.

Deeper still, the subcutaneous fat resides beneath the dermis and is home to medium-sized vessels and nerves. Age-related changes in each of these structures are linked to the functional changes summarized in Table 38.1 and discussed below.[3–5]

Skin Function and Age-Related Changes

The epidermis, dermis, and subcutaneous fat all provide physical barrier function, with the epidermis on the front line. When functioning properly, this water-insoluble barrier prevents loss of moisture from the skin and protects it from environmental exposures. As the epidermis ages, it produces fewer lipids and vital proteins such as filaggrin, which result in increased transepidermal water loss, leading to dry skin that appears rough and scaly.

Decreased collagen production by fibroblasts and increased matrix metalloproteinase activity, which is enhanced by UVR, result in a 20% decrease in dermal thickness in elderly compared to younger patients.[6] This thinning and loss of elasticity increase susceptibility to trauma.

Given its constant interaction with environmental antigens, it is not surprising that the skin is a major hub of immunologic activity. As the skin ages, immune surveillance and T-cell stimulation are reduced, making the elderly subject to infection and immune dysregulation, which may manifest as inflammatory skin disease.[7,8]

Protection from UVR is achieved through melanin production, though with increased age melanocyte numbers and melanin production decrease and melanin distribution becomes irregular. This manifests with mottled skin and fewer nevi. Melanocyte senescence also leads to reduced keratinocyte proliferation, contributing to epidermal atrophy.[9]

Thermoregulation via vasodilation and vasoconstriction also becomes more limited as dermal vessels decrease over time.[10] Loss and redistribution of subcutaneous fat also leads to decreased insulating properties, and fewer eccrine glands with aging impairs sweat-induced cooling.[10]

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Table 38.1 Summary of changes in skin structure and function with aging

Change in skin structure or composition	Location	Effect on function
Decreased production of lipid and filaggrin	Epidermis	Xerosis (dry skin), pruritus, decreased physical barrier function
Decreased keratinocyte proliferation	Epidermis	Skin fragility, delayed wound healing
Decreased collagen production and quality	Dermis	Dermal atrophy, skin fragility, easy bruising
Decreased elastin production and quality	Dermis	Decreased skin elasticity, increased fragility
Decreased Langerhans cell numbers and function	Epidermis, dermis	Increased skin infection with candida, dermatophytes, cellulitis Increased risk of cutaneous neoplasm
Fewer melanocytes	Epidermis	Mottled pigmentation, sallow color, increased photosensitivity
Decreased dermal blood vessels	Dermis	Impaired thermoregulation
Decreased subcutaneous fat	Subcutis	Decreased insulation for thermoregulation Decreased cushioning, resulting in increased bruising and risk of pressure ulcers
Decreased eccrine glands	Subcutis	Impaired sweating and thermoregulation
Loss of hair follicles and hair follicle melanocytes	Dermis and subcutis	Decreased hair density Graying of hair

Information from references.[2–5, 20]

Despite popular belief, wound healing is only moderately affected as part of normal aging. Fibroblast function, crucial to wound maturation and scar strength via collagen deposition, appears to be preserved. Wound epithelization and closure are slightly slowed, but tensile scar strength is relatively unaffected.[11]

Acquired and age-related comorbidities may delay wound healing or predispose to new skin wounds, such as pressure ulcers related to decreased subcutaneous fat and mobility. Venous incompetence increases extravasation of leukocytes and other blood cells, which propagate inflammation and delay wound healing. Diabetes and peripheral vascular disease also predispose toward chronic wounds, which are associated with substantial morbidity and high costs to the medical system.[12]

Perceived physical beauty has been shown to have a major influence on socioeconomic success and happiness. Photoaging, or dermatoheliosis, is propagated by UV exposure and leads to lentigines, seborrheic keratoses, rhytides (wrinkles), and many other benign skin changes. Many procedures and products have been developed in attempts to mask the visible signs of aging, and they vary widely in their costs and risks. It is important to realize that many patients undergo these procedures and may present with related complications.

Mitigating the Effects of Photoaging

Broad-spectrum sunscreens that block both UVA and UVB reduce the signs of photoaging and protect against melanoma and nonmelanoma skin cancer. Inorganic physical blockers such as zinc oxide and titanium dioxide are effective, broad-spectrum components of many commercially available sunscreens, and organic blockers in various combinations are typically also safe and effective.

Topical retinoids such as tretinoin are effective and frequently prescribed medications used to address the changes associated with photoaging. Long-term daily use moderately improves roughness, fine wrinkling, and dyspigmentation.[13] More invasive techniques include neurotoxins such as onabotulinumtoxin-A (Botox) that block acetylcholine release from motor neurons and improve rhytides that develop with repeated facial movement. Dermal fillers, such as hyaluronic acid, are also frequently used to treat superficial rhytides and replace volume where it has been lost over time. Various laser procedures, radiofrequency devices, dermabrasion, chemical peels, and surgical procedures carry potential for both risks and benefits. Only trained physicians should oversee such treatments and provide adequate counseling on risks.

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Appendages: Hair, Nails, and Glands

Hair has a major cosmetic function for most adults, and its distribution and thickness typically change with time. Most patients experience graying of >50% of their hair. Pattern hair loss is seen in nearly all males and more than half of females.[14] While scalp hair typically thins over time, hair density may increase on the upper lips and chins of females and on the ears, backs, and noses of males. Nails grow more slowly and become thinner and more brittle, while onychomycosis becomes more common.[10]

Lipid-secreting sebaceous glands typically increase in number over time as reductions in androgen levels slow cell turnover, but their function is reduced, leading to more frequent scaling and pruritis. On the face, proliferation of sebaceous glands may manifest as sebaceous hyperplasia.[15]

Benign Skin Lesions

Acrochordons

Acrochordons, commonly referred to as skin tags, are benign growths in elderly patients. They are soft, skin-colored, pedunculated papules in intertriginous areas such as the neck, axilla, and inguinal folds. They can be hyperpigmented or slightly verrucous. When numerous, obesity and acanthosis nigricans may be present and screening for diabetes mellitus is indicated.[16] If they are frequently traumatized or patients request removal, cryotherapy or removal with forceps and scissors are quick and effective when necessary. When larger, local anesthesia may be required at the base.

Cherry Angiomas

Cherry angiomas (also called cherry hemangiomas) begin arising in the third and fourth decades of life and are very common, especially in patients with fair skin. These bright-red vascular papules are benign, usually asymptomatic, can be scattered over any cutaneous surface, and are typically 1–5 mm in diameter, though are occasionally larger. Cosmetic treatment with vascular lasers or electrosurgery can be performed, but is typically unnecessary.

Seborrheic Keratosis

Seborrheic keratoses are a frequent cause of concern among patients and medical providers. While these pigmented papules and plaques typically have a classic waxy or verrucous, "stuck-on" appearance (Figure 38.1), they occasionally raise concern for melanoma because of variations in pigment and growth. While most are less than a centimeter in diameter, they can grow to several centimeters in size. They can be few or numerous. Patients often report the ability to partially remove or "pick off" portions with their fingernails, which can be reproduced in clinic with a tendency of the lesions to crumble slightly with blunt manipulation.

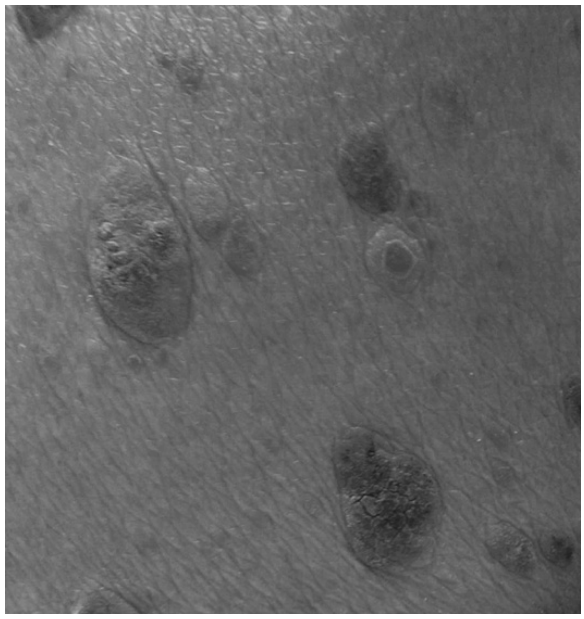

Figure 38.1 Seborrheic keratoses. Stuck-on, waxy, crumbling, hyperpigmented papules and plaques. (A black-and-white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

Recognition and reassurance are all that is typically required when counseling patients. While UV-induced signature mutations have been described in seborrheic keratoses and have a predilection for sun-exposed areas, they have no malignant potential. Inflamed seborrheic keratoses often have a pink or crusted appearance and may be confused for melanoma or nonmelanoma skin cancer in sun-exposed areas, but still tend to retain classic features and can be treated with cryotherapy, curettage, or sharp removal. No topical treatments are reliably effective.

Solar Lentigines

The solar or actinic lentigo results from increased melanin production limited to the basal layer of the epidermis. It is found with increased incidence in sun-exposed skin.[17] They are colloquially called "liver spots," but are not related to hepatic disease. The pigmentation is typically evenly distributed, with size ranging from a few millimeters to

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3–4 cm. The border often appears notched or "moth-eaten." Lentigines often darken with UV exposure, but do not fade completely without it. The most challenging differential is lentigo maligna, as both can show similar findings.

Actinic Purpura

Actinic purpura, also referred to as senile or solar purpura, are found in approximately 12% of patients over the age of 50 years and up to one third of elderly patients in long-term care facilities.[18] These asymptomatic, nonpalpable purpuras are most common on surfaces routinely exposed to minor trauma (Figure 38.2). With normal aging and UV light exposure, the skin and microvasculature become more susceptible to the routine shearing and forces that occur with daily activities that lead to purpura. Typical resolution occurs over the course of 3 weeks without the evolution in coloration seen in traumatic purpura. Repeated purpura can cause persistent hyperpigmentation. Anticoagulants and steroids can predispose patients to larger and more frequent purpura.

Other Common Benign Skin Lesions

Sebaceous hyperplasia is commonly noted on the face of elderly patients and occasionally confused with basal cell carcinoma (BCC). These symmetric yellow papules typically have a central punctum or dell. They are usually multiple and lack the arborizing vessels seen in basal cell carcinoma. Cosmetic destructive therapies can be pursued.

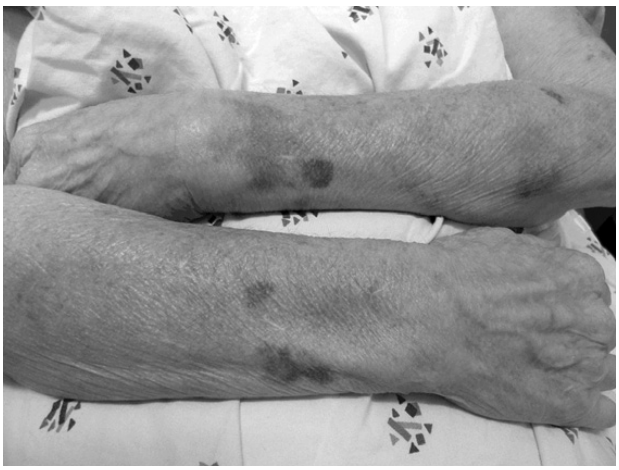

Figure 38.2 Actinic purpura. Purpura classically located on the forearm with absence of yellow-green hues. (A black-and-white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

Epidermal inclusion cysts (epidermoid cyst, sebaceous cyst, cyst of the follicular infundibulum) are derived from a wall of keratinizing follicular epithelium depositing keratinocytes into a central cavity. They present as dermal or subcutaneous mobile nodules with a central pore. While typically asymptomatic, manipulation and trauma may lead to rupture and subsequent sterile abscess formation. Incision and drainage usually leads to recurrence, so complete excision when asymptomatic is preferred definitive treatment. When inflamed, incision and drainage, intralesional triamcinolone, or antibiotics such as doxycycline may provide relief.

Malignant Skin Lesions

Skin cancers, affecting one in five Americans at some point in their life, are the most common malignancies encountered in the United States and have increased in frequency over the last three decades. BCC and squamous cell carcinoma (SCC), often described together as nonmelanoma skin cancer (NMSC), are not required to be reported to cancer registries, but an estimate based on Medicare databases and national survey data from 2006–12 suggested an incidence of 4.03 million in 2012 with annual cost of over $4.8 billion from 2007 to 2011.[19] NMSC make up the large majority. Most are due to sun exposure and are easily treated. Melanoma makes up a smaller proportion, but leads to the bulk of skin-cancer-related mortality. Routine screening skin exams are generally recommended every 1–3 years after age 40, depending on such risk factors as fair skin, family history, and prior UV exposure. Low-risk patients can be screened by primary care physicians comfortable providing full skin examinations. Patients with a history of skin cancer, multiple atypical pigmented lesions, >50 moles, or long-term immunosuppression should have an ongoing relationship with a dermatologist for routine monitoring.[20] Evidence supports sunscreen and sun avoidance to reduce risk of melanoma and NMSCs, including in patients with prior history of skin cancer.[21]

Basal Cell Carcinoma

Of the millions of nonmelanoma skin cancers that develop yearly, 75–80% will be BCC. Men are affected at a rate of 1.5–2:1 compared to women, with the majority on the head and neck.[22] UV exposure is the main contributor to BCC development, specifically via mutations in the gene *patched* (PTCH1).[23] Lightly colored hair and eyes, freckles, fair skin, immunosuppression, history of sunburns, radiation exposure, and family history can all contribute to increased risk.[20]

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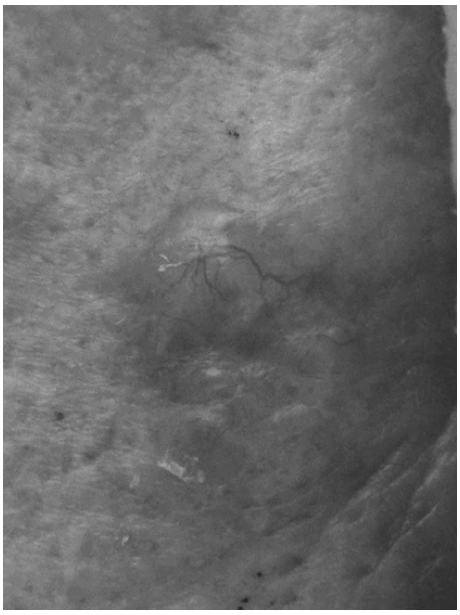

Figure 38.3 Basal cell carcinoma. Pearly papule in the retroauricular area with arborizing telangiectasias. (A black-and-white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

BCC classically appears as a pearly papule with arborizing telangiectasias and a rolled border, and ulceration may be present (Figure 38.3). The most common subtypes include superficial, nodular, and morpheaform. Up to 10% may have pigment, which may lead to confusion with melanoma. Metastasis and mortality is highly unusual as they often grow slowly for years, but morbidity is frequent because of bleeding and local invasion of deeper structures when left untreated. A shave biopsy is typically sufficient for diagnosis.[24]

Superficial BCC is contiguous with the epidermis and most easily treated. Electrodessication and curettage (E&C) allow for rapid treatment in the outpatient setting, little discomfort, and high cure rates. Topical therapy with imiquimod or 5-fluoruracil cream has lower cure rates than surgery, but is performed outside of the office with low morbidity and scar risk.[25] Nodular BCC is treated very effectively with E&C, but is less amenable to topical therapies. Standard excision with 4–5-mm margins is reliable in the outpatient setting for low-risk BCC.[25]

For poorly demarcated, large (>2 cm), or recurrent tumors, or those in cosmetically sensitive locations, Mohs surgery is the gold standard. The Mohs technique allows surgeons to evaluate the entire tumor margin on frozen sections for clearance prior to repair on the same day. Recurrence rates of <1% are typical. Table 38.2 summarizes common treatments for NMSC.

Actinic Keratosis and Squamous Cell Carcinoma

Actinic keratoses (AKs) are premalignant lesions arising on sun-damaged skin. There is an estimated rate of 1% per decade for evolution into invasive SCC, though some estimates are substantially higher. Since patients may present with multiple AKs, the risk quickly accumulates.

AKs present as erythematous macules or thin papules with sandpaper-like, gritty scale. Palpating high-risk areas such as the forehead, temples, cheeks, and nose is critical for detection. Cryotherapy is very successful in treating individual lesions (Grade A). Prescription topical treatment, such as 5-fluorouracil or imiquimod, is also highly effective for individual lesions or "field treatments" of widespread actinic damage over larger areas.[24,26]

AKs may evolve along a continuum to SCC in situ (SCCis), which can progress to invasive SCC (iSCC). SCCis and iSCC may also develop de novo. iSCC (Figure 38.4) should be suspected of lesions that develop thicker, nodular components, have rapid growth, bleed frequently, or are tender to palpation. Invasion of local nerves can cause persistent pain or paresthesia. Any lesion that is present or grows for more than 6–8 weeks, recurrently ulcerates or bleeds, and does not heal as expected should be biopsied.

A specific subtype of SCC, the keratoacanthoma typically arises and grows quickly, often reaching sizes of 2 cm or greater over the course of 4–6 weeks. It tends to have a characteristic central crater filled with keratinized debris. Most authors recommend surgical removal following diagnosis since it cannot be fully distinguished from invasive SCC.[27]

While SCCis does not have metastatic potential, iSCC can become aggressive in some instances. Estimates of metastatic disease developing in cutaneous SCC vary, but it is likely in the range of 3–5%. The lips, ears, genitals, and fingers carry an increased risk. SCCis can mostly be treated similarly to superficial or nodular BCC, whereas iSCC should be treated by standard excision or Mohs surgery depending on the size and location.[27] See Table 38.2 for additional information on the treatment.

Following the diagnosis of a first skin cancer, the risk of developing another skin cancer over the next 3 years increases to more than 10-fold that of the general population. Though standardized guidelines are lacking, most patients are followed every 6 months for full skin exams for 1–2 years

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Table 38.2 Common treatments for nonmelanoma skin cancer

	Description	Indications	Advantages	Disadvantages	Cure rates
Mohs micrographic surgery	Surgeon reads frozen section pathology and takes further sections until tumor is cleared	BCC, SCC, SCCis (all Grade A). Especially for high-risk locations, large tumors, and recurrent tumors	High clearance rates; single physician can often handle excision, pathology, and repair in the outpatient setting with local anesthesia	May require several hours in the office if multiple passes are needed to achieve clear margins	98–99%
Excision	Excision with local anesthesia, typically 3–4-mm margins with permanent section pathology in the following days	BCC, SCC, SCCis (all Grade A)	Short time in the office (30–90 min) with local anesthesia	May require larger margins and repairs than Mohs. Requires some restrictions in activities for 7–10 days. Not ideal for some tumors >2 cm or in high-risk locations	~95–98%
E&C	3 cycles of curettage followed by electrodessication	Superficial and nodular BCC, SCCis (all Grade A)	Fast (~10 min); local anesthesia in the office. Minimal wound care	Likely higher recurrence for more aggressive subtypes and high-risk areas such as face. Circular scar not ideal in some locations	~95%
Imiquimod	Topical immunotherapy activating Toll-like receptor-7. Applied 5x weekly for 6–12 weeks for skin cancer or 2–3x weekly for AKs	AKs (Grade A), superficial (Grade A) and nodular BCC (Grade B), SCCis (Grade B)	Patient-controlled, excellent cosmetic outcome	Higher recurrence rate than most physical modalities. May result in significant local inflammation. Chance of patient error	72–82% varies with duration and frequency. Lower responses for nodular BCC
5-fluorouracil	Pyrimidine analog that blocks DNA synthesis. Twice daily application for 3–6 weeks	AKs (Grade A), superficial (Grade B) and nodular (Grade D) BCC, SCCis (Grade B)	Patient-controlled, excellent cosmetic outcome	Higher recurrence than most physical modalities. May result in significant local inflammation. Chance of patient error	57–90%
Photodynamic therapy	Topical precursor of protoporphyrin IX applied and incubated. Red- or blue-light exposure results in reactive oxygen species production	AKs (Grade A), superficial BCCs (Grade A), SCCis (Grade A)	In-office treatment, more physician control than topical pharmacotherapy. Excellent cosmetic outcome	Higher recurrence than most physical modalities. May result in significant inflammation	73–98% clearance reported, protocols vary

Information from references 24–27

following the diagnosis of a nonmelanoma skin cancer and then annually.[28]

Melanoma

The least common and most lethal of the three main forms of skin malignancy, melanoma is the fifth most common cancer in men and the sixth most common in women. Incidence is steadily increasing, though advancements in diagnosis and treatment have resulted in a declining death rate over the past 5 years. Survival without nodal involvement approaches 99%, but 5-year mortality for metastatic disease is ~25%. The probability of developing invasive melanoma more than doubles in patients 70 and older.[22,29]

A number of mutations – including CDKN2A, melanocortin-1 receptor (linked to fair skin and red hair), and BRCA1/2 – are linked to higher rates of melanoma. Blistering sunburns, cumulative UV exposure, lighter skin types, presence of >50 nevi or atypical nevi, family history, and older age all increase melanoma risk.[30]

The clinical diagnosis of melanoma is challenging. Benign nevi (moles) are common, benign, melanocytic neoplasms that often have overlapping clinical features with melanoma. Provider familiarity with the ABCDEs of

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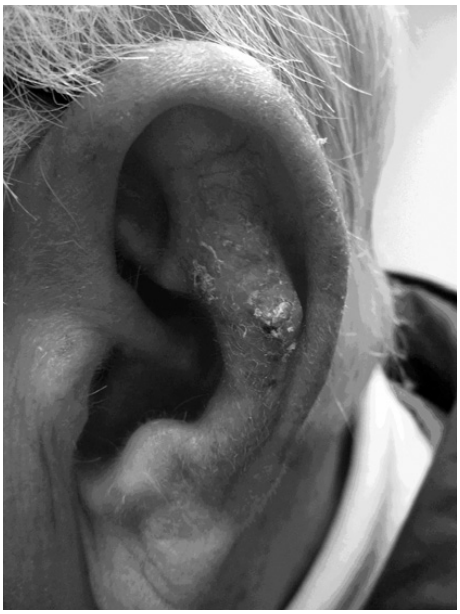

Figure 38.4 Squamous cell carcinoma. Crusted nodule on the antihelix, a high-risk location. (A black-and-white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

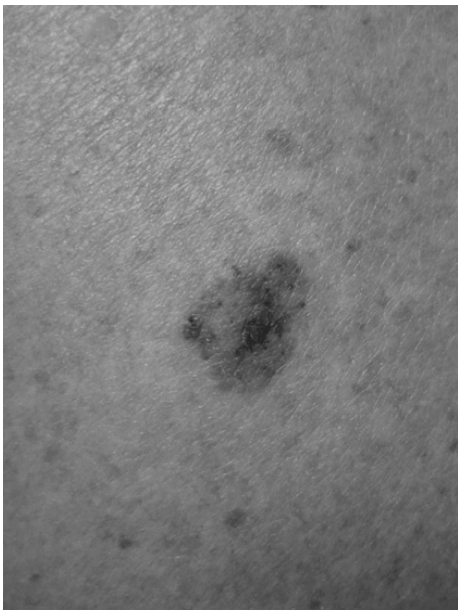

Figure 38.5 Superficial spreading melanoma. Irregularly shaped, pigmented plaque with variegated pigment. (A black-and-white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

melanoma (Asymmetry, Border irregularity, multiple Colors, Diameter >6 mm, and Evolution) is helpful. Benign nevi with one or two of these features, known as atypical nevi, are common. When patients have many atypical nevi, it is useful to recognize one or two patterns of "signature nevi" with similar appearance. An "ugly duckling" lesion that does not match the background pattern should raise suspicion for melanoma.[31]

Subtypes of melanoma include superficial spreading, nodular, lentigo maligna, and acral lentiginous. Up to 10% of all subtypes may be amelanotic or hypomelanotic.[32] The superficial spreading subtype (Figure 38.5) is the most common, but lentigo maligna melanoma increases in elderly, highly sun-damaged skin. Nodular melanoma is less common but has early vertical growth and often presents at more advanced stages. Acral lentiginous melanoma presents on the palms, soles, and within the nail unit. It is the least common type of melanoma overall, but makes up a higher proportion of melanomas in darker skin types.[30,33]

Urgent biopsy should be performed for suspected melanoma. Ideally, excisional biopsy is utilized to allow for adequate assessment of full tumor. The Breslow depth, a measure of invasive depth, is the most important prognostic factor and is essential for appropriate staging. Melanoma in situ, which is limited to the epidermis, carries little to no risk of metastasis. When ulceration is absent and Breslow depth is <1 mm, 10-year mortality is <5% following excision. Tumors with Breslow depth >4 mm with ulceration have 10-year survival of ~50%. Advanced age is associated with more advanced disease at diagnosis and poorer prognosis.[30,33]

The typical standard of care for melanoma is surgical excision with margins based on the depth of tumor invasion. There is an expanding role for Mohs surgery in cosmetically sensitive areas for thin melanomas, such as lentigo maligna, because of high clearance rates and excellent cosmetic and functional outcomes.[34] In tumors with >1 mm Breslow depth, a sentinel lymph node biopsy is generally indicated for prognostic purposes and to guide adjuvant treatment, though the benefit of complete lymph node dissection is controversial given the associated morbidity. Some recent studies have advocated instead for lymph node surveillance.[35,36] Metastatic melanoma has a poor prognosis, but, after decades of stagnation, major breakthroughs have emerged. In BRAF-mutated tumors, initial targeted therapy with BRAF and MEK inhibitors has led to dramatic responses, though recurrence over 1–2 years is frequent.[37] Immunotherapy blocking CTLA-4 and PD-1 allow activation of a patient's immune system to aid in tumor destruction and have led to

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improved survival and durable remissions for a substantial minority of treated patients. When used in combination for metastatic melanoma, PD-1 and CTLA-4 inhibitors have been shown to extend progression-free survival by more than 5 months and increase 2-year overall survival to 63.8%.[38]

Following invasive melanoma diagnosis, follow-up surveillance with review of systems, complete skin exam, and lymph node palpation should occur every 3 months for 12 months, every 6 months in years 2–5, and then annually.[33]

Inflammatory Skin Diseases

Rosacea

Rosacea is a common, chronic inflammatory condition that predominantly involves the central face and has a predilection for patients with fair skin and females.[39] There are four morphological subtypes.[40] Erythematotelangiectatic rosacea (ETR) is characterized by erythema, flushing, and scattered telangiectasia on the forehead, nose, and malar cheeks. For patients with skin of color, these changes are more subtle, so detailed history is essential. Exacerbating factors include alcohol, ultraviolet radiation, caffeine, heat, emotion, smoking, and spicy foods. Papulopustular rosacea presents with papules and pustules of the central face, though comedones typically seen in acne are absent (Figure 38.6). Phymatous rosacea is recognized by marked, nodular tissue hypertrophy that distorts the facial contours; nose involvement (rhinophyma) is most common, followed by chin, cheeks, and ears. Ocular rosacea affects up to 50% of patients with skin manifestations[39] with symptoms including redness, burning, watering, dryness, and light sensitivity. Ocular symptoms warrant referral to an ophthalmologist for management.

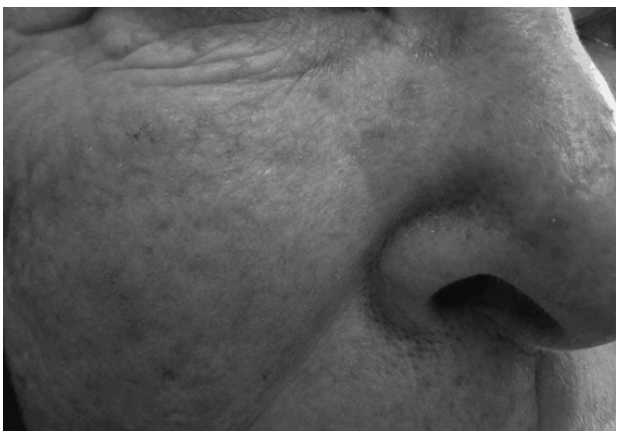

Figure 38.6 Rosacea. Erythema, telangiectasias, and scattered papules and pustules. (A black-and-white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

Dysregulated innate immunity and genetics likely play a role in pathogenesis, but no specific genes have been implicated. *Demodex folliculorum* infestation and *Helicobacter pylori* infection may also play a role in some patients.[41–42] Initial management includes counseling on avoidance of triggers and camouflage with green-tinted cosmetics.

Topical metronidazole, azelaic acid, and sulfacetamide-sulfur formulations applied daily are effective in mild disease, and oral tetracyclines are the next-line therapy. Ocular rosacea typically requires long-term control with oral tetracycline class antibiotics. For patients with ETR or persistent erythema, topical brimonidine is an alpha-2 adrenergic receptor agonist and works immediately to vasoconstrict dilated vessels and diminish redness. Laser is efficacious for erythema and telangiectasias. In severe papulopustular or early phymatous rosacea, oral retinoids such as acitretin or isotretinoin may be beneficial. Surgical or laser contouring may be required for severe phymatous deformity. Topical steroids are generally avoided because of dependence and a rebound effect.[39]

Seborrheic Dermatitis

Seborrheic dermatitis is a chronic inflammatory condition characterized by relapsing erythema and greasy, adherent scale that involves the glabella, eyebrows, alar creases, and nasolabial folds. Other frequently involved areas include the chest, frontal hairline, and ear canals (Figure 38.7). In patients with darker skin, scaly macules and patches may be hypopigmented and in arcuate formation. The etiology is thought to be due to the host response to commensal saprophyte *Malassezia*.[4,5,20,43] Patients with Parkinson's disease or HIV may have severe seborrheic dermatitis.

Mild disease is usually controlled with antifungals such as ketoconazole 2%, selenium sulfide 2.5%, or ciclopirox 1%. Creams are ideal for the face, whereas shampoos are used on the scalp. Suggested use is two to three times per week until controlled, then weekly for maintenance. For the face, low-potency steroids, such as hydrocortisone 2.5% or desonide 0.05% cream, are common adjuncts. For the scalp and body, mid- to high-potency steroids can be safely used, and patients with skin of color may prefer fluocinonide oil for the scalp. Short or intermittent courses of oral fluconazole can be beneficial for flares or severe disease.[43]

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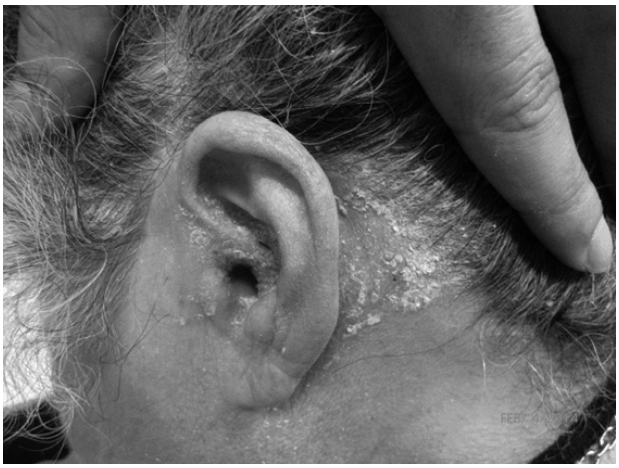

Figure 38.7 Seborrheic dermatitis. Erythematous plaques that are flaky, adherent, and slightly greasy affecting the scalp, ear, and retroauricular sulcus. (A black-and-white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

When seborrheic dermatitis is resistant to therapy, consider other diagnoses such as rosacea, psoriasis (particularly for scalp involvement), tinea corporis, systemic lupus erythematosus (SLE), and periorificial dermatitis.

Psoriasis

Psoriasis is an immune-mediated disorder typically presenting with erythematous papules and plaques with silvery scale (Figure 38.8). Subtypes include plaque, guttate, inverse, erythrodermic, pustular, and nail psoriasis. It affects 1–3% of the world's population, and 3.2% of patients have onset after age 60.[44] Diagnosis is usually clinical, but biopsy can confirm. Since 5–15% of patients develop psoriatic arthritis, it is important to screen for symptoms such as morning stiffness, joint swelling, and chronic tendonitis.[45] A rheumatologist should evaluate patients with suspected joint involvement. Psoriasis is also independently associated with elevated risk of coronary artery disease, type 2 diabetes mellitus,[46] and nonalcoholic fatty liver disease in patients over 55 years old.[47]

Streptococcus pyogenes has been associated with new-onset guttate psoriasis. Known medication triggers include antimalarial drugs, beta-blockers, and lithium, followed by angiotensin-converting enzyme inhibitors, NSAIDs, and terbinafine.[48] Abrupt cessation or rapid tapering of corticosteroids may provoke erythrodermic and pustular flares, and TNF-α antagonists have triggered palmoplantar and diffuse pustular psoriasis in patients without a prior history of disease.

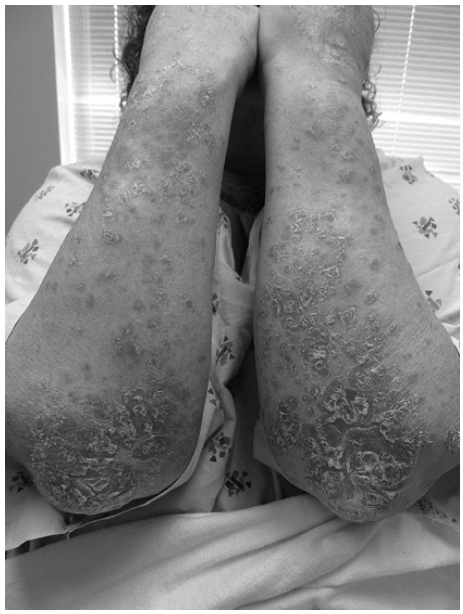

Figure 38.8 Psoriasis. Scaly, erythematous plaques with a predilection for extensor surfaces. (A black-and-white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

The first step in treatment is medium- to high-potency topical steroids. For facial or intertriginous areas, low- to mid-potency steroids, calcipotriene, or topical calcineurin inhibitors may be used. For patients with suboptimal control with topicals, nbUVB (311 nm) phototherapy and/or first-line oral therapies including methotrexate and acitretin can be considered, though lab monitoring is required.[49] Biologics options include TNF-α inhibitors, anti-IL-17 monoclonal antibodies, and anti-IL-23 monoclonal antibodies. Since these may be associated with a small infection risk, which increases with age,[50] the patient should be evaluated for latent tuberculosis and viral hepatitis prior to initiation. Specifically, a 1.5–3-fold increase in likelihood of developing herpes zoster was reported in psoriasis patients on systemic corticosteroids or immunotherapy.[51] It is recommended that all patients >50 years old, regardless of systemic therapy, be administered the recombinant zoster vaccine.[51]

Atopic Dermatitis

Atopic dermatitis (AD) is characterized by pruritic, erythematous, scaly plaques and a waxing and waning course. Onset is typically in early childhood, but it can continue through adulthood or occasionally present at

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older ages. The estimated prevalence of adult AD is 7%, with 25% reporting adult onset.[52] In adults, the hands, feet, and flexural surfaces are frequently involved. Less commonly, nonflexural morphological variants may occur. These include nummular (Figure 38.9), follicular, seborrheic dermatitis-like, and prurigo-like patterns.[53] Constant rubbing and inflammation may lead to lichenification of the skin, and secondary bacterial superinfection (most commonly *Staphylococcus aureus*), dermatophytosis, and eczema herpeticum can occur.

Diagnosis is usually clinical, although biopsy may help rule out other diagnoses. Contact dermatitis may have similar clinical and histologic findings, so patch testing is often performed in new adult-onset disease or with acute worsening, as up to one third of nummular dermatitis patients had a reaction to one or more allergens (nickel sulfate was highest at 10.2%).[54] Other conditions to consider include pityriasis rosea, lichen simplex chronicus, scabies, tinea corporis, and seborrheic dermatitis.

The discovery of the filaggrin mutation in some patients indicates that at least a subset is caused by a skin barrier dysfunction. Other subtypes may be predominately due to immune system dysregulation. Both of these ultimately result in abnormalities in cutaneous immunity, which is critical for protection against pathogens as well as tolerance to benign antigens.

First-line treatment includes generous emollient use and avoidance of triggers such as heat, low humidity, and identified contact allergens. Food and aeroallergens are infrequent triggers, and food-elimination diets are not generally recommended. The mainstay for mild disease is mid- to high-potency topical steroid preparations on the trunk and extremities, and low- to medium-potency topical steroids on the face. Antihistamines can be useful for associated pruritus, particularly for nocturnal itching. For disease that is recalcitrant to topical regimens, narrowband UVB (311 nm) or immunomodulating medications are warranted. Historically, first-line maintenance therapies have included methotrexate, azathioprine, and mycophenolate mofetil, though they are best prescribed by providers familiar with their use. The first biologic medication approved for AD, dupilumab, leads to substantial improvement in most patients, with one third having clear or almost clear skin with durable response to maintenance treatment. The only reported adverse effect compared to placebo in trials was conjunctivitis.[55]

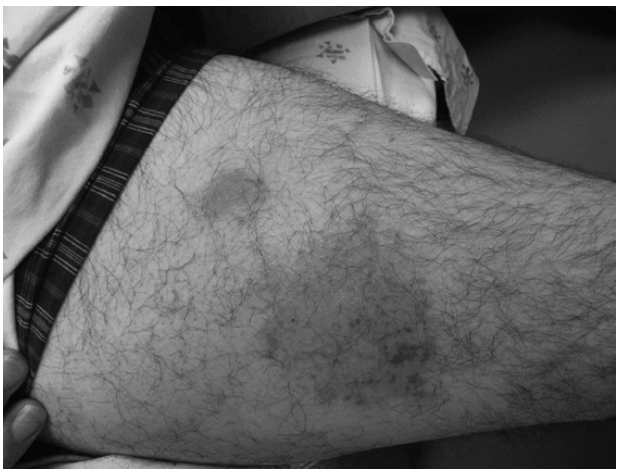

Figure 38.9 Nummular dermatitis. Annular, slightly scaly, pruritic plaques on the extremities. (A black-and-white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

Pruritus

Pruritus is a common and challenging complaint in the elderly population. Each year in the United States, more than 7 million patients complain of itching at outpatient visits, including 2 million patients 65 years or older.[56] Pruritus, though rarely life-threatening, can be a serious detriment to quality of life, sleep, and mental health.

Pruritus can be grouped into pruritus with skin disease, pruritus without skin disease, and pruritus resulting from secondary skin changes such as chronic rubbing or scratching (Table 38.3). If there is primary skin pathology, treatment should focus on the underlying condition. Common pruritic conditions include AD, Grover's disease, infectious diseases, and medication reactions.

Impaired skin barrier function and immunosenescence with aging may contribute to pruritus. Immunosenescence results in impaired cellular immunity, increased risk of infection, and a cytokine profile that favors inflammation and itch. Skin dryness, or xerosis, worsens over time with more than 50% of elderly patients affected. Xerotic itch often temporarily improves during bathing, worsens in winter months, and favors extremities over the trunk.[56] Harsh soaps, hot water, detergents, and astringents should be avoided. Application of petrolatum or ceramide-containing moisturizers should be performed immediately after bathing and throughout the day.

Chronic rubbing or picking may lead to well-demarcated, lichenified papules or plaques termed lichen simplex chronicus (Figure 38.10) and prurigo or

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Table 38.3 Differential diagnosis and evaluation of pruritus

Groups	Subgroups	Examples	Possible evaluation
Pruritus with skin disease	Primary dermatologic conditions	Atopic dermatitis Xerosis Urticaria Allergic/irritant dermatitis Lichen planus Cutaneous T-cell Lymphoma Grover's disease Bullous pemphigoid	Skin biopsy Patch testing
	Infectious diseases	Scabies Head or pubic lice Dermatophytoses (Tinea pedis, capitus, corporis, cruris, versicolor, Majocchi granuloma) Id reaction at distant site	KOH evaluation Mineral oil preparation Skin biopsy with special stains Tissue culture
	Medications	Photosensitizers (Thiazides, tetracycline, ACE inhibitors, CCBs, NSAIDS, quinine, and amiodarone)	Evaluation of medications started within 6 weeks of itch Skin biopsy
	Physiologic changes of aging skin	Impaired skin barrier function Immunosenescence	Diagnosis of exclusion
Pruritus without skin disease	Systemic disease	Thyroid, liver, and renal disease Hematopoietic disorders (lymphoma, polycythemia vera) Sensory neuropathy due to diabetes	Screening lab-work: CBC with differential, fasting plasma glucose, TFTs, LFTs, BUN/Cr, Ca, Phos, TSH, PTH, ferritin
	Neuropathic	Genital pruritus Forearms (brachioradial pruritus Midback (nostalgia paresthetica) Herpes zoster (dermatomal)	Nerve impingement, particularly C5–C8 in brachioradial pruritus and T2–T6 in nostalgia paresthetica Examination for lichenification or hyperpigmentation of involved areas
	Medications	ACE inhibitors, salicylates, chloroquine, CCBs	Evaluation of medications started within 6 weeks of itch
Pruritus with secondary skin disease	Due to chronic scratching or rubbing	Lichen simplex chronicus Prurigo nodularis	Psychological evaluation Exclusion of systemic disease Intralesional triamcinolone injections Topical steroids Unna boot wraps Oral antihistamines Systemics: methotrexate, CsA, thalidomide, MMF

Information from reference 56
CBC (complete blood count), TFT (thyroid function tests), LFT (liver function tests), BUN (serum urea nitrogen) Cr (creatinine), Ca (calcium), Phos (phosphorus), TFTs (thyroid function tests), PTH (parathyroid), CsA (cyclosporine A), MMF (mycophenolate mofetil)

"pickers" nodules. Once established, these reactive changes perpetuate an "itch-scratch" cycle that can be difficult to break. In severe, recalcitrant cases, a psychiatric evaluation may be necessary.

If no rash or skin pathology is evident on exam, this typically indicates a systemic or neurologic cause, and the focus should be directed at patient comorbidities and medications (see Table 38.4).

In some patients, no clearly identifiable cause will be found despite an appropriately thorough workup. For these patients, a combination of age-related skin changes is often the diagnosis of exclusion. Management is then centered on symptom management and mitigation of triggers or exacerbating agents.

Lower-Extremity Stasis-Related Skin Changes

Lower-extremity chronic venous insufficiency is a common finding in the elderly and is associated with a number of dermatoses, the most common of which is stasis dermatitis.

Stasis dermatitis affects a reported 6.2% of patients over the age of 65 and results from chronic venous hypertension.[57] The skin overlying the affected areas can be

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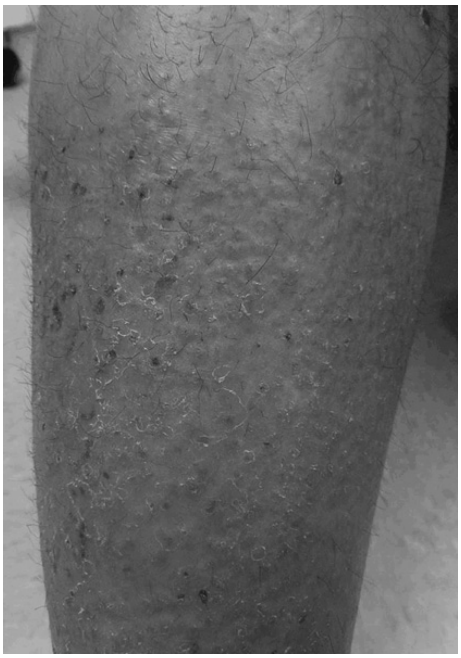

Figure 38.10 Lichen simplex chronicus. Thick, excoriated, lichenified plaque on the lower leg at a site of frequent scratching due to contact dermatitis. (A black-and-white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

brightly erythematous or violaceous, or it can appear rust-colored because of red blood cell extravasation and hemosiderin deposition in the dermis. Eczematous patches or plaques with scaling are typical, and in acute cases the skin can be weeping and vesicles may be present. It is not uncommon to have skin breakdown with ulcerations or widespread weeping of serosanguinous transudate.

The treatment for stasis dermatitis is targeted at improving venous return and edema. This is best accomplished by leg compression, elevation, and daily exercise. Mid- to high-potency corticosteroids such as triamcinolone or clobetasol ointments are a mainstay of therapy for active inflammation. For ulceration, barrier creams like zinc oxide are important to protect the skin from further trauma or secondary infection. Zinc oxide-impregnated compression wraps (Unna boots) are highly effective at reducing edema and promoting wound healing.

It is important to note that patients with stasis dermatitis are prone to developing contact allergies, so topical neomycin, bacitracin, and gentamicin should be avoided.[58] Secondary infection can be treated with antiseptic wraps or topical mupirocin, which is least likely to cause cutaneous sensitization. Secondary cellulitis requires oral antibiotic therapy, though this type of infection may mimic stasis dermatitis with warmth, erythema, and edema. The key differentiating feature is the distribution: stasis dermatitis is almost invariably bilateral, and it is exceedingly rare for cellulitis to be bilateral. (See Erysipelas and Cellulitis section for further reading.)

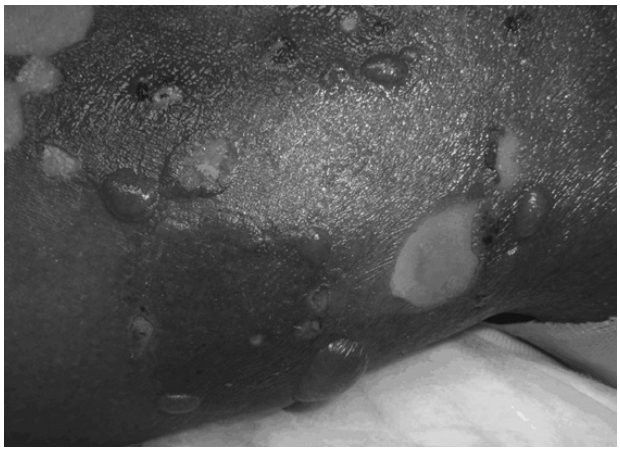

Figure 38.11 Bullous pemphigoid. Widespread, tense bullae erupting diffusely on the trunk and extremities. Shallow ulcers are present where bullae have ruptured. (A black-and-white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

Autoimmune Blistering Disorders

Autoimmune blistering disorders (ABDs) are characterized by the development of autoantibodies against cell–cell adhesion molecules and are broadly divided into pemphigus and pemphigoid. Pemphigus antigens are located within intercellular epidermal junctions called desmosomes, and the blisters are shallow and clinically flaccid. Pemphigoid antigens are located at the basement membrane where the epidermis and dermis are anchored together by hemidesmosomes, and the blisters are therefore deeper and tense when intact[59] (Figure 38.11). Both pemphigus and pemphigoid have subtypes that are clinically distinct. These subtypes affect different epithelial surfaces, have different responses to therapy, and have different natural courses. (See Table 38.5 for a brief review of these disorders.)

Diagnosis of ABDs is usually made using a combination of diagnostic tools, including skin biopsies for traditional hematoxylin and eosin (H&E) as well as direct immunofluorescence (DIF), which looks for the presence of autoantibodies deposited in the skin. Serum is also collected for enzyme-linked immunosorbent assay (ELISA) and indirect immunofluorescence to detect circulating autoantibodies.

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Table 38.4 Treatment of pruritus

Agent	Examples*	Applications
Topical steroids	Desonide Triamcinolone Clobetasol	Primary skin diseases excluding infectious Secondary skin diseases
Topical calcineurin inhibitors	Pimecrolimus Tacrolimus	Alternative to steroid for chronic use or for use on face/genital/intertriginous areas
Topical cooling agents	Menthol Camphor Phenol	For soothing effect. Can be kept in the refrigerator for added cooling effect
Topical anesthetics	Pramoxine 1% cream Eutectic mixture of lidocaine 2.5% + prilocaine 2.5% (EMLA) Lidocaine in acid mantle cream Lidocaine patch	Focal pruritus and neuropathic etiologies
Other topicals	Topical doxepin 5% Topical capsaicin 2–6%	Atopic dermatitis Neuropathic itch Neuropathic itch
Systemic antihistamines	Diphenhydramine Cetirizine Fexofenadine Loratadine	Focal or systemic pruritus. Used frequently despite lack of evidence of efficacy for diseases that are not specifically histamine mediated Use in geriatrics may be limited by anticholinergic effects and confusion
Other systemic medications	Gabapentin 100–300 mg and increased up to 1,800 mg/day. SNRI, esp. mirtazapine SSRI such as paroxetine, sertraline, fluvoxamine Doxepin	Neuropathic itch Some primary skin disease Itch associated with systemic disease Nocturnal pruritus
Adjuvant therapy	nbUVB	ESRD

Information from reference 56
* Topical steroids are typically applied twice a day to affected areas until improvement or up to 2 weeks.
Note: Topical antihistamines generally not recommended because of the risk of irritant or contact dermatitis and lack of efficacy.
Selective norepinephrine reuptake inhibitors (SNRIs), Selective serotonin reuptake inhibitors (SSRIs)

Prior to the use of immunosuppressants, pemphigus vulgaris (PV) was invariably fatal within 5 years of onset. Prednisone and long-term immunomodulators such as methotrexate, azathioprine, and mycophenolate mofetil were the mainstays of therapy for many years with relatively good overall success. More recently, the use of rituximab, an anti-CD20 antibody, in moderate to severe disease has revolutionized treatment with complete remissions off therapy in up to 90% of patients.[60] Bullous pemphigoid (BP) is associated with relatively low mortality, and topical steroids are a reasonable first-line therapy, assuming the patient is able to apply the medicine twice a day for at least several weeks.[61] Oral glucocorticoids are used to establish control in BP with the addition and gradual tapering of an adjunct immunosuppressant such as methotrexate or mycophenolate mofetil. Though its use is more controversial in BP than PV, rituximab has shown promising results in recalcitrant cases of pemphigoid.[62] For both pemphigus and pemphigoid variants with mucosal involvement in particular, additional measures include observation for secondary infection (especially herpes simplex virus and candida); avoidance of sharp, spicy, or abrasive foods; and good oral hygiene. The goal for all ABDs is long-term remission off of therapy.

Infectious Skin Diseases

Erysipelas and Cellulitis

Erysipelas is an acute, superficial subtype of cellulitis, usually caused by group A beta-hemolytic streptococcus, that involves both the upper dermis and superficial lymphatics.

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Table 38.5 Classification and presentation of autoimmune blistering disorders (ABDs)

ABD	Subtype	Distribution	Other features
Pemphigoid	Bullous Pemphigoid	Generalized or focal May present as itchy urticarial or eczematous plaques Trunk and extremities Rarely involves mucosa	Most common ABD Primarily affects the elderly Chronic, relapsing course, but can have long-term remission Non-scarring
	Mucous Membrane Pemphigoid (cicatricial pemphigoid)	Any mucosal surface, including oropharyngeal, conjunctiva, nose, esophagus, anal, and genital Cutaneous involvement is less common	Scarring
	Drug-induced pemphigoid*	Trunk and extremities Mucosa not uncommon	Acute and resolves within months after discontinuation of offending drug Non-scarring
Pemphigus	Pemphigus vulgaris	A. mucosal only B. mucosal and skin	Most common pemphigus Painful oral lesions. Significant morbidity. Long-term remission possible with immunosuppression. May be drug-induced**
	Pemphigus foliaceous	Superficial blisters not usually intact Seborrheic distribution	May mimic Hailey-Hailey or Grover's disease May be drug-induced**
	Paraneoplastic pemphigus	Characterized by painful, intractable stomatitis. Life-threatening upper airway and pulmonary involvement can occur	Most commonly associated with non-Hodgkin's lymphoma, chronic lymphocytic leukemia, Castleman's disease, and thymoma
	IgA pemphigus	Grouped vesicles that evolve into pustules, sometime in annular or circinate patterns Usually trunk and proximal extremities	May be pruritic

Information from references 59–62

* More than 50 different medications have been implicated, including antibiotics, NSAIDs, antiarrhythmics/antihypertensives, diuretics, salicylates, TNF-alpha inhibitors, and diabetes medications, among others.

** Penicillamine, captopril, penicillins, cephalosporins, enalapril, rifampin, and NSAIDS

It typically involves the face or a lower extremity and appears clinically as well-defined, bright-red, warm plaques with pain usually preceding skin findings, and possible acute onset with fever and chills. Diabetes is a significant risk factor. Mild cases can be treated with oral penicillin V or amoxicillin with more severe cases requiring hospitalization for IV therapy such as cefazolin, which provides additional coverage against some staphylococci – useful in cases where the diagnosis is unclear. Treatment for 5–10 days is recommended.

Cellulitis generally extends deeper than erysipelas and classically has a more ill-defined border, along with erythema and tenderness. There may be fluctuance, purulent drainage, or occasionally crepitus. Cellulitis can complicate a surgical wound infection, pressure ulcer, vascular ulcer, or sites of trauma. In the elderly population, sores and fissures between the toes due to tinea pedis and macerated skin are important access portals for pathogens.[63] The lower extremities are a common site of involvement, and a population-based study reported the incidence of lower-extremity cellulitis to be 199 per 100,000 person-years. The incidence of cellulitis was found to increase with age.[64]

Crepitus, central necrosis, or pain out of proportion to physical exam findings may suggest deeper infection, such as gangrenous cellulitis, necrotizing fasciitis, synergistic gangrene, or myonecrosis. These can develop and progress rapidly and are highly lethal. Identification of the responsible pathogen with wound gram stain and culture is the gold standard in effective antimicrobial therapy in soft-tissue infections, though deeper infections may have relatively few organisms and require empiric broad-spectrum coverage. Radiographic examination is not indicated.

Cellulitis is most often due to *Staphylococcus aureus*. Purulent cases are often due to MRSA, whereas non-purulent cases are usually due to beta-hemolytic streptococci or MSSA. Gram-negative aerobic bacilli are less

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common. Patient characteristics – such as immunodeficiency, comorbidities, history of hospitalization or surgical procedure, and the location of the infection – should prompt consideration of other pathogens. Pressure ulcer cellulitis can be due to both typical skin organisms and facultative and anaerobic microorganisms from the bowel. Cultures are not usually useful for mild infection,[65] which can be treated empirically. Patients with signs of systemic toxicity should be treated parenterally. Cultures of blood, pus, or bullae are indicated for patients with systemic toxicity, underlying comorbidities, persistent or extensive disease, buccal and periorbital cellulitis, and special exposures such as animal bites and salt or freshwater.[65]

Herpes Zoster

Primary varicella-zoster virus (VZV) infection results in varicella, or chickenpox, which is well recognized as a diffuse vesicular rash. Herpes zoster, commonly known as shingles, is due to the reactivation of latent VZV from the sensory dorsal root ganglia. It is estimated that 32% of the population will experience zoster over their lifetime.[66] Increasing age, malignancy, and conditions resulting in impaired cellular immunity significantly increase risk.[67]

Prodromal neuropathic pain and paresthesia in a dermatomal distribution for 1–2 days is an important clinical clue. Vesicles with an erythematous base develop within a few days (Figure 38.12), which evolve into pustules that dry and crust over within 7–10 days. Lesions are infectious to those without previous varicella exposure or vaccination for the first 7 days in uncomplicated cases, but in disseminated cases, the virus can be aerosolized, requiring airborne precautions. Diagnosis can be made clinically, but surface swab for polymerase chain reaction (PCR) is rapid, inexpensive, and highly sensitive and specific. Tzank smear can be performed at bedside, but is less reliable, and direct immunofluorescence testing is usually slower to result and more expensive.

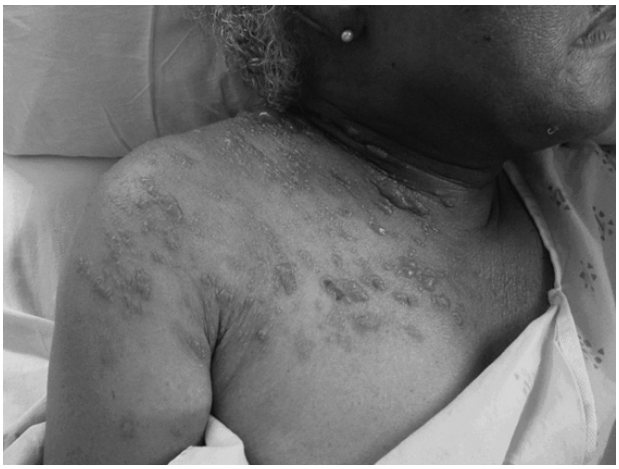

Figure 38.12 Herpes zoster. Clustered vesicles on an erythematous base in a dermatomal distribution. Note the sharp demarcation at midline. (A black-and-white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

Postherpetic neuralgia (PHN) is a common complication defined as pain lasting at least 90 days after resolution of skin lesions. Older age is a risk factor and it occurs in 20% of patients over age 80 years.[68] Development of PHN correlates with the presence of prodromal pain, initial pain severity, and patient age. Herpes zoster ophthalmicus (HZO) occurs when VZV reactivates in the ophthalmic division of the trigeminal nerve. Fifty to seventy-two percent of those cases have eye involvement. Nasal tip involvement (Hutchinson's sign) suggests nasociliary nerve branch involvement, which can lead to neurotrophic keratitis and chronic corneal ulceration, or acute retinal necrosis. Herpes zoster oticus (Ramsay Hunt syndrome) involves facial and auditory nerves leading to ipsilateral facial palsy, ear pain, and vesicles on the external ear canal or tympanic membrane. Tinnitus, vertigo, taste perception, lacrimation, and deafness may also occur. Ramsay Hunt syndrome can also be due to herpes simplex type 2 infection. Immunosuppressed patients are at risk for central nervous system involvement, including leptomeningitis, meningoencephalitis, segmental or transverse myelitis, or local palsies. Stroke syndromes due to involvement of the cerebral arteries have been reported.[68–69]

Antiviral therapy with acyclovir, famciclovir, or valacyclovir initiated within 72 hours of the onset of the rash can reduce healing time and duration and severity of acute pain,[68–70] though the impact on PHN is unclear. Early treatment with gabapentin may reduce risk and severity of PHN. Systemic corticosteroids are controversial, but can be considered with severe pain, cranial polyneuritis, or motor neuropathy. Treatment for acute pain or PHN includes lidocaine 5% patches, gabapentin, pregabalin, opioids, and tricyclic antidepressants. Zoster vaccination is indicated in patients who are 50 years of age or older regardless of history of zoster.[71] Vaccination has been shown to decrease rates of herpes zoster and risk of PHN, and a recently developed recombinant vaccine reduces rates of zoster by nearly 90% in the elderly population.[71,72]

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Scabies

Scabies is an infestation by the mite *Sarcoptes scabiei.* Though usually transmitted by direct contact, it can survive on fomites for 24–36 hours.[73] The characteristic intense itch is due to a delayed type IV hypersensitivity reaction to the mite as well as its feces and eggs.[74] Close-quarter living arrangements, such as in nursing facilities, increase the risk of outbreaks, as does limited mobility that is more common in elderly patients.[75]

It is critical to consider scabies in all elderly patients complaining of intense or new-onset itch out of proportion to skin findings, particularly with involvement of finger web spaces, axillae, waist, and genitals. In the elderly, there may be complete sparing of finger web spaces, but face and scalp involvement, and burrows on the soles of the feet.[75] Physical findings including erythematous papules, excoriations, small vesicles or pustules, and linear burrows 2–15 mm long are pathognomonic, though not always evident. A positive scabies preparation confirms the diagnosis, but is not required for treatment.

Two doses of permethrin 5% cream applied 1–2 weeks apart should be massaged thoroughly into the skin from scalp to toes, including the areas under the finger and toenails, and washed off in 8–14 hours. This is highly effective, though limited mobility and ability to apply to full body must be considered in elderly patients.[75] Oral treatment with ivermectin 200 mcg/kg given 2 weeks apart is similarly effective and may be warranted for patients with persistent or nodular infections or for those who cannot apply the cream appropriately.[76] The patient's close contacts should also be treated, even if not symptomatic. Recently worn clothing and linen should be washed in hot water, and the home vacuumed and cleaned thoroughly. It is important to counsel that itching typically persists for up to 4–6 weeks despite effective treatment. Medium- or high-potency topical steroids and second-generation antihistamines can be used to manage associated pruritus.

Onychomycosis

Onychomycosis is a chronic infection of the nailbed and plate with fungi and increases with age. The dermatophyte, *Trichophyton rubrum*, is responsible for the majority of finger and toenail infections.[77] *Candida* infection can also occur when hands are frequently in wet environments.[77] Other risk factors include diabetes, obesity, and smoking.[78]

Onychomycosis presents clinically as dystrophy of the nail plate with yellow discoloration due to subungual keratinaceous debris. Superficial white onychomycosis has dull, chalky white spots on the surface of the nail plate that spread centrifugally to involve the whole nail plate if left untreated. Trauma and inflammatory conditions such as psoriasis can lead to similar nail changes. Concomitant tinea of the hands and feet makes onychomycosis more likely.

Tinea unguium can be diagnosed in the office with potassium hydroxide (KOH) or chlorazol black examination of subungual debris, nail clippings sent for histology, or with culture. Culture may help identify resistant organisms when typical treatment fails. Histologic evaluation is more costly, but more sensitive than either KOH or culture, with one study reporting sensitivities of PAS stain, culture, and KOH at 88.2%, 29.4%, and 55.9%, respectively.[77]

Topical treatment is generally safe, but effectiveness is disappointing. Examples include topical terbinafine, azole creams, and ciclopirox. Estimated mycologic and complete cure rates for ciclopirox are around 30% and 5.5–8.5%, respectively. Efinaconazole fared better with mycologic cure rates near 55% and complete cure rates of only 15.2–17.8%, though cost is often prohibitive.[79]

Oral treatment is generally recommended based on patient preference and for patients who have pain due to onychodystrophy and those with a history of, or risk factors for, lower-extremity cellulitis. Terbinafine 250 mg daily is the first-line agent for oral therapy and is given for 6 weeks for fingernails and 12 weeks for toenails. Alternative systemic therapies include itraconazole and fluconazole. Fingernails and toenails have complete cure rates of 59% and 38%, respectively, after a course of terbinafine.[79] Though usually well tolerated, severe drug-induced liver injury has been reported at rates of 1 in 50,000–120,000 patients. Recent data suggests routine lab monitoring is likely unnecessary since it is unlikely to detect or predict liver injury. Recurrence may occur in 20–25% of patients, but may be reduced with topical antifungal prophylaxis.[79]

Intertrigos

Intertrigo is a descriptive term used to describe any inflammatory condition affecting the intertriginous area, typically due to friction, moisture, and poor aeration. Drying powders and barrier creams containing zinc oxide can keep the high-risk areas dry and protected. The most common secondary infection is with candida or dermatophytes, though *group A streptococcus* or *Corynebacterium minutissium* can occur less frequently.[80] Candidal

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intertrigo is brightly erythematous, weeping, and macerated with superficial erosions and classic satellite papules or pustules. KOH preparation can be confirmative. Antifungal azole creams or nystatin powders are highly effective. When unresponsive to treatment, alternative diagnoses such as inverse psoriasis, allergic contact or irritant dermatitis, tinea cruris, seborrheic dermatitis, bacterial intertrigo, and erythrasma should be considered.[81] Erythrasma is caused by a *Corynebacterium* infection and can be treated with topical clindamycin or oral macrolide antibiotics.

Summary

The geriatric population seeks care for a variety of skin conditions. Many skin issues encountered in the elderly can be attributed to normal physiologic changes of aging. Immunosenescence, a lifetime of UV exposure, comorbid conditions, and polypharmacy all contribute to the increased burden of skin conditions presenting uniquely within their demographic. Being mindful of how to best address these skin conditions and their impact on quality of life is essential to the care of our elderly patients.

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Chapter 39

Pressure Injuries

Practical Considerations in Prevention and Treatment

Barbara M. Bates-Jensen and Stephanie L. Woelfel

Pressure injuries, previously known as pressure ulcers, are areas of local skin and tissue damage, usually developing where soft tissues are compressed between bony prominences and any external surface at high intensity and/or for an extended time.[1] They are most common over bony prominences or under medical devices where skin and tissues are subject to extreme or prolonged pressure or pressure in combination with shear. A pressure injury is a sign of local damage; the skin may be involved, or there may be muscle and subcutaneous fat tissue destruction underneath intact skin.[1] The injury can present as discolored intact skin or an open wound. Once a wound develops, pressure injuries often fail to proceed through normal wound healing, often stalling in the inflammatory phase of healing and remaining in a chronic inflammatory state.[2]

Pressure injuries are a complex, multifactorial, and costly global health problem. The likely worldwide pressure injury prevalence rate in acute care settings is between 6% and 18.5%.[3,4] Globally, superficial pressure injury stages 1 and 2 are the most frequently occurring, with sacrum, heels, and hip the most affected body sites.[4]

Morbidity and mortality related to pressure injuries is significant.[6–8] Sepsis is the most serious complication of pressure injuries.[9–12] There are 3.5 episodes of pressure injury-associated bacteremia per 10,000 discharges. Of these episodes of pressure injury-associated bacteremia, the pressure injury is the probable source in nearly half the cases.[9,11] In these cases, 76% had pressure injuries colonized and/or infected with either *S. aureus* (21%), Gram-negative bacilli (32%), or both (47%), and most were multidrug-resistant organisms (MDROs) (65%).[16] When the pressure injury is the source of bacteremia, in-hospital mortality is nearly 60%.[11]

Pressure Injury Healing Time

Healing times for pressure injuries vary according to the severity of the damage. Stage 2 partial-thickness pressure injuries have reported healing times ranging from 18 to 52 days, with 69% of stage 2 pressure injuries healed within 12 months.[1,25] Healing times for full-thickness stage 3 and 4 pressure injuries range from 62 to 150 days, with 41% of stage 3 and 21% of stage 4 pressure injuries healed within 12 months.[1,26,27] Others have suggested that with standard treatment, 70% of stage 2, 50% of stage 3, and 30% of stage 4 pressure injuries should heal within 6 months.[1,2] Pressure injuries that decrease in size by 45% by week 2 of treatment or 77% by week 4 of treatment are more likely to heal completely within 15 months.[28] Thus, pressure injuries should show evidence of healing or improvement with 2 weeks of treatment.

Quality of Life and Pressure Injury

Pressure injuries impact quality of life. In interviews of hospital patients with pressure injuries (stage 2 and greater), most indicated the pressure injury and its treatment affected their lives emotionally, mentally, physically, and socially.[29] Nearly all described pressure injury pain with ranges from extreme pain to "little shooting pains." Many described fluid leakage, odor, discomfort, and mobility issues associated with the pressure injury and treatments and suggested the pain, discomfort, and distress of pressure injuries was not acknowledged by staff.[29] Health-related quality of life (HRQL) issues in older patients with pressure injuries involve physical and social impact, psychological effect, pressure injury symptoms, general health, health-care provider–patient relationships, need for versus effect of interventions, impact on others and finances, perceived etiology, and need for knowledge.[30]

Pressure Injury Stages

Pressure injury severity is categorized by skin and tissue damage visible according to the International Guideline six-stage classification system.[1] Table 39.1 presents the pressure injury classification definitions.[1]

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Table 39.1 Pressure injury classifications

National Pressure Injury Advisory Panel, 2019 Pressure Injury Staging Classifications	
	Definition and clinical description
Stage 1	Intact skin with a localized area of non-blanchable erythema, which may appear differently in darkly pigmented skin. Presence of blanchable erythema or changes in sensation, temperature, or firmness may precede visual changes. Color changes do not include purple or maroon discoloration; these may indicate deep tissue pressure injury.
Stage 2	Partial-thickness loss of skin with exposed dermis. The wound bed is viable, pink or red, moist, and may also present as an intact or ruptured serum-filled blister. Adipose (fat) is not visible and deeper tissues are not visible. Granulation tissue, slough, and eschar are not present.
Stage 3	Full-thickness loss of skin, in which adipose (fat) is visible in the ulcer and granulation tissue and epibole (rolled wound edges) are often present. Slough and/or eschar may be visible. The depth of tissue damage varies by anatomical location; areas of significant adiposity can develop deep wounds. Undermining and tunneling may occur. Fascia, muscle, tendon, ligament, cartilage and/or bone are not exposed. If slough or eschar obscures the extent of tissue loss, this is an Unstageable Pressure Injury.
Stage 4	Full-thickness skin and tissue loss with exposed or directly palpable fascia, muscle, tendon, ligament, cartilage or bone in the ulcer. Slough and/or eschar may be visible. Epibole (rolled edges), undermining and/or tunneling often occur. Depth varies by anatomical location. If slough or eschar obscures the extent of tissue loss, this is an Unstageable Pressure Injury.
Unstageable	Full-thickness skin and tissue loss in which the extent of tissue damage within the ulcer cannot be confirmed because it is obscured by slough or eschar. If slough or eschar is removed, a Stage 3 or Stage 4 pressure injury will be revealed. Stable eschar (i.e., dry, adherent, intact without erythema or fluctuance) on the heel or ischemic limb should not be softened or removed.
Deep tissue injury	Intact or non-intact skin with localized area of persistent non-blanchable deep red, maroon, purple discoloration or epidermal separation revealing a dark wound bed or blood-filled blister. Pain and temperature change often precede skin color changes. Discoloration may appear differently in darkly pigmented skin. This injury results from intense and/or prolonged pressure and shear forces at the bone-muscle interface. The wound may evolve rapidly to reveal the actual extent of tissue injury, or may resolve without tissue loss. If necrotic tissue, subcutaneous tissue, granulation tissue, fascia, muscle or other underlying structures are visible, this indicates a full-thickness pressure injury (Unstageable, Stage 3, or Stage 4). Do not use DTI to describe vascular, traumatic, neuropathic, or dermatologic conditions.

NPIAP, https://cdn.ymaws.com/npiap.com/resource/resmgr/online_store/npiap_pressure_injury_stages.pdf. Accessed on 03/26/2021.

Pathophysiology of Pressure Injury Development

Pressure injuries result from mechanical loads of high-intensity or prolonged duration of pressure force on the tissues with deformation of cells and/or parallel shear force of the skin and underlying tissues.[1] The pathophysiologic injury mechanisms proposed include ischemia,[31,32] cell deformation damage,[33,34] local inflammation,[34] reperfusion injury,[35,36] impaired lymphatic function,[37] increased capillary permeability and localized edema,[34] accelerated cell senescence,[2] and altered skin microclimate (temperature, friction, and moisture).[38,39] These mechanisms interact, causing direct and indirect cell and tissue damage.

Pressure (stress) and deformation (strain) are involved in all pressure injury development hypotheses. Pressure is the perpendicular force or load exerted on a specific area, causing hypoxia and tissue ischemia. The gravitational pull on the skeleton causes loading and deformation of the soft tissue between the bony prominence and external support surface. Shear is force applied against a surface as it moves or slides in an opposite but parallel direction, stretching tissues and displacing blood vessels laterally. Deformation stretches and pulls cells and at sufficient force damages cell walls.[33] Superficial pressure injuries are caused primarily by shear and friction forces at the skin surface, while deeper pressure injuries are a result of high pressure and shear with cell and tissue deformation within tissues at the surface over bony prominences.[1,33]

High-pressure areas in the supine position are the occiput, sacrum, and heels. In the sitting position, the ischial tuberosities exert the highest pressure, trochanters are affected in the side-lying position, and the forehead, cheeks, chin, chest, and iliac crest are most affected in the

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prone position. The mechanical physical forces of pressure and shear are not always sufficient to cause skin and tissue damage. It is the combination of mechanical forces along with impaired or compromised skin and tissues intolerant to the forces that leads to pressure injury. Tissue tolerance differs by tissue type and condition and is affected by the microclimate of the skin, perfusion, age, general health, and comorbidities.[1] Thus, events surrounding pressure injury development include a variety of intrinsic and extrinsic factors.

Most pressure injuries occur over bony prominences where there is less tissue for compression and the pressure gradient within the vascular network is altered. Direct deformation damage from pressure and/or shear forces leads to micro- and macroscale damage in the least amount of time, while micro- and macroscale damage due to ischemia requires longer time periods to develop.[1,31,33]

Pressure occludes blood and lymphatic circulation, causing tissue hypoxia and metabolic waste accumulation. If pressure is relieved before a critical time period is reached, reactive hyperemia, a normal compensatory mechanism, restores tissue oxygenation. With unrelieved pressure, the increased pressure gradient leads to vascular changes. Interstitial fluid pressure increases, exceeding and inhibiting venous flow, increasing the pressure gradient throughout the vascular network, and impeding arteriolar circulation. Increased capillary arteriole pressure leads to increased capillary permeability, tissue edema, and subsequent autolysis. Lymphatic flow is also decreased, increasing tissue edema and contributing to tissue necrosis. As ischemia and lymph obstruction progress, the extracellular pH decreases and cell migration to the area is slowed.[41] Muscle tissue is more sensitive to ischemia and hypoxia, possibly because of the increased vascularization and higher metabolic demand compared to skin and subcutaneous tissue.[42] Tissue damage apparent on the skin surface may be minimal, but damage to the deeper structures can be severe.

Deformation of cells and tissues is the underlying mechanism in the damage seen in deep-tissue injury and full-thickness pressure injuries. Tissues are deformed because of the bone's compression of the soft tissue against the external surface, leading to cell death. How the tissues are deformed depends on the tissues (i.e., size and shape of the different tissue layers), the mechanical properties of the involved tissues (e.g., stiffness, strength), and the magnitude and distribution of the external mechanical force applied to the tissues.[1] Tissue ability to withstand deformation can change with time because of aging, lifestyle changes, injury, or disease. The sustained mechanical deformation damages cells directly and also obstructs blood flow.[33,34] Muscle tissue is also more sensitive to deformation forces; irreversible damage may be present at the muscle layer without such damage occurring in the skin or subcutaneous layers. The cell death and local tissue necrosis change the geometry and characteristics of the tissues, which further increases the deformation force exacerbating the pressure injury. Animal models have shown irreversible ischemic damage to muscle tissues after 2–4 hours of sustained deformation forces. Further, muscle deformation greater than 50% leads to microscale tissue damage within minutes.[43,44] Heat accumulation or increased skin temperature intensifies the effects of ischemia and hypoxia on tissues.[40]

As the vascular network is relieved of pressure, the tissues are reperfused and reoxygenated. The sudden entry of oxygen into previously ischemic tissues releases oxygen-free radicals, which induce new endothelial damage and decrease microvascular integrity, causing reperfusion injury.[35,36] These cellular changes result in inflammation and edema locally at the site of injury. These inflammatory changes exist in the tissues before any damage is visible on the skin surface.[45,46,47] This is the *nonvisible spectrum of pressure-induced tissue damage*, the preclinical stage of disease in the physiology cascade leading to pressure injury. These inflammatory changes with tissue edema can occur from 3–10 days before visible skin breakdown.[45,46] The activation of inflammation leads to vascular dilation, increased membrane permeability, and movement of fluid from the vascular space to the interstitial space.[48] The end result is increased water and fluid in the tissues or subepidermal moisture (SEM) at the site of injury.[49]

It is both the intensity and duration of pressure that cause tissue damage. Pressures lower than those that cause immediate tissue damage are capable of causing damage when present over a long time period. The critical time-frame is 1–3 hours for both pressure and deformation. Deformations below 65% (of normal tissue formation) are only tolerated by muscle cells for periods of time less than 1 hour.[50]

Pressure Injury Detection

Stage 1 and 2 pressure injuries account for the majority of all pressure injuries in elders.[20,21] Many stage 1 pressure injuries progress to more severe injuries, and they are considered a strong risk factor for development of

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stage 2 or greater pressure injuries.[1,51,52] In hospitalized patients, 26% of stage 1 pressure injuries deteriorate to more severe injuries in as little as 7 days.[53] Nursing homes report pressure injury deterioration among residents with stage 1 pressure injuries ranges from 9% to 58% in as little as 2 weeks.[54] Thus, detection of subclinical pressure-induced tissue damage is important because early intervention may prevent development of severe pressure injuries.

The standard method of detection is visual skin assessment, observing for blanchable and non-blanchable erythema. While visual skin assessment has some reliability for individuals with light skin tones,[54] by the time non-blanchable erythema is evident, tissue damage has already occurred. Blanchable erythema alone has been suggested as a strong predictor of later-stage injuries.[54] Visual skin assessment fails to detect skin discoloration in persons with dark skin tones, missing both pre-stage 1 pressure damage and stage 1 pressure injuries.[1]

Differential Diagnosis

Superficial stage 2 pressure injuries and incontinence-associated dermatitis (IAD) or intertriginous dermatitis are often confused. Table 39.2 presents differential characteristics of IAD, intertriginous dermatitis, and superficial pressure injuries. Differentiating between skin failure and a terminal pressure injury is also difficult. Persons at life's end, during an acute critical illness, or with severe trauma may experience skin failure or terminal pressure injuries. Skin failure is defined as an acute episode where the skin and subcutaneous tissues become necrotic because of hypoperfusion that occurs concurrent with severe dysfunction or failure of other organ systems.[56,57] Hypoperfusion creates an extreme inflammatory reaction along with severe dysfunction or failure of multiple organ systems that compromises the skin.[57] Skin failure may present anywhere on the body. One manifestation of skin failure may be a terminal or Kennedy pressure injury.[58] Kennedy described a specific subgroup of pressure injuries that some individuals developed as they were dying. Typically, over the sacrum, shaped like a pear, butterfly, or horseshoe with a variety of colors including red, yellow, or black, Kennedy pressure injuries are very sudden in onset, typically deteriorate rapidly, and usually indicate that death is imminent, with just over half (56%) dying within 6 weeks of discovery of the injury.[58]

Risk Screening and Assessment

Pressure injury risk screening involves identification of individuals for whom a more complete risk assessment is needed. Certain risk factors can be easily identified and used to screen individuals quickly in order to place them immediately on a general prevention program. For these individuals, a more complete risk assessment is necessary to identify specific factors that are amenable to more targeted prevention strategies. Screening factors range from individual factors such as age to factors that are more related to procedures or medical conditions such as emergent admission to the hospital or prolonged surgical time.

Risk assessment should include use of a risk assessment scale or tool to identify specific factors that place an individual at increased risk, skin assessment to determine existing skin condition, and clinical judgment based on specialty-specific indicators related to a person's risk status.[1]

Risk factors that increase mechanical boundary conditions include activity and mobility limitations, and sensory perception limitations.[1] Risk factors such as skin status, perfusion, circulation and oxygen factors, nutrition, temperature, and blood markers increase susceptibility and reduce tolerance of the individual. Some risk factors affect both mechanical boundary conditions and susceptibility and tolerance of the individual, including moisture, older age, and general and mental health status.

Immobility, inactivity, and sensory loss. Immobility or severely restricted mobility is the most important risk factor for all populations and a necessary condition for the development of pressure injuries. Closely related to immobility is limited activity. Patients who are bed- or chairbound are thus at greater risk for pressure injuries.[61]

Sensory loss increases the risk for compression of tissues and pressure injury development, because the normal mechanism for translating pain messages from the tissues is dysfunctional. Patients with intact nervous system pathways feel continuous local pressure, become uncomfortable, and change their position before tissue ischemia occurs. These responses are reduced or absent in patients with spinal cord injury and many patients with altered mental status. Thus, these patients have a higher incidence and prevalence of pressure injuries.

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Table 39.2 Differential diagnosis factors for incontinence-associated dermatitis, intertriginous dermatitis, and superficial pressure injury

Assessment factors	Incontinence-associated dermatitis (IAD)	Intertriginous dermatitis (ITD)	Superficial pressure injury
Location	Groin, buttocks, sacral/coccygeal area, gluteal fold, inner thighs, perianal and perineal area	Intergluteal cleft, under pannus or breasts, groin crease	Over bony prominence, sacrum, coccyx, ischial tuberosities
Depth and shape	Superficial, partial thickness, blisters, typically multiple lesions	Superficial, partial thickness initially, linear	Shallow, superficial, partial thickness, round or oval shape
Characteristics	• Diffuse blanchable erythema • Papulovesicular reaction • Vesiculation • Oozing, crusting, and scaling • Erosions and denudation • Copy lesions (one lesion on buttock mirrors a second on the opposite buttock) • Multiple partial-thickness ulcers • Ulcer edges diffuse, irregular • Fungal infection may be present (C. albicans) with rash and satellite lesions NOTE: in addition to above, in dark skin tones may present as white, dark red, purple, or yellow skin discoloration	• Surrounded by maceration • Ulcers edges clear • Fungal infection may be present (C. albicans) with rash and satellite lesions	• Stage 1: non-blanchable redness over bony prominence. • Stage 2: Partial-thickness shallow open ulcer with a red-pink wound bed, without slough. May also present as an intact or open/ruptured serum-filled blister.
Symptoms	• Pain • Itching • Burning • Tingling	• Pain • Itching	• Pain
History	Urinary and/or fecal incontinence	Sweating or diaphoresis, skin-to-skin contact	Patient immobile, issues with friction/shearing
Pathophysiology	▪ Inflammatory response to external surface irritants – stool/urine ▪ Inflammation involves upper dermis and epidermis (stratum corneum) ▪ Epidermal and dermal injury	▪ Inflammatory response to internally *produced* surface irritants – sweat ▪ Inflammation involves upper dermis and epidermis ▪ Epidermal and dermal injury ▪ Friction may intensify effects ▪ Occlusion may be a factor	▪ Inflammatory response to mechanical forces of shear and friction ▪ Shear and friction (the resistance to motion or rubbing of one object or surface against another in a parallel direction) forces at the skin surface abrade or blister the epidermal surface ▪ Epidermal and dermal injury over bony prominences ▪ Inflammation involves dermis, upper dermis, and epidermis

Skin status. Presence of non-blanchable erythema (stage 1 pressure injury) is a strong risk factor for development of more severe pressure injury.[1,60,62] The odds ratios of developing a stage 2 or greater pressure injury after the presence of stage 1 pressure injury range from 1.9 to 7.[1] In fact, erythema alone can be considered a risk factor for pressure injury development.[47,55]

Perfusion, circulation, and oxygen. Factors that reduce oxygenation and perfusion of the skin and tissues increase the likelihood of pressure injury development. Reduction in arteriolar flow and the resultant skin compromise typically occurs in critically ill patients with cardiovascular instability, and these persons are at higher risk for pressure injuries.[104]

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Nutrition. Weight loss, cachexia, and malnutrition are all commonly identified as risk factors predisposing patients to pressure injury development.[1,63] Measures of nutritional intake, weight, or use of a nutrition screening tool are commonly used indicators of nutritional status.[1] Body mass index (BMI) has a U-shaped relationship with pressure injury development, with those at the extremes of weight at risk for pressure injury.[1,60]

Temperature. Elevated body temperature has been shown to be a risk factor for pressure injury development.[1,64–66] It is likely the elevated temperature increases metabolic demands of the tissues, impacting the susceptibility and tolerance of the individual by affecting physiology and repair mechanisms.[1]

Blood markers. Several laboratory blood markers have been associated with pressure injury development: low serum albumin, low hemoglobin, and elevated C-reactive protein.[67,68] Low serum albumin may be related to inflammatory response with an increase in acute phase proteins, malnutrition, or other factors. Regardless of cause, low serum albumin leads to interstitial edema, which slows transmural pressure and perfusion to tissues.[1] Low hemoglobin reduces the oxygen-carrying capacity of the blood and reduces the health of the tissues.

Moisture. Excess moisture may be due to wound drainage, diaphoresis, and fecal or urinary incontinence. Incontinence contributes to pressure injury formation by creating excess moisture on the skin and by chemical damage to the skin. Fecal incontinence has an added detrimental effect, as enzymes in stool also contribute to skin breakdown.[69–71] In the presence of both urinary and fecal incontinence, the pH in the perineal area is increased by the fecal enzymes' conversion of urea to ammonia. The elevated pH increases the activity of proteases and lipases found in stool, which, in turn, increases skin permeability, leading to irritation by other agents.[69,70] Inadequately managed incontinence poses a significant risk factor for pressure injury development.[71]

Age. Advanced age is a risk factor for pressure injury development.[1,60] The skin and support structures undergo changes in the aging process. There is a loss of muscle mass, a decrease in serum albumin levels, diminished inflammatory response, decreased skin elasticity, and reduced cohesion between the dermis and epidermis.[72]

General and Mental Health Status. Certain medical conditions or disease states are also associated with pressure injury development. Orthopedic injuries, specifically hip fractures and spinal cord injury, are such conditions.[1,73,74] General health status has also been associated with pressure injury development. Higher illness severity scores or increased acuity,[62,75,76] acute versus elective admission to the hospital,[62] mechanical ventilation, and length of stay all increase risk for pressure injury development.

Risk assessment. The most common risk assessment tool in use in the USA is the Braden Scale for Predicting Pressure Sore Risk (Braden Scale).[77,78] The Braden Scale is composed of six subscales: sensory perception, moisture, activity, mobility, nutrition, and friction and shear. All subscales are rated from 1 to 4, except for friction and shear (rated from 1 to 3). The subscales may be summed for a total score, with a range from 6 to 23, with a lower score indicating higher risk. Scores of 18 and below are considered "at risk" for hospitalized adults. Use of a standardized risk assessment is recommended in clinical practice guidelines.[1,79]

Critical care risk assessment. Patients in critical care settings often experience uncontrollable risk factors, placing them at very high risk for pressure injuries. Uncontrollable factors specific to intensive care that increase risk include: multiple surgical procedures, mechanical ventilation, sedation or paralytics, traction and/or external fixators, weeping anasarca, shock, multiorgan dysfunction syndrome, cardiac arrest, multiple vasopressors, drive lines, nitric oxide ventilation, proning, and use of high-flow oxygen devices.[75,76]

Persons admitted to general medical-surgical units of hospitals should have risk assessment conducted on admission and, if at risk, every 48 hours after admission. Those persons admitted to critical care units should have a risk assessment that includes specialty-specific risk factors conducted on admission and at least daily thereafter. In nursing homes, risk assessment should be conducted on admission to the facility and, if at risk, then weekly for 4 weeks, and for all residents quarterly or whenever a change in status occurs. For home-care patients, risk should be assessed on admission to home health care, weekly for the first 4 weeks, and every other week thereafter until day 62, depending on patient condition and frequency of home visits. Risk assessment should be performed whenever a significant change occurs in the patient's general health and status.

Skin assessment. A complete skin assessment is a critical part of risk assessment, as persons with existing pressure

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injuries or other skin damage are at higher risk for new pressure injury development. Key areas for skin assessment are the common bony locations for pressure injury development: sacrum, coccyx, ischial tuberosities, trochanters, buttocks, ankles, and heels. Medical devices should be removed and the skin examined for discoloration, wounds, abrasions, or signs of pressure damage. Other areas for examination include the occiput of the head, shoulder blades, elbows, hips, and knees. It is essential to document the skin assessment on admission to the health-care facility. Documentation ensures that damage present on admission is communicated to the health-care team.

Pressure Injury Prevention

Pressure injury prevention is the most cost-effective treatment for pressure injuries. Prevention is best accomplished with a multidisciplinary team focused on eliminating specific individual risks and aligned with individual goals of care.[1,2] Prevention includes attention to six key areas: risk assessment, daily skin assessment, tissue load management with offloading and support surfaces, mobility and activity interventions, nutrition assessment, and incontinence management. Several studies have identified the importance of managing patients' pressure injury risk based on their Braden subscale scores in addition to overall Braden score.[78,79]

Physical therapy and pressure injury prevention. Physical therapists are especially helpful with managing tissue loads with offloading and support surfaces and interventions directed at activity and mobility deficits. Patients with activity and mobility limitations must have local pressure alleviated with scheduled repositioning and use of pressure redistribution support surfaces for the chair and bed.

Scheduled repositioning. Repositioning schedules are usually interpreted as every 2 hours for full-body change of position and more often for small shifts in position. Full-body change of position involves turning the patient to a new lying position; for example, turning the patient from the right 30-degree side-lying position to the left 30-degree side-lying position or the supine position. Frequency should be based on the patient's tissue tolerance, level of mobility and activity, medical condition, skin integrity, treatment goals, and support surface use. High-level studies have demonstrated 2-, 3-, and 4-hour repositioning are all effective to a certain extent in decreasing pressure injuries.[80,81] Four-hour repositioning with use of viscoelastic support surfaces has been shown to decrease frequency and time to occurrence of stage 2 or greater pressure injuries compared to: standard care (no turning schedule), those on standard hospital mattresses repositioned every 2 and 4 hours, and those on viscoelastic support surfaces repositioned every 6 hours. No differences in pressure injury development existed between 2- and 4-hour turn groups on viscoelastic support surfaces in a follow-up study.[80,81]

In nursing homes, 3-hour repositioning using a 30-degree tilt position is more effective at reducing pressure injuries compared to 6-hour repositioning using a 90-degree lateral rotation position.[82] Examining pressure injury development with 2-, 3-, and 4-hour repositioning for residents identified as moderate or high risk and placed on high-density foam support surfaces found no difference in pressure injuries.[83] Further, 3- and 4-hour repositioning was cost-effective and showed an increase in quality-adjusted life years (QALYs).[84,85]

In critical care, 2-hour repositioning with the 30-degree tilt position compared to 4-hour repositioning among ventilated patients showed no difference in pressure injury incidence. While repositioning was interrupted for hemodynamic or respiratory instability repositioning, compliance was not different between the groups.[86]

Similar approaches are useful for patients in chairs. Full-body change of position for persons in chairs involves standing the patient and re-seating him or her in the chair or having the patient use arm push-ups to completely relieve pressure. Small shifts in position for those in chairs might be changing lower-extremity position or tilting the chair. Patients with upper-body strength should be taught to shift weight every 15 minutes to allow for tissue reperfusion. Weight shifting can occur with chair push-ups, backward chair tilts, leaning side to side, or forward leans. Physical therapy can assist in body alignment strategies with even the most contracted patient.

The best prevention strategy for eliminating pressure injuries on the heels is to keep the heels off the surface of the bed. Use of pillows under the lower extremities, if they remain in place, can keep the heel from making contact with the bed. Specialized heel pressure-redistributing devices are effective in reducing pressure on heels.[87] Use of prophylactic dressings are effective in preventing heel pressure injuries in high-risk hospital and critical care patients and nursing home residents.[88]

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Pressure redistribution support surfaces. A support surface is a specialized device for pressure redistribution developed for managing tissue loads, microclimates, or other therapeutic functions.[1] Pressure redistribution support surfaces assist in pressure injury risk reduction by managing tissue loading either by reducing the load or duration of loading. Support surfaces are used to redistribute pressure in bed and chairs, and during operative procedures. Advanced support surfaces are superior to standard hospital beds in preventing and managing pressure injury.[1,2] There are two basic types of pressure redistribution support surfaces: non-powered and powered. Pressure redistribution non-powered support surfaces such as high-specification foam mattresses or overlays increase the body surface area that comes in contact with the support surface to decrease the pressure between the body and the support surface interface with immersion and envelopment (i.e., the body sinks into or is engulfed by the surface). There is no difference between different high-specification foam mattresses.

Powered redistribution support surfaces work by sequentially altering the parts of the body that bear load and so reduce the duration of loading on the tissues at any given anatomic location such as alternating pressure air mattresses. Alternating pressure support surfaces reduce pressure injury incidence in hospitalized and critical care patients compared to standard hospital mattresses.[89] There is limited evidence of differences between alternating pressure support surfaces.

Providing pressure redistribution for chairbound patients is critical because of the high pressures across the ischial tuberosities when sitting upright. Most pressure-redistributing support surfaces for chairs are non-powered overlays, such as those made out of foam, gel, air, or some combination. Patients with spinal cord injury spend considerable time in a chair and should have high-level pressure redistribution support surfaces.

Pressure redistribution support surfaces should be used on the operating table for persons determined at risk for pressure injuries and those undergoing prolonged procedures or at risk of hypotensive episodes during surgery.[1] Use of high-specification foam, viscoelastic support surfaces, or medical grade sheepskin on the operating table as a pressure-redistributing support surface can reduce the incidence of operative-acquired pressure injuries.[1,90]

Effective use of support surfaces requires choosing support surfaces based on multiple factors, use of a multidisciplinary team to finalize selections, and periodic reevaluation of products and patient/institution needs. These steps are critical, as limited evidence exists to support use of one specific device versus another.

Prophylactic dressings. Use of a silicone foam dressing on sacrum and heels is recommended prophylactically to prevent pressure injuries among at-risk hospitalized patients, critically ill patients, and nursing home residents, and under medical devices.[1] The dressings should be applied early in the prevention process. In critical care units use of prophylactic dressings has reduced pressure injuries by 10%, with a number needed to treat to prevent one pressure injury of 10 patients.[88] The dressings have been shown to be cost-effective.[92] Findings also show a decrease in sacral and heel injuries with silicone foam dressing use prophylactically in general hospital populations[91] and among nursing home residents.[88] In nursing homes, use of the dressings decreased pressure injuries with a relative risk reduction of 80%. Importantly, cost-effectiveness analysis showed the average net cost of the dressings was lower than net costs for residents in the no-dressing group.[88,93]

Use of prophylactic dressings, both hydrocolloid and silicone foam dressings, under medical devices is recommended. Use of a hydrocolloid dressing is generally recommended to help prevent pressure injuries in high-risk patients, including pressure injuries caused by non-invasive ventilation (NIV). Patients who used a hydrocolloid dressing had a decreased incidence of pressure injuries compared to those with no dressings or gauze dressings.[1,94]

Mobility and activity interventions. Overhead bed frames with trapeze bars are helpful for patients with paraplegia, stroke patients with upper-body strength, and obese patients, and may increase mobility and independence with body repositioning. Wheelchair-bound patients with upper-body strength can be taught and encouraged to do wheelchair push-ups and body tilts/leans to relieve pressure and allow for reperfusion of the tissues at the ischial tuberosity. For patients who are weak from prolonged inactivity, providing support and assistance for reconditioning and increasing strength and endurance will help to prevent future disability and dysfunction. Mobility plans are the responsibility of the multidisciplinary team working together in all healthcare settings. Even frail elder nursing home residents are capable of improving mobility and activity levels and wheelchair use.[95]

Nutrition assessment. Nutrition assessment should be performed by a nutritionist or dietician on all patients

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determined to be at risk for pressure injury. If the patient is diagnosed as malnourished, nutritional supplementation may be indicated to help achieve a positive nitrogen balance. The goal of care is to provide approximately 30–35 kcal/kg of weight per day and 1.25–1.5 g of protein per kg of weight per day for patients at risk and those with pressure injuries.[1]

Incontinence and moisture management. To minimize skin exposure to excessive moisture, assess for excessive moisture by determining the cause for the specific patient; for example, urine, feces, perspiration, wound exudate, and/or saliva. General skin hygiene and skin barrier creams and ointments are useful regardless of moisture cause. Use of a pH-balanced skin cleanser with a surfactant is most effective for incontinence skin hygiene. Moisture barrier creams and ointments protect the skin from the effects of moisture.[96] The success of the particular product is linked to its formulation and hydrophobic properties. Dimethicone (3%) skin protectants have been shown to decrease pressure injury incidence at the sacrum and buttocks in incontinent nursing home residents.[96]

Pressure Injury Assessment

Routine pressure injury assessment is the base for maintaining and evaluating the therapeutic plan of care. Initial assessment and weekly follow-along assessments to monitor healing progress are necessary to determine the effectiveness of the treatment plan. Monitoring can be performed by less skilled caregivers at each dressing change; however, assessment should be performed on a routine basis by trained clinicians. Minimally, the pressure injury should be assessed for location, depth and stage, size, and general wound-bed description. Other macroscopic wound characteristics important for determining treatment include necrotic tissue, exudate, wound edges for undermining and tunneling, and the presence or absence of granulation and epithelialization.

Clinical practice guidelines, expert panels, and federal nursing home guidelines recommend standardized assessment of pressure injuries, and use of a standardized tool is recommended.[1] One such instrument is the Bates-Jensen Wound Assessment Tool (BWAT) (wwwoundcare.ca). The BWAT has been incorporated in a variety of electronic medical records and has demonstrated reliability with a variety of users across anatomic locations and severity levels and among patients with various skin tones.[97,98]

Pressure Injury Management

Pressure injury management is focused on wound-bed preparation to promote healing. Wound-bed preparation incorporates aspects of wound care represented by the acronym TIMERS:[1,99]

Tissue management
Infection and inflammation control
Moisture balance
Epithelial edge advancement
Repair and regeneration
Social factors and those related to the individual

Tissue management. Tissue management includes debridement of necrotic tissue and adequate wound cleansing. Wound debridement is necessary to reduce the necrotic tissue burden, decrease risk for infection, and promote granulation tissue formation. Benefits of debridement may also include removal of senescent fibroblasts and nonmigratory hyperproliferative epithelium, and stimulation of blood-borne growth factor production. Debridement is not indicated for dry eschar presenting on the heel or when the pressure injury presents on an ischemic limb. Five methods of debridement (e.g., surgical or sharp, mechanical, autolytic, enzymatic, biological) are available. Physical therapists are able to selectively debride wounds of necrotic tissue using low-frequency contact ultrasound, maggots, and/or forceps/scalpel/scissors. Clinical practice guidelines on pressure injury treatment recommend wound debridement with surgical or sharp debridement for extensive necrosis or when obtaining a clean wound bed quickly is important. More conservative methods (autolytic and enzymatic) are usually frontline for those in long-term care or home-care environments.[1,100] Adequate wound debridement is essential to wound-bed preparation and healing. Initial debridement with maintenance debridement at intervals is often necessary to maintain a biofilm-free wound bed and support healing.[1,100]

Wound cleansing removes surface debris, dressing residue, and microorganisms from the wound and surrounding tissues. Cleansing can be accomplished using clean technique by irrigating the wound with fluid or use of pulsatile lavage. Irrigation pressures between 4 and 15 psi are acceptable and can be realized using a 35-cc syringe with a 19-gauge angiocatheter attached.[1] Pressure injury cleansing at each dressing change with normal saline or potable water is recommended in clinical practice guidelines.[1,100] Use of antiseptic solutions

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such as 5% mafenide acetate (Sulfamylon solution), 10% povidone with 1% free iodine (Betadine), 0.25% sodium hypochlorite ("half strength" Dakin's solution), 3% hydrogen peroxide, and 0.25% acetic acid have varying effects on wound-healing parameters and are not recommended for *clean* necrotic-free pressure injuries.[1,100,101] Antiseptic and antimicrobial solutions for cleansing pressure injuries with *necrotic* debris and/or confirmed high bacterial colonization should be used with caution and attention to the solution chosen, the characteristics of the microorganisms present in the wound, and duration of use (e.g., course of therapy for 2 weeks with evaluation for continuation at that time). If the pressure injury contains necrotic debris or is infected, then antimicrobial activity is more important than cellular toxicity. The chemical and mechanical trauma of wound cleansing should be balanced by the dirtiness of the wound. One recommendation is use of solutions with surfactant antimicrobials (e.g., Polyhexamethylene Biguanide [PHMB], Octenidine dihydrochloride [OCT]). These agents lower the surface tension and promote spread of the fluid across the wound bed, facilitating separation of loose nonviable tissue and bioburden.[1,102]

Infection and inflammation control. Stage 3, 4, and unstageable pressure injuries should be evaluated for infection. Most chronic pressure injuries are contaminated or colonized with microorganisms. Infection may occur locally and/or systemically. Predominant organisms present in infected pressure injuries include *Staphylococcus aureus* (including methicillin-resistant), *Proteus mirabilis*, and *Pseudomonas aeruginosa*.[1] One difference in dealing with infection in pressure injuries is the assessment and treatment of bacterial biofilms. Biofilms are the critical colonization of microorganisms on the wound bed that develop support structures that protect the bacteria. Biofilms lead to chronic inflammation and failure to heal. Bacterial biofilms have enhanced resistance to endogenous antibodies and phagocytic cells, and exogenous antibiotics and antimicrobial solutions.[1,102] Approximately 60% of chronic wounds contain bacterial biofilms, and this may be the underlying pathology preventing wounds like pressure injuries from healing.[1,102] Classic signs and symptoms of acute wound infection including heat, erythema, edema, and purulent exudate have poor ability to determine a true positive diagnosis of wound infection in pressure injuries.[1] Biofilm presence and infection in pressure injuries should be suspected when any of the following exist:

- Delayed healing
- Lack of signs of healing for 2 weeks with appropriate care
- Larger size or depth
- Wound breakdown or dehiscence
- Friable granulation tissue
- Malodor
- Increased pain, heat, exudate, or necrotic tissue
- Change in exudate character
- Pocketing or tunneling or bridging in the wound bed.

Bacterial burden of the pressure injury should be determined by tissue biopsy or quantitative swab technique (do not swab necrotic tissue or exudate; rotate end of swab over 1 cm^2 area for 5 seconds with sufficient force to cause tissue fluid expression). Use of topical antibiotic and antiseptic solutions for a 10- to 14-day course of therapy may be beneficial in reducing and/or preventing bacterial biofilms and supporting granulation tissue development and wound healing.

Moisture balance. Topical therapy for pressure injuries should be provided using moisture-retentive dressings.[1,2,103] Randomized controlled trials as well as several comparative studies provide compelling support for use of moisture-retentive dressings instead of any form of dry gauze dressings (e.g., wet to dry gauze, dry gauze dressing, or impregnated gauze dressing) for pressure injuries. Moist wound healing allows wounds to re-epithelialize faster than wounds left open to air or treated with dry gauze dressings, and the use of moisture-retentive dressings has lower treatment costs per patient compared with gauze dressings.[1,100,104] Table 39.3 presents general categories of moisture-retentive dressings and ability to manage moisture.

Stage 3 and 4 pressure injuries are full-thickness wounds often with a cavity. To prevent premature superficial closure and to allow the wound to heal from the inside out, these pressure injuries may require packing with a wound filler (e.g., amorphous gel, alginate, impregnated gauze) under a secondary dressing.[1]

Epithelial edge advancement. Failure of the epithelial edge to advance is evidence that healing obstacles have not been dealt with adequately. Non-proliferative epithelial edges may indicate issues with cellular matrix, hypoxia, or abnormal protease activity.[1] Infection and inflammation control, adequate initial and maintenance debridement, and effective moisture management promote epithelial advancement.

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Table 39.3 General categories of moisture-retentive dressings

Dressing category	Definition	Moisture management
Composite dressings	Combine one dressing type with another to address wound characteristics. For example, foam or hydrocellular and transparent film dressing properties, hydrocolloid and alginates, etc.	Minimal to moderate exudate management Absorbency depends on combination of dressing types used in the composite
Transparent film dressings	Polyurethane and polyethylene membrane film coated with a layer of acrylic hypoallergenic adhesive. Moisture vapor transmission rates (MVTRs) vary	No exudate management Partial-thickness wounds Protects against friction
Hydrocolloids Regular or thin wafers, different shapes for specific anatomic locations (sacral, heel)	Gelatin, pectin, carboxymethylcellulose in a polyisobutylene adhesive base with a polyurethane or film backing	Low exudate management
Hydrogels Sheets, wafers, amorphous gels, impregnated gauze	May or may not be supported by a fabric net, high water content, varying amounts of gel-forming material (glycerin, copolymer, water, propylene glycol, humectant), hydrated hydrophilic polymers	Low to minimal exudate management Nonadherent, may have adhesive borders Carrier for topical medication
Wound fillers (exudate absorbers) Beads, flakes, pastes, powders	Consist of copolymer starch, dextranomer beads or hydrocolloid paste that swell on contact with wound fluid to form gel, dextranomers, polysaccharides, starch, natural polymers, and colloidal particles	Moderate to large exudate management Useful to fill cavities, pockets, undermining Can be used with topical medications
Alginates Ropes, pads, wafers	Calcium-sodium salts of alginic acid (naturally occurring polymer in seaweed)	Moderate to large exudate management Dressing of choice for cavities, pockets, undermining Can use with topical medications or on infected wounds
Foams Wafers (thick or thin), pillows, composite dressings with thin film covers, gelling foam, available with surfactant impregnated or charcoal layer	Inert material that is hydrophilic and modified polyurethane foam, may include hydropolymers	Moderate to large exudate management Can be used with topical medications and on infected wounds Nonadherent, may have adhesive borders
Hydrofibers Pads, wafers, ropes	Soft nonwoven pad or ribbon dressings made from sodium carboxymethyl-cellulose fibers, similar absorbent material used in hydrocolloid dressings	Moderate to large exudate management
Polymeric membranes	Hydrophilic polyurethane matrix that contains wound cleanser, glycerin, and absorbent polymer	Moderate to large exudate management

Repair and regeneration. Adjunctive therapies and advanced wound-healing dressings can be considered when standard care and conventional moisture-retentive dressings fail to result in healing or improvement.[1,2] There is limited evidence supporting use of advanced wound therapy in stage 3 and 4 pressure injuries, and the evidence that exists is not definitive. However, these therapies can stimulate the wound-healing process by promoting development of extracellular matrix and stimulating cell activity. In some patients, surgical repair may be required.

Drugs

Pharmacologic interventions for pressure injuries focus on antibiotics and pain management. Antibiotics may be

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systemic or local. Clinicians should institute systemic antibiotics for patients exhibiting signs and symptoms of systemic infection such as sepsis or cellulitis with associated fever and an elevated white blood cell count. Systemic antibiotics should be initiated for osteomyelitis. Because of the high mortality of sepsis associated with pressure injuries despite appropriate antibiotics, broad-spectrum coverage for aerobic gram-negative rods, gram-positive cocci, and anaerobes is indicated pending culture results in patients with suspected bacteremia. Topical antibiotics or antimicrobial topical dressings are most appropriate for stage 3 or 4 injuries when there is evidence of local infection. A 2-week trial of a broad-spectrum topical antibiotic can be considered for clean pressure injuries that are not healing after 2 to 4 weeks of optimal management. Cadexomer iodine dressings, silver impregnated dressings, or cleansing with hypochlorous acid solution may be used.

Lower levels of pain may be manageable with use of moisture-retentive dressings and topical wound analgesia. Nonpharmacological techniques useful for noncyclic and cyclic wound pain associated with procedures (e.g., debridement, dressing changes, repositioning) include electrical stimulation, use of distraction (e.g., talking to the patient while performing the procedure), allowing the patient to call a "time-out" during the procedure, and allowing the patient to control and participate in the procedure. Pharmacological strategies for pressure injury pain include providing nonsteroidal anti-inflammatory drugs 30 minutes prior to the procedure and afterwards, and administering topical anesthetics or topical opioids using hydrogels as a transport media. Low-dose topical morphine (diamorphine) has been used to successfully control pressure injury pain.[1,106]

Nutrition

Moderately strong evidence exists that use of high-protein high-density nutritional supplements (24–25% protein) improves pressure injury healing. Providing 30–35 kcal/kg and 1.25–1.5 g/kg of calories and protein daily has been shown to significantly improve pressure injury healing.[107] Use of oral nutritional supplement enriched with arginine, zinc, and antioxidants for at least 8 weeks with patients with pressure injuries showed higher reduction in ulcer area, and more patients had a 40% or greater reduction in pressure injury size compared to standard nutritional supplements.[107] No evidence exists for use of supplemental vitamins or minerals (e.g., vitamin A, E, C, zinc) in persons with pressure injuries with no coexisting specific vitamin/mineral deficiency to improve pressure injury healing.

Summary and Conclusion

Pressure injuries are the result of multiple interacting factors and require a multifaceted, multidisciplinary approach for care. Development of a pressure injury occurs with mechanical forces of pressure, shear, and deformation. A wide variety of factors place patients at risk for pressure injuries. Timely recognition of risk is essential to promptly intervene with prevention strategies. Prevention involves attention to risk assessment, skin assessment, management of tissue loads with repositioning and use of pressure redistribution support surfaces, nutrition assessment, and incontinence management. Pressure injuries should be assessed initially and at least weekly for macroscopic wound characteristics to monitor healing and direct treatment. Pressure injury treatment is best represented by wound-bed preparation, which includes attention to: tissue management with debridement and cleansing, infection and inflammation control with attention to bacterial biofilms, moisture balance with use of moisture-retentive dressings, and epithelial edge advancement, which requires frequent and consistent assessment of the healing progress to identify obstacles to timely epithelial migration, repair, and regeneration, including use of advanced wound dressings and adjunct therapies to support healing in recalcitrant wounds. It is also important to pay attention to social factors and those related to the individual.

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Chapter 40

Anemia and Other Hematological Problems

Satish Shanbhag and Rakhi P. Naik

Anemia in the Elderly

Definition of Anemia and Prevalence

Anemia is defined by the World Health Organization (WHO) criteria as a hemoglobin concentration of less than 12 grams/deciliter (g/dL) in adult women and less than 13 g/dL in men.[1] Based on recent large population cohort studies, nearly 10% of adults over the age of 65 years are anemic,[2,3] with prevalence estimates as high as 20–30% in adults over 80 years of age.[3,4]

Common Etiologies of Anemia

The most common identifiable causes of anemia in older persons include iron deficiency, chronic kidney disease (CKD), myelodysplastic syndrome (MDS), and anemia of inflammation.[3,5] However, despite thorough investigation, in nearly 20–40% of elderly individuals, the underlying etiology of anemia may never be identified.[3,5] This phenomenon, known as unexplained anemia of the elderly (UAE), may be due to the underlying biology of aging and may encompass elements from the known forms of anemia such as erythropoietin deficiency,[6] inflammation,[7] or poor marrow reserve.[8,9]

Clinical Consequences of Anemia

Regardless of the underlying etiology of anemia, low hemoglobin is clearly associated with an increased risk of hospitalization and overall mortality in elderly individuals.[2,10–13] In addition, anemia in older adults appears to be associated with poor physical functioning,[13,14] including an increased risk of falls and increased frailty.[15,16] Neurologic dysfunction is also more prevalent among older individuals with anemia and is associated with an increased risk of cognitive decline,[17] poor cognition,[18] and dementia.[19] As a result, diagnosis of the etiology of anemia and targeted strategies to improve low hemoglobin has become an essential component of the care of geriatric populations.

Approach to Anemia

Classification of Anemia

Traditional classification of anemia involves use of the mean corpuscular volume (MCV), which characterizes anemia based on the predominant red blood cell size. MCV categorization was initially developed (1) to identify probable differential diagnoses for the underlying etiology of the anemia, and (2) to provide a guide for targeted workup for anemia. Although tremendous overlap exists between MCV categories and the underlying cause for anemia, especially in the elderly population where the etiology may be multifactorial, MCV still remains a useful paradigm to guide workup and diagnosis of anemia in older individuals. A general scheme using MCV includes:

- Microcytic anemia, defined as MCV <80 fL, which generally points to underlying etiologies such as iron deficiency, thalassemia trait, or anemia of inflammation.
- Normocytic anemia, defined as MCV 80–100 fL, which is associated with anemia of inflammation, CKD-associated anemia, or unexplained anemia.
- Macrocytic anemia, defined as MCV >100 fL, which can be related to vitamin B12 deficiency, alcoholism, medications, or MDS.

In addition to MCV, reticulocyte percentage and absolute reticulocyte count are important laboratory parameters for the workup of any anemia, as a decreased reticulocyte count suggests a hypoproliferative anemia, and increased count may suggest peripheral destruction. The reticulocyte count is often used in conjunction with additional laboratory tests to make a final diagnosis. Specific laboratory tests will be outlined further in each relevant subsection.

Initial Evaluation of Anemia

Anemia in the elderly is often detected on routine laboratory examination, since a complete blood count (CBC) is

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an essential part of routine follow-up of geriatric patients. Anemia may be transient, as can be observed with acute illness, or may be longstanding, as in individuals with underlying comorbidities. The need for additional workup or referral to hematology requires an assessment of the (1) trajectory of hemoglobin values, (2) severity of anemia, and (3) presence of additional cytopenias.

The stability of anemia can be an important clue about the etiology of low hemoglobin in older individuals. For patients with a long history of laboratory records, persistent and stable hemoglobin values often point to anemia of inflammation, CKD, or UAE. Thorough evaluation of underlying comorbidities is warranted for those with stable anemia, as low hemoglobin may be the first clue to subclinical disease, especially renal dysfunction. Other causes such as nutritional deficiencies or bone marrow disorders are usually associated with a clear downtrend in hemoglobin, since they are generally caused by a progressive decline in red cell production. These disorders are also associated with a progressive trend in MCV, such as the new development of microcytosis with iron deficiency or the new development of macrocytosis with MDS.

Assessment of the severity of anemia is also important to guide the need for further workup, referral, or urgent management. Although there is no clear cutoff for the definition of significant anemia, generally a new anemia of <10–11 g/dL warrants further investigation. Abrupt severe anemia may indicate acute bleed, infection, or acute illness. Progressive severe anemia may occur in the context of underlying bone marrow disorders such as MDS or multiple myeloma, nutritional deficiency, or worsening CKD. Anemia that becomes transfusion-dependent always warrants thorough workup and treatment. While it is not unusual for severe anemia to develop during a hospitalization secondary to inflammation, daily phlebotomy, and medications, hemoglobin values should be expected to rebound to baseline values within several weeks post-discharge.

The evaluation of anemia must also involve an assessment for abnormalities in the white blood cell count (WBC) and platelet count. The presence of one or more additional cytopenias may point toward an underlying bone marrow process and warrants investigation. Obtaining a WBC differential is particularly useful in the setting of leukopenia or leukocytosis, as it can often direct the diagnostic workup. A further discussion of WBC and platelet disorders is detailed later in the chapter.

Microcytic Anemias

Iron Deficiency Anemia

The workup of any degree of anemia in the elderly should involve evaluation for iron deficiency anemia, given its high prevalence, association with underlying gastrointestinal malignancy or disease,[20] and ease of management. Although iron deficiency anemia is one of the most common reasons for low hemoglobin in the elderly population, it is notoriously one of the most difficult etiologies to diagnose.[21] Classically, iron deficiency is associated with microcytosis; however, it may also occur in the absence of anemia or may manifest as normocytic anemia.[22] Additional parameters in the CBC that may point to a diagnosis of iron deficiency include an elevated red cell distribution width (RDW) and/or thrombocytosis, although these are not always present.

In addition to a CBC, the relevant laboratory studies that should be sent in the evaluation of iron deficiency include serum iron, total iron-binding capacity (TIBC), transferrin saturation, and ferritin. Iron deficiency occurs in discrete stages starting with the depletion of iron stores (ferritin), followed by decrease in available iron (iron, percent transferrin saturation), and finally by the development of anemia and microcytosis. The definitional cutoffs for iron deficiency vary; however, a serum iron <60 mcg/dL, transferrin saturation <16–20%, and ferritin <40 ng/mL is usually indicative of absolute iron deficiency. Concurrent chronic inflammation, which is present in a significant proportion of older individuals, may result in false increases in the ferritin level,[23] such that empiric iron therapy may be the only reliable way to diagnose deficiency.[21] Functional iron deficiency in the setting of chronic inflammation also presents a diagnostic conundrum, as inflammation may cause an iron-deficient state secondary to iron sequestration and inability to utilize available iron stores.[6]

Because anemia occurs in the late stages of iron deficiency, patients may describe symptoms preceding the development of hematologic abnormalities. Symptoms that should be elicited during a targeted history for iron deficiency should include fatigue, pica (especially pagophagia, i.e., ice chewing), and evidence of bleeding. Fatigue and pica symptoms may occur in a majority of individuals with absolute iron deficiency related to blood loss or malabsorption;[24] however, patients may not recognize their decreased energy or craving behaviors until these symptoms improve with iron therapy. Bleeding assessment should include history of

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hematochezia, melena, symptoms of peptic ulcer disease, or other abnormal mucosal blood loss.

Gastrointestinal (GI) blood loss due to chronic gastritis, arteriovenous malformation (AVM), and colorectal cancer remains a common cause of iron deficiency in the elderly population, with nearly half of anemic individuals demonstrating an identifiable GI lesion on endoscopic evaluation.[20] Baseline screening with upper endoscopy and colonoscopy is, therefore, recommended for evaluation of iron deficiency in all older individuals.

Management of iron deficiency involves oral or IV iron supplementation. Because iron deficiency may be difficult to diagnose in the presence of other comorbidities in elderly individuals, empiric iron therapy for isolated anemia can often be trialed and may result in discernible hemoglobin response.[21] IV iron formulations may be needed in those with severe iron deficiency, oral iron intolerance, or ongoing blood loss. In patients with CKD, especially those requiring erythropoietin-stimulating agents (ESAs), iron supplementation is often recommended for individuals with a transferrin saturation <20–30% and ferritin <100–500 ng/mL to allow for maximal ESA response.[25]

Thalassemia

The thalassemias are a clinically heterogeneous group of disorders caused by reduced or absent production of either the α- or β-chain of hemoglobin. Clinically significant thalassemia syndromes are usually diagnosed in childhood or adolescence; therefore, geriatricians are unlikely to encounter a patient with anemia due to thalassemia who will require intervention or treatment. However, clinically silent α- and β-thalassemia syndromes known as thalassemia trait or minor are common in certain populations, especially those of Mediterranean, African, Middle Eastern, Indian, and Southeast Asian backgrounds.[26] Although it does not require specific management, thalassemia trait results in microcytosis, which may confuse the diagnosis of iron deficiency. A careful review of historical lab values can often help in distinguishing thalassemia trait from iron deficiency, since microcytosis in thalassemia is lifelong and is associated with mild or no anemia, normal RDW, and MCVs that are generally <75 fL.[27] Hemoglobin electrophoresis may be useful in detecting β-thalassemia trait, as hemoglobin A2 levels will be increased; however, a similar test does not exist to diagnose α-thalassemia trait. In those cases, a trial of empiric iron therapy with normalization of iron studies but persistence of microcytosis may suggest thalassemia trait.

Normocytic Anemias

Anemia of Inflammation

Anemia of inflammation (AI), also known as anemia of chronic disease, is a term used to describe the low hemoglobin that has been observed with acute and chronic illness. While initially thought to be associated with conditions with overt inflammation such as infectious, rheumatologic, and neoplastic disease, it is now clear that AI occurs in many circumstances, including aging itself. In fact, it is thought that unexplained anemia of the elderly may be a subset of AI with a similar underlying pathophysiology.[5,28] The mechanism of this type of anemia is thought to be multifactorial and may involve relative erythropoietin deficiency, decreased erythropoietin responsiveness, impaired iron utilization, and shortened red cell survival.[6,29,30] The pathophysiology also likely involves impaired bone marrow response secondary to the suppressive effect of proinflammatory cytokines, which appear to be upregulated with aging.[28,31]

By recent estimates, AI is thought to be the underlying reason for low hemoglobin in approximately one third of persons older than 65 years.[3] Laboratory evaluation usually reveals a normocytic anemia, although microcytosis with AI can also be observed in severe cases. Functional iron deficiency may coexist with AI; therefore, features of iron deficiency anemia such as increased RDW are also common.[32] Iron studies may reveal a low serum iron, low transferrin saturation, and normal or elevated ferritin. Hepcidin, a peptide hormone produced by hepatocytes, is a major regulator of iron metabolism in AI. Upregulation of hepcidin in response to inflammation results in impaired iron absorption and iron sequestration, leading to the observed changes in iron studies.[33] However, commercially available hepcidin assays are not yet routinely used in clinical practice. Direct measurement of inflammatory markers such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) is not usually indicated, given the variability of these cytokines in AI; [34] however, they may be useful for older individuals suspected of having rheumatologic disease or chronic infection. In patients suspected of having AI, a thorough evaluation for chronic disease (such as diabetes mellitus, congestive heart failure, and CKD) is warranted, as anemia is common in these conditions and may demonstrate pathophysiologic and morphologic features typical of AI.[33]

The mainstay of treatment for AI is management of the underlying condition; however, anemia secondary to

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ongoing inflammation or to changes secondary to aging itself may persist despite optimal therapy. Blood transfusion may be required in severe cases, although consensus transfusion goals in the elderly have not been established. Restrictive transfusion for hemoglobin <7 g/dL in asymptomatic individuals appears to be safe.[35] However, older persons – especially those with underlying cardiovascular disease – may become symptomatic at a high hemoglobin threshold. Generally, transfusion is reserved for patients who are thought to have symptomatic anemia characterized by increased fatigue, dyspnea on exertion, or palpitations, which usually does not occur until hemoglobin levels drop below 9–10 g/dL.[36]

Given the high prevalence of functional iron deficiency in AI, empiric iron therapy may be warranted in a majority of affected individuals. Treatment with ESAs may also be used in AI to decrease transfusion requirements and improve symptoms, especially in those with underlying CKD or malignancy.

Anemia of Chronic Kidney Disease

CKD is defined as a reduced estimated glomerular filtration rate (eGFR) of <60 mL/min per 1.73 m^2 and is associated with anemia even in early-stage disease. The prevalence and degree of anemia is directly correlated to the severity of CKD, with nearly 90% of individuals with an eGFR <25–30 mL/min demonstrating low hemoglobin levels.[37] The mechanism of anemia related to CKD involves components of chronic inflammation, functional iron deficiency, and erythropoietin deficiency. Erythropoietin is a glycoprotein growth factor produced by the kidney in response to hypoxemia. The primary role of erythropoietin is to stimulate maturation of erythrocytes,[38] thereby increasing red blood cell production and promoting oxygen delivery. Impaired erythropoietin synthesis in the setting of CKD is, therefore, an important cause of anemia in kidney disease.

Laboratory values often reveal a normocytic anemia that is virtually indistinguishable from AI, given the overlap in pathophysiology. Similar to AI, iron studies may reveal functional iron deficiency with reduced serum iron and percent saturation and normal ferritin levels. Serum erythropoietin levels are not generally required for diagnosis of anemia in kidney disease, since absolute deficiency may not be present despite a relative decrease in production.[39]

Treatment of anemia in patients with CKD involves iron supplementation and ESA support with the goal of decreasing transfusion requirements, alleviating symptoms, and improving patient outcomes.[40] Absolute iron deficiency is likely to be present in individuals with CKD at a transferrin saturation <20% and ferritin levels <100 ng/mL; [41] therefore iron supplementation and workup for underlying cause is required in those patients. In addition, functional iron deficiency may result in ESA non-responsiveness, especially in dialysis-dependent kidney disease, therefore higher goals of transferrin saturation of <30% and ferritin <500 ng/mL may be appropriate.[25] ESA support is recommended for those on dialysis for a hemoglobin <10g/dL after adequate iron repletion. However, because high hemoglobin goals are associated with adverse outcomes including death,[42] ESA support should be withheld for hemoglobin >11 g/dL. For CKD patients not on dialysis, ESA support can be initiated on an individual basis to improve symptoms that are related to the underlying anemia.

Macrocytic Anemias

Vitamin B12 Deficiency

Cobalamin (vitamin B12) deficiency is associated with macrocytic anemia due to impaired DNA synthesis and ineffective erythropoiesis. Deficiency of vitamin B12 is common in individuals older than 65 years and may be associated with dementia, although the etiologic link is not yet clear.[43] Reduced vitamin B12 levels in older populations are often due to malabsorption in the setting of hypochlorhydria, gastric or intestinal surgery, or pernicious anemia. Dietary deficiency is rare, except in the case of strict vegetarianism or chronic malnutrition.

Laboratory evaluation in individuals with vitamin B12 deficiency reveals isolated macrocytic anemia, although pancytopenia may be present in severe cases and may be indistinguishable from MDS. In true deficiency, serum vitamin B12 levels are generally lower than 200 pg/mL; however, levels between 200 and 300 pg/mL may also be associated with true vitamin B12 deficiency, and elevated levels of methylmalonic acid (MMA) and homocysteine are required to verify the diagnosis.[44] Antibodies to intrinsic factor (IF) should be checked in patients with verified vitamin B12 deficiency, as they are highly specific for pernicious anemia.[45] Evaluation of the peripheral blood smear may reveal macro-ovalocytes and hypersegmented neutrophils, which are a hallmark of this condition.

In individuals with pernicious anemia or an anatomic cause for vitamin B12 deficiency such as gastric bypass surgery, treatment with cobalamin supplementation is

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required lifelong. The most commonly prescribed regimen includes intramuscular injection of vitamin B12 at 1,000 mcg daily for 1 week, followed by 1,000 mcg weekly for 4 weeks, then 1,000 mcg monthly. Alternative regimens using oral cobalamin supplementation at supranormal doses of 1,000–2,000 mcg daily may also be considered and can overcome deficiency due to malabsorption.[46] Full hematologic responses usually do not occur for approximately 2 months, although a reticulocyte response and normalization of MMA and homocysteine levels should be noted within the first week of therapy.[44]

Myelodysplastic Syndrome

MDS is a clonal disorder of hematopoietic stem cells resulting in impaired production and maturation of bone marrow elements. The median age at diagnosis of MDS is 65–70 years, with less than 10% of cases occurring in individuals younger than 50 years of age.[47] The pathophysiology of MDS is due to a host of acquired genetic, epigenetic, and chromosomal alterations,[48] and as a result, there is significant clinical heterogeneity in presentation and disease course.

Initial presentation of MDS can vary widely from asymptomatic isolated anemia on routine testing to debilitating disease. Symptoms, when present, are secondary to the degree and type of cytopenias, although constitutional symptoms such as anorexia and weight loss may also occur in severe cases. Anemia may result in significant fatigue, weakness, and dyspnea. Thrombocytopenia may lead to petechiae, easy bruisability, and mucosal bleeding. Neutropenia may result in recurrent bacterial or fungal infection such as pneumonia and sinusitis. Because MDS is often associated with functional impairment of platelets and neutrophils, symptoms can occur with even modest levels of thrombocytopenia and leukopenia.

Macrocytic anemia may be the first manifestation of MDS in the elderly. Macrocytosis in older individuals without any obvious underlying cause such as nutritional deficiency or excessive alcohol intake should be considered suspicious for MDS.[49] Elevated RDW is usually present due to heterogeneity of red cell production, and reticulocyte count is usually inappropriately low, reflecting ineffective erythropoiesis. While anemia is found in nearly all patients with MDS, thrombocytopenia and neutropenia is present in only about a third of individuals at diagnosis.[47] Peripheral blood smear examination may reveal features suggestive of MDS such as hypogranular neutrophils and abnormal nuclear segmentation; however, definitive diagnosis always requires bone marrow examination. Typical features of the bone marrow include hypercellularity, dysplasia, increase in blast percentage, and karyotypic abnormalities. However, in early or mild disease, bone marrow examination may be equivocal, especially if dysplasia is not prominent and chromosomal mutations cannot be identified.

Prognosis in MDS is generally dictated by the risk of progression to acute leukemia. Median survival can range from less than 1 year to more than 5 years, depending on the stage of disease at presentation. Cytopenias, cytogenetic abnormalities, and blast percentage are generally used to determine severity of MDS at diagnosis and have been found to predict survival in MDS.[50]

Treatment for MDS in older individuals depends on the risk category of disease and baseline performance status. Management may include the use of growth factors such as ESAs, chemotherapy, hypomethylating agents, or allogeneic stem-cell transplantation. Generally, aggressive therapy is reserved for high-risk MDS, with the overall goal of modifying disease course by decreasing the risk to progression to acute leukemia and improving overall survival. Low-risk disease, on the other hand, can often be treated with supportive care alone to improve quality of life and reduce transfusion requirements.[51]

Autoimmune Hemolytic Anemias

Warm autoimmune hemolytic anemia (AIHA) is characterized by IgG antibodies that react with red cells antigens at room temperature and cause hemolysis. Reticulocytosis, microspherocytes on peripheral blood smear, a positive direct Coombs test, elevated lactate dehydrogenase (LDH) levels, and reduced-to-absent serum haptoglobin are all lab abnormalities typical for this disorder. Patients with ongoing hemolysis can compensate for weeks to months before presenting to medical care and usually have splenomegaly (red cell destruction occurs mainly in the spleen in warm AIHA) along with the sequelae of anemia.[52] AIHA is often associated with underlying lymphoproliferative disorders such as chronic lymphocytic leukemia (CLL) and autoimmune disease, therefore a workup for underlying etiology is warranted.

Cold agglutinin disease is a type of autoimmune hemolytic anemia characterized by predominantly IgM antibodies binding to the red cell surface and causing complement-mediated red cell destruction at below-normal body temperatures. This is a disease of the elderly

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with a median age at diagnosis of 67 years. Similar to AIHA, cold agglutinin disease is often associated with underlying malignancy. Non-Hodgkin lymphomas, monoclonal gammopathies, and CLL are present in a large majority of patients diagnosed with cold agglutinins, thereby making the recognition of a background inciting factor important when this diagnosis is made.[53–55]

Characteristic features of cold agglutinin disease are symptoms related to anemia along with acrocyanosis and livedo reticularis, both phenomena related to cold exposure in the peripheries. The blood smear has a characteristic look of clumped red cells, and the automated cell counter is often unable to give an accurate read on the hemogram until the sample is warmed to room temperature. There is limited evidence on therapeutic efficacy in this disorder, but the monoclonal anti-CD20 antibody rituximab seems to have the best initial response rates in cases that require treatment.[53,55]

Thrombocytopenia

Overview

Platelets are the dominant cells involved in the primary hemostatic process that occurs in response to vascular injury. The major functions of platelets include adherence, activation, aggregation, and initiation of coagulation; therefore, an overall reduction in platelet number may result in bleeding, especially at sites of frequent vascular injury such as mucosal surfaces.[56] However, although absolute platelet count is clearly a major determinant of hemostatic function, the rate of production and function of platelets also plays a role in determining bleeding risk.

Thrombocytopenia is defined by a platelet count below 150,000/μL. The severity of thrombocytopenia is often categorized by absolute platelet count, with values between 100 and 150,000/μL defined as mild, 50–99,000/μL defined as moderate, and <50,000/μL defined as severe.[57] The underlying mechanisms of thrombocytopenia most commonly include impaired platelet production, peripheral platelet destruction, and splenic sequestration; however, rarely platelet consumption may occur in micro-thrombotic diseases such as thrombotic thrombocytopenia purpura (TTP) or disseminated intravascular coagulation (DIC). Processes that result in impaired platelet production or function, such as MDS, generally lead to bleeding at moderate counts, whereas those that cause thrombocytopenia primarily by peripheral destruction, such as immune thrombocytopenic purpura (ITP), result in bleeding only at severely low counts, since platelet production is preserved.

The evaluation of thrombocytopenia in older individuals should involve an assessment of the trend in platelet count, severity of disease, and associated cytopenias. Stable mild to moderate thrombocytopenia often points to systemic disease (such as liver disease) or medications, whereas progressive, severe thrombocytopenia may suggest a more acute process such as ITP. The presence of additional cytopenias may suggest an underlying bone marrow disorder and should prompt a thorough evaluation. Symptoms, if present, may include mucosal bleeding such as recurrent epistaxis, gum bleeding, GI bleeding, or hematuria. Skin findings include petechiae and spontaneous bruising. Wet purpura (blood blisters on the mucosal surfaces of the mouth) are a marker of severe thrombocytopenia or severely impaired platelet function and, therefore, require immediate evaluation and management.

Isolated thrombocytopenia may be the first manifestation of chronic liver disease or cirrhosis – including virus-associated hepatitis, alcoholic liver disease, or nonalcoholic fatty liver.[58,59] The pathophysiology of thrombocytopenia in liver disease appears to be related primarily to hypersplenism in the setting of portal hypertension, as the spleen can sequester up to 90% of platelet mass; [60] however, decreased thrombopoietin synthesis may also play a role in severe disease.[61]

Immune Thrombocytopenic Purpura

ITP is an acquired disorder caused by the development of autoantibodies against platelet antigens, leading to the premature destruction of platelets in the peripheral circulation by splenic macrophages.[62] ITP is quite common in older individuals, and the incidence appears to increase steadily with age. The average incidence rate for ITP among adults is about two cases per 100,000 person-years, increasing to nearly five cases per 100,000 person-years in people over the age of 60.[63]

ITP should be suspected in any elderly patient with isolated thrombocytopenia and relatively minimal bleeding symptoms compared to the platelet count. Clinically apparent symptoms of thrombocytopenia usually do not occur until the platelet count is lower than approximately 20,000/μL, given the robust bone marrow response that often accompanies this condition. The degree and course of low platelets, especially in the elderly, may vary from chronic moderate thrombocytopenia to a severe acute drop in platelet count.

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An underlying etiology of ITP cannot be found in many cases; however, secondary ITP may occur in the setting of viral infection – especially hepatitis C and human immunodeficiency virus (HIV) – and lymphoproliferative disorders such as CLL. All adult patients suspected to have ITP should undergo testing for hepatitis C and HIV, given the strong association with ITP.[64] Antiplatelet antibody testing has limited utility in ITP, since its predictive value is generally poor.[65] Peripheral flow cytometry should usually be reserved for patients who have an absolute lymphocytosis or suspicion of CLL based on physical examination or blood smear. In addition, bone marrow biopsy is usually not routinely warranted even in older individuals unless additional cytopenias are present or symptoms are concerning for a bone marrow process.[64] Evaluation of the peripheral smear in ITP usually reveals normal erythrocyte and WBC morphology with occasional large (young) platelets. Unlike in children, ITP tends to be chronic in adult patients; therefore, treatment is often aimed at stabilizing the platelet count and preventing symptoms, rather than curing the disease itself. Because bleeding risk does not usually increase until severe thrombocytopenia develops, treatment is generally initiated only if the platelet count is below 30,000/μL. First-line therapy usually involves the use of prednisone at a dose of 1 mg/kg that is tapered slowly over a course of weeks to months. Intravenous immunoglobulin (IVIg) may also be used in severe cases. If clinically significant thrombocytopenia recurs after this initial course, second-line therapy with immunosuppressive agents such as rituximab may be considered.[66]

Hematologic Malignancies in the Elderly

Multiple Myeloma

Myeloma, a plasma-cell neoplasm, is one of the most common hematologic malignancies in the elderly, responsible for an estimated 32,000 new cases and 12,800 deaths per year in the United States for 2020. Despite the 5-year survival in myeloma improving dramatically in the last three decades (from 30.7% in 1994 to 45.1% in 2006 and 53.9% in 2010), myeloma remains a major cause of morbidity in the elderly, with an estimated 140,000 people living with myeloma in the United States in 2017.[67] Myeloma is a disease of the elderly, and the incidence of the malignancy increases significantly with age; in fact, the median age of patients diagnosed with myeloma is 69 years.[68] The prevalence of myeloma is also much higher in African Americans, who have about twice the incidence rates as Caucasians across all age-groups (Figure 40.1).

Multiple myeloma commonly presents with a hypoproliferative anemia, which is present in about 75% of patients at the time of diagnosis. The anemia is typically normocytic, but macrocytosis may be observed in about 10% of patients.[69] The mechanism of anemia can be multifactorial, including impaired hematopoiesis secondary to marrow infiltration by clonal plasma cells and impaired renal function due to direct effects of the monoclonal protein. In addition to anemia, patients with myeloma may develop bony lytic lesions, hypercalcemia, and renal damage from the monoclonal protein.

Given the high prevalence of myeloma in the elderly, a workup for anemia should include an evaluation for monoclonal gammopathy. About 80% of patients with

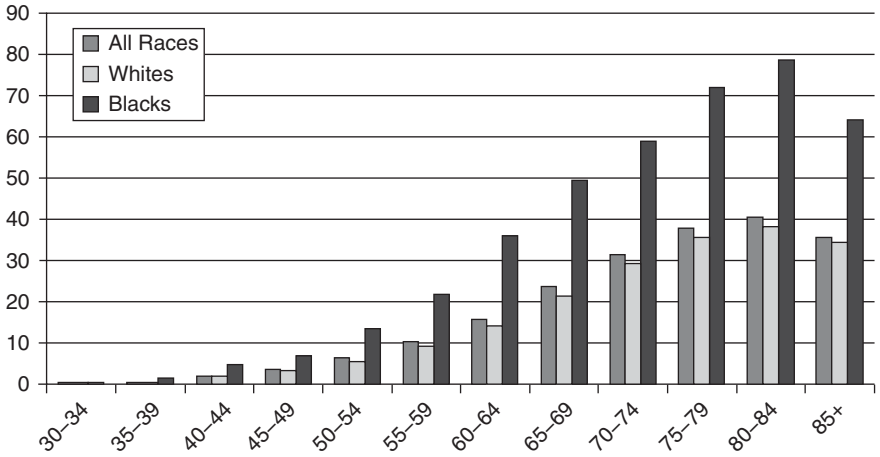

Figure 40.1 Age-specific Surveillance, Epidemiology, and End Results (SEER) incidence rates, 2007–11. Data from the SEER program.

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multiple myeloma have clonal plasma cells that secrete the entire immunoglobulin molecule, which consists of both a heavy and light chain, while 20% of patients have cells that produce only the light-chain fragment of the immunoglobulin molecule.[69] This can make the diagnosis of myeloma somewhat challenging, as patients with light-chain only secretory myeloma do not present with increased gamma globulins (gamma gap) and cannot be diagnosed easily with serum protein electrophoresis alone.

While serum protein electrophoresis and immunofixation (SPEP and SIFE) can be used to detect clonal immunoglobulins, they are not sensitive for detecting light-chain only disease. In the past, urine studies (urine protein electrophoresis and immunofixation) were required to detect urinary light chains, also known as Bence-Jones proteinuria. The advent of the serum-free kappa and lambda light-chain assay, however, has considerably improved the diagnostic ease in detecting patients with light-chain only secretory myeloma and assessing response to therapy.[70] Because myeloma is a clonal disease, the plasma cells secrete only either kappa or lambda restricted immunoglobulin molecules, and an abnormal ratio of kappa to lambda light chains can be seen in the serum at diagnosis. The light-chain assay is also a very useful tool to assess burden of disease, as improvement in the kappa/lambda ratio denotes response to therapy (Table 40.1).

Another important plasma-cell dyscrasia that is frequently found in the elderly is monoclonal gammopathy of undetermined significance (MGUS). MGUS is an asymptomatic, premalignant clonal plasma cell or lymphoproliferative disorder defined by the presence of a serum M-protein <3 g/dL, bone marrow biopsy demonstrating <10% clonal plasma cells, and the absence of end-organ damage. MGUS is not considered a cancer; however, it is a premalignant state. MGUS always precedes the development of multiple myeloma, initially in the form of smoldering myeloma and ultimately in the development of symptomatic myeloma with end-organ damage. However, a majority of elderly patients will never progress to multiple myeloma in their lifetime and will remain in the category of MGUS.

In a predominantly Caucasian population in Olmsted County, Minnesota, the prevalence of MGUS was about 4.6% in those between the ages of 70 and 79 years and rose to 6.6% in those over 80 years.[71] This prevalence is about two to three times higher in African Americans. Risk stratification of MGUS is based on quantification of the monoclonal protein, the free light-chain ratio, and characteristics of the clonal immunoglobulin molecule. These factors can help define the risk of progression to a symptomatic multiple myeloma. The risk of progression at 20 years can be as high as 58% in high-risk MGUS, 37% in high-intermediate-risk MGUS, 21% in low-intermediate-risk MGUS, and 5% in low-risk MGUS.[72] The International Myeloma Working Group updated criteria for the diagnosis of multiple myeloma include validated biomarkers (clonal bone marrow plasma-cell percentage ≥60%, involved: uninvolved serum-free light-chain ratio ≥100, >1 focal lesions on MRI) in addition to attributable CRAB features (hypercalcemia, renal failure, anemia, and bone lesions).[73] This has led to a rise in the number of patients diagnosed with and treated for multiple myeloma, who may have been monitored for smoldering myeloma in the past.

A discussion of therapies for myeloma is out of the scope of this chapter, but suffice it to say that myeloma therapy has evolved to include novel agents such as lenalidomide (immunomodulators), Daratumumab (CD38 monoclonal antibodies), and bortezomib (proteasome inhibitors), which in combination can result in >95% response rates in patients with newly diagnosed myeloma.[74] A majority of elderly patients with myeloma

Table 40.1 Light-chain ratio showing improvement with institution of therapy in a patient with lambda light-chain secretory myeloma with response in quantitative serum-free lambda light-chain levels and improvement of the kappa/lambda ratio

	Normal range	Diagnosis	1 month into therapy	2 months into therapy	6 months into therapy
Serum-free kappa chains	3.3–19.4 mg/L	7.2	1.6	2.5	10.8
Serum-free lambda chain	5.7–26.3 mg/L	2890	564	260	148
Kappa/lambda ratio	0.26–1.65	0	0	0.01	0.07

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are not considered eligible for high-dose chemotherapy and autologous stem-cell transplantation because of their age and comorbidities.

The Leukemias

Chronic lymphocytic leukemia (CLL) is one of the most commonly encountered leukemias in clinical practice in the elderly. Of an estimated 60,500 cases of leukemia diagnosed annually in the United States, approximately 34% of cases are CLL and 14% are chronic myeloid leukemia (CML).[75] CLL is a B-cell chronic lymphoproliferative neoplasm characterized by progressive accumulation of mature, clonal lymphocytes in the bone marrow and peripheral blood. The natural history of the disease, as defined in the 1970s by Rai and colleagues, can vary widely, with median survival times from diagnosis ranging from >150 months in patients presenting with stage 0 disease (defined by lymphocytosis alone) to only 19 months in patients with Stage III and IV disease (characterized by the presence of anemia and thrombocytopenia, respectively).[76] Since then, major improvements including chemoimmunotherapy and targeted therapy have steadily chipped away at CLL-related mortality – 5-year net survival improved from 70.9% in 1995–9 to 77.3% in 2005–9.[77] The incidence of CLL increases dramatically with age from seven cases per 100,000 person-years in those between 50 and 64 years old to 20 cases per 100,000 person-years in those between 65 and 74 and 30 cases per 100,000 person-years in those older than 75.[78]

Anemia itself is not the usual presentation in patients with newly diagnosed CLL, as it can take several years to get to the level of marrow infiltration required to cause disruption of normal hematopoiesis. Thus, hypoproliferative anemia, marked by a low reticulocyte count, usually manifests many years after lymphocytosis initially manifests. Other mechanisms of anemia include autoimmune hemolysis, which can occur in about 4% of patients with CLL and can be seen as a presenting sign of CLL.[79] Autoimmune hemolysis detected in patients with CLL is generally characterized by an elevated reticulocyte count (indicative of a bone marrow responsive to anemia) and the presence of an IgG warm autoantibody detected on a direct Coombs test. Other immune phenomena associated with CLL include ITP and pure red cell aplasia (PRCA), both thought to be the result of immune dysregulation.[80]

The distinction between a hypoproliferative anemia and hemolysis is important, as the treatment can be very different. Steroids and the anti-CD20 monoclonal antibody rituximab form the backbone of therapy for autoimmune hemolysis in CLL, while targeted or combination chemotherapy aimed at the CLL clone forms the mainstay of treatment for anemia related to dense marrow infiltration by the leukemia. The introduction of new oral agents such as the Bruton's tyrosine kinase inhibitor ibrutinib and bcl2 inhibitor Venetoclax have dramatically altered the natural history of CLL, even in patients with high-risk disease and those who relapse after prior therapy.[81] CML is a myeloproliferative neoplasm characterized by unregulated growth of cells in the granulocytic lineage (Table 40.2) and is commonly accompanied by thrombocytosis. Hypoproliferative anemia can be seen in advanced cases of patients presenting in accelerated or blast phase. In contrast to CLL, patients with CML are treated rather than monitored, even when asymptomatic. Highly effective tyrosine kinase inhibitors such as imatinib and dasatinib have revolutionized CML care; a large percentage of patients in complete molecular remission are now able to come off therapy, thereby achieving a functional cure.[82]

Acute myelogenous leukemia (AML) is characterized by accumulation of undifferentiated myeloid precursors (blasts) in the bone marrow and blood with rapidly progressive anemia and thrombocytopenia. It is responsible for about 32% of newly diagnosed leukemias in the United States.[75] Incidence of AML increases with age, with incidence rates of 10 cases per 100,000 person-years in those aged 65–69 years, 15 cases per 100,000 person-years in those aged 70–74 years, and 20 per 100,000 person-years in those aged 75–79 years.[83] AML is a highly aggressive malignancy that is rapidly fatal when untreated. Treating AML in the elderly is challenging, as the aggressive inpatient therapy required to "cure" this cancer is very poorly tolerated in the older adult. Biologic rather than chronological age comes into play to select those few elderly able to tolerate the rigors of months of intensive chemotherapy regimens required to induce a remission. However, the discovery of driver mutations such as FLT3, IDH1, and IDH2 and the subsequent development of targeted agents against them (Midostaurin and Ivosedinib, for example) have revolutionized targeted therapeutics in this space. A combination of the BCL2 inhibitor Venetoclax along with a hypomethylating agent is now approved in the USA for treatment of older adults with AML. It is highly effective in eliciting responses, well tolerated, and treatment can be delivered in an outpatient setting. AML remains incurable with these strategies alone in the majority of patients; recent advances in stem-cell transplant technology

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Table 40.2 Contrasting hemograms in the leukemias

	Normal range	Stage 0 CLL	CML	AML
White blood count	4.5–11 k/cu mm	33	82	60
Hemoglobin	13.9–16.3 g/dL	15.7	14.4	8
Platelets	150–350 k/cu mm	195	895	30
Neutrophils (%)	40–70%	18%	63%	20%
Lymphocytes (%)	24–44%	70%	5%	5%
Bands, myelocytes, and metamyelocytes	0–1%	0%	22%	5%
Atypical lymphs	0–1%	8%	0%	0%
Basophils	0–2%	0%	7%	0%
Blasts	0–1%	0%	1%	70%

have made allogeneic transplants accessible to the select fit elderly with minimal comorbidities.[84]

Myeloproliferative Neoplasms

Polycythemia vera (PV), essential thrombocytosis (ET), and primary myelofibrosis (PMF) are all myeloproliferative neoplasms (MPNs) that exhibit clonal proliferation and differentiation of mature terminal myeloid cells. PV is unique in its epo-independent expansion of the red cell mass with incidence rates that rise with age; in fact, the highest incidence rate of PV is for men aged 70 to 79 years old (24 cases per 100,000 persons per year).[85]

Virtually all patients with PV have the V617F mutation (a change of valine to phenylalanine at the 617 position) in the Janus Kinase 2 (JAK2) gene.[86] This mutation leads to constitutive tyrosine phosphorylation activity that promotes hypersensitivity to cytokines/growth factors and induces epo-independent erythrocytosis. Approximately half the patients with ET and PMF also have the JAK2 V617F mutation.

Classic clinical findings in these patients with PV include thrombocytosis, leukocytosis/neutrophilia, and thrombocytosis with palpable splenomegaly and symptoms of itching and flushing. They have a significant thrombotic risk that can be reduced by phlebotomy (hematocrit goal of 45% in men and 42% in women), antiplatelet agents, and cytoreductive therapy.[87–89]

ET presents with chronic unexplained thrombocytosis; young patients with ET may be asymptomatic and not require therapy, but patients over the age of 60 years are considered high-risk for thrombotic events. The incidence of ET rises with age, with the median age of presentation at 72 years.[90] Patients at high risk of thrombosis may benefit from cytoreductive therapy with hydroxyurea or anagrelide in addition to antiplatelet agents.[91–94]

PMF is the rarest of the three JAK2-mutations associated with MPNs; it remains a disease of the elderly, with a median age at presentation of about 67 years.[90] Common signs at presentation include leukocytosis with a neutrophil predominance, teardrop cells on the peripheral blood smear due to marrow fibrosis, and extramedullary hematopoiesis, splenomegaly, pruritus, and macrocytic anemia. Ruxolitinib, a JAK1/JAK2 inhibitor, has shown activity in patients with PMF and can be beneficial in reducing splenomegaly and symptomatology.[95,96]

In summary, we have outlined here the approach to anemia and hematological disorders in the elderly, with an emphasis on the common causes of anemia and general guidelines for initial therapy.

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Chapter 41

Cancer

Caroline Mariano, Hyman B. Muss, and Chelsea Osterman

Introduction

Cancer represents one of the most common clinical problems encountered in the care of older adults. Cancer is the second leading cause of death in North America. Currently about 50% of cancer diagnoses occur in patients over 65, but this number is expected to increase to 70% by 2030. This is driven in part by the shifting demographics, increasing life expectancy, and improved mortality from cardiovascular disease.[1]

The association of cancer with aging also has biologic foundations. The cellular and genetic changes that occur in cancer development parallel those associated with the aging process. Accumulation of genetic instability, telomere shortening, and epigenetic alterations all contribute to cancer risk. Chronic inflammation is also increasingly understood as an important contributor to cancer risk in older adults.[2]

The aging cancer population has impact across the treatment spectrum from screening decisions, multimodality treatment options, and survivorship to end-of-life care. The older patients are increasingly recognized as victims of health-care disparities during each stage of care. The management in the older adult cancer population is further compounded by the rapidly evolving treatment landscape in oncology. Treatment is becoming increasingly complex, involving newer technologies in surgery and radiation and novel targeted chemotherapeutic options. While many of these treatments have improved toxicity profiles, they are inevitably more costly. Thus, value in cancer care is becoming an important focus moving forward and is one of the major priorities of the American Society of Clinical Oncology (ASCO).

This chapter will review the approach to the principles of geriatric oncology and how these are applied in the most commonly encountered tumor types.

Principles of Geriatric Oncology

Life Expectancy, Geriatric Assessment, and Defining Goals of Treatment

Older cancer patients are different than younger cancer patients, as they frequently have functional loss and other comorbid illnesses that compete with cancer as a cause for mortality. Knowing just the patient's age, tumor characteristics, performance status, and organ function provides only a small snapshot of the patient (Figure 41.1). Frequently the effect on survival of comorbid illness far exceeds the risk of dying of cancer. Brief geriatric assessment (GA) tools that include evaluation of activities of daily living, instrumental activities of daily living, falls, and psychosocial status – including anxiety and depression, social support, nutritional status, and medication use – have been developed for use in the oncology setting and shown to be feasible in both academic and community settings.[3] In addition, a detailed list of comorbid illnesses and their effects on function is obtained. A brief GA can identify problems that when addressed can be improved by utilizing proven interventions (for example, identifying falls and referring for evaluation and management). Multiple studies have shown that GA variables combined with clinical information can predict severe chemotherapy toxicity in elders[3,4] and that these measures are superior to standard assessment of performance status.[5] Furthermore, GA variables, particularly physical function, have been shown to be predictive of mortality in older cancer patients.[6] Randomized trials have shown that GA-based interventions in cancer patients can improve communication among patients and their physicians and lower treatment-related toxicity.[7,8] Performing the GA can be time consuming, and to help overcome this barrier, screening tools have been developed that identify vulnerable or frail patients who can then be referred for a more detailed assessment.[5,6] When possible, care providers are best advised to learn

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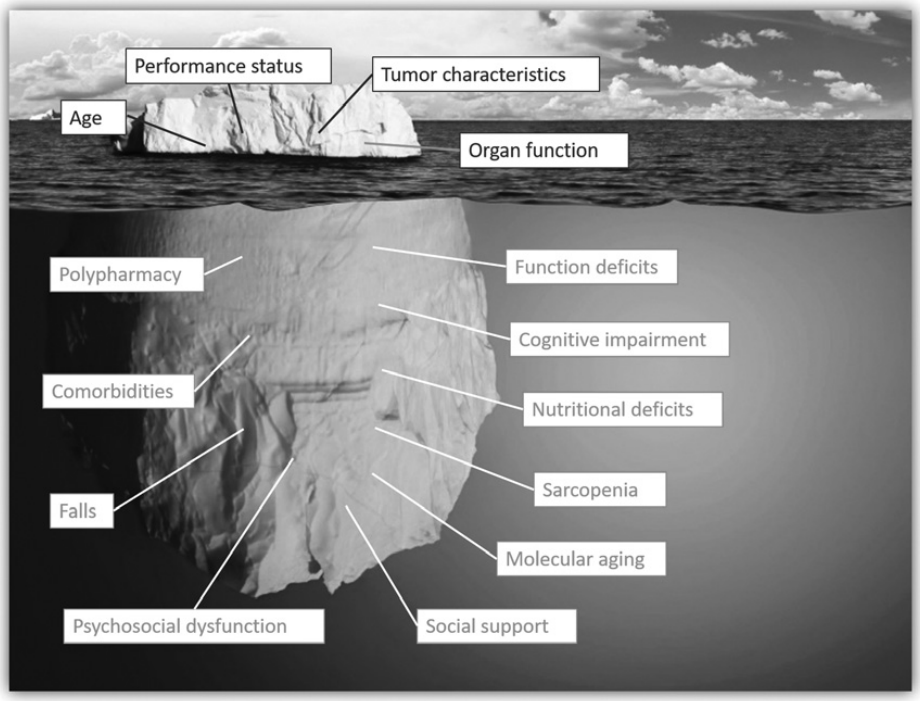

Figure 41.1 The geriatric oncology "iceberg."

how to do a GA; user-friendly, web-based tools are available for this (www.mycarg.org).

Using information from the GA, the first issue to address in caring for the older cancer patient is life expectancy exclusive of the cancer diagnosis. Life expectancy varies greatly among older patients of similar age and must be factored into all treatment decisions. Several user-friendly models are available on the Internet (https://eprognosis.ucsf.edu) that can estimate life expectancy in a variety of settings using clinical and GA parameters. An example using two patients of similar age but with markedly different clinical and geriatric assessment variables shows the value of one of these models in predicting survival (Table 41.1).

After completing the GA and estimating life expectancy, the goals and options of treatment can be better defined. The goals of treatment of older cancer patients also differ from their younger counterparts; older patients with serious illness are much less likely to accept treatment that would result in loss of independence or cognition.[4] One classic study of older patients noted that about 75% of patients would decline therapy that improved their survival if it resulted in severe functional impairment, while almost 90% would decline if it affected their cognitive function.[4] For patients with potentially curable cancers who have long estimated life expectancy but whose cancers are likely to recur or need immediate treatment, standard therapies should be considered. The goal here would be to cure or improve the chances of cure. When cure is not an option, the goals of therapy are palliation of symptoms (if any) and controlling the cancer for as long as possible. Depending on the results of the GA and the potential toxicity of the specific cancer treatment options on physical and cognitive function, the most advantageous treatment recommendations can be offered. Final decision-making should be patient-centered with the patient's (and their family if appropriate) choice taking precedence.

Clinical Trials

In spite of the fact that older patients represent the majority of those with cancer, they are woefully underrepresented in clinical trials.[9,10] Although there is now an expanding literature on outcomes for older patients with cancer treated with surgery, radiation therapy, and chemotherapy, the poor accrual of older patients to newer large Phase III practice-changing trials continues to limit

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Table 41.1 Five- and ten-year all-cause mortality risk for two patients (one well and one unwell) using the combined Schonberg-Lee calculator

Variable	Well	Unwell
Age	75–79 years	75–79 years
Gender	Male	Male
BMI	≥25	<25
Patient's self-reported health	Good	Fair
Chronic lung disease	No	Yes
Prior cancer	No	No
Congestive heart failure	No	No
Diabetes or high blood sugar	No	No
Describe cigarette use	Former smoker	Current smoker
Difficulty walking a ¼ mile without help	No	Yes
Overnight hospitalization in last 12 months	No	One
Help in routine daily activities	No	No
Memory problems interfering with managing finances	No	No
Memory problem interfering with bathing or showering	No	No
Difficulty pushing or pulling large objects	No	Yes
Risk of 5-Year Mortality	23%	69%
Risk of 10-Year Mortality	34–43%	93%

(Modified from www.eprognosis.org)

the generalizability of these results in older patients. Older cancer patients with life expectancies exceeding 5 years and who are fit should be offered participation in clinical trials. Although there are several well-defined barriers to trials participation among older patients, an inherent age bias among oncologists is probably the major reason for not offering older patients clinical trials.[11,12] A paucity of trials exists for patients with poor performance status and/or frailty. Specific trials need to be devised for these groups, as findings from mainstream Phase III trials are not generalizable to this population. In addition, different endpoints may be more relevant for older patients than standard outcome variables such as response rates, progression-free status, and overall survival. Such trials might focus on the effects of treatment on function, quality of life (QOL), and other endpoints that are of major concern for elders.

Breast Cancer

Epidemiology and Risk Factors

Breast cancer is the most common cancer in women, with an estimated 2 million cases diagnosed in 2018. The highest rates are in North America and Western Europe, with age-standardized incidence rates of 85 per 100,000. In the USA, the average age of breast cancer diagnosis is 62, and 44% of new breast cancer diagnoses and 60% of deaths are in women 65 and older.[13]

Traditional risk factors are less relevant in older women, as increasing age becomes a dominant risk factor for developing new cancers. While 1 in 28 women aged 40 to 59 will be diagnosed with breast cancer, that number increases to 1 in 15 for women over age 70.[13] It is common for older women to have a family history of breast cancer; however, BRCA1 and BRCA2 mutations are identified in only a small number of patients. In postmenopausal women, increased body mass index (BMI) is associated with an increased risk of breast cancer. Conversely, physical activity has been shown to lower the risk by as much as 25%.[14,15] The Gail prediction model (https://bcrisktool.cancer.gov) incorporates age and many of the traditional risk factors and can calculate 5-year and lifetime risk, but is less reliable in older women.

Characteristics of Breast Cancers in Older Women

Breast cancers in older women are more likely to be estrogen receptor (ER) positive, progesterone receptor positive, human epidermal growth factor receptor 2 (HER-2) negative, and of lower grade compared to younger women. These factors are typically associated with a less aggressive phenotype, which is substantiated by genetic subtype analysis.[16] However, older women are more likely to present with larger tumors and tumors that have spread to regional lymph nodes. These observations are partially explained by later presentation and detection, but older women tend to develop axillary lymph node spread with relative small primary tumors.[17]

Screening and Prevention

The use of screening mammography in older women is extremely controversial. Only one major screening trial included women aged 70–74 and failed to show a statistically significant improvement in breast cancer mortality in the screened group. Mammographic false-positive

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rates decrease with increasing age.[18] However, for women with decreasing life expectancy, the number needed to screen to prevent a breast cancer death increases exponentially.

Different agencies and societies have different recommendations for screening older women. We suggest that clinicians have an informed discussion of the pros and cons of screening with their older patients who have life expectancies of at least 5 to 10 years with recommendations made on an individual basis.

For postmenopausal women with a high risk of breast cancer, chemoprevention has been shown to decrease new cases of breast cancer by as much as 50%. Tamoxifen, raloxifene, exemestane, and anastrozole have all proven to be effective in lowering the risk of hormone-receptor-positive breast cancer in randomized controlled trials. However, there is no evidence that such treatment improves survival, and potential adverse effects have prevented widespread use.[19,20] Thus, chemopreventive strategies, like screening, should only be offered for very high-risk women with long life expectancy. Older women can reduce their risk for breast cancer by engaging in exercise and maintaining a healthy weight.

Clinical Presentation and Diagnosis

The vast majority of breast cancer patients will present as a result of screening or with a palpable mass. A breast mass in an older woman is more likely to be malignant than in a younger woman. Although additional imaging with targeted mammography, magnetic resonance imaging (MRI), or ultrasound may be helpful to further define the extent of the lesion, core needle biopsy is the optimal method to establish the diagnosis. About 5–10% of women in developed countries will present with metastatic disease at the time of their initial diagnosis. Common sites of metastases include bone, lung, liver, and brain.

Staging

Breast cancer is staged using the TNM, which describes the primary tumor, affected lymph nodes, and distant metastasis. Recently, pathologic characteristics (tumor grade, the presence or absence of estrogen and/or progesterone receptors, and whether the HER-2 gene is amplified) have been added to the traditional staging parameters. For most women newly diagnosed with breast cancer (Stage 0–II), extensive staging investigations are not required. All women should have a detailed history and physical examination (with careful assessment of breast, skin, and lymph nodes), bilateral mammography, complete blood count (CBC), liver function tests, and testing of alkaline phosphatase (ALP). For women with Stage III or locally advanced disease imaging of the chest and liver, and a bone scan are recommended.[21]

Prognosis

Overall, the prognosis for breast cancer is excellent, with close to 90% of patients alive 5 years after diagnosis. When assessing prognosis for older patients, competing causes of mortality must also be considered. Several online tools have been developed to help assess the relative benefit of various treatment strategies. Predict Breast (https://breast.predict.nhs.uk) provides accurate estimates of breast cancer-specific and overall survival at 5, 10, and 15 years, based on age, tumor characteristics, and potential treatment options for patients in average health.[22]

Management

Early-Stage Disease

DCIS

Ductal carcinoma in situ (DCIS) is a precursor to invasive ductal carcinoma and rarely metastasizes. DCIS is most commonly treated with breast-conserving surgery (BCS) and radiation. There is little data about management of older women with DCIS. Older women with DCIS are less likely to have recurrence[17] and almost all will die of non-breast cancer causes. For some older women, particularly those with small amounts of low-grade disease, surgery alone can be adequate. In women with ER-positive DCIS, adjuvant therapy with tamoxifen or aromatase inhibitors can reduce the risk of ipsilateral and contralateral DCIS and breast cancer, but evidence to date shows no improvement in survival.

Local Therapy for Stage 0–III Breast Cancer: Surgery and Radiation

The standard of care for early-stage operable breast cancer is mastectomy or BCS followed by radiation therapy. For lymph node-negative patients, sentinel lymph node biopsy (SLNB) is the procedure of choice. For those with clinically involved nodes, axillary dissection should be considered. Recent data has shown that women with minimal involvement of sentinel nodes by cancer can be spared axillary dissection without increasing their risks of local recurrence or compromising survival.

Both BCS and mastectomy have very low operative mortality rates, with morbidity and mortality driven by

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the presence of comorbidities and not age. For women with limited life expectancy (i.e., less than 5 years), extreme surgical risk, or who refuse surgery, treatment with primary endocrine therapy for women with hormone-receptor-positive tumors is an option that can provide disease control.[23]

Radiation therapy to the breast after BCS is generally safe and well tolerated in older women. Several trials have questioned the benefit of radiation in older women with low-risk hormone-receptor-positive tumors. The CALGB 9343 trial randomized women aged 70 and older with Stage I, ER-positive breast cancer treated with BCS plus tamoxifen versus tamoxifen plus radiation. In long-term follow-up patients treated with radiation had a 2% risk of local-regional recurrence compared to 10% in those treated with tamoxifen alone, but there was no difference in survival.[24] A similar trial in the United Kingdom showed that treatment with radiation improved the risk of ipsilateral breast cancer recurrence (4.1 vs. 1.3%) but other outcomes were similar.[25] It is important that patients who forgo radiation are compliant with their endocrine therapy.

Systemic Adjuvant Therapy

Systemic adjuvant treatment with endocrine therapy, chemotherapy, or both is frequently recommended in addition to surgery in potentially curable patients to lower the risk of recurrence and improve survival. Endocrine therapy is the mainstay of therapy in women with hormone-receptor-positive tumors. The addition of chemotherapy is appropriate for some older women with hormone-receptor-positive tumors and many older women with hormone-receptor-negative or HER-2 positive tumors.[26] Recently, validated genetic assays that can be done on paraffin-embedded tumor tissue can more precisely determine the potential benefits of chemotherapy in patients with lower-risk hormone-receptor-positive tumors.[27] About 10–15% of older patients have Her-2-positive tumors and should be considered for chemotherapy and anti-Her2 therapy depending on risk of recurrence. About 10–15% of older patients have "triple-negative" breast cancer (estrogen and progesterone receptor negative and no amplification of the Her2 gene) and should be considered for chemotherapy. In general, older patients should not be offered chemotherapy unless the estimated survival benefit is 5% or more at 10 years. For those where estimates suggest a survival benefit of 3–5% at 10 years, a balanced discussion of the risks and benefits of chemotherapy is necessary.

Endocrine Therapy

Adjuvant endocrine therapy has been consistently shown to improve outcomes in women with ER-positive breast cancers regardless of age. Tamoxifen has been used for this purpose for decades. Although generally well tolerated, it carries a small risk of endometrial carcinoma (less than 2% over 10 years of use) and venous thromboembolic disease, and has drug interactions with some selective serotonin reupdate inhibitors (SSRIs). In postmenopausal women, aromatase inhibitors (exemestane, letrozole, and anastrozole) have shown improvement in disease-free survival compared to tamoxifen alone. However, they carry a risk of arthralgias and loss of bone density.

Careful discussion and assessment of cancer recurrence risk, life expectancy, and comorbidities is necessary when selecting endocrine therapy in older women. All women treated with aromatase inhibitors should be counseled about bone health and bone density and closely monitored. Endocrine therapy is recommended for at least 5 years after diagnosis. In higher-risk patients 10 years may be superior; recently, genetic tests that can be done on archival tumor tissue can help in making the decision for prolonged endocrine therapy.[28]

Toxicity Considerations

The most effective chemotherapy regimens are multiagent, including an anthracycline and a taxane, but are also the most toxic.[29] Although fear of toxicity in older women is widespread, the decision to offer chemotherapy should not be based on age. The pivotal CALGB 49907 study randomized women over 65 to capecitabine versus multiagent chemotherapy and found that women randomized to the capecitabine arm had inferior overall survival.[30] Trastuzumab in combination with chemotherapy dramatically improves outcomes for Her-2-positive breast cancer patients, and the effect appears independent of age. Non-anthracycline regimens combined with anti-Her-2 targeted therapy are as effective as anthracycline-based therapy and minimize the cardiac risks of anti-Her-2 therapy. All women treated with anti-Her-2 therapy require a baseline cardiac evaluation and routine monitoring during treatment.

"Chemo brain" is a common concern among breast cancer patients. Although data has been mixed, patients may experience cognitive changes with chemotherapy that persist in long-term follow-up,[31,32] and this is an active area of ongoing research. Taxane therapy can lead to severe peripheral neuropathy that is frequently

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irreversible, and patients receiving this treatment require close monitoring.

Locally Advanced Disease

Women who present with locally advanced or inoperable cancer require careful multidisciplinary evaluation. The best outcomes are achieved using neoadjuvant systemic therapy, surgery, and radiation. However, this approach may not be feasible in frail older adults. Neoadjuvant endocrine therapy for those with ER-positive tumors may be effective in this situation as well as primary radiation therapy. For those with triple-negative cancers who are fit, chemotherapy should be considered.

Metastatic Disease

Metastatic breast cancer is incurable and the median overall survival remains about 2–3 years; however, the course is extremely variable between patients. For all women, previous therapy, comorbid disease, functional status, and goals of care must be considered when making treatment decisions.[26] For women with ER-positive disease, hormonal therapy can provide disease control with minimal toxicity for months to years. In patients with ER-negative or organ-threatening disease, chemotherapy, including anthracyclines, taxanes, vinca alkaloids, and capecitabine, can be used. Typically, sequential single agents are used to minimize toxicity. Trastuzumab and other anti-Her-2 therapies are also extremely effective in women with Her-2-positive metastatic disease. For women with bone metastases, bisphosphonates and denosumab prevent skeletal-related events including fractures, hypercalcemia, and bony pain.

Survivorship

After curative treatment, breast cancer survivors should be followed with regular history, physical examination, and mammography. Monitoring for recurrence with blood work, tumor markers, and other imaging studies is not recommended.[33] Women should be encouraged to participate in regular physical activity, maintain a healthy weight, and follow a diet low in fat. There is evidence that these lifestyle interventions improve treatment-related side effects and may decrease risk of recurrence.[34,35]

Prostate Cancer

Epidemiology

Prostate cancer is the third most common cancer globally, with an estimated 1.2 million cases diagnosed in 2018.[36] Incidence rates vary widely across regions depending on use of prostate-specific antigen (PSA) testing. In North America it is the most common cancer diagnosed in men, with an age-standardized incidence rate of 73.7 per 100,000, and it is the second leading cause of male cancer-related death.[37] Prostate cancer is a disease of older men. In autopsy series up to 73% of men over 80 have prostate cancer.[38] The median age at diagnosis in the United States is 66 years old, and over 66% of prostate cancer deaths occur in men aged 75 and older.

Screening and Prevention

PSA is a glycoprotein produced exclusively by prostate epithelial cells. It can be elevated in benign and malignant conditions. Unfortunately, its use as a screening test has been plagued by lead time bias and length time bias.

The two largest PSA screening trials, enrolling over 200,000 men, have now reported long-term outcomes with conflicting results. The European trial found a decrease in prostate cancer mortality of 20% in the screened group aged 55–59, with a number needed to invite to screen of 1,055. In the American trial, which used a combination of PSA screening and digital rectal exam (DRE), no difference in prostate cancer mortality was found. However, 52% of patients in the control group received at least one PSA test.[38,39]

Neither trial enrolled men aged 75 and older, and most consensus groups do not endorse PSA screening in this age group. However, PSA testing remains common practice in the United States even among patients with limited life expectancy.[40] In our opinion, the potential harms, including complications from biopsy and long-term side effects from unnecessary prostate cancer treatment, outweigh any potential benefits of PSA screening in patients 70 and older, and the United States Preventive Services Task Force (USPSTF) recommends against PSA screening in this group.[41]

Clinical Presentation and Diagnosis

Most men presenting with prostate cancer are asymptomatic. Urinary symptoms are more likely to be caused by concurrent benign prostatic hyperplasia. Finding cancer tissue in transurethral prostate resection (TUPR) specimens is another common presentation; 5–10% of patients present with metastatic disease. Prostate cancer usually spreads to regional lymph nodes, bladder, bone, and lung.

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Staging

Workup for prostate cancer should include history, physical examination, DRE, and PSA. Abnormal PSA or DRE are typically followed up with transrectal ultrasound guided biopsy. Although prostate cancer can be staged using the TNM system, a more useful classification creates prognostic groups based on PSA, Gleason score, and T stage (Table 41.2). Additional staging with bone scan and computed tomography (CT) scan of the pelvis is only necessary for patients with intermediate- or higher-risk disease.

Prognosis

Overall, the prognosis for prostate cancer is excellent, with a relative 10-year survival of 99%.[37] Men over 70 tend to present with more aggressive cancers, and have worse survival.[42] Long-term side effects from prostate cancer treatment are also more common in older patients.[43,44] The challenge is to identify patients who are likely to die from prostate cancer rather than other causes. Assessment of tumor biology and GA is helpful in this regard.

Table 41.2 Prognostic grouping for prostate cancer[21]

Risk group	PSA	Gleason score	Stage
Low risk	<10	≤6	T1, T2a
Intermediate risk	10–20	7	T2b, T2c
High risk	≥20	8–10	T3a
Very high risk	Any	Any	T3b, T4
Metastatic	Any	Any	N1 or M1

Management

Localized Disease

For patients with early prostate cancer, a number of treatment options are available, which can be daunting for both clinicians and patients (Table 41.3). Few head-to-head trials exist to compare strategies.

Watchful Waiting

In watchful waiting, patients are treated with palliative therapy only if their disease becomes symptomatic. This is a strategy typically reserved for patients with limited life expectancy who are not felt to be candidates for curative treatment.

Active Surveillance

Active surveillance is an increasingly utilized option in patients with low-risk prostate cancer. It involves a fixed schedule of PSA monitoring and prostate biopsies. Cancer is treated if there is clear evidence of progression. About half of patients end up receiving treatment, and outcomes do not appear to be compromised by the delay in treatment.[44]

Radical Prostatectomy (RP)

RP involves removal of all prostate tissue and seminal vesicles. Open, laparoscopic, and robotic techniques have similar rates of complications. Surgeon experience most consistently predicts the rate of surgical complications and cancer recurrence.[45] Impotence is the most common side effect; rates vary depending on preoperative sexual function and tumor location. Urinary incontinence is common postoperatively, but most patients recover within 2 years.

Table 41.3 Strategies to treat localized prostate cancer

Strategy	Patient selection	Advantages	Disadvantages
Watchful waiting	Frail, limited life expectancy	Avoids treatment in patients likely to die from noncancer causes	Risk of undertreating elderly patients
Active surveillance	Low-risk disease Educated about disease process	Spares treatment in many patients	Risk of biopsy-related complications Anxiety of living with cancer
Radical prostatectomy	Acceptable surgical risk, Tumor not fixed to surrounding tissue	Excellent long-term cancer control Bowel dysfunction rare	Surgical risks Impotence common
Radiation therapy	Able to lie flat and tolerate daily treatments	Excellent long-term cancer control No operative risk Can treat disease beyond prostate bed	Gastrointestinal side effects High symptom burden during treatment Impotence common
Brachytherapy	Low or intermediate risk Prostate volume <50 cc Able to tolerate anesthesia	Excellent long-term cancer control Single treatment	Risk of long-term urinary dysfunction Impotence common

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Radiation Therapy

External beam radiation therapy (EBRT) delivers a therapeutic dose of radiation to the prostate bed, typically given in daily fractions over 6 weeks. Fields can be extended to cover extra-prostatic disease. Sexual dysfunction is a common side effect but rates are generally lower compared to RP.[46] About 50% of patients will experience urinary symptoms during treatment, but most resolve. Radiation proctitis occurs in about 20% of men, with a small risk of long-term gastrointestinal side effects.

Brachytherapy

In brachytherapy, a radioactive source is implanted into the prostate to deliver a high dose of radiation directly to the tumor. With appropriate patient selection and operator experience it offers similar cancer control outcomes to EBRT and RP. Sexual dysfunction is common, again with rates lower than surgery. Gastrointestinal side effects are less common than with EBRT, but about 15% of patients report urinary dysfunction in long-term follow-up.[46]

Advanced or Recurrent Prostate Cancer

Advanced prostate cancer is a heterogeneous disease. Many patients have only biochemical evidence of cancer, detected by rising PSA after curative treatment, and will die from other causes. Other patients have diffusely metastatic disease, leading to a high burden of morbidity and mortality.

Hormone-Sensitive Disease

The first-line treatment of advanced prostate cancer is androgen deprivation therapy (ADT), most commonly with luteinizing hormone-releasing agonists. Long-term ADT is associated with loss of libido, fatigue, decreased bone mineral density, decreased muscle mass, reduced insulin sensitivity, increased cholesterol, and subtle changes in cognition.[47] The PR7 trial compared intermittent with continuous ADT in patients with biochemical recurrence only.[48] Overall survival was similar between arms, but intermittent therapy was associated with lower costs and a small improvement in QOL. Only 45% of deaths in the trial were due to prostate cancer. A similar trial in men with documented metastatic disease also showed some improvement in QOL with intermittent ADT, but median overall survival was shorter (5.1 vs. 5.8 years).[49]

Randomized Phase III trials have demonstrated an overall survival benefit with the addition of abiraterone, an inhibitor of androgen synthesis, or enzalutamide or apalutamide[50,51], androgen receptor inhibitors, to ADT over ADT alone in men with metastatic hormone-sensitive prostate cancer. QOL is generally preserved with the addition of these agents to ADT, but many of these studies had a nonsignificant hazard ratio for death in patients ≥70 or ≥75, and so uncertainty remains regarding the risks versus benefits of these therapies in older men.[52]

Castrate-Resistant Prostate Cancer (CRPC)

Patients will eventually fail ADT and progress to a hormone-resistant state. Until 2011, docetaxel was the only drug shown to improve survival in CRPC. With the development of newer agents, the median overall survival is now over 2 years. Docetaxel has been consistently shown to benefit older men,[53] and fit older patients were represented in clinical trials of most novel agents (Table 41.4). Since many of these therapies are less toxic than traditional chemotherapy, a wider spectrum of patients can be offered treatment.[54] In the absence of data directly comparing these agents, selection is often based on the toxicity profile of each drug and patient comorbidities. Because of the rapidly changing therapeutic landscape, patients with reasonable functional status should be referred to medical oncology for a discussion about their options. Additionally, for men with bone-metastatic CRPC, treatment with a bone-modifying agent such as denosumab or zoledronic acid is recommended to prevent skeletal-related complications.

Colorectal Cancer

Epidemiology

Colorectal cancer (CRC) is the fourth most common cancer worldwide. It is the third most common cause of cancer-related death in the USA, with 70% of deaths occurring in patients over 70. In North America the age-standardized incidence rate is 30 per 100,000. The average age at diagnosis is 69.[37]

Screening and Prevention

CRC screening recommendations have evolved considerably, and differ between major consensus groups.[55,56] Most screening tests have not been extensively validated in older adults. Because of lag time and the long natural history of CRC, models suggest that it takes 10 years for patients to ultimately derive benefit from CRC screening.

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Table 41.4 Agents used in treatment of advanced castrate-resistant prostate cancer (adapted from [54])

Drug	Mechanism of action	Older patients in registration trial	Benefit/advantages	Side effects/ disadvantages
Docetaxel	Taxane-type chemotherapy	20% age ≥75	Improved survival by 2.4 months. Improved QOL	Fatigue, neuropathy, myelosuppression
Abiraterone	Oral androgen biosynthesis inhibitor	28% age ≥75	Improved survival by 4.8 months. Well tolerated, improved QOL	Fluid retention, hypokalemia
Enzalutamide	Oral anti-androgen	25% age ≥75	Improved survival by 4.8 months. Well tolerated, improved QOL	Seizures (rarely), fatigue, hot flashes
Radium-223	Alpha-emitting radiopharmaceutical targeting bone	Median age 71	Improved survival by 3.6 months, Improved pain, QOL	Mild diarrhea, thrombocytopenia
Cabazitaxel	Taxane-type chemotherapy	9% age ≥75 max age 80	Improved survival by 2.4 months	Neutropenia common, 6% treatment-related deaths in older adults
Sipileucel -T	Immunotherapy	73% age ≥65	Improved survival by 4.1 months	Infusion reactions Very costly

Therefore, fit older patients may benefit from screening, if life expectancy is more than 10 years.[57]

Both endoscopic screening and stool-based tests are used for CRC screening. Sensitive fecal occult blood testing (FOBT) annually or biennially decreases CRC mortality by 15–25%.[58] Fecal immunochemical-based testing (FIT) offers improved sensitivity and specificity compared to guaiac-based FOBT. Endoscopic screening offers both diagnostic and therapeutic opportunities. Flexible sigmoidoscopy every 5 years or colonoscopy every 10 years are both options. While colonoscopy has the advantage of evaluating the whole colon, older patients are more likely to suffer from complications of colonoscopy.[59]

We recommend that clinicians offer CRC screening to patients aged 50–75 either with FIT testing (or FOBT if not available) yearly, or endoscopic screening, if estimated life expectancy is more than 10 years. For patients over 75 with a long life expectancy, the decision to screen must be individualized.[60]

Meta-analysis of chemoprevention trials has shown that aspirin use is associated with a 6–7% absolute decrease in the risk of developing CRC or adenomas. However, this must be weighed against potential adverse events and does not replace screening.[61]

Clinical Presentation

Only about 30% of CRCs are diagnosed though screening in North America. Common presenting symptoms include iron deficiency anemia, change in bowel habits, abdominal pain, and weight loss; 5–10% of patients will present with an acute obstruction. Patients with rectal cancers are more likely to present with hematochezia, tenesmus, and pain; a small percentage will be palpated on DRE. In symptomatic patients the next step should be referral for colonoscopy. Screening tests such as FOBT are not appropriate for diagnostic evaluation.

About 20% of patients in developed countries will present with metastatic disease. Common sites of CRC spread are intra-abdominal lymph nodes, liver, lung, and peritoneum.

Staging

CRC is staged using the TNM system. All patients with suspected CRC should have a complete colonoscopy and biopsy of suspicious lesions. Rectal cancers are defined as tumors arising below the peritoneal reflection, which is usually 12–15 cm from the anal verge.

Preoperatively all patients with CRC Stage II and above should have a CT scan of the abdomen and pelvis, CBC, and carcinoembryonic antigen (CEA) level. The role of chest imaging is controversial as it often picks up indeterminate lesions, particularly in older patients. Positron emission tomography (PET) scanning offers increased sensitivity but is less reliable in lesions under 1 cm and should not be routinely performed.

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Surgical planning for rectal cancers is more complicated, so additional local-regional staging including pelvic MRI and endorectal ultrasound is often required.

Prognosis

Overall, the 5-year relative survival for CRC is 65%. In early-stage disease other factors such as grade, genetic features of the tumor, and comorbidities are important determinants of outcome.

Management

Stage I–III (Early-Stage) Disease

Local Therapy: Surgery and Radiation

The goal of oncologic surgery is complete removal of the tumor and regional lymph nodes, typically with partial colectomy. Although operative mortality is low, frail older patients have a higher risk. A preoperative geriatric assessment is helpful to counsel patients and optimize comorbid conditions. Unfortunately, older patients remain undertreated and are more likely to undergo noncurative or emergency surgery.[62]

Local management of rectal cancer is complex and requires multidisciplinary evaluation. The surgical procedure of choice is a total mesorectal excision (TME). In cancers stage T3 and above the addition of pelvic radiation (often with concurrent chemotherapy) improves outcomes.

In frail older patients with symptomatic CRC, surgical consultation is still warranted, as minimally invasive procedures can sometimes be performed to relieve bleeding or obstruction.

Adjuvant Chemotherapy

The use of adjuvant 5-flurouracil (5-FU)-based chemotherapy is well tolerated, and this treatment has been shown to decrease recurrence rates and improve survival in colon cancer for several decades. The vast majority of Stage II colon cancers are cured with surgery alone, and adjuvant chemotherapy provides little benefit. However, in selected patients with high-risk disease it may be considered.

For Stage III colon cancer the standard of care is adjuvant chemotherapy with a combination of 5-FU/leucovorin and oxaliplatin. The 5FU component can be given as the oral derivative – capecitabine. The relative risk reduction of cancer relapse with the use of 5-FU alone is 35% compared with placebo. In a landmark clinical trial the addition of oxaliplatin in Stage III colon cancer improved overall survival at 5 years to 73% compared to 69% with 5-FU alone.[63] There is ongoing debate about the benefit of oxaliplatin in older patients. In subgroup analyses of two randomized control trials, patients over 70 did not derive any benefit from oxaliplatin.[64] However, in large cohort analyses older patients have similar outcomes to younger patients treated with adjuvant chemotherapy.[65] Neuropathy occurs in 90% of patients during treatment with oxaliplatin and persists at 1 year in about one third of patients. This can ultimately result in functional limitations. For older adults with contraindications to oxaliplatin, single-agent capecitabine (an all-oral regimen) is often chosen. The duration of combination chemotherapy was traditionally 6 months. However, recent trials suggest that 3 months of therapy is adequate in low-risk patients (i.e., T2-3, N1).[66]

Older adults with CRC remain undertreated with adjuvant chemotherapy.[67] Treatment decisions should be based on life expectancy, tumor biology, functional status, and patient preference, not age alone.[62]

Follow-Up

For the first 5 years after curative treatment, patients should be routinely followed with clinical evaluation, colonoscopy, CEA levels, and imaging of the liver and chest. This allows for early detection of recurrent disease that could be amenable to resection. For patients who would not be candidates for further surgery or chemotherapy, active surveillance should not be pursued.[68]

Stage IV (Advanced-Stage) Disease

The median survival of advanced CRC is approximately 2–3 years, and many patients maintain good QOL.

A subset of patients with isolated liver or lung metastases may be cured with surgical resection or high-dose radiation therapy to the metastatic site. Careful multidisciplinary evaluation is necessary to plan and sequence treatment. Surgical approaches can also be helpful in palliation of symptoms such as obstruction or bleeding in advanced disease.

Chemotherapy remains the mainstay of treatment for most patients with metastatic CRC. Several targeted biologic agents have been developed that improve outcomes (Table 41.5). Typically, agents are used sequentially, with 5-FU remaining the backbone. Chemotherapy "breaks" can be used safely to preserve QOL and improved toxicity. Frail older patients may also benefit from dose modifications to avoid serious toxicity.[69] Unfortunately, older patients have been underrepresented in metastatic

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Table 41.5 Chemotherapy commonly used in treatment of colorectal cancer

Agent	Mechanism of action	Common toxicities
Infusional 5-FU	Cytotoxic antimetabolite	Diarrhea, mucositis
Capecitabine	Oral 5-FU derivative, renally cleared	Hand-foot syndrome, diarrhea
Oxaliplatin	Cytotoxic platinum derivative	Neuropathy, myelosuppression
Irinotecan	Cytotoxic DNA topoisomerase inhibitor	Diarrhea, myelosuppression
Cetuximab Panitumumab	Monoclonal antibody against epidermal growth factor receptor	Infusion reactions, rash, hypomagnesemia
Bevacizumab	Monoclonal antibody targeting vascular endothelial growth factor (VEGF)	Hypertension, proteinuria, impaired wound healing. Rare arterial thromboembolism but higher risk in elderly

CRC trials, but the existing evidence suggests fit patients derive similar benefit to their younger counterparts.[62]

Lung Cancer

Epidemiology

Lung cancer is the most common cancer diagnosed worldwide, with over 2 million new cases in 2018.[36] It is also the leading cause of cancer-related deaths. In developed countries overall rates are decreasing; lung cancer is more common in men, but rates in women are increasing. In the USA the average age of lung cancer diagnosis is 71; 85% of lung cancer cases are classified as non-small-cell lung cancer (NSCLC), which can be further divided into subtypes including adenocarcinoma, squamous cell carcinoma, and large cell carcinoma. Small-cell lung cancer (SCLC) accounts for the majority of remaining cases.

Screening and Prevention

The best and most cost-effective preventive measure for lung cancer is smoking cessation. In the USA, approximately 8% of people age 65 and older are current smokers. Worldwide, tobacco use continues to increase, particularly in developing countries, where there are fewer limits on advertising and lower rates of taxation on tobacco products.[70] Smoking cessation gradually decreases lung cancer risk over 15 years, which then plateaus at approximately two times baseline risk. Even in patients diagnosed with lung cancer, smoking cessation improves mortality.[71]

Screening for lung cancer was not thought to be effective until the publication of the National Lung Cancer Screening Trial (NLST). This trial enrolled 53,000 people aged 55–74 at high risk for lung cancer. Patients were deemed high risk if they had at least a 30-pack-year history of smoking, were current smokers, or had quit within 15 years. Participants were randomized to low-dose CT chest versus chest X-ray annually for 3 years. In the CT group, 24% of patients had an abnormal result, of which 96% were false positives. Diagnostic evaluation for positive screenings was performed at high-volume centers, with a 1.4% rate of complication. There was a 20% decrease in lung cancer mortality in the CT group, with a number needed to screen of 320.[72] More recently, models have been developed to predict probability of cancer in nodules identified on CT screening.[73]

Several organizations including the USPSTF have recommended screening for patients who fit NLST criteria.[74] We recommend that lung cancer screening be discussed with these high-risk patients who have life expectancy more than 5 years and would be able to tolerate lung cancer treatment. Patients who elect to be screened should be referred to a center with expertise in interpretation and evaluation of lung nodules. Smokers should be counseled about cessation, regardless of age or comorbidities.

Clinical Presentation

Many patients with early lung cancer are asymptomatic. Local symptoms can include cough, hemoptysis, chest pain, and dyspnea.[75] More than 50% of patients will present with advanced lung cancer; metastases to liver, bone, brain, and adrenal glands are most common. Weight loss at presentation is associated with adverse prognosis. A variety of paraneoplastic phenomena are associated with lung cancer. Hypercalcemia and hypertrophic osteoarthropathy are commonly associated with NSCLC. Small-cell lung cancer is associated with SIADH (syndrome of inappropriate antidiuretic hormone) and a variety of neurologic and endocrine paraneoplastic syndromes.

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Staging and Diagnosis

NSCLC is staged using the TNM system, updated in 2018. Clinically, SCLC is divided into limited-stage disease, cancer limited to the lung that can be encompassed in one radiation port, and extensive-stage disease.

All patients with suspected lung cancer should have a detailed history and physical examination. Initial imaging should include a CT scan of the chest and upper abdomen. This can usually identify a site for biopsy. A core biopsy, rather than fine needle aspiration, should be obtained whenever possible. Brain imaging should be performed in all patients with SCLC, and patients with Stage III or IV NSCLC.[21]

Patients with potentially resectable NSCLC should be referred for multidisciplinary evaluation. Further staging investigations include PET scan, mediastinal lymph node evaluation (either with endobronchial ultrasound or mediastinoscopy), and full pulmonary function tests.

Management of Non-Small-Cell Lung Cancer

Early Stage

Surgery is the standard of care in patients with Stage I and II NSCLC and offers the potential for cure in 60–70% of patients. Careful preoperative assessment is crucial, including mediastinal staging, detailed assessment of lung function, and functional capacity. Fit patients should be considered for partial or complete pneumonectomy, depending on tumor location. For patients not eligible for surgery, treatment with stereotactic radiation therapy is another potentially curative modality.[76,77]

In patients with Stage II and some patients with Stage IB NSCLC, the addition of four cycles of cisplatin-based adjuvant chemotherapy has been shown to improve 5-year overall survival by up to 15%.[78] Subset analyses of clinical trials showed that while older patients often received lower doses or fewer cycles of chemotherapy, they still derived survival benefit, without compromise in long-term QOL.

Locally Advanced Lung Cancer

The management of locally advanced lung cancer is complex, and practices vary widely between centers. In general, multimodality therapy including surgery, radiation, and chemotherapy offers the best outcomes. Unfortunately, few older patients were enrolled in clinical trials for Stage III lung cancer. The existing evidence suggests that fit older patients still benefit from aggressive therapy, but may be at higher risk for pneumonitis, myelosuppression, and cardiac complications.[79]

Metastatic NSCLC

Outcomes for advanced NSCLC are generally poor, with a median overall survival of 6–12 months, but the introduction of immunotherapy has greatly extended survival for a subset of patients. Initial treatment for fit patients is typically a combination of platinum-based chemotherapy and immunotherapy, while more vulnerable patients may be treated with single agents. Although many of the trials evaluating the use of immunotherapy in NSCLC included few patients over age 75, retrospective analyses suggest that older patients derive similar benefit from immunotherapy to younger patients.[80,81]

In an important trial of patients ≥70 years old with Stage IV NSCLC, patients were randomized to treatment based on Comprehensive Geriatric Assessment (CGA) versus performance status and age.[82] Patients in the CGA arm experienced less toxicity (85.6% vs. 93.4%) and fewer treatment failures due to toxicity (4.8% vs. 11.8%) compared to the standard care arm. Although the median treatment failure-free survival and overall survival were not different between groups, this study underscores the importance of the CGA in treatment decision-making.

Fortunately, recent advances have allowed for identification of "driver mutations" in some NSCLC patients, which can be effectively treated with oral tyrosine kinase inhibitors (TKI) targeting these mutations. One example is the epidermal growth factor receptor (EGFR) mutation, which occurs in about 15% of patients with adenocarcinoma. In these patients, treatment with TKIs targeting EGFR has proven more effective and far less toxic compared to cytotoxic chemotherapy. Similarly, a small percentage of adenocarcinoma patients will have an anaplastic lymphoma kinase (ALK) rearrangement and can be effectively treated with oral therapy. Therefore, even older patients who refuse or are unfit for chemotherapy should be referred to medical oncology to discuss other therapeutic options.

Patients with metastatic lung cancer often have a high symptom burden. Many interventions are available to address symptoms, including radiation therapy and indwelling pleural catheters. In a pivotal study, early referral to specialist palliative care was shown to improve both QOL and survival in patients with Stage IV NSCLC, despite lower utilization of chemotherapy in the intervention group.[83]

Management of SCLC

SCLC is a very aggressive disease, with a tumor doubling time of approximately 60 days. Standard treatment of

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limited-stage disease is a concurrent radiation and platinum-based chemotherapy. This offers a response rate of 80–90%, but only 10–15% of patients will ultimately be cured.[84]

Initial therapy for extensive-stage SCLC is platinum-based chemotherapy with immunotherapy, followed by maintenance immunotherapy. With treatment, the median survival is approximately 12 months. As in NSCLC, older patients are often undertreated, but evidence suggests that they derive similar benefit to their younger counterparts. Patients with limited-stage SCLC and extensive-stage SCLC without brain metastases have improved survival when treated with prophylactic cranial radiation. The benefit is consistent in older patients; [85] however, whole brain irradiation is associated with neurocognitive side effects,[86] which may have a more significant impact in the frail elderly.

Immunotherapy in Older Adults

Immunotherapy is an emerging category of anticancer therapy. Immunotherapy agents are being used across tumor types. These agents have the advantage of being less toxic than traditional chemotherapy and potentially more effective. Immunotherapy has become the standard of care for treatment of melanoma, lung cancer, bladder cancer, kidney cancer, and head and neck cancer, with new indications emerging rapidly.

The agents used are primarily immune checkpoint inhibitors. They are antibodies directed against proteins in the T-cell immune cascade that effectively "unleash" the T-cell response to cancer cells. The first approved agent was ipilimumab, which acts against CTLA-4. Other commonly used agents include nivolumab (anti-PD-1) and pembrolizumab (anti-PDL1), and there are six other agents the Food and Drug Administration (FDA) had approved at the time of writing. Response rates to these agents are typically 20–50%, with a smaller number of patients achieving long-term durable responses. These agents have consistently shown better side-effect profiles compared to chemotherapy, with decreased risk of fatigue, nausea, and infectious complications. However, they do have a risk of autoimmune side effects such as colitis and pneumonitis, which can be life-threatening. Prompt treatment with corticosteroids is warranted.

Data to date suggests that older adults with good performance status have similar rates of response to immunotherapy compared to their younger counterparts. They do not seem to have higher rates of toxicity; however, the frail elderly may have more difficulty recovering from serious adverse events.[87]

Conclusions

The management of cancer in the older patient remains a challenge, which has been complicated by gaps in education and resource availability. There is a major gap in geriatric knowledge and training among oncologists; likewise, primary care physicians, including geriatricians, are frequently unaware of the potential benefits of modern cancer treatments. Coupled with the projected shortage of oncologists and geriatricians in the years to come, this lack of training in geriatrics will further compound the challenges for a health-care system currently in crisis. Education of cancer care providers is a pressing need and is currently being addressed by many organizations. Ideally, such training in geriatrics should begin in medical school, continue throughout residency, and be part of maintenance of certification. Outstanding resources exist to train practitioners about key issues related to the care of older patients, and several are listed in Table 41.6.

Geriatric oncology is a rapidly growing field, and new research will undoubtedly lead to more rational assessment and management of older cancer patients. We believe that interdisciplinary care and education are the keys to better outcomes in this population.

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Table 41.6 Internet resources helpful in the management of older cancer patients

Name	Description
ASCO University http://university.asco.org/geriatric-oncology	Online modules that explore different care options for older patients with various malignancies. Also has maintenance of certification (MOC) module on Geriatric Oncology.
International Society of Geriatric Oncology (SIOG) www.siog.org	Website provides useful links to geriatric oncology guidelines and other educational materials.
ePrognosis http://eprognosis.ucsf.edu/default.php	A series of calculators for estimation of life expectancy based on studies of older adults in different health-care settings. Includes smartphone application that can be applied to screening decisions.
CARG (Cancer and Aging Research Group) www.mycarg.org	A group of researchers with major interest in geriatric oncology research. Opportunities for mentoring. Also online chemotherapy toxicity calculator and geriatric assessment tools.
Moffitt Cancer Center Senior Adult Oncology Program Tools http://moffitt.org/cancer-types–treatment/cancers-we-treat/senior-adult-oncology-program-tools	The CRASH score – online tool for estimating the toxicity of chemotherapy. Site also has other geriatric calculators.
Lineberger Comprehensive Cancer Center Geriatric Oncology http://unclineberger.org/geriatric	Free PowerPoint slide sets of core lectures in geriatrics as well as other resources.

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Chapter

42 Ocular Disorders

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The management of ophthalmic conditions in the aged population poses challenges to primary care physicians, emergency medicine teams, and ophthalmologists because of a diverse spectrum of ophthalmologic conditions.[1,2] The USA defines visual impairment as vision worse than 20/40 with corrective lenses, and legal blindness is defined as vision worse than 20/200 with corrective lenses. Vision loss in the elderly has been deemed a significant public health problem by the Centers for Disease Control, as at least one third of the American population over the age of 65 has a vision-compromising condition.[3] The management of ocular health in the rapidly aging US population poses a significant economic and societal burden.[4]

Vision is imperative for an autonomous and independent aging process, and reduced vision may result in significant impairment, limitations on activities of daily living, and a negative impact on quality of life. There are significant associations between decreased vision, chronic medical disease, social isolation, and depression. It is imperative that ocular pathologies are identified, monitored, optimized, and treated to prevent vision loss, and to provide our patients with the best quality of life. The American Academy of Ophthalmology recommends comprehensive ophthalmic examination every 1–2 years in otherwise healthy, asymptomatic individuals over the age of 65.[5]

Eyelids and Lacrimal System

The eyelid is anatomically delineated into anterior and posterior lamellae, divided by the tarsal plate, and is lined internally with palpebral conjunctiva. The eyelid structure is integral in protecting and nourishing the ocular surface. Effective blinking and complete eyelid closure facilitate distribution of tear film over the entire ocular surface and prevent desiccation of the cornea and conjunctiva. Additionally, the tear film has important utility in ocular health and optical quality. Tear film maintains an antimicrobial environment, delivers growth factors and nutrients, and lubricates and protects the ocular surface. As eyelid tissues age, changes in tissue laxity, tensile strength, and fat-pad alterations may alter lid function, which may lead to ocular surface dysregulation.

Structural Changes to the Eyelid

Age-related structural changes to eyelid anatomy can lead to visual deterioration by physically occluding the visual axis, or by altering the ocular surface. Structural and positional changes to eyelid anatomy alters blink function, making the surface of the eye susceptible to pathology. Alterations in blink efficacy or incomplete closure of the eyelids can cause surface exposure, and may result in corneal breakdown, leading to ulceration and severe infection.

Ectropion, Entropion

Lid malpositions such as ectropion (external rotation of the lid margin away from the eye), or entropion (internal rotation of the lid margin toward the surface of the eye) are common conditions seen in the elderly. Ectropion can result in excessive copious tearing because the punctum drainage system of the eye is no longer flush and in contact with the ocular surface. The tear lake is unable to effectively drain, and therefore tears spill over the margin of the eyelid onto the face. Additionally, ectropion can cause severe dry eye disease (explained further in the Cornea section) because of poor seal of the eyelids. Entropion may cause contact between the abrasive eyelashes and the delicate cornea and conjunctiva. Entropion often leads to surface discomfort and irritation, and may lead to severe corneal breakdown and infection. Lid malposition can be managed conservatively with lubrication, or surgically with lid repositioning surgery.

Ptosis

Ptosis, an abnormally low-lying upper eyelid, is a common finding in the elderly and can be congenital or acquired.

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Acquired ptosis subtypes include myogenic, neurogenic, mechanical, and traumatic ptosis. All acquired ptosis requires thorough workup to evaluate for underlying systemic etiologies such as myasthenia gravis, Horner's syndrome, cranial nerve palsies, masses, or trauma. Ptosis may be corrected by treating the underlying medical disease, or with surgery to reposition the lid.[6]

Dermatochalasis

Dermatochalasis, loose eyelid skin, is a common accompaniment to the normal aging process. Dermatochalasis is benign and initially may be considered a cosmetic issue, but can progress to compromise the visual field. Skin that is obstructing the visual field can be removed surgically. The procedure to repair dermatochalasis is called blepharoplasty.

Epiphora

Epiphora is copious unilateral or bilateral tearing. Epiphora may be due to eyelid malposition, conjunctivochalasis, or nasolacrimal duct obstruction. Conjunctivochalasis, redundant folds of conjunctiva, can precipitate tears falling out of the eye by blocking the inferior punctum and by disrupting the inferior tear lake. Nasolacrimal duct obstruction occurs when the tear drainage pathway is obstructed. Obstruction can be assessed by in-office irrigation of the nasolacrimal duct, and can occur because of recurrent infection or medication side effects, or can be idiopathic. Mechanically obstructed nasolacrimal duct obstructions or stenosis may require surgical repair with stenting or other surgery to provide patent outflow drainage of tears into the nasal cavity. Patients with obstructed drainage are at higher incidence of localized infection, called dacryocystitis, which may warrant antibiotic therapy.

Floppy Eyelid Syndrome

Floppy eyelid syndrome (FES), a structural abnormality of the eyelids, is due to a deficiency in elastin in the tissue, which creates excessive lid laxity. The anatomic changes of FES lead to chronic ocular surface discomfort, redness, and papillary conjunctival reaction of the upper palpebral conjunctiva. Primary care physicians should be aware of FES because of the association between FES and obstructive sleep apnea (OSA). A patient diagnosed with FES warrants investigation into OSA.

FES can be managed conservatively with ocular lubrication, antihistamine drops, and eyelid taping if lids are too mobile when sleeping. Surgical intervention with horizontal eyelid shortening can help to relieve eyelid irritation by restoring the appropriate anatomic structure of the lids.[7]

Eyelid Neoplasms

Eyelid growths are commonly encountered in the elderly population.[8,9] Over 80% of all eyelid lesions are benign. The most common neoplasms seen in the elderly are basal cell carcinoma, squamous cell carcinoma, sebaceous cell carcinoma, and melanoma. Rarely, Merkel cell carcinoma, metastasis, and lymphoma can be seen involving the eyelid.[10] Suspicious eyelid lesions should be referred to subspecialty care and may require excisional biopsy or complete surgical excision, ideally with Mohs, rapid fixation, or frozen section methods.[11,12]

Basal Cell Carcinoma (BCC)

BCC is the most common eyelid neoplasm. Risk factors for BCC include fair skin, sun exposure, and a history of smoking. The most common location is the lower eyelid. BCC has a predilection for individuals of European descent, specifically those of Celtic ancestry. Orbital invasion occurs in <5% of BCC.

Squamous Cell Carcinoma (SCC)

SCC is the second most common eyelid neoplasm and, like basal cell, it occurs on the lower lid most often. SCC is more common in males and in patients with a history of previous skin cancer. Risk factors for SCC are UV light, human papilloma infection, and burns. SCC metastasizes through the lymphatic system, and can also infiltrate cranial nerves.

Sebaceous Cell Carcinoma

Sebaceous cell carcinomas most often occur in women in their 70s, and are often misdiagnosed as blepharoconjunctivitis or chalazia. This neoplasm is highly malignant and aggressive, and therefore warrants clinical awareness. The lesions are typically located on the upper lid, arise from sebaceous glands, and can produce a chalazion-like round or yellow exudative-like lesion. Suspicion for sebaceous cell carcinoma should arise when chalazia are not resolving, or in the presence of any chronic dysmorphic eyelid lesion.[13]

Awareness of eyelid neoplasms is imperative, as early recognition and subsequent treatment allow for better medical and cosmetic outcomes.

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Blepharitis

Blepharitis is a common inflammatory condition caused by accumulation of skin flora and debris at the base of the eyelashes. Nearly 50% of adult patients examined by ophthalmologists are reported to have blepharitis. The inflammatory reaction causes erythematous, edematous, and uncomfortable eyelids. Blepharitis may cause ineffective or altered tear film due to inflammation impacting the meibomian gland structures of the eyelids. Tear film alterations lead to secondary ocular surface irritation in addition to eyelid symptoms. Blepharitis can be subdivided into separate etiologies: infectious and noninfectious.

Staphylococcal blepharitis causes lash loss and/or lash misdirection, and is the most common type of infectious bacterial blepharitis. Demodex mites are another common infectious etiology. Microscopic evaluation of the lash base may reveal Demodex mites and larvae, which can lead to chronic blepharoconjunctivitis.

There are noninvasive, commercially available regimens effective in managing blepharitis and Demodex mites. The standard routine to control eyelid inflammation from blepharitis involves lid-hygiene regimens, including warm compresses, lid scrubs, and washes. If patients fail primary management, oral or topical medical management may be warranted.[14]

Conjunctiva

The conjunctiva is a translucent mucous membrane overlying the sclera and inner part of the eyelids. The conjunctiva is composed of an epithelium, goblet cells, blood vessels, and lymphatic channels. Conjunctivitis is inflammation and hyperemia of the conjunctiva. Conjunctivitis occurs because of conjunctival vessel dilation. Conjunctivitis can be infectious, due to bacteria or viruses, or can be inflammatory, from allergens, chemicals, or physical irritation.

Conjunctivitis

Bacterial

Bacterial conjunctivitis is subdivided into subacute, acute, and chronic, depending on duration, and is less common in the elderly population than viral conjunctivitis. Bacteria can be transmitted to the conjunctiva from contaminated fingers or fomites, and is prevalent in crowded living or social conditions. In rare cases, bacterial conjunctivitis may spread from nasal or sinus mucosal colonization. Elderly patients are more likely to develop bacterial conjunctivitis if immunosuppressed, following trauma, in the presence of atypical or altered eyelid anatomy, or with compromised tear production.

The most common pathogens causing bacterial conjunctivitis in adults are staphylococcal species, streptococcus pneumoniae, and haemophilus.

Bacterial conjunctivitis presents similarly to other forms of conjunctivitis, with redness, irritation, tearing, and light sensitivity; however, the hallmark of bacterial conjunctivitis is copious, mucopurulent discharge. Bacterial conjunctivitis may be unilateral or bilateral. The duration of bacterial conjunctivitis is usually 7–10 days and is often self-limited.

Bacterial conjunctivitis can often be managed conservatively, but topical antibiotic drops may decrease the duration of symptoms and speed the eradication of microorganisms. Common antibiotic drops used for bacterial conjunctivitis include topical fluoroquinolones or polymyxin combination drops. Patients should be encouraged to adhere to strict hygiene precautions, including frequent hand-washing, and should remain out of work until the infection clears. Cold compresses and artificial tears may be helpful for irritation.

Bacterial conjunctivitis that does not resolve by 7–10 days may warrant culture and further subspecialty management.

Viral

Viral conjunctivitis manifests as follicular conjunctivitis, which can be acute or chronic. Viral conjunctivitis is highly contagious and is transmitted via endogenous spread from an underlying viral illness, or via direct contact with contaminated medical instruments, infected personal items, and through swimming pool water. Incubation periods are typically 5–11 days and patients are deemed contagious for up to 2 weeks.

Viral conjunctivitis may present in patients with concurrent or recent upper respiratory infections, and the hallmark symptoms include red, infected eyes and watery discharge, and may include mild/moderate itchiness. Viral conjunctivitis may be part of clinical syndromes such as epidemic keratoconjunctivitis and pharyngoconjunctival fever. Epidemic keratoconjunctivitis most commonly presents with severe conjunctivitis, watery discharge, hyperemia, chemosis, and prominent lymphadenopathy. Pharyngoconjunctival fever is characterized by abrupt onset of high fever, pharyngitis, bilateral conjunctivitis, and preauricular lymph node enlargement.

The most common viral pathogen in viral conjunctivitis is adenoviral conjunctivitis, which is highly contagious

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and most prevalent in the summer months. Other viral etiologies include herpetic or cytomegalovirus conjunctivitis. Herpes simplex can create a primary unilateral conjunctivitis and may account for 5% of acute conjunctivitis cases in adults.

Allergic

Allergic conjunctivitis occurs because of allergen exposure. Allergic conjunctivitis can be transient, episodic, or persistent. Allergic conjunctivitis typically manifests as acute, predominantly itchy, red eyes, which may be accompanied by chemosis of the conjunctiva or periocular area. Allergic conjunctivitis is classified as a type 1 hypersensitivity reaction involving mast cell degranulation.

Allergic patients may benefit from alteration of their environment, minimized contact with and exposure to their allergic triggers, cold compresses, and mast cell stabilizer-antihistamine topical ophthalmic medications. Persistent or resistant atopy may require subspecialty care to prevent sequelae of allergic conjunctivitis, such as corneal ulceration and conjunctival or corneal scarring.

Chronic Conjunctivitis

Chronic conjunctivitis is defined as conjunctival reaction for more than 4 weeks. The differential for chronic conjunctivitis includes chlamydia/trachoma, molluscum contagiosum, conjunctival malignancy, ocular cicatricial pemphigoid, sebaceous cell carcinoma, eyelid malpositions, and irritation from medicated drops, such as glaucoma medications. Often, chronic conjunctivitis warrants culture and/or biopsy. If a neoplastic or cicatricial systemic process is suspected, a conjunctival biopsy should be performed.

Dry Eye

Dry eye is one of the most common yet underdiagnosed ocular surface conditions. Dry eye syndrome is multifactorial in etiology, and manifests as symptoms of intermittent blurry vision, foreign body sensation, stickiness, and sandy or gritty feeling of the eyes. Dry eye has been identified in nearly 60% of established ophthalmology patients, and the prevalence is higher in the elderly. Underlying autoimmune or inflammatory conditions are known to contribute to dry eye syndrome, especially rheumatologic disease and Sjogren's syndrome. Many systemic medications have also been implicated in exacerbating dry eye disease, such as beta-blockers and selective serotonin reuptake inhibitors (SSRIs).

Dry eye management can be initiated by a primary care provider with artificial tears, but patients with persistent symptoms may benefit from subspecialty evaluation and care. Given the multitude of etiologies for dry eye syndrome, a multidisciplinary approach may be required to accurately diagnose and treat underlying systemic conditions.[15]

Cornea

The cornea is the clear, avascular central surface of the eye, and is primarily responsible for allowing the unimpeded passage of light. It provides a significant amount of optical power to the eye, allowing light to be focused through the pupil and ultimately onto the retina. The cornea is made of five layers: epithelium, Bowman's layer, stroma, Descemet's membrane, and endothelium. The cornea is densely populated with nerves, and even a small abrasion of the corneal epithelium can result in significant pain, photophobia, and tearing. Alterations to the structure of the cornea often manifest with significant visual changes.[16]

Corneal Abrasions and Trauma

Corneal abrasions disrupt the corneal surface epithelium. The cornea is highly innervated, and even minor abrasions can result in significant ocular pain, photophobia, and tearing. Alterations of the surface epithelium make the eye susceptible to superimposed infections.

Patients with significant ocular trauma with decreased visual acuity or concerning mechanism of injury should be evaluated emergently to rule out lacerations or ruptured globes. Abrasions are diagnosed by fluorescein staining and slit-lamp examination, which is within the scope of practice of emergency room physicians, optometrists, and ophthalmologists.

Corneal abrasion management depends on the mechanism of injury, but can often be managed symptomatically with topical lubrication, and may require antibiotic prophylaxis via ophthalmic antibiotic drops.

Infectious Keratitis

Herpetic eye disease is extremely common in the elderly. Herpes simplex I and II (HSV I and HSV II) in addition to varicella zoster (VZV) cause ocular manifestations with very serious complications and sequelae.

Herpetic keratitis is an ocular manifestation of HSV I or HSV II. It is reported that HSV is present in the trigeminal ganglion on autopsy in 100% of patients older than 65 years of age, which supports the approximate

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60,000 cases of herpetic keratitis seen annually. The presentation of herpetic keratitis is typically unilateral, painful, red eye that occurs in the absence of trauma or other precipitating events. This disease can be recurrent and often requires topical and systemic therapy, in addition to long-term systemic suppression with antiviral medications. The sequelae can lead to neurotrophic corneas, which can ultimately result in ulceration and corneal decompensation.[17]

Herpes zoster ophthalmicus (HZO) is a manifestation of varicella zoster virus, or shingles, on or around the eye, involving dermatomes of the trigeminal nerve. Varicella zoster virus occurs because of reactivation from dormancy in the dorsal root ganglion, and is seen in immunocompromised or immunosuppressed patients, with very high incidence and increasing severity in patients over 60. HZO can cause lesions on the cornea, conjunctiva, and eyelid skin and can also cause viral damage to the retina and structures of the posterior eye. One clinical indicator suggestive of ocular involvement is the Hutchinson sign – vesicles on the tip, side, or base of the nose. The Hutchinson sign represents nasociliary nerve involvement and is highly predictive of ophthalmic involvement, including corneal lesions, decreased corneal sensation, and ocular inflammation. Patients with suspicion for HZO should be referred to an ophthalmologist urgently for complete dilated fundus examination.[18]

Presbyopia

Presbyopia is a natural, age-related process of irreversible loss of accommodation of the natural lens of the eye, which leads to the inability to focus on near objects. The natural crystalline lens of the eye has innate accommodative ability, which changes the refractive power to allow eyes to focus at a distance and near.

Until the age of 20, the natural crystalline lens has 7–10 diopters of accommodative power to focus near, but by the age of 50, the lens can only accommodate up to 0.50 diopters, which necessitates reading glasses or visual aids for near work. Over time, patients often find they need increased power to continue to see well for near tasks and may occasionally need to adjust their prescription to maintain adequate near vision.

The exact mechanism of presbyopia remains unknown; however, many patients do well with over-the-counter reading glasses, bifocal glasses, or progressive lenses. There are refractive surgery options for patients who desire independence from glasses.[19]

Cataract

Cataracts, clouding of the natural crystalline lens of the eye, are the number one cause of preventable blindness worldwide, and disproportionately affect the elderly.[20] The crystalline lens of the eye is responsible for providing refractive power to the eye. As we age, the clear lens becomes opacified. Cataract formation is accelerated by diabetes, smoking, UV exposure, trauma, previous eye surgery, and genetic predisposition.

A typical age-related cataract develops gradually over time, and the clouding of the lens results in progressive visual symptoms including decreased acuity, difficulty reading in dim light, glare, and halos around lights. There are no medical therapies that slow cataract development.

Cataract surgery is an elective outpatient procedure, and is one of the most common elective surgeries performed worldwide. Cataract surgery is the process of removing the natural crystalline lens and replacing it with a synthetic plastic intraocular lens that replicates the natural lens of the eye. Presently, the most common technique for cataract extraction is via ultrasound phacoemulsification.[21,22]

Given the prevalence of cataract surgery, providers may encounter patients during their postoperative course. While most patients have an unremarkable postoperative course, the most serious yet rare complication of cataract surgery is bacterial endophthalmitis. Bacterial endophthalmitis occurs following cataract surgery in 0.04–0.2% of cases.[23,24] Patients with bacterial endophthalmitis typically present within 3–5 days but up to 2 weeks after cataract surgery, and have new onset of pain, redness, and decreased vision. Bacterial endophthalmitis is an emergency that should be referred immediately for ophthalmologic care.

Glaucoma

Glaucoma is defined as optic neuropathy with corresponding visual field loss. Glaucoma is most often attributed to elevated intraocular pressure; however, glaucomatous optic neuropathy may occur with normal intraocular pressure, and can occur with or without pain. Glaucoma is the second most common cause of blindness worldwide.

Diagnosis of glaucoma involves serial examinations to evaluate the structure and function of the optic nerve, including direct slit-lamp biomicroscopy, gonioscopy, visual field testing, and ultra-structural assessment of the optic nerve with optical coherence technology

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(OCT). Undiagnosed, untreated, or undertreated glaucoma can lead to irreversible vision loss.

Patients with suspicious-appearing optic nerves and risk factors for glaucoma without optic neuropathy are termed "glaucoma suspects." Glaucoma suspects may be initiated on topical therapy to reduce intraocular pressure to prevent future visual field loss.[25,26]

Primary Open-Angle Glaucoma

Primary open-angle glaucoma (POAG) is the most common type of glaucoma, and disproportionately impacts the elderly population. Patients with POAG develop optic nerve damage due to chronically elevated intraocular pressure. Risk factors for POAG include advancing age, African ancestry, myopia, and medical conditions including diabetes and hypertension. POAG is an insidious disease, and patients often have optic nerve damage and concurrent visual field loss before becoming symptomatic.

Patients with glaucomatous optic nerve damage are initiated on topical eye medications, and may also require laser and/or surgical intervention including trabeculectomy, or tube surgeries, to attempt to lower intraocular pressure. POAG requires routine lifelong ophthalmic care.

Angle-Closure Glaucoma

Acute angle closure is a less common subtype of glaucoma, but requires emergency ophthalmic care. Acute angle-closure glaucoma occurs when the anatomic angle between the cornea and iris becomes blocked, which acutely inhibits the outflow of aqueous humor from the eye. The blocked outflow creates a dramatic, sudden rise in intraocular pressure and induces acute pain, severe headache, and notable vision changes. The sudden elevated intraocular pressure can create optic nerve damage, and may compromise the visual field.

Angle closure can occur because of the anatomic structure of the eye, such as anatomical narrow angles related to hyperopia, advanced cataracts, or as a result of systemic medications that are commonly used in the aging population, such as antidepressants (amitriptyline, imipramine, paroxetine, fluoxetine, venlafaxine, citalopram, escitalopram), anti-Parkinsonians (levodopa), and anticonvulsants (topiramate). Treatment focuses on addressing the underlying etiology and rapid administration of intraocular pressure-lowering medications. Often, laser surgery such as an iridectomy may be performed to create a new pathway for aqueous humor to drain out of the eye.

Glaucoma Medications

Given the prevalence of glaucoma, practitioners taking care of the elderly should be familiar with the medication classes and known adverse effects of glaucoma medications. Topical glaucoma medications are formulated to lower intraocular pressure; however, despite topical application, they may be systemically absorbed. Table 42.1 highlights common and/or significant adverse effects.

Vitreous, Retina, and Choroidal Disorders

The retina is a neurosensory membrane found along the posterior internal aspect of the globe. The retinal substructure consists of many layers of neuronal cells, axons, and photoreceptors, which are responsible for converting light into electrical impulses for interpretation by the brain. The retina has a rich vascular supply from the internal carotid artery and is drained by a network of ophthalmic veins.

Posterior Vitreous Detachment

The vitreous is a gel-like substance that contributes to globe structure, and also has optical properties. The vitreous is adherent to the neurosensory retina at the ora serrata, along the optic disc and along retinal vessels. The natural aging process induces a liquification of the gel, and subsequent vitreous syneresis. This normal aging process generates density changes throughout the vitreous. Ultimately, the liquified vitreous separates from the adherent sites along the retina, creating a posterior vitreous detachment (PVD). PVD occurs commonly, typically without complication. Many patients are asymptomatic during or after PVD; however, some may experience new, significant floaters and/or photopsias. When PVD occurs in areas of firm vitreous adhesion, there is a small risk of associated retinal tear, with or without associated retinal detachment.

Retinal tears secondary to PVD occur in approximately 3.7–5.2% of symptomatic patients.[27] Patients reporting "flashing lights" or "lightning bolts," new or worsening floaters, and visual field defects described commonly as a "curtain" or "veil" should have dilated fundoscopy performed. The incidence of PVD increases with age, with the average age of onset typically between 60 and 70 years old, but often earlier in myopic eyes.

PVD is considered an ongoing process that is hypothesized to occur over 2–3 months, therefore patients often require interval assessment to ensure the retina is without tears or detachment.[28]

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Table 42.1 Glaucoma medications

Class	Mechanism	Common trade name(s)	Adverse effects
Topical beta-blockers /beta-adrenergic antagonists	Suppress aqueous humor production	Betagan, Timoptic, Timoptic XE, Betopic	Bradycardia, arrhythmia, heart failure exacerbation, heart block, syncope, bronchospasm, depression, weakness, fatigue, hallucination, impotence, elevation of blood cholesterol levels, reduced glucose tolerance, masking of hypoglycemia signs
Adrenergic agonists	Suppress aqueous humor secretion, increase uveoscleral outflow	Brimonidine	Ocular irritation, follicular conjunctivitis, corneal surface irregularities Dry mouth
Prostaglandin analogs	Increase uveoscleral outflow	Latanoprost Travoprost Bimatoprost	Periocular skin hyperpigmentation Hypertrichosis Iris pigment darkening
Topical carbonic anhydrase inhibitors	Suppress aqueous production	Dorzolamide	Ocular surface irritation, metallic taste
Systemic carbonic anhydrase inhibitors	Suppress aqueous production	Diamox	Metallic taste, paresthesias, malaise, metabolic acidosis, renal lithiasis, electrolyte imbalance, diarrhea, pancytopenia

Retinal Detachment

The neurosensory retina may become detached from the retinal pigment epithelium in a process called retinal detachment. Retinal detachments are sight-threatening and require immediate ophthalmologic intervention.

Retinal detachments present with a visual distortion, photopsias described as "flashing lights" or "lightning bolts," new or worsening floaters, and visual field defects described commonly as a "curtain" or "veil." Retinal detachments typically occur as a result of PVD with subsequent retinal tear, or may occur after trauma or intraocular surgery. Patients suspected of retinal detachment should be referred to ophthalmology for a dilated fundus examination. Retinal detachments require surgical repair to mechanically facilitate the reattachment of the neurosensory retina.

Retinal detachment repair options include office-based procedures such as laser barricade, which is best for localized, smaller retinal detachments, or pneumatic retinopexy, gas, and cryotherapy tamponade of a retinal break(s), best for superior detachments in phakic eyes. Additional procedures for retinal detachment include pars plana vitrectomy, removal of the vitreous coupled with laser and/or gas or oil fill, or scleral buckle, which is a supportive band placed around the eye to facilitate reattachment of the neurosensory retina.

Surgical intervention for retinal detachments may entail long postoperative recovery, and vision may not return to baseline, based on the extent of detachment, time to surgical repair, and postoperative complications.[29,30]

Diabetic Retinopathy

Diabetic retinopathy describes a pattern of retinal damage that occurs from chronic elevated blood glucose. Chronic hyperglycemia damages pericytes and vascular endothelium of the retinal vasculature, resulting in ischemic retinal damage. The incidence of diabetic retinopathy increases as disease years progress. Type 1 and type 2 diabetics are both at risk for diabetic retinopathy. After 20 years of diabetes, approximately 99% of type 1 diabetics and 60% of type 2 diabetics will have retinopathy.[31]

Diabetic retinopathy is classified as either non-proliferative or proliferative. Non-proliferative diabetic retinopathy is a less severe stage of disease, and is characterized by dot blot hemorrhages, microaneurysms, cotton wool spots, and venous beading. Patients may also have diabetic macular edema. The chronic vascular damage and resultant ischemia of retinal tissue causes the production of vascular endothelial growth factor (VEGF), which generates new, abnormal blood vessels called neovascularization. Proliferative diabetic retinopathy is characterized by aberrant neovascularization of the retina, vitreous, optic disc, and iris. Proliferative diabetic retinopathy has vision-threatening consequences,

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including vitreous hemorrhage, tractional retinal detachments, and neovascular glaucoma.

From an ocular health standpoint, all patients with diabetes are encouraged to maintain strict blood glucose and blood pressure control, commensurate with their overall health and goals of care. Ophthalmic interventions are aimed at preventing or treating the complications of proliferative diabetic retinopathy. Current treatment modalities include laser, intravitreal injections of anti-VEGF or steroid medications, and surgery.

Retinopathy can be asymptomatic, and current interventions are aimed at preventing vision loss, therefore it is imperative that diabetics incorporate ophthalmic evaluations into their routine medical care. All diabetics should have a complete eye exam at least annually, including dilated fundoscopy.[32,33]

Retinal Vascular Events

Retinal vascular events cause ischemia to neurosensory retinal tissue. The retinal arterial system is supplied by the central retinal artery, a branch of the ophthalmic artery, which supplies the optic nerve and the inner layers of the neurosensory retina. Venous drainage is via the ophthalmic vein.

Retinal artery occlusions (RAO). These are obstructions of retinal blood flow due to embolism, thrombus, vasculitis, trauma, or vasospasm. RAO are subdivided into central retinal artery occlusions (CRAO) and branch retinal artery occlusions (BRAO). Patients may present with precedent transient vision loss, called amaurosis fugax, or sudden, painless, persistent vision loss. Diagnosis is confirmed via direct ophthalmoscopy and ancillary testing such as fluorescein angiography and electroretinography.

Risk factors for RAO are similar to ischemic intracerebral stroke, and include advanced age, smoking, hypertension, diabetes, hyperlipidemia, cardiovascular disease, underlying coagulopathy, and male gender. In the elderly, the most common etiology of RAO is embolic phenomenon originating from carotid atherosclerotic disease. CRAO may also occur in the setting of giant cell arteritis (GCA), and a thorough review of systems should be elicited in all cases of CRAO to determine if there are systemic signs and symptoms consistent with GCA (see GCA section).

Retinal artery occlusions are an ophthalmic emergency and are managed as expeditiously as an acute cerebral stroke. Patients identified as having RAO are referred immediately to stroke centers.

Unfortunately, there are no evidence-based therapies that have demonstrated efficacy in improving visual outcomes in RAO. Patients with RAO require frequent ophthalmic follow-up to identify and manage sequelae, including neovascular complications, which can induce further vision loss and pain.[34,35]

Retinal vein occlusions (RVO). These are ischemic events from thrombus formation, resulting in impeded venous drainage out of the eye. There are two types of RVO: central RVO (CRVO), which occurs at the central retinal vein near the lamina cribrosa, and branch RVO (BRVO), where arterial compression onto veins creates thrombus formation and prevents antegrade venous blood flow.

Patients with RVO present with monocular acute central or peripheral vision loss. Risk factors for RVO include advanced age, hypertension, atherosclerosis, diabetes, hyperlipidemia, hypercoagulability, and elevated intraocular pressure.

There are no available treatments to reverse the ischemic damage from RVO; however, there are therapies to treat sequelae of neovascularization and macular edema. Patients are treated with anti-VEGF intravitreal injections, and often may require intraocular steroids or laser therapy.[36]

Patients with RAO and RVO are encouraged to work with their primary care physicians to optimize modifiable risk factors such as blood pressure, hyperlipidemia, smoking status, diabetes, cardiovascular disease, or hypercoagulable states.

Intraocular vascular events can be visually catastrophic. The visual consequences range from mild visual impairment to blindness, and both RAO and RVO lead to ischemic complications such as macular edema, aberrant blood vessel regeneration, and neovascularization of the anterior chamber, retina, or optic nerve. Anomalous blood vessels may cause neovascular glaucoma and different degrees of vitreous or retinal hemorrhages, ultimately resulting in further compromise of vision and painful, blind eyes.

Age-Related Macular Degeneration (ARMD)

ARMD is an acquired retinal degeneration resulting in significant, irreversible central vision loss. Studies report that the prevalence of ARMD worldwide is 1.6%, with a sharp increase in those 75 years or older. According to the Framingham Eye Study, there is a threefold increase

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in ARMD in patients over 75 years old compared to 65–74 years of age. The prevalence of ARMD continues to increase with longevity, and it is estimated that within 25 years the disease burden will increase to impact 288 million individuals worldwide.[37]

There are dry and wet forms of ARMD. The mechanism of vision loss in ARMD is multifactorial. In a healthy retina, photoreceptors and retinal pigment epithelium (RPE) work in synchrony to facilitate visual phototransduction and to maintain the metabolic environment of the neurosensory retina. ARMD results in damage to photoreceptors and RPE, which results in accumulation of metabolic waste products under the RPE, compromising the structure and function of the neurosensory retina. The metabolic waste products are called drusen, which are a hallmark feature of dry ARMD. As ARMD progresses, drusen and atrophic areas of retina increase in size, resulting in large areas of damaged neurosensory retina, called geographic atrophy. This progressive process ultimately deprives the macula of functional central vision because of scarring and loss of viable tissue structure.[38,39]

Wet ARMD results from pathological choroidal neovascularization (CNV) that penetrates retinal tissue. Neovascular changes result in hemorrhage and exudation that damage the neurosensory retina, leading to distortion of tissue architecture, and causing further scarring and retinal atrophy.[40]

Risk factors for ARMD include age, race, smoking, and genetic susceptibility. Age is the strongest demographic risk factor associated with the development of ARMD.[41] The Baltimore Eye Study reported that individuals of Caucasian ancestry are four times more likely to develop any form of AMD, including progression to wet AMD, as compared with individuals of other ancestry. ARMD is prevalent in those who have smoked, with a statistically significant increase in patients with a 10-pack-year tobacco smoking history. Current smokers are twice as likely to have ARMD-related vision loss when compared to non- or ex-smokers who have quit over 20 years prior.[42]

The genetics of ARMD are of ongoing interest, with at least 40 genes identified that may contribute to the development of AMD. Mutations and polymorphisms in the complement system likely result in immune dysfunction that contributes to the pathogenesis of ARMD, specifically complement factor H (CFH). Individuals homozygous for complement factor H (CFH) were found to have 7.4-fold risk in the development of ARMD.[43] In addition to environmental and genetic factors, epigenetic mechanisms, such as microRNA regulation, are hypothesized to further influence the development and severity of this multifactorial disease.

Treatment for ARMD is limited, and currently exists for wet ARMD only. The neovascular process of wet ARMD is mediated by VEGF, a potent angiogenic factor. Patients with wet ARMD may benefit from intravitreal injections with anti-VEGF. Intravitreal anti-VEGF therapy is effective at treating active CNV. Research is ongoing to attempt to modify genetic factors and immune processes, to mitigate or halt the development of dry ARMD.[44,45]

Charles Bonnet Syndrome

Charles Bonnet syndrome (CBS) is a condition characterized by visual hallucinations reported in patients with significant vision loss due to known ocular pathology, with otherwise good baseline cognition. CBS is reported in as many as 15% of elderly patients with low vision. It is important for primary care physicians to be aware that visual hallucinations in the setting of profound vision loss may not be due to cognitive alterations, dementia, or psychosis. Reassurance, eye closure, social support, and limiting social isolation have been found to be helpful at improving visual hallucinations in this condition.[46]

Optic Nerve Disorders

Neuro-ophthalmic diseases more prevalent in the elderly include giant cell arteritis (GCA) and non-arteritic ischemic optic neuropathy (NAION).

Giant cell arteritis (GCA). This is an autoimmune vasculitis that affects large- and medium-sized arteries. It is imperative to have high clinical suspicion for GCA because of permanent, progressive visual and systemic implications, including myocardial infarction, aortic aneurysm, stroke, and death.[47]

GCA is the most prevalent autoimmune vasculitis in the elderly and is characterized by granulomatous inflammation affecting medium-sized cranial arteries, and larger systemic vasculature.[48] Patients with GCA may present with transient or sudden monocular vision loss, diplopia, cranial nerve palsies, headache, scalp or temple tenderness, jaw claudication, weight loss, proximal muscle weakness, malaise, and fatigue. GCA may present concurrently with polymyalgia rheumatica.

In addition to the signs and symptoms of GCA, laboratory analysis with elevated inflammatory markers – elevated ESR, CRP, and platelets – is suggestive of GCA.

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Definitive diagnosis is confirmed by temporal artery biopsy; however, the presumptive diagnosis is clinical, and treatment should not be delayed pending biopsy. It is generally accepted that temporal artery biopsy can be performed within 2 weeks of initiating steroid therapy to maintain sensitivity.

Patients with suspicion for GCA must be treated rapidly and aggressively with high-dose intravenous steroids because the granulomatous inflammation may impact the vasculature supplying the fellow eye, leading to sequential, permanent vision loss. Vision loss from GCA is most often due to arteritic anterior ischemic optic neuropathy (AAION), but may also be due to central retinal artery occlusion. Immediate ophthalmic examination is warranted in any patients with spontaneous, painless vision loss, especially if they have systemic signs and symptoms suspicious for GCA.[49]

Non-arteritic ischemic optic neuropathy (NAION). This is sudden painless monocular vision loss due to optic nerve infarction. NAION is the most common cause of acute optic neuropathy in patients over the age of 50.

It is estimated that NAION occurs at a frequency of 1/10,000 and has a predilection for elderly women of Northern European ancestry.[50] Typical presentation is acute, painless vision loss, often first noted when awakening from sleep. The mechanism of NAION remains disputed but is thought to involve reduced perfusion or an ischemic event to the anterior portion of the optic nerve. The mechanism of ischemia in NAION is different from that of GCA, which is vasculitic, and patients presenting with painless vision loss and systemic signs and symptoms suspicious for vasculitis warrant a workup for GCA, as management and sequelae are different.

Risk factors for NAION include obesity, obstructive sleep apnea, diabetes, hyperlipidemia, and dysregulation of blood pressure. In addition to sudden painless vision loss, patients with NAION often manifest a stereotypical visual field defect of altitudinal or arcuate defect, and also have signs of optic neuropathy including decreased color vision and a relative afferent pupillary defect. On examination, patients have unilateral optic disc edema, with peripapillary retinal hemorrhages that ultimately progress to optic nerve pallor over the course of weeks to months after infarction. Patients diagnosed with NAION have variable visual outcomes. There is no definitive treatment for NAION; however, risk factor reduction is imperative to minimize the risk of fellow eye optic neuropathy.[51]

Low Vision

Low vision is defined by the American Academy of Ophthalmology (AAO) as significant visual field loss or best spectacle corrected visual acuity of 20/70 or worse in the better eye.[52] Patients with low vision may benefit from the expertise of low-vision specialists and low-vision rehabilitation services.[53] Low-vision specialists offer tools and resources to aid in better vision so that individuals with low vision can continue to participate in their activities of daily living and hobbies. Modern technological advancements such as computers with magnification features, enhanced lighting, large print resources, talking devices, and techniques on environment optimization help patients function as optimally as possible. The elderly population with visual impairment is at additional risk of falls, and therefore may benefit from social services and safety accommodations in order to facilitate functional, safe living spaces. Accommodations include enhanced lighting, handrails/grab bars, safe transition spaces, including elimination of area rugs, and audio aids.

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Chapter 43

Geriatric Otolaryngology

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Introduction

Many common complaints that present to a community otolaryngologist office, including hearing loss, vertigo, dysphagia, and dysphonia, become increasingly prevalent with age and can profoundly affect the functioning of older adults. This chapter discusses the expected anatomic and physiologic changes seen with aging as well as the pathophysiology, presentation, and treatment of common head and neck conditions that geriatricians will encounter.

Ear

Outer Ear

Age-Related Changes

The external auditory canal changes with age. The lateral cartilaginous ear canal weakens, reducing structural rigidity, and outgrowths in the medial bony ear canal (e.g., benign exostoses) can further narrow the canal, leading to cerumen impaction and difficulty fitting a hearing aid.[1,2] Atrophy of the surface epithelium and decreased apocrine gland function of the ear canal predisposes to dryness, scaling, and trauma.

Common Pathologies

Cerumen impaction can be a consequence of age-related changes in the external auditory canal.[3–5] Cerumen protects the ear with antimicrobial secretions and captures foreign particles, which are eliminated from the ear canal with epithelial migration. In older adults, apocrine glands atrophy, decreasing the watery component of cerumen, producing cerumen that tends to be drier, harder, and less likely to be expelled.[2,6] Hearing aids and the thicker, fuller tragal hairs associated with aging can further impede the canal's normal "self-cleaning" mechanism.[6] Every patient should be regularly evaluated for impaction at clinic visits, particularly if they use hearing aids.[7] Cerumen can be removed with curettage, ideally under binocular microscopy. Other options include suction, topical cerumenolytics, and/or irrigation with warm water. However, irrigation should be avoided in patients who are diabetic, immunocompromised, or those with perforated tympanic membranes because of the risk of acute otitis externa or media.[8] Preventive measures include counseling against cotton swab use and encouraging the regular use of emollients like mineral oil or glycerin.[7]

Squamous cell carcinoma, basal cell carcinoma, and actinic keratoses can affect the pinna.[9] With age, skin becomes atrophic because of dermal thinning, disorganized collagen deposition, and decreased elastic tissues and mitotic divisions. Free radicals produced secondary to ultraviolet light exposure promote crosslinking of fibrous proteins and DNA. Risk factors for skin cancer include age, sun exposure, type I–II skin types on the Fitzpatrick scale, and a history of multiple sunburns.[10,11] Squamous cell carcinoma may present as a red patch, often preceded by actinic keratosis. Basal cell carcinoma can present as an elevated, pink, waxy nodular lesion with a clearly demarcated capillary bed. It is paramount to maintain a high level of suspicion for malignancy and refer for biopsy for any suspicious lesion, especially when accompanied by persistent otalgia, granulation tissue, recurrent otitis externa, bleeding, hearing loss, or facial nerve weakness.[12]

Acute otitis externa (AOE) is a bacterial infection of the external auditory canal often precipitated by a break in the skin secondary to water exposure or manipulation, such as with cotton swab use.[13] The setting of a warm, moist ear canal encourages bacterial growth of common pathogens like *Pseudomonas aeruginosa* and *Staphylococcus aureus*. Presenting symptoms include pruritus, purulent discharge, severe otalgia, and, at times, conductive hearing loss due to canal narrowing.[13,14] On physical exam, pulling the pinna elicits significant pain, distinguishing AOE from otitis media. Topical treatment with antiseptic or antibiotic

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eardrops, with or without steroids, is first-line treatment. Although several meta-analyses of randomized controlled trials have not demonstrated clinically significant differences between topical treatments, ciprofloxacin and steroid preparation is a common, although expensive, treatment.[14,15] Cheaper options include a neomycin, polymyxin, and hydrocortisone preparation or antiseptic treatment with acetic acid 2.0% with or without hydrocortisone 1.0%. If soft-tissue swelling limits drug delivery, eardrops may be administered with a wick, along with pain medication and frequent debridement.[15] Systemic antibiotic treatment, with coverage of *Pseudomonas aeruginosa* and *Staphylococcus aureus*, is reserved as adjunctive treatment if the patient is diabetic, immunocompromised, the infection has spread beyond the ear canal, or topical therapy is unable to be delivered effectively because of significant inflammation of the ear canal.[15] If significant debris, impaction, or failure of initial antibiotic course occurs, referral to an otolaryngologist is needed.

Malignant otitis externa (MOE) is a feared complication of AOE and is a rare, potentially lethal infection of the soft tissue and bone of the external auditory canal and skull base, most often by *Pseudomonas aeruginosa.*[13,16] Classically, this pathology occurs in older, diabetic, and immunocompromised patients. Symptoms can include severe otalgia, purulent otorrhea, and, occasionally, cranial neuropathies, particularly facial paralysis.[13] On physical exam, granulation tissue is seen along the ear canal floor. Long-term intravenous antibiotics and correction of immunosuppression (e.g., glucose control in diabetics) are the primary treatment, while surgery is reserved for debridement. In a recent study exploring characteristics of MOE patients, older patients were more likely to experience in-hospital complications and mortality than their younger counterparts.[17] Suspicion for malignant otitis externa requires emergent referral to an otolaryngologist.

Herpes zoster oticus is a vesicular rash of the external ear canal accompanied by severe pain and hearing loss; when accompanied by facial paralysis, it is called Ramsay Hunt syndrome.[16] Caused by reactivation of latent herpes virus, the incidence increases with age because of the decline of cellular immunity.[18] With a worse prognosis than Bell's palsy in terms of return to normal facial function, early treatment with steroids and antiviral medication and an urgent appointment with an otolaryngologist are essential.[19–21] Close monitoring, early audiometric testing, and documentation of the degree of facial paralysis are important in following disease progression and establishing prognosis.

Middle and Inner Ear

Age-Related Changes

The middle and inner ear undergo changes with age, including stiffening and thinning of the tympanic membrane and middle-ear ossicles, death of inner and outer cochlear hair cells, atherosclerotic changes of inner-ear vessels, and devascularization of the spiral ligament, which impact a patient's auditory and vestibular systems.[1,2,22]

Common Pathologies

While otitis media is a common pediatric problem, it may also occur in older adults, often coinciding with upper respiratory tract infections or allergic rhinitis.[23] Acute otitis media presents with rapid onset of otalgia and possibly fever, accompanied by fullness and erythema of the tympanic membrane and possible effusion. Treatment includes amoxicillin for uncomplicated otitis media, and amoxicillin/clavulanate for diabetic or immunocompromised patients.[24] In complicated cases, myringotomy may be performed in the acute setting by an otolaryngologist in the office to provide immediate symptomatic relief.[23] Referral to an otolaryngologist is needed if middle-ear effusions are persistent for ≥8 weeks, particularly if unilateral, to rule out a nasopharyngeal lesion.[25,26]

In older adults, acquired cholesteatomas may present as a chronically draining ear with purulent otorrhea, hearing loss, tinnitus, vertigo, or facial nerve palsy.[27,28] On otoscopic exam, there can be tympanic membrane retraction, white debris, granulation tissue, and ossicular erosion. Cholesteatomas are due to squamous epithelium in the middle ear, mastoid, or epitympanum. The pathophysiology of cholesteatomas is debated, but hypotheses involve epithelial migration or implantation. Cholesteatomas are benign masses, but may lead to significant morbidity secondary to erosive expansion and damage to the delicate middle- and inner-ear structures. A computed tomography (CT) temporal bone scan without contrast and audiogram are essential to early workup and can be ordered before referral to an otolaryngologist. Treatment by an otolaryngologist generally involves surgical removal. Untreated cholesteatomas can lead to serious extracranial and intracranial complications, including cerebrospinal fluid (CSF) otorrhea, labyrinthine fistulas, ossicular erosion, facial nerve paralysis, mastoiditis, and meningitis.

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Obstructive eustachian tube dysfunction is also fairly common among older adults, and the prevalence increases with age.[29] Eustachian tube dysfunction can present with aural fullness, otalgia, or ear popping.[30] On exam, the tympanic membrane may be retracted, and tympanogram can show negative middle-ear pressures or an effusion. There are many contributing factors including inhalant allergies, sinus disease, and enlarged adenoids. Medical management includes intranasal steroids. Surgical treatment for eustachian tube dysfunction has historically involved placement of tympanostomy tubes; however, a new procedure involving eustachian tube balloon dilation has shown efficacy and is gaining popularity.[31,32]

Auditory

Age-Related Hearing Loss

Treatment of age-related hearing loss generally involves evaluation by an audiologist, hearing instrument specialist, or hearing aid dispenser. Nearly all insurance programs will cover the cost of audiologic testing by an audiologist with a physician's referral but will not cover hearing aids, which average $4,700 for bilateral fitting and follow-up care.[33] Several need-based outreach programs provide access to discounted or free hearing aids through an application process that can be coordinated by the referring audiologist. Over-the-counter assistive listening devices and personal sound amplifiers (devices worn on or in the ear similar to a hearing aid but sold as a consumer electronic device not explicitly for the treatment of hearing loss) can also provide amplification and may be a good option, particularly when hearing aids are not possible because of limited time or financial resources. A number of high-quality, low-cost, over-the-counter options exist on the market and are equivalent in sound quality to traditional hearing aids.[34] Over the past several years, national-level efforts have translated to significant advances in the affordability and accessibility of hearing care as a public health priority.[33,35] These efforts have resulted in federal legislation passed in 2017 that allowed for the creation of over-the-counter hearing aids, which will be on the market by 2022.[36]

Several recent studies have shown associations between age-related hearing loss and several negative outcomes across almost all domains of aging, including dementia.[37–46] Given its prevalence and the severity of association, hearing loss is the largest potentially modifiable risk factor for dementia. However, studies are limited on whether hearing care can affect cognitive decline. The ACHIEVE trial is a National Institutes of Health (NIH)-funded multicenter randomized controlled trial that is examining the efficacy of hearing aids on slowing cognitive decline, with results expected in 2022.[47] Hearing care is increasingly viewed as an essential tool for aging well, and patients should be appropriately counseled regarding the potential benefits of this low-risk, nonpharmacological intervention as well as the limitations of current evidence.[48]

For older adults with more severe hearing loss who no longer obtain significant benefit from hearing aids, cochlear implantation is an option.[49,50] Advances in cochlear implant technology and surgical technique have expanded to include individuals with less severe hearing loss given increasing ability to preserve residual hearing.[51] Cochlear implantation among older adults is a routine outpatient procedure, typically <2 hours under anesthesia, with a safety profile comparable to implantation in younger adults and children and associated with significant gains in speech perception and quality of life, including among octogenarians and nonagenarians.[52] A cochlear implant directly stimulates the cochlear nerve, bypassing the impaired cochlea, and can restore access to sound and language for many adults with severe hearing loss. Cochlear implantation is covered by Medicare and most other private insurance programs. Single-sided deafness has recently been added as an indication for cochlear implantation because of studies proving significant benefits for patients.[53] If there is concern for a hearing loss where the patient is no longer receiving benefit from their hearing aid, referral to an otolaryngologist who performs cochlear implants is warranted.

Sudden Sensorineural Hearing Loss

Sudden sensorineural hearing loss can be distinguished from an acute conductive hearing loss by history, otoscopy, and the Weber and Rinne tests.[54–56] Sudden sensorineural hearing loss involves at least a 30-decibel hearing loss over three contiguous frequencies that occurs acutely.[54,57] Patients may notice pressure in the affected ear and transient vertigo. History and physical should include information about trauma, ear pain, drainage, and fever. Sensorineural loss is caused by pathology to the inner ear or auditory nerve from viral infection, tumor, or other etiology that requires immediate referral to an otolaryngologist for audiologic confirmation, workup, and treatment. Treatment within the first several days is critical for the efficacy of oral

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corticosteroids and intratympanic steroids in helping aid hearing recovery.[54,57]

Tinnitus

Tinnitus is the perception of sound in the absence of external noise.[58] The physical exam and history can differentiate between pulsatile and non-pulsatile tinnitus.[59] Pulsatile tinnitus presents with a rhythmic whooshing sound corresponding to the heartbeat, while non-pulsatile tinnitus is described as a constant ringing or buzzing sound. Persistent pulsatile tinnitus could reflect vascular or other pathologic etiologies and should be further worked up.[60] Initial workup may include listening for carotid bruits, obtaining a carotid ultrasound, and brain MRI with contrast to evaluate for intracranial masses. Referral to an otolaryngologist is indicated if pulsatile tinnitus is persistent. Non-pulsatile tinnitus is more common and often seen in the setting of hearing loss. It can be exacerbated by neck or back strain, stress, or temporomandibular joint problems.[61] If non-pulsatile tinnitus is sporadic and not bothersome, generally no further evaluation is needed. If it is occasionally bothersome, ambient stimulation, which includes using a sound generator when sleeping or having a radio on in the background, is a simple, low-cost approach to decrease attention directed at tinnitus. Tinnitus is commonly associated with hearing loss, and amplification can assist with symptom management. Tinnitus retraining therapies (e.g., Neuromonics) are an option for patients whose daily function is severely impacted but must be used daily for weeks to months with the aim of decreasing loudness and annoyance. If tinnitus worsens, is very bothersome, or is accompanied by vertigo or change in hearing, evaluation by an otolaryngologist is warranted with further referrals for specialized tinnitus management, including tinnitus retraining, biofeedback, and cognitive behavioral therapy.[61]

Balance

Age-Related Changes

Presbytasis is the loss of balance due to aging and is caused by changes in mobility, vision, proprioception, and vestibular function.[2] Recent evidence has shown an association between vestibular impairment and poor spatial cognition in older adults, and a spatial subtype of Alzheimer's disease may be made worse by age-related vestibular impairment.[62,63] Balance disorders are worsened by polypharmacy and decreased muscle tone of older adults, which increases the risk of falls and worsens health outcomes.[2,9]

Common Pathologies

Vertigo is the sensation of movement in the absence of movement. Benign paroxysmal positional vertigo (BPPV) is the most common cause of vertigo among older adults and is caused by the movement of otoconia into the semicircular canals.[64] This is in part due to aging contributing to the fragmentation of the otoconia, which are then more likely to be displaced.[65] The displacement leads to brief, episodic vertigo lasting 1 minute or less precipitated by head movements, most commonly turning over in bed. Performing the Dix-Hallpike maneuver and observing for nystagmus can diagnosis posterior semicircular canal BPPV.[64,66,67] A head-roll test can identify BPPV of the horizontal semicircular canal. Treatment involves maneuvers that shift the otoconia back into place, such as the Epley maneuver.[68] Older adults are more likely to experience a longer course and recurrent symptoms than younger patients.[65] If refractory to treatment, referral to an otolaryngologist is necessary.

Another common peripheral cause of vertigo, Ménière's disease, is characterized by spontaneous episodes of vertigo accompanied by fluctuating low-frequency hearing loss, tinnitus, and a sense of aural fullness.[69] The natural history of Ménière's disease can range from rare episodes to extended periods of vertigo.[64] Individual episodes can last hours. The disorder is thought to originate from excess endolymph within the inner ear that leaks into perilymph, which excites, then inhibits, cranial nerve VIII, and basal hair cells.[64,70] If Ménière's disease is suspected, referral to an otolaryngologist is needed. Initial treatment involves a low-salt diet and diuretics, with intratympanic steroids reserved for more refractory cases.[9,64,70]

Migrainous vertigo or vestibular migraine occurs in roughly 25% of migraine patients and is the second most common cause of episodic vertigo.[64,69,71] Symptoms can include vertigo and/or a sense of disequilibrium and can last from seconds to days to weeks at a time. Symptoms may occur in the absence of or prior to traditional headache symptoms. Pharmacologic treatment consists of migraine treatment with triptans, calcium channel blockers, and beta-blockers. Lifestyle measures include identification and elimination of dietary triggers, which can include a range of foods, such as processed meats, onions, and avocados.[72,73]

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Throat

Swallowing

Age-Related Changes

Swallowing is a synchronized series of muscle contractions that allows a food bolus to safely enter the stomach.[2,74] With aging, this process can become problematic because of weakness of masticatory, tongue, and facial muscles. Older patients experience longer swallowing phases that lead to an increased risk of aspiration. This is compounded by the high prevalence of neurologic disorders and polypharmacy in older adults.

Common Pathologies

Dysphagia is common in the older population and can be secondary to gastroesophageal reflux disease (GERD), Zenker's diverticulum, autoimmune disease, and neurologic diseases like cerebrovascular accidents.[9,74,75] Further functional tests include the modified barium swallow and flexible endoscopy while swallowing. Referral to a speech-language pathologist is often warranted when dysphagia persists, with further evaluation by an otolaryngologist as needed. Treatment of the underlying pathological process is necessary along with rehabilitation. For instance, Zenker's diverticulum is commonly treated with surgery by otolaryngologists.[76] Moreover, simple measures like a soft diet, swallowing exercises and maneuvers (e.g., chin tuck), and head-of-bed elevation can help tremendously.

Voice

Age-Related Changes

The voice undergoes age-related changes including calcification of the laryngeal cartilage, decreased muscular tone, joint stiffening, and bowing of the vocal folds.[77,78] These changes are compounded by age-related decreases in secretions, nerve conduction speed, and pulmonary function.

Common Pathologies

There are numerous etiologies of voice disorders, including GERD, chronic obstructive pulmonary disease (COPD), vocal cord paralysis, and neurologic disorders.[2,77] Treating the underlying cause and rehabilitation of the voice often involves a multidisciplinary approach with speech pathologists, otolaryngologists, neurologists, and pulmonologists. Treatment of vocal cord paralysis and presbylaryngis with injectables can be done in an office setting or the operating room. Furthermore, voice hygiene with hydration and avoidance of tobacco are essential. Warning signs for neoplasm include progressive hoarseness, dysphagia, hemoptysis, weight loss, and a palpable mass, particularly in a smoker.[77] Any concern for malignancy requires urgent referral to an otolaryngologist for further workup.

Nasal Cavity and Sinus

Age-Related Changes

Aging of the nasal cavity and sinuses involves decreased smell and taste secondary to loss of olfactory epithelium.[2,9] Drugs, radiation, trauma, and infections can compound this loss. Loss of moisture due to atrophy of glandular tissue can lead to excessive nasal dryness and increase the risk of epistaxis.[79] This dryness is exacerbated by common medications prescribed to older adults. Lower mucociliary clearance and immunosenescence increase the risk of infection and inflammation.[79–81]

Common Pathologies

Rhinitis is common among older adults and can be classified into allergic and nonallergic types.[79,82] General rhinitis symptoms include rhinorrhea, sneezing, coughing, nasal drainage, difficulty smelling, and postnasal drip. Because of the phenomenon of immunosenescence, nonallergic rhinitis has a higher prevalence in older adults. These two entities are differentiated by skin or blood allergen testing but may coexist in certain patients. Vasomotor rhinitis, a type of nonallergic rhinitis, is thought to be caused by a dysregulated autonomic nervous system, leading to overstimulation of the parasympathetic system.[79,83] Rhinitis medicamentosa stems from overuse of intranasal decongestants, such as oxymetazoline. Patients should be cautioned against using oxymetazoline for more than 3 days, as this may cause rebound nasal congestion. Atrophic rhinitis involves degenerative changes of the mucosa, excessive cholinergic activity, and architectural changes of the nasal cavity's blood vessels and connective tissue. Gustatory rhinitis involves copious rhinorrhea during meals. Initial treatment modalities can include intranasal steroids, antihistamines, and anticholinergics.[79,84]

Sinusitis is inflammation or infection of the sinus cavities; it may occur independently of or concurrent with

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rhinitis.[85] Bacteria, viruses, and other noninfectious etiologies can cause acute sinusitis. A bacterial cause should be suspected when symptoms are present for 10 or more days or worsen after a period of initial improvement. Symptoms can last up to 4 weeks and involve congestion, sneezing, purulent nasal discharge, epistaxis, and facial pain and fullness. For acute uncomplicated cases, imaging is not recommended. Amoxicillin with or without clavulanate is the treatment of choice for acute bacterial sinusitis and trimethoprim-sulfamethoxazole or macrolides for penicillin allergies, while symptomatic relief is recommended for viral causes.[86] Treatment can also include intranasal steroids and hypertonic saline nasal irrigation. Antihistamines, decongestants, anticholinergics, leukotriene inhibitors, corticosteroids, and mucolytics can also be employed.[85]

Chronic sinusitis is common among older adults because of the decreased effectiveness of the aging immune system.[9] Chronic rhinosinusitis is defined as 12 weeks or longer of two or more of the following: mucopurulent drainage, congestion, facial pain or pressure, and decreased smell, as well as documentation of purulence or edema in the nasal cavity, polyps, or radiographic imaging of sinus inflammation.[85] Chronic sinusitis results from allergies or viral infections that lead to increased secretions, poor mucociliary function, and obstruction with possible superimposed bacterial infection. If obstruction continues, the sinus mucus membranes can undergo fibrosis, which further impairs the ability to clear pathogens. When symptoms of headache and facial pain predominate, physicians must differentiate chronic sinusitis from migraine, which is challenging given overlap in symptoms and patient demographics.[87] Referral to an otolaryngologist is needed if symptoms persist for more than 4 weeks.

Invasive fungal sinusitis is potentially life-threatening, but generally only needs to be considered in diabetic and immunocompromised patients.[88] Mucormycosis is a rare, potentially fatal infection that occurs in the setting of ketoacidosis, hematologic malignancy, and neutropenia. Mucosal gangrene results from infiltration of vasculature with fungi. Symptoms include congestion, fever, sinus pain, rhinorrhea, cranial nerve palsies, abrupt change in vision, and alterations in consciousness. The infection can erode through adjacent bone, spread along blood vessels and nerves, and lead to intracranial involvement and death. A CT and nasal endoscopy with biopsy are needed for diagnosis, and treatment is intravenous antibiotics and urgent surgery. These patients are critically ill and often in an intensive care unit. In a review of over 100 invasive fungal sinusitis patients, worse survival was associated with recent chemotherapy or bone marrow transplant, hematologic malignancy, and infection with atypical fungal organisms.[89]

Epistaxis has a bimodal distribution with peaks in children and older adults.[90,91] With aging, the nasal cavity experiences loss of fat and sclerosing of small vasculature.[79] Older adults also experience increased nasal dryness, atrophic rhinitis, neoplasms, nasal continuous positive airway pressure (CPAP) use, and drugs that predispose to bleeding.[90] Secondary causes of epistaxis include trauma, iatrogenic injury, and systemic bleeding disorders. An often overlooked exacerbating factor is hypertension. Most bleeding occurs from the anterior septum and can easily be treated by having the patient put his/her chin to the chest (which avoids blood going into the nasopharynx), liberal application of topical decongestant (e.g., oxymetazoline), and firmly pinching the lower lateral cartilages of the nose (i.e., the lower third of the nose) to fully obstruct the nostrils for 5–10 minutes (10–15 minutes if the patient is taking an anticoagulant). Prevention of epistaxis with nasal moisture is essential and includes regular nasal saline sprays with daily application of petroleum-based jelly to the nares. Use of humidifiers at night can also be helpful to promote moisture of the nasal mucosa. Control of hypertension, transitioning to anticoagulants that have available reversal agents, and avoiding nasal sprays like Flonase may also help to prevent future epistaxis.

Lastly, anosmia is a common complaint in older adults and can be due to rhinosinusitis, loss of olfactory neuroepithelial cells, drugs, neoplasms, trauma, and Alzheimer's and Parkinson's disease.[92] If symptoms worsen, are particularly bothersome, or have no clear cause, referral to an otolaryngologist may be warranted.

Oral Cavity

Age-Related Changes

The oral cavity tends to become drier with age because of decreased glandular acinar tissue, making dry mouth a common complaint.[2,79] Older adults are at risk for dental caries and gum disease because of decreased yet thicker saliva and lower IgA levels. There is also noted mandibular and maxillary bone resorption, which can worsen the fit of dentures. Ill-fitting dentures can cause paresthesia and a burning sensation from persistent contact with the mental nerve. Sensory abilities decline with decreased olfactory cells and tastebuds, which can result in malnutrition.

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Table 43.1 Characteristics of common oral lesions[93,94,97]

Lesion	Characteristics	Management
Candidiasis	White plaques with an erythematous foundation that can be removed; angular cheilitis may be present	Topical nystatin or azole class antifungals
Leukoplakia	White patch that cannot be removed	Refer for biopsy and removal
Erythroplakia	Red velvety lesion	Refer for biopsy and removal
Neoplasm	Persistent ulcer or protruding mass	Refer for biopsy, removal, and management

Common Pathologies

White lesions are often found in the oral cavity of older adults and have a wide differential diagnosis (Table 43.1).[2,79,93] Oral candidiasis is most commonly due to a fungal infection by *Candida albicans* and frequently seen in older people because of the high prevalence of immunocompromised states such as diabetes. Candidiasis can also be secondary to inhalational steroid use, dentures, or antibiotic treatment. On oral exam, there are white plaques with erythematous foundations. Cytology or culture can be performed but are not necessary for diagnosis. Treatment is with topical antifungals in the form of lozenges, liquids, or creams.[93,94] Oral leukoplakia is a white patch that cannot be rubbed away and generally results from persistent irritation leading to hyperkeratosis.[95,96] Oral leukoplakia is a diagnosis of exclusion, and malignant transformation is uncommon. Similarly, erythroplakia is an oral red, velvety lesion that is a diagnosis of exclusion. However, it is less common but with higher malignant potential.[96] Referral for biopsy by an otolaryngologist or oral surgeon can rule out malignancy, and subsequent management involves removal of the lesion with surgical or laser treatments and avoidance of risk factors.[97]

The parotid gland can be bilaterally enlarged secondary to viral infections or autoimmune disorders like Sjogren's syndrome.[98,99] Bacterial parotitis is common among older adults, particularly in those residing in nursing homes, and can be secondary to dehydration or obstruction of Stensen's duct by a sialolith or inflammation. Presentation involves sudden onset of unilateral warmth, erythema, pain, and edema over the cheek and can be accompanied by fever, purulent exudate, and, often, trismus. With milking of the parotid (i.e., firmly stroking the parotid from the anterior tragus to the corner of the mouth), cultures can be obtained from the Stensen's duct for suspected bacterial parotitis to aid in antibiotic selection. Treatment involves intravenous hydration, antibiotics, regular use of sialagogues, aggressive external and bimanual cheek massage two to three times daily to "milk" the gland, application of heat, and, if needed, surgical drainage of an abscess or stone removal.[100] Empiric treatment for *Staphylococcus aureus* with vancomycin and clindamycin may be warranted, particularly given the prevalence of MRSA among nursing home residents.[100,101] Acute bacterial parotitis in frail, older adults has a poor prognosis, so early intervention is vital.[98,102]

Facial Nerve Palsy

Bell's palsy is a sudden-onset, idiopathic facial paralysis and is thought to be secondary to reactivation of latent herpes simplex virus.[19] The incidence of Bell's palsy increases with age. As Bell's palsy is a diagnosis of exclusion, other etiologies of facial palsy should be ruled out including cerebrovascular accidents, cholesteatomas, trauma, infectious diseases including Lyme disease, rheumatologic disease, and parotid gland and skull base tumors.[19,103] Loss of facial sensation in the affected area, otalgia, hyperacusis, taste changes, and decreased lacrimation are common. Evaluation includes thorough head and neck examination involving palpation of the parotid gland and neurologic testing focusing on cranial nerves and cerebellar function. Treatment consists of high-dose oral steroids within 72 hours of symptom onset.[66,104] The efficacy of antivirals is controversial, but can be considered.[105] To prevent corneal damage, it is essential to use moisturizing eye drops frequently and protect the involved eye at night. The degree of facial paralysis is an important prognostic indicator, where total paralysis is less likely to regain full function. However, the overall prognosis of Bell's palsy is good, with 85% of patients' facial function returning.[106] If a patient is suspected to have Bell's palsy, but experiences no improvement in facial movement or worsening of facial paralysis in the ensuing weeks, then referral to an neurologist or otolaryngologist for further workup is recommended.[104,107] If patients continue to exhibit bothersome facial weakness on exam 6 months after diagnosis, it is warranted to refer to a facial plastic surgeon who specializes in facial reanimation; a range of in-office procedures and surgeries exist that can improve function and quality of life.[108,109]

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Head and Neck Cancer

The risk of head and neck cancer increases with age because of the cumulative exposure of carcinogens and immunosenescence.[2] Presentation of head and neck cancer varies widely depending on the site of lesion. Thyroid cancer tends to be more aggressive in older adults, so midline neck masses should be treated with high suspicion, and an ultrasound is the imaging study of choice. Nasopharyngeal cancers can present with a variety of symptoms, including nasal obstruction, epistaxis, obstructive eustachian tube dysfunction, otalgia, and cranial neuropathies.[110] Oral cavity carcinoma presents with a persistent, ulcerative lesion, while swollen lymph nodes or neck masses can be the presentation of lymphoma or a nonspecific finding of many head and neck cancers.[95] Oropharyngeal cancer associated with HPV, a common sexually transmitted infection, has risen significantly over the past decades and is associated with a younger age of diagnosis than non-HPV-associated cancers.[111] These patients typically do not have the traditional risk factors of smoking and alcohol history associated with head and neck cancers and have a better prognosis.[112] Dysphonia or dysphagia are common presenting symptoms of laryngeal cancer. Referral to an otolaryngologist is necessary in any case concerning possible head and neck cancer.

Further Reading

Key references include:

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- *Cummings Otolaryngology – Head and Neck Surgery*, 6th edition, 2015.
- American Academy of Otolaryngology – Head and Neck Surgery Guidelines, www.entnet.org/guidelines.

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Chapter 44

Oral Health

Sarah G. Lowman, Jonathan M. Reside, and Ingeborg J. De Kok

Introduction

Oral health is an important consideration in the holistic care of older adults. Mounting evidence suggests a connection between the oral cavity and common systemic diseases.[1] Furthermore, poor oral health may negatively impact quality of life[2] and oral pain may contribute to malnutrition[3] among community-dwelling older adults. Residents of long-term care communities have unique oral health needs and may face increased risk of poor oral health compared to adults who live independently.[4]

Common oral diseases, including dental caries and periodontitis, are prevalent among older adults and may result in tooth loss. Tooth loss is a serious occurrence that impacts speaking, eating, smiling, and quality of life and may also be associated with cognitive decline. Edentulism (the loss of all one's natural teeth) has been shown to be independently associated with cognitive decline in people aged 51 and older.[5] Additionally, cancers of the oral cavity, oropharynx, and larynx significantly impact this population.

A central goal of this chapter is to prepare health professionals who care for older adults to appreciate the prevalence and etiology of oral diseases, identify common signs and symptoms of disease, and engage in disease prevention and management strategies.

Dental Caries

Dental caries, commonly known as cavities or tooth decay, is a chronic process that causes local destruction of a tooth's natural enamel, dentin, and cementum layers. If it progresses, caries may cause inflammation of the dental pulp and infection in the surrounding bone.[6] In rare cases, serious systemic infection may occur.

Dental caries is the most prevalent oral disease and a common cause of tooth loss. In addition, caries may cause pain or discomfort, diminished function, and increased susceptibility to tooth fracture. Initial changes to tooth structure due to dental caries present as white spots and are reversible; however, as progression occurs, caries result in irreversible damage to tooth structure.[7] The crowns of teeth are susceptible to caries (coronal caries), as are root surfaces (root caries).

Prevalence in Older Adults

Among people aged 65 years and older with teeth, more than 90% have had dental caries in their permanent dentition and nearly 20% have untreated tooth decay.[8] State-level surveillance data shows that nearly half (48.6%) of adult assisted living residents of all ages had untreated tooth decay at the time of examination.[9]

Root caries is common among older adults, and prevalence may increase with advancing age. Findings from the 1999–2004 National Health and Nutrition Examination Survey (NHANES) show that nearly a third of adults aged 65 to 74 years had root caries, and more than 40% of adults over age 75 had the condition.[10] A prospective longitudinal study of community-dwelling older adults in Ireland (mean age of 69 years) found that more than half had root caries.[11]

Despite advances in caries prevention and treatment during the past several decades, inequities of race, ethnicity, and income persist in untreated disease,[8] tooth loss,[12] and oral health status.[9]

Etiology and Risk

Dental caries involves a dynamic relationship between the oral microbiome and physical, behavioral, environmental, and social factors that influence disease onset and severity.[13,14] Host factors and behaviors that influence caries occurrence include salivary production and flow, salivary composition, fluoride exposure, diet, and oral hygiene practices.[7] Of these, older adults may experience aging-related changes in oral hygiene, saliva, and diet, which potentially impact oral health status and caries development.

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Bacteria. Caries develops from dental plaque, a biofilm that forms on the surface of teeth and soft tissue. Research suggests that the bacteria *Streptococcus mutans*[15] and *Lactobacillus* are leading etiologic considerations in dental caries. However, less is known about the role of other oral microorganisms,[16] biological mechanisms, and microbial–host interactions.[14]

Saliva. Saliva protects against coronal and root caries by controlling pH and promoting antimicrobial activity in the oral cavity.[17] The production and flow of saliva may be diminished in older age, often resulting in xerostomia (the feeling of dry mouth).[18] People with normal salivation rates may experience xerostomia as well.[19] The use of common prescription and over-the-counter medications (e.g., anti-histamines, anticholinergics, daily aspirin) is a risk factor,[20] likely contributing to the high occurrence of xerostomia among older adults. The prevalence of xerostomia among community-dwelling older adults is unknown, but estimated to be more than 20% and strongly associated with taking several medications and increasing age.[21]

Diet and nutrition. Malnutrition is common among older adults and may be related to loss of appetite, food insecurity, difficulty obtaining or preparing food, difficulty eating, and depression.[22] Imbalanced diets that lack protein, dairy, and fresh vegetables may be high in cariogenic foods, including sugars and carbohydrates. Additionally, approaches to managing malnutrition, like increased frequency of eating and the use of oral nutritional supplements, may have negative impacts on oral health. Oral nutritional supplements often contain added sugar and carbohydrates, and frequent consumption of cariogenic foods (sipping or snacking throughout the day) increases caries risk compared to consumption mainly at mealtimes.[23,24]

Periodontal Diseases

Teeth are surrounded, supported, and protected by the gingiva, alveolar bone, and periodontal ligament.[6] These tissues may be affected by several diseases and conditions, including gingivitis and periodontitis.

Gingivitis is a nondestructive and reversible inflammatory condition confined to the gingiva, or soft tissues, immediately surrounding the teeth. It is most often associated with the presence of dental plaque. Clinical signs of inflammation, including gingival erythema and edema, are diagnostic of gingivitis. In addition, bleeding is expected in patients with gingivitis when the gingival tissues around the teeth are gently probed with a dental instrument. Left untreated, gingivitis may lead to the progressive resorption of the tooth-supporting bone, a condition known as periodontitis (Figure 44.1).

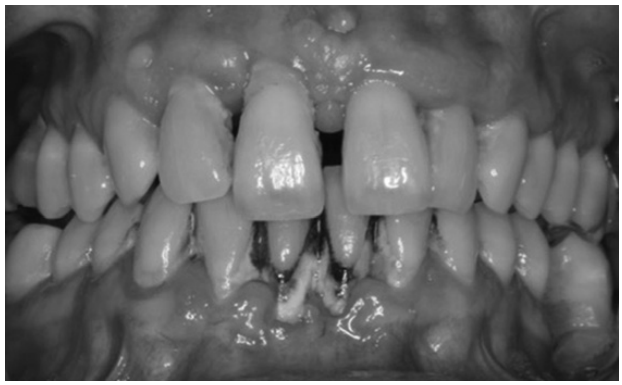

Figure 44.1 Patient with poorly controlled periodontal disease as evidenced by gross dental plaque and calculus deposits, gingival erythema, and gingival recession.

Changes in the oral cavity due to periodontitis are irreversible and may go undetected without a comprehensive oral exam. While often painless, common signs and symptoms of periodontitis include gingival erythema and bleeding, dental calculus (tartar), gingival recession, halitosis, an unpleasant taste in the mouth, and loose teeth. Periodontitis may be categorized and diagnosed based on disease severity and distribution.[25] Like dental caries, periodontal diseases (specifically periodontitis) may cause tooth loss.

Periodontitis shares possible mechanistic pathways with other systemic diseases, offering promise for future preventive and management interventions. Specifically, increasing evidence suggests a relationship between periodontitis and diabetes, as well as cardiovascular, immunoinflammatory, neurologic, metabolic, and respiratory diseases, and some cancers.[1,26–28]

Prevalence in Older Adults

Periodontal disease is common among older adults, but is not necessarily a normal consequence of aging. Prevalence estimates suggest that 42% of dentate adults over 30 had periodontitis and more than 60% of people aged 65 years or older had the disease. Severe periodontitis was most prevalent among adults aged 65 or older, those who smoked, and racial/ethnic minorities.[29]

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Etiology and Risk

Periodontitis has been defined as a "chronic multifactorial inflammatory disease associated with dysbiotic plaque biofilms resulting in chronic non-resolving and destructive inflammatory responses."[1,30,31] Like dental caries, periodontitis involves complex interactions between the oral microbiome and other physical, genetic, behavioral, environmental, and social factors to influence disease onset and progression.

Bacteria. The bacteria *Treponema denticola*, *Porphyromonas gingivalis*, *Tannerella forsythia*, *Prevotella intermedia*, and *Aggregatibacter actinomycetemcomitans* are leading etiologic agents in periodontitis that may result in a hyperinflammatory host response and eventual destruction of periodontal tissues. These periopathogenic bacteria are commonly found in dental plaque, which may accumulate under and above the gum line.

Diabetes. The relationship between diabetes mellitus and periodontitis is bidirectional. Diabetes is a risk factor for periodontitis, likely due to diabetes-related risk factors (diet, alcohol consumption, low income/socioeconomic status) and mechanisms related to the disease process itself. Research suggests that periodontitis and tooth loss are significantly greater in adults with elevated hemoglobin A1c when compared with those who have normal glycemic control. In addition, periodontitis negatively impacts glycemic control, diabetes outcomes, and all-cause mortality.[32,33]

Smoking. Cigarette smoking is a well-defined risk factor for periodontitis. Smoking may disrupt and alter the oral microbiome and influence host responses to bacteria.[34] In addition, smoking compromises periodontal treatment outcomes and periodontal surgical site healing. The role of e-cigarette use, vaping, and chewing tobacco in periodontal disease risk remains unclear.

Oral Cavity, Pharyngeal, and Laryngeal Cancers

Oral cavity, pharyngeal, and laryngeal cancers (referred to in this chapter as oropharyngeal cancer) include a variety of malignancy types that present on the lip, tongue, mouth, oropharynx, and larynx. The majority of oropharyngeal cancers are squamous cell carcinomas (SCC). Oropharyngeal cancers are frequently asymptomatic. The first signs of disease may be a lump in the neck or an ulceration in the oral cavity, most commonly on the floor of the mouth or lateral border of the tongue.

Tobacco use, alcohol consumption, and human papillomavirus (HPV) are established risk factors associated with oropharyngeal cancer.[35] In particular, HPV-associated oropharyngeal SCC rates are on the rise among both sexes.[36] Independent of treatment modality, patients with HPV-positive cancers have better survival compared to those with HPV-negative tumors.[37] However, survivors may experience diminished quality of life and increased risk of xerostomia, dysphagia, and chewing difficulties.[38]

Incidence among Older Adults

Annual US estimates for 2020 predict approximately 53,000 new cases of oropharyngeal cancer and 10,750 deaths (among men and women) from the disease, with greater incidence among men.[39] Between 2007 and 2016, oropharyngeal cancer rates were stable among people aged 80 and over, increased among people aged 50 to 79, and decreased among the 40 to 49 age group in the United States.[35] HPV-negative tumors are most common in individuals aged 60 years and older, but the incidence of this tumor type is decreasing, likely because of changing tobacco use patterns. In contrast, HPV-associated SCC cases among older adults have increased in recent years, and age at diagnosis (median age of 58 years) has trended upward as well. Similar to younger patients, older adults with HPV-positive oropharyngeal tumors have more favorable prognosis compared to patients who have HPV-negative tumors.[40]

As with other cancer types, racial disparities in oral cavity, pharyngeal, and laryngeal cancers in the United States are notable and necessitate solutions that address socio-structural inequalities. Laryngeal cancer incidence is higher in Black men compared to Whites, but the opposite is true for oral cavity and oropharyngeal cancer incidence.[41] The Black–White disparity in incidence has been narrowing over time.[41,42] Still, Black men experience higher mortality compared to White men from both laryngeal and oropharyngeal cancers.[41,43] Later-stage diagnosis in Black patients may explain this disparity, in part,[39] as well as racial differences in HPV-positive tumor prevalence.[44]

Etiology and Risk

Oropharyngeal cancers are thought to be caused, in part, by an interplay of genetic, epigenetic, behavioral, and environmental factors. Specific risk factors associated with oropharyngeal cancers include tobacco use, alcohol consumption, and high-risk strains of HPV.

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Tobacco and alcohol use. Smokeless and smoking tobacco products, as well as excessive alcohol consumption, are well-established risk factors for HPV-negative tumors of the oropharyngeal region. Among smokers, oropharyngeal cancer risk is three to six times higher than in nonsmokers.[45] A proposed mechanism is "the field effect," which posits that carcinogenic mediators (tobacco and alcohol, among others) predispose an area of epithelium, as opposed to a single cell, for later transformation into carcinoma.[46] Ethanol may also increase risk through local and systemic bodily processes.[47]

HPV. HPV infection has the potential to cause benign and malignant lesions in the oral cavity and oropharynx. The benign condyloma acuminatum and verrucus vulgaris lesions are generally caused by low-risk subtypes of HPV[48] and cause minimal morbidity. High-risk HPV subtypes are those with oncogenic potential. The majority of HPV-positive oropharyngeal tumors are associated with HPV16 and HPV18 subtypes. However, presence of HPV alone may not cause cancer. A proposed mechanism of oncogenesis is that the expression of certain high-risk HPV proteins induces malignant transformation.

Prevention and Treatment

A number of preventive strategies exist for common oral diseases, including fluoride exposure, diet modification, and smoking cessation. Addressing the common risk factors shared with other oral and chronic conditions is an efficient and effective way for the primary care provider to support oral health.[13,49,50]

Prevention of Dental Caries

The use of fluoride, a common ingredient in toothpaste and municipal water systems, is a safe and effective caries prevention strategy for adults of all ages.[51,52] A recent Cochrane Review cites benefits of fluoridated toothpaste in the prevention of dental caries when compared to nonfluoridated toothpaste; however, the optimal fluoride concentration remains undetermined.[53] Beyond primary prevention, fluoride products have demonstrated effectiveness in the nonsurgical management of active caries. Two products, 5% NaF varnish and 38% silver diamine fluoride (SDF), may be applied topically to tooth structure to arrest both coronal and root caries.[54,55] Lesion location and severity dictate optimal approaches to caries management.

Diets high in protein, dairy, and vegetables and low in fermentable carbohydrates and sugar-sweetened beverages are ideal for caries prevention. Frequency of food consumption is also a factor in dental caries, with frequent sipping and snacking potentially increasing risk. While diet and frequency of eating are important, they are not the only factors to consider in caries prevention. In patients who experience dry mouth, lubricating rinses, gels, frequent water sipping, and reduced alcohol consumption may be helpful. If xerostomia is induced by medications, it may be appropriate to consider alternative pharmaceuticals or deprescribing approaches.

Routine plaque removal reduces the threat of harmful bacteria in the oral cavity. To aid in plaque removal, toothbrushing is recommended at least twice daily for 2 minutes. Adults who use electric toothbrushes may experience better plaque control compared to those who use manual brushes,[56] although either is effective when used properly. Adjunctive aids that facilitate cleaning between teeth (floss, interproximal brushes) are often useful for maintaining oral hygiene. Flossing has been shown to slow the progression of oral disease (dental caries, periodontal disease, and tooth loss) in older adults.[57] However, flossing and other self-care activities may present a unique challenge for older adults because of loss in manual dexterity[58–60] and other conditions including depression, arthritis, and complications from diabetes. Interproximal brushes, end-tufted brushes, rubber tip stimulators, gauze squares, and wide-handled toothbrushes may be effective at removing plaque, and easier to manipulate, for patients with restricted mobility or dexterity. Oral hygiene education may benefit caregivers who assist with these activities.

Treatment of Dental Caries

In addition to nonsurgical management with fluoride products, dental caries is often treated with surgical approaches. These involve removing decayed tooth structure with a dental instrument and replacing it with amalgam or composite materials (dental filling). If the decayed portion of the tooth is large, this treatment approach may leave the tooth severely weakened and at risk of fracture. In this case, a restorative option that fully covers and protects the natural tooth may be preferable. This procedure involves preparing a tooth with a dental instrument to receive a dental crown. Crowns are affixed to natural tooth structure with dental cement or through a chemical bonding process.

If decay has reached the dental pulp, which runs through the center of the tooth and houses nerves and blood vessels, endodontic treatment may be necessary to

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save the tooth and minimize pain and infection in the supporting tissues. The most well-known endodontic treatment is root canal treatment, or root canal therapy. This procedure involves removing infected dental pulp tissues with instruments, cleaning and disinfecting the canals, and filling the empty space with an inert material (often gutta percha) and a sealing cement. After root canal treatment, a dental crown is often used to cover and protect the remaining tooth structure.

When carious lesions are severe and extend beneath the gum line, root canal treatment alone may yield unpredictable outcomes. Additionally, this treatment approach may not be desired by the patient because of the time commitment involved (multiple dental visits) and high cost of treatment. In such cases, tooth extraction is a treatment option.

Prevention of Periodontal Diseases

Prevention of periodontal disease and mitigation of disease burden may be accomplished through behavior change interventions that target risk factors, several of which are shared with other oral and systemic diseases. As with dental caries, routine plaque removal is essential for periodontal disease prevention. Patients with disease may have more complex home care needs that go beyond toothbrushing and flossing. The addition of oral hygiene adjuncts is often recommended; however, the manipulation of these aids may be compromised by reduced manual dexterity. Management of hemoglobin A1c through diet, exercise, and medication is another key strategy for minimizing the impact of diabetes on periodontal health. Finally, smoking cessation interventions are important for oral and systemic health.

Treatment of Periodontal Diseases

The management of periodontal disease may involve nonsurgical or surgical intervention. The first step in reestablishing periodontal health often involves removal of dental plaque and calculus from under the gums (scaling and root planing). In more advanced cases, periodontal surgery may be indicated to improve access for debridement (open-flap debridement), eliminate deep periodontal pockets by removing gum tissue (gingivectomy), recontour underlying bony defects (osseous surgery), or rebuild lost periodontal tissues (periodontal regeneration). Tooth extraction may be needed as well. The use of lasers and biologics-based regenerative technologies are novel approaches that may enhance treatment outcomes. Systemic antibiotic use in any of the aforementioned therapies is governed by the severity of periodontal disease and therapeutic goals.

With any periodontal treatment, long-term follow-up (periodontal maintenance) is recommended. Care typically occurs at a 3- to 4-month interval and is focused on supportive therapy. In addition to periodontal evaluation, biofilm disruption, and tailored oral hygiene instructions, special care is given to sites where progressive periodontal breakdown continues to occur. In these cases, additional scaling and root planing may be recommended. The local delivery of antibiotics beneath the gum line may be considered for sites resistant to traditional periodontal therapies. Finally, pharmacologic host modulation may be necessary in severe cases or for patients with a hyperinflammatory immune response. These medications alter the host immune response to microbial challenges and external environmental factors, minimizing periodontal tissue damage. Subantimicrobial dose doxycycline is the most popular host modulation agent currently available, but is typically reserved for patients with severe forms of periodontitis or when other treatment options have been unsuccessful.

Replacement of Missing Teeth

Compared to previous generations, older adults today are retaining more of their natural teeth. Still, tooth loss is common, and patients continue to request replacement options. There are different treatment alternatives for tooth replacement, including resin-bond prostheses, fixed partial dentures, removable partial dentures, complete dentures, and implant-supported restorations. The condition of the remaining dentition, health and height of alveolar bone, timing of the procedure, financial considerations, and ability to clean around the prosthesis are often considered when deciding the best option for a patient. Long-term survival and complication rates of each treatment modality should also be considered.

Complete dentures replace all the teeth in an edentulous dental arch and are a popular option for tooth replacement among older patients. Complete dentures require daily maintenance and care, much like natural teeth, including removal of the bacterial biofilm. This can be accomplished by soaking, brushing, and rinsing the dentures with a nonabrasive denture cleanser when they are out of the mouth. If the dentures remain outside the oral cavity, they should be stored in water. To minimize the risk of developing denture stomatitis and candidiasis on the oral soft tissues, dentures should not be worn for 24 hours at a time. Also, prostheses may be damaged if

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placed in boiling water or sodium hypochlorite bleach solution.

Oral Health in Long-Term Care Settings

Oral health and hygiene are of particular concern for long-term care residents, especially those with dementia or other disorders that compromise self-care abilities. In addition, several studies suggest an association, and possible bidirectional relationship, between cognitive decline and oral disease.[61–65] However, more research is needed in this area.[66]

Nursing home residents requiring supportive oral care may not consistently receive adequate assistance from care staff. In one nursing home study of five US communities, investigators found that toothbrushing occurred less than 20% of the time, and never for a full 2 minutes. A lack of flossing and glove changing were also reported. Also, residents frequently engaged in care-resistant behaviors during oral care activities.[67] Unfortunately, little is known about care aide perceptions of barriers and facilitators to providing oral care to nursing home residents.[68] This lack of an informed perspective poses a challenge for intervention development. Oral health education interventions for long-term care staff offer promise for improved resident oral health, as well as reduced pneumonia incidence,[69] but more evidence is needed to better understand staff and resident outcomes related to these initiatives.[70]

Denture loss has financial, functional, and psychosocial implications. Unfortunately, it has been identified as a challenge in hospital and long-term care settings.[71,72] Denture loss may be exacerbated by hiding and hoarding behaviors related to dementia. Additionally, denture cleaning and maintenance activities are often limited and inconsistent in long-term care settings. There is a need for denture care protocols in both nursing homes and assisted living communities, as well as efforts to minimize the loss and theft of oral prostheses.

The delivery of oral health-care services in long-term care settings varies by state and community type (nursing home vs. assisted living). Non-standardized policies across states pose a challenge for the large-scale implementation and evaluation of oral health-care delivery to long-term care residents. Examples of non-standardized policies include professional scope of practice for the dental team, assisted living community policies, adult dental benefits in the Medicaid program, and reimbursement rates. Dental benefits for adult participants in the Medicaid program are discretionary and vary across states, and few older adults have private dental insurance. In 2012, only 12% of Medicare beneficiaries reported having dental insurance.[73] Workforce training is another barrier to providing oral health-care services in long-term care settings. The USA has a dearth of oral health-care professionals with education and expertise in geriatrics.[74]

Opportunities exist to broaden oral health-care access to long-term care residents. The dental therapist model introduces a new advanced practice professional to the dental team, which may result in expanded access to services for this underserved patient population. Today, dental therapists work safely in multiple states, while other states are piloting dental therapy initiatives and considering changes to workforce legislation. Expanded function dental hygienists are another promising addition to the oral health workforce. Additional strategies for expanding access to care include mobile dental services and telehealth capabilities.

Oral Health at the End of Life

Optimal oral health care for the frail older adult population focuses on pain and symptom management, quality of life, and oral function. Dental problems are widespread at the end of life, and oral health-related quality of life may be compromised.[75] However, a minority of people in the last year of life receive supportive palliative care for oral health problems; most do not receive any oral health care, while others undergo aggressive and comprehensive treatment.[76] Current practice patterns suggest a need for the development of oral care standards for end-of-life care.[77]

Implications for Primary Care Practice

Oral disease risk assessment, nonsurgical intervention, and referral to a dental home may be incorporated into primary care and the specialty care of geriatric patients. One approach is the Oral Health Delivery Framework, which encourages clinicians to ASK-LOOK-DECIDE-ACT-DOCUMENT the oral health needs of patients.[49] The first step of the framework involves asking patients and caregivers about oral health behaviors and risk factors, including oral self-care activities, diabetes management, smoking status, and alcohol use. Questions to assess oral health behaviors, and signs and symptoms of disease, may include:[78]

- Have you been to a dentist or dental hygienist in the past 12 months?

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- Are you having pain on your teeth or gums? Sensitivity to hot or cold?
- Do your gums bleed after brushing? Do you wake up with blood on your pillow?
- Do you have difficulty taking care of your (teeth, gums, denture) at home?
- Do you use products that contain fluoride (community water, toothpaste, rinse)?
- What do you eat/drink in a typical day (soda, juice, oral nutritional supplements)?
- Do you experience feelings of dry mouth?
- Have you had any cavities, or lost any teeth, in the past 3 years?
- What concerns, if any, do you have regarding your oral health?

Next, a visual assessment of the oral cavity[49] may help determine the presence of dental plaque buildup, broken or missing teeth, discolored teeth with active caries, halitosis, presence or absence of saliva, redness or inflammation of the soft tissue, and gross asymmetry of the tongue, tonsils, uvula, and other oral structures. A head and neck exam may reveal lymphadenopathy, tenderness, or lumps in the region. Risk assessment and exam findings will inform an action plan. A plan may target risk factors and involve oral health education (for patient or caregiver), application of topical fluoride agents in accordance with professional scope of practice, and referral to a dental home.

Real-world clinical demands may limit the feasibility of implementing risk assessment and screening protocols in nondental practice settings. As an alternative, discussing and addressing risk factors that are common to other chronic conditions may be an efficient and effective way of addressing oral health.[13,49,50] In addition, enhanced health professional partnerships and innovative interprofessional practice models may further benefit older adult patients and care providers moving forward.

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Chapter 45

Foot Health

Diana Homeier and Araz Melkonian

Overview

Foot health is important to the overall well-being of the older adult. Foot problems can significantly affect mobility, function, and quality of life. Painful foot problems occur in approximately 24% of older adults[1] and have been shown to increase the risk of falls.[2] Because the foot can impact overall function, the podiatric exam for either preventive or problem-related issues is an important part of geriatric care.[3] Once problems are identified, management may include definitive treatment, proper footwear, and referral to an appropriate specialist.

The foot undergoes some changes with increasing age. The skin of the feet is drier, there is an increased risk of infection, and wound healing is more prolonged. The toenails undergo trophic changes and may become deformed. There is loss of muscle mass and atrophy of the soft tissues, leading to increased risk of injury. Vascular changes and age-related changes to the peripheral sensory system can predispose the feet to infection or injury. Age-related loss of muscle strength in the foot and ankle can affect the overall function of the foot and increase risk for falls.[4,5]

Foot Examination

The foot examination is an important part of health care for an older adult. Older adults may not be vigilant about their feet and may lack the vision, sensation, or flexibility to adequately check their own feet.[6] The foot examination may begin with the evaluation of the patient's shoes. The type of shoe and ability to ambulate in it can give clues to underlying foot problems.[7] The foot exam then includes a dermatologic, musculoskeletal, vascular, neurologic, and functional assessment.[8] The dermatologic exam is an evaluation of the condition of the skin and the presence of discoloration, hyperkeratotic lesions, ulcerations, infection, and nail abnormalities. The musculoskeletal assessment detects foot deformities (hallux valgus, digiti flexus) and arthritic changes. A vascular assessment includes the palpation of the posterior tibial and dorsalis pedis pulses and inspection of skin and temperature for signs of vascular disease. The neurologic exam includes motor function, reflexes, and sensation, including a monofilament test when indicated. The functional exam is an assessment of foot motion, gait, and mobility.[4,8]

Skin and Nail Conditions

Skin changes that occur with aging and skin pathologies that occur elsewhere on the body can also occur on the foot. Xerosis (excessively dry skin) is a very common problem for older adults (see Figure 45.1). Xerosis is due to a lack of hydration and lubrication of the skin. The skin may form fissures or cracks (common on the heel), which increase the risk of possible ulceration and infection. Management includes the use of emollients applied daily after bathing. Urea cream/solution or ammonium lactate can be used as a mild keratolytic. A heel pad (mineral oil pad) or cup can also be used to minimize trauma to the heel.[6]

Pruritis is common in older patients and may be related to dry skin and cold weather. The patient may complain of itching and scratching, and the foot examination may reveal excoriations. An examination should be done to rule out tinea, urticaria, or other skin lesions.[4] Treatment of pruritis consists of skin lubricants and topical steroids if warranted. Antihistamines can be helpful, but must be used in low doses with caution because of potentially dangerous side effects.

Venous stasis dermatitis is caused by venous insufficiency and can manifest as edema, induration, discoloration, and ulcerations of the skin. It starts most commonly in the medial aspect of the ankle. Management includes elevation and compression stockings to improve venous return and manage edema. Emollients and topical corticosteroids can help to manage the dermatitis. Antibiotics may be indicated when infection is present.[9]

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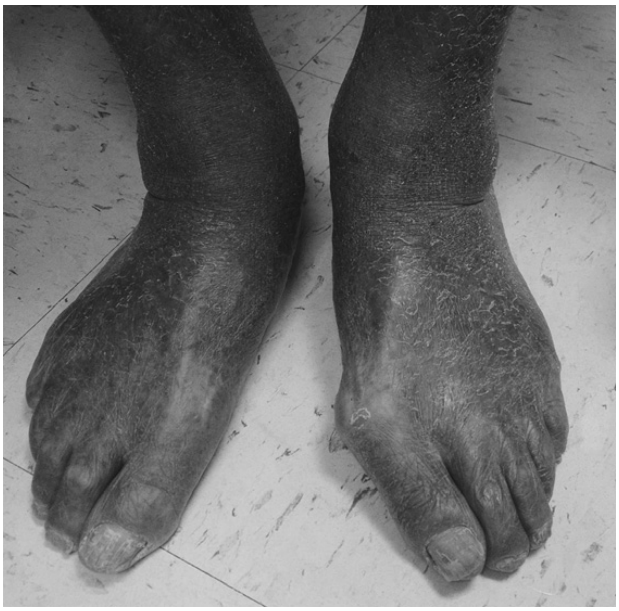

Figure 45.1 Feet of an older adult showing many of the conditions that can happen with aging. Xerosis is present on both legs and feet. Onychomycosis is present bilaterally. Hallux valgus (bunion) is noted on the left foot. Digiti flexus (hammertoe) is present in the right second toe. (A black-and-white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

Hyperkeratotic Lesions

Hyperkeratotic lesions such as tyloma (callus) and heloma (corn) are thickened and hardened areas of the skin that have developed over time because of constant stimulation of the epidermis through friction or pressure.[10] Ill-fitting shoes may be a factor in the development of these lesions. Other factors include contractures, gait changes, deformities, loss of skin tone and elasticity, loss of soft tissue with age, and atrophy of the plantar fat pad.[11] Generally, these are not harmful, but patients often present with pain, ulceration, or infection that can limit their ambulation.[12]

It is often hard to differentiate between corns and calluses. Corns are usually smaller than calluses and have a "central core" that is surrounded by irritated skin. Corns are often painful with pressure. Calluses are rarely painful. These usually develop on the stratum corneum, the outermost layer, on the sole of the foot, under the heel or ball of the foot.[13] It is important to differentiate corns and calluses from verruca (warts) or other similar appearing lesions. Plantar verruca are caused by a strain of the human papilloma virus. These lesions are circular, punctated, flat, and may contain thrombosed blood vessels. To differentiate these from hyperkeratotic lesions, when debrided, warts will disrupt the normal skin markings and may have dark specks representing capillary thromboses. Skin lines are not disrupted in corns or calluses.[14]

Treatment for hyperkeratotic lesions is dependent upon the patient and the effect on function. The primary focus should be placed on removing pressure and preventing ulceration or further injury. Considerations for treatment include padding, shoe modification, emollients, debridement, and orthoses. When the lesion is on the plantar aspect of the foot, treatment should be aimed at dispersing the weight. Donut pads and metatarsal bars may help to redistribute pressure or contact. The lesions can be treated with salicylic acid plasters and debridement. There are also many salicylic acid solutions available over the counter that have been shown to be beneficial.[10] Surgical revision may be indicated in some cases. The problem may be recurrent or persist because of the residual deformity and underlying conditions such as diabetes.[12] The treatment of verruca is based on creating an immune response by irritating the verruca cells. Salicylic acid and cryotherapy have both been found to be useful in this treatment. There are over-the-counter alternatives for these that have also been found to be effective.[12]

Ulceration

The term "ulcer" describes destruction of the epidermis that extends into the dermis and may reach subcutaneous fat or deeper tissues. An ulceration is a soft-tissue injury that leads to an open wound that is difficult to heal. It can result in complete loss of the epidermis and may go as deep as the dermis and subcutaneous fat.[15] Ulcers can lead to poor quality of life, high mortality due to sepsis, and increased hospitalization. Determining the cause of the ulcer is important in determining the treatment.

There are three main types of ulcerations: venous, arterial, and neuropathic ulcers. The ulcers are defined by their appearance, location, and the surrounding tissue.

Venous stasis ulcers occur in patients with poor venous circulation in the leg. They primarily occur below the knee, just above the ankle; the medial malleolus is the most common site. These ulcers are typically irregular and shallow and may have yellow, fibrinous exudate. Pain is often mild and the arterial pulses are usually normal. Additional findings might include varicosities, edema, and stasis dermatitis. Because of poor circulation, risk of infection and prolonged healing often occur.

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Arterial (ischemic) ulcers are often found distally on the toes or on pressure points, like the heel or anywhere there are bony prominences. Arterial ulcers have a "punched-out" appearance, well-demarcated edges, and may have overlying necrotic eschar. Patients complain of severe pain, especially at night. Other findings consistent with arterial disease may include decreased hair density, shiny and thin skin, diminished peripheral pulses, or prolonged capillary refill time.[16] Patients may feel better after hanging the leg over the side of the bed. There is usually very poor circulation, causing poor tissue granulation and color changes (pale white/yellow/gray color) when the leg is elevated. Peripheral arterial disease can be confirmed with the Ankle-Brachial Index.[16]

Neurotrophic ulcers are usually located on the bottom of the feet or heels beneath pressure points or hyperkeratotic lesions. These ulcers have a punched-out appearance often within a callous and are painless. Additional clinical findings may include findings of diabetic neuropathy, poor sensation in the involved area, and dryness or scaling. Patients may complain of tingling, numbness, or a burning sensation. Ulcers can become deep, and underlying osteomyelitis should be considered when ulcers do not heal with offloading therapies.[15]

Treatment should be focused on reducing pressure to the area and preventing infection. Pressure reduction can be obtained with dressings, orthoses, shoe modifications, and special depth shoes. In order to prevent infection of the ulcerated area, wound care is essential. The wound must be kept clean and the wound base must be healthy to permit healing. Appropriate wound care may include debridement and/or antibiotics (when indicated). Vascular or arterial ulcers may warrant further evaluation with a vascular specialist. It is essential to improve vascular supply to the area and minimize worsening of conditions.[11]

Tinea Pedis

Tinea pedis, otherwise known as Athlete's foot, is caused by contagious dermatophytic fungal infection, such as *T. rubrum or T. mentagrophytes*. It affects the skin and causes scaling, flaking, and itching. Tinea pedis is more common in warmer climates and is easily transmitted in moist communal areas where people walk barefoot (locker rooms and showers).

Tinea pedis can occur in the interdigital spaces and on the sole of the foot. The diagnosis can usually be made clinically through history and physical. Treatment should include conservative measures such as allowing the feet to ventilate and remain dry with good hygiene. Topical antifungal medication is usually effective. There are many options for this, including clotrimazole, miconazole, ketoconazole, econazole, and terbinafine. A Cochrane Review found that the fungicidal allylamines (such as terbinafine) have a higher cure rate and require less time to cure than other topical medications.[17] For severe or refractory infections, oral terbinafine may be effective, but its safety must be considered.[18]

Onychomycosis

Onychomycosis is one of the most prevalent diseases of the toenail. Its incidence increases with age.[19] Blood circulation diminishes and nail growth becomes retarded, increasing the susceptibility to infection.[18] It is estimated that one third of the older population has onychomycosis[6] and one study of institutionalized elderly persons found that 43% had onychomycosis.[20] It is usually caused by dermatophytes (*Trichophytum rubrum* or *T. mentagrophytes*),[18] but can also be due to yeasts and nondermatophyte fungi. Factors that contribute to this disease can include occlusive footwear, repeated trauma, sports participation, comorbid diseases such as diabetes, peripheral arterial disease, chronic tinea pedis, genetic predisposition, and foot hygiene.

The patient often presents with discoloration and increased thickness in the toenail (see Figure 45.1). The infection may cause distal subungual, white superficial, proximal subungual, or total dystrophic changes. The superficial variety does not involve the nail plate, whereas the distal and proximal kind affect both the nail bed and the nail plate. When the nail plate is involved, hypertrophy and deformity of the nail occur and external pressure to the nail may cause pain. It is important to rule out any other diseases that may mimic onychomycosis such as trauma, lichen planus, and psoriasis. Only approximately half of the suspected onychomycosis is truly due to a fungal infection, and the other half is due to other causes that may lead to variation of nail morphology.[21] Therefore, the clinical presentation, culture, and KOH preparation may all be helpful in diagnosis.

Diagnosis can be made based on laboratory confirmation with potassium hydroxide smear, culture, and nail biopsy, and, less frequently, immunohistochemistry, restriction fragment length polymorphisms, and polymerase chain reaction assays. It is important to get the correct specimen sampling and to use proper technique to prevent false negatives.[19]

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Options for management include systemic antifungals, topical medications, keratolytic agents, debridement, photodynamic therapy, and surgical removal. After diagnosis is confirmed, a decision to treat may be based on discomfort, risk of infection (as in a diabetic patient), or cosmesis. Systemic antifungals include terbinafine, itraconazole, and fluconazole. These medications can be effective but must be used with caution in older adults, as the side-effect profile, duration of therapy, and drug interactions can be problematic. Topical antifungal agents such as ciclopirox, amorolfine, and tioconazole have a much lower cure rate and require 24–48 weeks of treatment but are well tolerated.[6] Serial nail debridement can decrease the risk of infection, ingrown toenails, corns, and ulceration.[22] Treatment is often challenging, as the infection is usually embedded within the nail and difficult to reach. Factors that can indicate poor prognosis include the amount of nail involvement, nail dystrophy, mold, dermatophytoma, immunosuppression, and poor circulation.[19]

Other Nail Disorders

Ingrown toenail (onychocryptosis) is an incurvation of the edge of the nail into the nail groove. This is usually due to poor self-care (nail-cutting), trauma, or narrow-fitting shoes.[18] When this occurs, an abscess or an infection may result. If treatment is not initiated, periungual granulation tissue forms and complications such as deformity and involution may occur. Treatment may include excision, partial avulsion, fulguration, desiccation, or caustics and astringents.[12]

Paronychia is a localized infection involving the lateral or medial nail wall, most often caused by a spike of ingrown nail. This requires incision and drainage of the abscess with removal of the nail tissue. Antibiotics may also be necessary if there is surrounding cellulitis or if the patient is diabetic. Nonhealing granulation tissue in the nail groove may be an indication for a biopsy, as Kaposi sarcoma, melanoma, and squamous cell carcinoma can present in this manner.[22]

There are many nail dystrophies that can occur in the older adult. They are usually the result of repeated trauma, underlying disease, or degenerative changes. These nail conditions can be a source of pain, ulceration, and infection. Treatment should be individualized and may include specialized shoes, orthoses, debridement, and possible excision.

Subungal hematomas can occur in older adults secondary to microtrauma. However, when pigmented lesions are noted under the nail (especially if not associated with trauma), melanoma must be considered.[22]

Common Foot Disorders in the Elderly

Forefoot

Hallux Valgus (Bunion)

Hallux valgus, commonly known as bunion, affects up to one third of people over age 65, with a greater prevalence in women. It is characterized by subluxation of the first metatarsal-phalangeal joint and lateral deviation, greater than 15 degrees, of the great toe toward the lesser toes[23] (see Figure 45.1). The tissues around the joint may become swollen and sore. It may lead to restricted movement and thickening of the skin. Patients often complain that it is difficult to wear shoes or find shoes that are comfortable. Bunions may eventually lead to bursitis, which is inflammation of small fluid-filled pads (bursae); hammertoe, a bend in the middle joint of the toe, usually the one next to the big toe; and metatarsalgia (inflammation of the ball of the foot). Weight-bearing radiographs may be helpful in grading the deformity. Conservative management may include adaptive shoes (wider shoes or shoes with wider toe), proper padding and taping, anti-inflammatory medication, and applying ice. Although there is no evidence that over-the-counter splints or interdigital pads correct the deformity or improve function,[10] they are frequently used and may provide some pain relief.[24,25] Some shoemakers will stretch shoes for a fee to accommodate a bunion deformity.[10] If the bunion is significantly symptomatic, surgical management may be required. There are over 100 different surgical techniques described to correct bunion deformities. Retrospective studies have shown that 85% of patients have a good result and are satisfied, 10% are less satisfied with the outcome, and 5% have a poor surgical result.[24,26] Possible complications include recurrent deformity, wound-healing problems, pain, pseudoarthrosis, and rarely necrosis of the metatarsal head.[26] Although bunion surgery may reduce symptoms and improve patient ability to comfortably wear shoes, there is some evidence that after 2 years surgery may be no better than orthoses or placebo.[27]

Hallux Limitus and Hallux Rigitus

Hallux limitus is a condition in which there is restriction in the range of motion of the first metatarsophalangeal (MTP) joint. It is thought to affect 40% of men and 50% of women by age 70 and is often a result of osteoarthritis in

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the joint.[10] Hallux rigitus is the end stage of this condition in which there is very little motion and significant arthritic changes in the MTP joint.[6] On examination, the first MTP joint may be painful, swollen, and have a limitation in range of motion. Radiographs (dorsoplantar and lateral) may show joint space narrowing and an osteophyte on the dorsal aspect of the first metatarsal head.[28] This condition can be mistaken for gout. Orthotics and hard soled shoes can restrict motion at the joint and decrease pain.

Digiti Flexus (Hammertoe Deformity)

A digiti flexus (hammertoe) or contracted toe is a deformity arising from increased extension at the metatarsophalangeal joint (MPJ), flexion of the proximal interphalangeal joint (PIPJ), and hyperextension of the distal interphalangeal joint (DIPJ) of the lesser toes, causing it to be permanently bent, resembling a hammer[28] (see Figure 45.1). Two other types of toe deformities are claw toe and mallet toe. Claw toe deformity is an extension contracture of the MPJ and flexion of both PIPJ and DIPJ. Mallet toe consists of flexion at the DIPJ only. A "crossover" second toe deformity occurs when the hallux valgus deformity crosses over the second MPJ, causing subluxation.[29] Hammertoes may be flexible (easily moved and reduced into position) or rigid (nonreducible). Rigid hammertoes are often painful and may be associated with hyperkeratotic lesions from abnormally shaped toes rubbing on shoes.[6]

Treatment should be focused on alleviating pain and caring for keratotic lesions. Management may be nonsurgical or surgical. The conservative approach includes physical therapy, proper-sized shoes (wider and deeper), padding, and orthotic devices. Debridement of hyperkeratotic lesions can also be beneficial, along with corticosteroid injections to decrease inflammation or bursitis. Taping and splinting mild crossovers to prevent further worsening of conditions may be helpful. A crest pad may help bring the toe into a more neutral position and alleviate pressure on the tip of the toe.[6] Surgical correction may be necessary for more severe cases.[29] A hammertoe deformity may progress to metatarsalphalangeal dislocation. Usually this is a dislocation of the phalanx on top of the metatarsal head. This can result in chronic pain and possibly be a source of callus or ulceration.

Sesamoiditis

Sesamoiditis is the inflammation of the sesamoid bones and usually affects those that are under the first metatarsal joint of the hallux. There are normally two sesamoid bones, sometimes comprising two separate pieces each. The function of the sesamoid bones is to act as a hinge for the flexor tendons, which allows the big toe to bend downwards.[30]

Sesamoiditis is caused by repetitive motion, sudden bending upwards of the big toe, direct trauma, osteoporosis, or osteoarthritis. Treatment involves rest, proper-fitting shoes, and padding with proper arches to prevent continual impact to the sesamoid bones. Anti-inflammatory medications, corticosteroid injections, and immobilization may also be used.[30]

Fracture of Phalanx/Stress Fracture

Stress fracture can develop from overuse and occurs in the weight-bearing bones of the foot. It is more common in patients with chronic inflammatory arthropathies, severe osteoporosis, joint deformities, or chronic corticosteroid injections.[29] This happens when overused muscles are unable to minimize the shock of repeated impacts and thus transfer the stress to the bones, leading to small fractures. The most common sites of stress fractures are the second and third metatarsals of the foot.[29] The patient may complain of gradually increasing pain and swelling on top of the foot. The patient may also notice bruising. A stress fracture is often difficult to see on X-ray and may require a bone scan, magnetic resonance imaging (MRI), or computed tomography (CT) scan.

A conservative approach is to wear proper footwear and orthoses. It is important to immobilize and modify activities. Normal activities can be resumed after adequate healing, which may take 6 to 8 weeks.[29] Strength training and physical therapy will be of great benefit for these patients after recovery.

Other fractures may occur from falls or trauma in older adults. This may be due to instability or neuropathy. When there are fractures of the phalanges, this can affect balance and ambulation. Referral to a specialist may be necessary, depending on complications such as osteomyelitis and displacement. If the fractures are nondisplaced, often buddy taping and immobilization with boot or cast are appropriate. A displaced fracture may be reducible or irreducible. If reducible, a cast will be placed to prevent movement. If irreducible or open fracture, open reduction may be required.

Morton's Neuroma (Intermetatarsal Neuroma)

Morton's neuroma is a benign lesion that involves the compression of the common digital nerve occurring in the third intermetatarsal space. The patient will complain of numbness and tingling and/or radiating, shooting, or

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burning pain on weight-bearing, relieved with rest or removal of footwear. Occasionally the pain may be described as "walking on a lump."[31] The symptoms may be replicated with direct pressure at the intermetatarsal space. A click may be felt or heard with compression to the forefoot and applying plantar pressure, known as Mulder's sign. Other maneuvers that can be used are Gauthier's test (applying medial to lateral pressure of the forefoot) and the Bratkowski test (hyperextending the toes, rolling the thumb over the symptomatic area, and feeling a mass).[29]

It is important to differentiate Morton's neuroma from other diseases such as stress fracture, osteoarthritis, neoplasm, bursitis, or capsulitis. An X-ray, ultrasound, or MRI may be helpful to rule out other musculoskeletal pathology.[32] Initial treatment should be focused on conservative measures: proper footwear, orthoses, corticosteroid injections, and sclerosing alcohol injections to decrease pressure and irritation of the nerve. Cryogenic neuroablation is an option to preserve structures, but is less effective on large neuromas.[31] For some patients, surgical neurectomy or nerve decompression may be required and has been found to be successful, with 80% of patients satisfied after surgery.[6]

Midfoot

Pes Planus

Pes planus (also known as flatfoot) is a postural deformity in which the arch of the foot is touching or nearly touching the ground. This can be congenital or acquired. When the patient is standing, the longitudinal arch collapses. Many patients with pes planus are asymptomatic and untreated. However, as they age, the deformity can become more rigid and cause pain and stress on foot and ankle joints. The primary goal of management is the reduction of pressure through weight loss and arch support orthoses. Surgery is reserved for serious cases when other measures have failed.[33]

Posterior Tibial Tendon Dysfunction

Posterior tibial tendon dysfunction is the most common cause of acquired flatfoot in the older adult.[34] It is caused by the weakness, elongation, and gradual tearing of the tibialis posterior tendon. The tibialis posterior tendon lies directly behind the medial malleolus and passes to the plantar surface of the foot to insert primarily onto the navicular bone. Its function is to invert and plantar flex the foot. As the tendon is serially damaged, there is collapse of the longitudinal arch. This can lead to subluxation of the tarsal joints and even the ankle.[6]

Most patients present with an insidious onset of unilateral flatfoot deformity. Initially, the patient may report vague pain in the medial malleolar region. This may progress to pain and swelling along the medial foot and ankle and pain in the arch that radiates to the medial calf muscle. The arch can progressively collapse, changing the shape of the foot. There are four stages described. Stage I is tendinitis of the tibialis posterior tendon without foot deformity. Stage II describes a ruptured tendon that is functionally incompetent with a reducible foot deformity. In Stage III the deformity becomes rigid and fixed. In Stage IV there is a valgus ankle deformity.[35]

Treatment is dependent on the stage of the disease at the time of presentation. Stage I deformity may be treated with orthoses, casting, bracing, physical therapy, and cautious use of nonsteroidal anti-inflammatory drugs (NSAIDs) or corticosteroid injections. Stage II may involve tendon transfer or calcaneal osteotomy.[6] Stages III and IV usually require arthrodesis and complex management.[35]

Heel

Plantar Fasciitis

Heel pain is a common complaint among older adults. Plantar fasciitis is the most common cause of heel pain. It is caused most frequently by an overuse injury at the origin of the plantar fascia due to excessive stress or pronation. The typical history is sharp pain localized to the anteromedial aspect of the heel (insertion of the fascia) that is severe with ambulation after a period of inactivity, then will improve or resolve as the activity progresses. Classically, patients complain of pain with the first step in the morning.[36] If the condition becomes chronic, the pain may become dull and constant.[37] Radiographs are not necessary for diagnosis, but may be done to rule out other causes in patients who have had symptoms for several months. A plantar calcaneal spur may be visible on radiographs, but this is not pathognomonic for plantar fasciitis, as they are reported to be present in 27% of people without any symptoms.[37]

The differential diagnosis includes calcaneal stress fracture, plantar fascial tear, tarsal tunnel syndrome, plantar calcaneal bursitis, and bone contusion. Approximately 80% of patients with plantar fasciitis will improve within 12 months with conservative management, including

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activity modification, ice, massage, heel lift, stretching of the plantar fascia, and proper footwear.[36] Orthoses, anti-inflammatory medication, taping, dry needling/acupuncture, physical therapy, and steroid injections can also be helpful. Difficult cases may require night splints, extracorporeal shock wave therapy, or possibly surgery.[37] Surgery now consists of open, percutaneous, and endoscopic options. Endoscopic fasciotomy has shown 97% pain relief within 7 days of the procedure.[38]

Heel-Pad Syndrome (Fat-Pad Atrophy)

Heel-pad syndrome is described as a bruising pain in the heel. The pain is present while walking, especially when walking barefoot, and is somewhat relieved by shoes. On examination, there may be little to no fat pad present on palpation over the heel, palpation may elicit tenderness, and there may be hyperkeratotic lesions overlying the heel. Treatment is with proper shoe support, rest, ice, taping, heel cups, and shoe inserts.[39]

Achilles Tendinitis

The Achilles tendon (formed from the merging of the soleus and gastrocnemius muscles) inserts into the posterior calcaneus. Inflammation and degeneration of the tendon can cause posterior heel pain that may run along the course of the tendon. This tendinitis is typically described as achy and worsening with activity or pressure. Fluoroquinolone use has been shown to precipitate Achilles tendinopathy in older adults.[40] The tendon may be tender to examination, and thickening of the tendon may be palpated. Treatment consists of activity modification, anti-inflammatory medication, warm compresses, orthoses or heel lifts, and physical therapy exercises.[39] Steroid injections should be avoided because of the risk of rupture. Achilles tendon tear or rupture should be considered if the patient had trauma or sudden onset of pain with push-off of the foot.

Tarsal Tunnel Syndrome

The tarsal tunnel is located posterior and inferior to the medial malleolus. Within the tarsal tunnel lies the tibialis posterior tendon, flexor digitorum longus tendon, flexor hallicus longus tendon, and posterior tibial nerve.[41] Tarsal tunnel syndrome (TTS) is a compressive entrapment neuropathy of the posterior tibial nerve or one of its branches.[42] It is an uncommon condition that occurs more often in patients with a history of trauma or rheumatoid arthritis. The patient with TTS complains of burning, tingling, or shooting pain in the posteromedial ankle, heel, and sometimes into the distal sole and toes. The pain is usually worse with standing or walking and relieved with rest. The differential diagnosis includes plantar fasciitis, Morton's neuroma, and systemic diseases that can cause a neuropathy. On examination, there may be tenderness over the nerve, elicited by tapping along the nerve (Tinel sign), and reproduction of the pain with stress or compression maneuvers.[39] There may also be decreased sensation and some muscle wasting and weakness in the region of the nerves affected.[42] The diagnosis is difficult and MRI, ultrasound, and electrodiagnostic testing may be helpful. Treatment includes activity modification, orthoses, steroid injections, and careful use of anti-inflammatory or neuromodulator (antiepileptics) medications. Surgical release can be considered if warranted.[39]

Systemic Diseases in the Foot

Diabetes

Diabetes is the number one systemic condition affecting foot health in the elderly.[6] It is estimated that 15% of diabetics will develop foot ulceration[43] and 10% of ulcers will lead to amputation.[6] The National Diabetes Advisory Board has stated that 50–75% of amputations could be prevented with proper surveillance, education, and early intervention.[43] It is imperative that healthcare providers have a high level of surveillance and care for diabetic feet. Older diabetics may suffer from neuropathy, vascular disease, and skin changes due to their diabetes. Because of sensory, vision, and mobility changes, it may be more difficult for an older diabetic to note changes to their feet.

Prevention of foot ulcers in older diabetics is crucial. Older diabetics should have their feet examined at every visit, and a thorough diabetic foot exam should be done at least annually. This should include a skin assessment, neuropathic assessment (pressure sense with 10-gm monofilament test, vibration sense using a 128-Hz tuning fork, position, pain, and temperature), structural assessment for abnormalities, reflex testing, and vascular assessment.[44] Diabetic patients and caregivers should receive education regarding self-examination, proper footwear, and reduction of pressure points. Additional keys to prevention of foot ulcers include glycemic control and treatment of neuropathy, foot deformities, hyperkeratotic lesions, nail deformities, poor joint mobility, and arterial disease.[6] In diabetics with high risk for ulcer formation, early intervention with proper footwear and foot care reduces complications.[45]

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Once a diabetic foot ulcer develops, a comprehensive assessment and proper management are key to reduce the potential for amputation.[43] Documentation of the wound and its progress should be carefully undertaken. The presence of infection must be considered and treated appropriately. Imaging should be used to exclude osteomyelitis (plain radiographs, MRI, or leucocyte scans).[44] Vascular studies should be performed and consultation with a vascular specialist if abnormal. Management consists of offloading pressure, debridement, management of ischemia, antibiotics for infection, and hospitalization and surgery when necessary.

In patients with poorly controlled diabetes and peripheral neuropathy, a swollen, warm, red foot without ulceration could indicate Charcot neuroarthropathy.[46] Charcot neuroarthropathy (or Charcot foot) begins with inflammation, bone resorption and fragmentation, and joint dislocation. It can progress to bony consolidation and fusion as well as new bone formation. The end result is a deformed foot that is less functional. Because the patient has neuropathy, the stresses or injuries to the foot that occur over time may be painless. The diagnosis may be made by radiograph or by clinical presentation of an erythematous, warm, neuropathic foot with a change in shape, but without an open wound. The goals of treatment are foot stability and prevention of skin injury. Treatment may involve immobilization, bisphosphonates, and surgery. If treatment is initiated early in the course of the disease process, there is better probability of preserving function.[46]

Gout

Gout (podagra), the most common form of inflammatory arthritis in the United States, often affects the foot.[47] Most people affected are obese and have hypertension and renal disease.[48] Gout is characterized by a painful and often disabling form of arthritis in joints, most often in the big toe. In gout, an excess of uric acid (due to an increase in production or a decrease in renal clearance) collects in the blood and tissues and sodium urate crystals deposit in the joints and elsewhere. Acute gouty attacks occur when tophi, an accumulation of the crystalline and amorphous urate, are formed.[49]

Classic presentation of a gouty attack is an erythematous, swollen joint – often the big toe or foot with severe pain with weight-bearing. The most common location is the MTP joint of the great toe. Elderly people, particularly women who are taking diuretics, may present with urate deposit in preexisting Heberden's nodes that may or may not be painful.[49,50] Some other types of arthritis can mimic gout, so a thorough history and evaluation is imperative.

The diagnosis of gout is primarily clinical. Confirmation of monosodium urate crystals (strong, negative birefringent needle-shaped crystals) via needle aspiration of the affected joint is the gold standard for diagnosis. As joint aspiration is not done in most cases, a diagnostic rule has been developed to aid in the clinical diagnosis. This rule uses the following seven criteria: male sex, previous arthritis attack, onset within 1 day, joint erythema, first MTP joint involved, hypertension, and uric acid >5.88.[47] Although this clinical tool can be useful, an atypical presentation or lack of response to usual therapy should trigger thorough evaluation, including joint aspiration.[47]

The treatment of gout flares in older patients is complicated by comorbidities, renal dysfunction, and other medications. NSAIDs and colchicine can be used in patients without contraindications. (Indomethacin should be avoided in older adults because of its higher risk of adverse effects.) Glucocorticoids (systemic or intraarticular) can be used for gout flares in patients in whom NSAIDs or colchicine may pose an increased risk.[47]

It is also important to emphasize diet and lifestyle changes. Urate-lowering therapy (xanthine oxidase inhibitors and probenecid) can provide long-term prophylaxis for those with recurrent attacks, along with concurrent well-managed comorbidities.[47]

Foot Problems and Falls

In older adults, foot problems are associated with increased risk for falls.[51] The Centers for Disease Control and Prevention (CDC) guidelines for assessment and management of falls recommend evaluation of feet and footwear as part of a fall prevention strategy. The CDC STEADI algorithm (Stopping Elderly Accidents, Deaths & Injuries) specifically mentions assessing feet and footwear, and providing education on shoe fit, traction, insoles, and heel height.[52] Health-care practitioners should incorporate this practice into the evaluation of all older adults at risk for falls, and individuals with foot-related risk factors should be referred for management. Routine assessment and management of foot problems could help to retain motor performance, improve lower limb strength and balance, and decrease fear of falling in community-dwelling older adults.[53,54]

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Care of the Geriatric Foot

Foot care is an important part of overall health care for older adults. Healthy feet are fundamental to mobility, comfort, and independence in function. All older patients should have a foot examination performed at least annually. Health-care practitioners are in an ideal position to perform this examination, provide basic education and treatment, and refer the patient to a specialist when needed.

Patient education includes a discussion about self-examination (using a mirror if needed), foot hygiene, skin care, foot safety, and proper footwear. In discussing foot safety, the older adult may be reminded to wear shoes or slippers even when at home to prevent injury. Socks made of cotton or wool are preferred because they can provide a barrier against wetness. Some sources recommend white socks because they can show drainage, alerting the patient to a possible problem.

Proper footwear is important for foot health. Patients should be warned to break in new shoes slowly to prevent injury.[55] There have been multiple studies evaluating the ideal footwear type for older adults at risk of falls. Older adults should wear shoes that fit appropriately both inside and outside the house because walking barefoot or in socks alone increases the risk of falls.[56] Ideal footwear elements for older adults may include a proper anatomical fit, a well-fitting toe box, a limited heel height, a broad enough heel, a firm insole and midsole, an outsole with sufficient tread and width, a beveled heel and shoe nose, a firm heel counter with snug fit, and an easy and effective closing mechanism.[57] Breathable materials that can stretch such as leather and canvas are preferable to plastic.[58]

Shoe modifications and orthoses can be used for patients at higher risk for foot injury or with foot deformities. For example, extra-depth shoes can provide more space for deformed toes. Orthoses can be used to support a joint, accommodate or correct a deformity, and redistribute weight.[59] There are multiple forms of orthoses available, and they can be made to specifically fit the patient's foot and needs. Sophisticated technologies (such as computerized gait analysis) can be used for difficult or complex cases.

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Chapter 46

Surgical Principles

Krista Haines, Vishnu R. Mani, and Sasha Adams

Rapid advances in medical sciences have significantly increased life expectancy over the past century, especially in developed nations.[1–3] The trends are catching up in the developing and underdeveloped nations as well in a proportional manner. The average life expectancy in Western Europe alone is 79 years of age for males and 84 for females,[1] while Northern America expects 77 years and 81 years,[3] respectively. All of these are in sharp contrast to Sub-Saharan Africa with an average life expectancy of only 61 years.[4] The global population of individuals >65 years as of 2019 stood at 703 million out of 7.8 billion (9%), with estimated projections of >1.5 billion (15.3%) out of 9.8 billion by 2050.[5,6] In the USA, older adults are about 54 million (16.45%) of the total 328 million people.

It is estimated that one in four individuals living in the USA would be aged >65 years by 2050.[7] Their impact on health care is greater than their proportion of the population. Older adults account for about 20% of emergency department (ED) visits annually,[8] and their resultant hospital admissions increased proportionally with age – 32.4% admissions for those aged 65–74, 37.2% for 75–84, and 43.4% for >85.[9–11] Older adults account for more than 40% of hospitalized adults[12–13] and almost half of the 3.5-trillion-dollar health-care expenditure annually.[14–15] For surgery specifically, almost 53% of all surgical procedures are performed on older adults across the USA,[16] and it is projected that half of those aged >65 will require surgery once in their lives.[17] Advanced age is accompanied by physiological changes with altered intrinsic homeostasis due to the increased likelihood of chronic disease states, multimorbidity, and polypharmacy. This very understanding is inherent to the principles of surgical care and outcomes in this population. This chapter will summarize the essentials of perioperative care for older adults undergoing surgery, processes that can lead to optimal outcomes.

Preoperative care of the older adult. A thorough assessment of an individual's health is essential to providing ideal care for the older surgical patient. While preoperative management core principles will mirror that of any age group, certain special considerations are needed in this population.

History and physical exam. The history and physical exam, when performed comprehensively, will set the stage for the complete understanding of the disease process and goals of care, and direct the physician to appropriate referrals, laboratory and radiological tests, and required management strategy. Alcohol use needs to be recorded. In a study of community-dwelling older adults, 62% were found to use alcohol, and 13% abused it.[18] Hence it is important to use available tools such as the CAGE questionnaire[19] to identify those with alcoholism. Preoperative alcohol dependence has been linked to prolonged hospitalizations, increased postoperative complications, and morbidity, with added health-care expenditures.[20,21] Hence, identifying and implementing a pharmacological and behavioral strategic intervention to achieve abstinence and prevent withdrawal should be undertaken and carried forward for at least 4–8 weeks to significantly reduce postoperative complications.[22,23] Similarly, recognizing and addressing smoking and tobacco use is of paramount importance, as these are well known to be associated with increased complications, particularly pulmonary complications and poor wound healing.[24] An intervention of >6 weeks is of clinical benefit in reducing complications and decreasing associated mortality.[25] Social history must also include queries about recreational substance use without making the assumption that the older patient would not be involved in such activities.

Cognitive and psychological evaluation. Evaluation of cognitive function is a pivotal part of the preoperative evaluation in the older adult to assess for dementia or unrecognized delirium.[26,27] Any form of altered sensorium and declined neurological status contributes to worse postoperative outcomes, including increased length of hospital stay, ICU stay, pulmonary complications, infections, and in-hospital falls.[28,29] Delirium is a clinical diagnosis, and current reference standard

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diagnostic criteria are the 5th edition of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5)[30] and World Health Organization's *International Classification of Diseases*, 10th revision (ICD-10).[31] More than 24 delirium scales have been reported in the literature,[32,33] of which the confusion assessment method (CAM) is the most commonly used and validated.[34] There is a wide array of risk factors for the development of delirium, the most pertinent of which are dementia, comorbidities, polypharmacy, malnutrition, electrolyte abnormality, sensory, visual, and auditory impairment, substance abuse, depression, social isolation, sleep deprivation, urinary retention, occult disease, and medications.[35] Identification and treatment/optimization of risk factors will result in more rapid neurological recovery and better postoperative outcomes. Cognitive impairment can be evaluated with several available screening tools; the Mini-Cog is a widely accepted and pragmatic tool that can be used in the preoperative setting.[36] Similarly, screening for depression should be undertaken using an institution-specific patient questionnaire or the geriatric depression scale.[37,38] Patients with depression or related symptoms are more likely to develop postoperative delirium,[39] which is associated with poor functional recovery and increased likelihood of being discharged to a facility after surgery.[40,41] An assessment of a patient's decision-making capacity can be made at this point, and for those who lack capacity, a surrogate will need to assist with decision-making and informed consent.

Goals of care. Goals-of-care discussions are among the main tenets of older adult patients' perioperative management. This has become a lost art among some surgeons, and its importance in the surgical care of the elderly cannot be overemphasized. This is best addressed during the preoperative evaluation. The health-care team, patient, and family/caregivers should ensure that the patient's goals and treatment preferences are understood and align with the prescribed treatment plan. The treatment plan should be catered toward accomplishing the patient's goals, even if it means declining an otherwise medically recommended treatment. This is also the preferred time to discuss the possibility of specific outcomes postoperatively, such as the possible need for prolonged mechanical ventilation and/or long-term feeding access, postoperative functional decline, loss of independence, and the need for long-term skilled nursing facility placement. These conversations will allow providers to gauge the patient and caregivers' specific wishes in different clinical scenarios, especially in the event of postoperative complications. The physician should also initiate discussions that ensure all older adults undergoing surgery have an advance directive. The document should include designation of a health-care agent or durable medical power of attorney and preferences for "do not resuscitate" (DNR) and "do not intubate" (DNI) orders, and specify the extent of medical treatment that would be desired under various potential postoperative circumstances. Documentation of the discussion, preferences, and required forms should be entered into the electronic medical record as part of the preoperative evaluation. One study found that about half of older adult patients undergoing surgery required goals-of-care decisions in the final days of life,[42] indicating that older adult patients who need surgical intervention could benefit greatly from early goals-of-care discussions as part of the preoperative evaluation.[43]

Functional evaluation. Surgery results in stress and insult to the human body. An individual's homeostatic mechanisms help postoperative recuperation. However, surgical stress can be exacerbated in the elderly, and the ability to recover is primarily determined by the preoperative physiological reserve and comorbid conditions. Additional postoperative clinical challenges can also significantly alter the trajectory of the stress response recovery.[44,45] While the immunologic responses to surgical stress in older adults are beyond this chapter's scope, it is critical to recognize age-related impairment in homeostatic mechanisms that can cause an inadequate surgical stress response. These changes are best anticipated by preoperative functional status assessments and should be done preoperatively in all older adults. Traditional functional status measurements, including the number of blocks or stairs an individual can climb, might not be as relevant in older adult patients. Evaluation of the patient's ability to perform their activities of daily living (ADLs) is essential, using scales such as the Katz ADL index[46] and the Lawton scale to assess instrumental activities of daily living (IADLs).[47] The metabolic equivalent task score (METS) could also be employed.

As defined by Xue, frailty is a state of vulnerability caused by an age-associated decline in multiple physiologic systems, leading to decreased reserve and function as well as an alteration in the ability to cope with acute stressors.[48] Functional status and frailty are interrelated, therefore caregivers need to consider both of these within the preoperative evaluation. The two phenotypes of frailty described are the physical phenotype and the

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comprehensive multidomain frailty. While a comprehensive frailty evaluation using tools such as the Canadian Study of Health and Aging Frailty Index or the Edmonton Frailty Index might not always be possible in a preoperative setting,[49] shorter versions such as the modified frailty index (mFI) can be used. If functional deficits or frailty are identified preoperatively, especially before any elective nonemergent procedure, [50] preoperative interventions, including exercise programs and nutritional supplementation, may be useful.

Fall risk should be assessed with measures such as the Timed Get Up and Go test,[51] as a recent history of falls within 12 months is associated with increased postoperative complications, discharge to a rehabilitation facility, and high rates of hospital admissions.[52] Strategy to minimize these complications should be undertaken in the preoperative setting and tailored to the individual patient's needs, including but not limited to physical therapy and exercise to improve mobility and avoidance of medications that contribute to falls.[53] Such interventions can improve outcomes and quality of life after surgery.[54] Patients with functional impairment are at increased risk of postoperative complications such as further functional decline and institutionalization. An understanding of this relationship between function and outcomes should lead to a more thorough preoperative evaluation, including discussion of advance directives and the patient's goals of care.

Laboratory and radiology investigations. We suggest performing laboratory and radiological investigations as indicated by the history and physical exam and, most importantly, based on the scheduled procedure. It is also important to consider the patient's American Association of Anesthesiology classification class (ASA) and the presence of comorbidities when considering which tests will help prepare and optimize the patient for their procedure.[84]

Cardiopulmonary risk. Preoperative cardiac and pulmonary risk evaluation must be done for all older patients, as age >60 in itself is an independent risk factor for postoperative pulmonary complication.[55] Postoperative myocardial infarction in this age group is associated with elevated hospital mortality. The American College of Cardiology and the American Heart Association task force guidelines for perioperative risk determination for noncardiac surgery recommend judicious testing, curtailing unwarranted tests for low-risk procedures, and most importantly performing tests only if it would change the clinical management.[56]

Medication optimization and polypharmacy. Global studies indicate that older adults each take approximately 3–9 medications, and these numbers are even greater in North America.[57] The prevalence of inappropriate medication use in this population was found to be from 11.5–62.5%.[58] A comprehensive review of a patient's current medication is essential in the preoperative setting. This review must include over-the-counter medication, herbal supplements, vitamins, oral anticoagulants, aspirin (ASA), and statins. Over-the-counter medications must be expressly clarified, as patients might not disclose them initially, considering them to be irrelevant. All unwarranted medications need to be stopped; multiple tools are validated to assist in identifying inappropriate medications among older adults, including Beers Criteria,[59] Screening Tool of Older Persons' Prescription (STOPP), and Screening Tool to Alert Doctors to Right Treatment (START) criteria.[60]

While a comprehensive discussion of preoperative optimization of all medications is beyond this chapter's scope, we will briefly underscore pertinent medications. The following medications should be continued through the perioperative period: beta-blocker, calcium channel blocker, digoxin, statins, alpha-2-agonists like clonidine, inhaled beta-agonists, and inhaled and systemic glucocorticoids. Statins specifically are cardioprotective during the perioperative period by a meta-analysis of 28 randomized clinical trials.[61] Oral hypoglycemic agents can be continued until the morning of surgery; insulin dosing requires individual adjustments based on the type of insulin, type of surgery, duration of preoperative fasting, and expected length of surgery. Close monitoring of glucose levels during the perioperative period is generally recommended. The use/discontinuation of angiotensin-converting-enzyme (ACE) inhibitors needs to be individualized, especially for heart failure patients, while other patients might be able to stop these medications the night before surgery. No consensus statements currently exist on the perioperative use of diuretic medications, and these decisions must be individualized. Optimization of anticoagulation is best served with closed-loop communication with the surgeon and is based on the indication for which the patient is taking the medication. As a general rule of thumb, warfarin needs to be stopped 5 days before surgery, aspirin and clopidogrel 7 days, and direct oral anticoagulants (DOAC) comprising direct thrombin inhibitors and factor Xa inhibitors should be stopped at least 3–5 half-lives before elective surgery. These recommendations are not one-size-fits-all and require an individualized strategy for each patient.

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There are several exceptions concerning anticoagulation, including the need for bridging either in the outpatient or inpatient setting, or postponing surgery until it is safe to stop antiplatelet agents (i.e., recent coronary stents, based on stent type and duration). Aspirin use is permitted preoperatively by many surgeons based on the type of surgical intervention, but there is no overall consensus. Patient safety demands clear communication between the surgical team and preoperative providers regarding the best perioperative strategy for a patient on antiplatelet agents or anticoagulants.

Risk assessment and referrals. Individual system risk assessments are covered in the topics above. There are several preoperative and postoperative risk predictors; however, none has been shown to demonstrate superiority over other prediction models. The American College of Surgeons National Surgery Quality Improvement Program (ACS-NSQIP) preoperative risk calculator estimates surgical morbidity and mortality for patients. The risk calculator is part of a best practice guideline for preoperative assessment of older adults, developed by ACS in collaboration with the American Geriatric Society (AGS). Discussions of postoperative plans with patients should include setting expectations, planning for recovery time, discussing potential need for a rehabilitation facility versus home care, and other relevant social support topics. Such discussions are important in decreasing postoperative morbidity.[62] Providers must make appropriate specialty referrals for the patient for further testing or optimization as deemed appropriate. Lastly, consideration for prehabilitation, including physical therapy and nutritional assessment/optimization, can potentially enhance surgical outcomes.

Prehabilitation. This is described as a composite and comprehensive strategy to enhance the patient's preoperative condition and thereby improve postoperative outcomes.[63] Prehabilitation includes optimizing the patient's cardiorespiratory function, muscle function, and modifiable risk factors such as anemia, anxiety, smoking, comorbidities, and sarcopenia.[64] Improving cardiopulmonary status and planning a strategy for physical therapy in frail patients can significantly reduce postoperative complications and improve recovery and outcomes.[65] Preoperative malnutrition and sarcopenia are predictors of infection, delirium, anastomotic leaks, and wound dehiscence.[66] Decreased albumin levels are a strong predictor of 30-day mortality and morbidity post-surgery.[67] A nutritional assessment and preoperative optimization under the guidance of a dietician might include increasing dietary protein content; adding supplemental multivitamins, electrolytes, iron, or appetite stimulants; and improving the overall caloric dietary intake in a holistic preoperative approach to improving outcomes.[68]

Table 46.1 FIT CHAMP mnemonic for preoperative assessment of older adults

F – Frailty index, fall risk, functional status
I – INR – anticoagulants and titration and or bridging
T – Tests – lab, radiology, specialized testing
C – Cognitive evaluation, comorbidities
H – History and physical
A – Advance directives – goals of care
M – Medication review and optimization
P – Prehabilitation – risk prediction scores, optimization, nutritional, pulmonary referrals, preoperative pain control strategy

An easy-to-remember mnemonic that covers the essential aspects of preoperative care in the elderly as discussed above in this chapter is "FIT CHAMP" (Table 46.1). This could be used in locations where designated comprehensive preoperative geriatric assessment clinics are unavailable.

Intraoperative care of the elderly. A comprehensive discussion of intraoperative care is beyond the scope of this chapter. Physicians caring for the patients pre- and postoperatively should be aware of the impact of anesthetics, intraoperative medications, and procedures themselves on organ systems as well as the resulting clinical implications of postoperative physiologic alterations. The type of surgery, duration of surgery, amount and type of fluids received, blood products transfused, normothermia, normoglycemia, and intraoperative urine output are all important parameters that should be reviewed when providing peri- and postoperative care.

Postoperative care. In older patients, both physical and psychological postsurgical stress can lead to an imbalance in autonomic, endocrine, metabolic, and immune function.[69] Postoperative care must cater to the specific individual surgery performed and be based on the expected outcomes and complications of that particular surgery. Rather than delving into the specifics of care for individual surgical procedures, this section will discuss common postoperative complications that are responsible for major morbidity and poor outcomes in older patients.[70] These include: delirium, pulmonary complications, aspiration, malnutrition, urinary tract

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infections (UTIs), pressure ulcers, and functional decline (www.facs.org/~/media/files/quality%20programs/geriatric/acs%20nsqip%20geriatric%202016%20guidelines.ashx.)

Delirium is the most significant and major age-related cause of morbidity in the elderly during the postoperative period.[71] The reported prevalence ranges between 9% and 44%.[72] Similar to our detailed discussion in the preoperative section of delirium, postoperative delirium is best prevented by measures including pain control, removal of invasive lines and catheters, promoting normal sleep-wake cycles by minimizing overnight sleep interruptions, and optimizing medications. If delirium develops despite these interventions, timely identification can yield a more favorable prognosis. Identification is promoted by a screening strategy that must be performed as frequently as possible or at least twice daily with validated tools like the Confusion Assessment Method for the ICU (CAM-ICU).[85] Delirium may present as a hypoactive or hyperactive state. The etiology can be multifactorial, including postoperative pain, infection, electrolyte abnormalities, sleep deprivation, polypharmacy (especially psychotropic medications), constipation, obstipation, and urinary retention. Identifying the etiology, treating infections, correcting electrolyte or metabolic abnormalities, removing offending medications, assuring proper elimination, and providing multicomponent nonpharmacological therapy should be the first line of treatment.[73] Antipsychotics should be reserved for nonpharmacologic therapy failure or hyperactive delirium with a risk of harm to self or others.

Postoperative pulmonary complications are a major source of prolonged hospitalization and morbidity in any patient but more so in the older patient, as advanced age itself has been implicated as an independent predictor of postoperative pneumonia.[74] A common cause of postoperative pneumonia is atelectasis due to pain-restricted shallow breathing. Atelectasis can be prevented by routine incentive spirometry use, breathing exercises, chest physiotherapy, and ambulation. Pain-restricted shallow breathing can be prevented with optimization of pain control and epidural therapies where appropriate. The prevention of aspiration in this population is of critical importance. Aspiration risk can be reduced by promoting head-of-the-bed elevation, encouraging the patient to be out of bed to the chair during meals, and regularly performing bedside swallow assessments. For those patients with feeding tubes, residual volume measurements, close monitoring, and regular exams can give insight into possible pathology (i.e., gastroparesis or ileus), which could ultimately result in aspiration. Aspiration can occur in patients with feeding tubes who are intubated and mechanically ventilated. This risk must not be overlooked. Nutritional goals in the postoperative period are of paramount importance as undernutrition is a marker for adverse events, as previously described in this chapter.[75,76] Nutritional goals should include oral nutrition as early as possible with daily assessments, and where not feasible, feeding via nasogastric tube or parenteral nutrition should be started, especially for those expected to need prolonged bowel rest or inability to tolerate enteral food for more than 7 days.

UTIs represent roughly 32–40% of all nosocomial infections,[77] therefore urinary catheters should be removed as soon as possible, unless there is a surgical or pathological reason necessitating an indwelling catheter. Catheter alternatives are available and should be considered, including external female catheters and condom catheters.

Preventable pressure ulcers develop in up to 60% of hospitalized older adults.[78] Preventive measures should be employed for all hospitalized nonmobile patients, such as frequent turning, pressure-reducing mattresses, and soft pads on pressure points. Institutions should have pressure ulcer prevention protocols established. Objective assessments with an available tool such as the Waterloo, Norton, or Braden scale[79–81] should be done and recorded in the medical record.

Older adults have an increased risk of a conglomeration of frailty, cognitive impairment, depression, falls, incontinence, pressure ulcers, and decreased social supports that can render them functionally impaired postoperatively. A keen and thorough understanding of their postoperative risk and a multimodal clinical approach will yield the best possible outcomes.

Acute Care Surgery and Trauma

The approach to acute and trauma surgery in older adults will mirror that of any age during the preoperative phase because of their presentation's acuity. Nevertheless, providers and patients must understand that not all older adults presenting with critical life-threatening surgically correctable conditions will tolerate surgery in the same way as their younger counterparts. This concept must be quickly realized and prompt immediate goals-of-care discussions initiated with the patient and/or their family, if the patient lacks full decision-making capacity. While this might be a difficult decision, certain patients with multimorbidity and polypharmacy presenting with critical

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illness or injury, including severe septic shock and ischemic bowel, will likely not survive their surgery and may die in a way that does not align with their wishes. Other factors to consider include anticoagulant use, which may require emergent optimization before surgery. Patients on antiplatelet medications or direct oral anticoagulants with life-threatening head trauma or need for emergent surgery will need rapid reversal of these medications, and depending on the agent used, the options for reversal can be both expensive and difficult to obtain.

Common acute care surgeries in older adult patients include hip and knee surgery secondary to falls, or abdominal operations for pathologies such as bowel obstructions, diverticulitis, ulcer perforation, colonic volvulus, incarcerated hernia, gastrointestinal bleeding, or complications of diagnosed or undiagnosed malignancy. Older adults also experience higher mortality than their younger counterparts because of trauma regardless of mechanism. One third of all older adult trauma patients with a low to moderate level of injury severity may eventually succumb to their injury during hospitalization. Motor vehicle crashes are second only to falls as the most common cause of injury of older adults. However, motor vehicle crashes still are the most common cause of traumatic mortality.[82,83] All traumas in older adults, regardless of mechanism, require advanced postoperative care with a multimodal preventive approach for minimizing complications and achieving optimal outcomes. Regardless of whether emergent surgeries are due to traumatic injury or life-threatening pathology, the concepts of medical ethics, including sound communication, patient autonomy, and goals-of-care discussions, are just as important, if not more so, despite the time limitations to make informed decisions.

Summary

Given the rapidly growing medical needs of the increasing population of older adults, all physicians should prepare to provide holistic, comprehensive, age-specific care regardless of their specialty. This requires disseminating information for increased awareness among surgeons through official bodies like the American College of Surgeons and the American Geriatric Society. ACS and AGS have recognized this future challenge and have developed a collaborative approach – the 2016 ACS-NSQIP/AGS best practice guidelines: Optimal Preoperative Assessment of the Geriatric Surgical Patient (www.facs.org). A sound foundation of perioperative knowledge of the specific unique considerations for older adults, as described in this chapter, along with a comprehensive multidisciplinary team approach will yield optimal outcomes.

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Chapter

47 Rehabilitation

Paul Thananopavarn, John Baratta, and Tanya E. Zinner

Introduction

One of the most challenging and important aspects of geriatrics is maintaining an elderly person's function and quality of life. Functional independence is certainly one of the top priorities of people as they age. In a survey conducted by the National Council on Aging, Americans' greatest concerns about growing older were "not being able to take care of myself" and "being a burden."[1] Unfortunately, a multitude of physiologic changes that come with age lower a person's functional reserve capacity to withstand and recover from insults due to illness or accidents. However, the resulting impairments are often amenable to rehabilitation in an effort to restore function and independence.

Age and Function

Functional aging may follow one of four models or paths.[2] The first model represents a person of good health and a large functional reserve who never becomes dependent with age, but then has a sudden catastrophic event or illness that leads to a quick demise (Figure 47.1). The second model describes a person who suffers from a series of debilitating events, but is temporarily able to recover most, but not all, function through healing and rehabilitation (Figure 47.2). The third model is of a person with a low functional reserve due to aging and disease who slips easily into a state of dependence (Figure 47.3). These models emphasize: (1) the importance of maintaining a large functional reserve through preventive care, exercise, nutrition, and good fortune, and (2) that rehabilitation can be useful whenever there is a loss of function, but has the greatest potential in those with a high baseline functional status. The fourth model (Figure 47.4) relates to persons aging with a developmental disability. Rehabilitation medicine helps maximize a disabled person's potential early in development and may also be necessary later in life to recover function and quality of life. As more people survive and live longer with

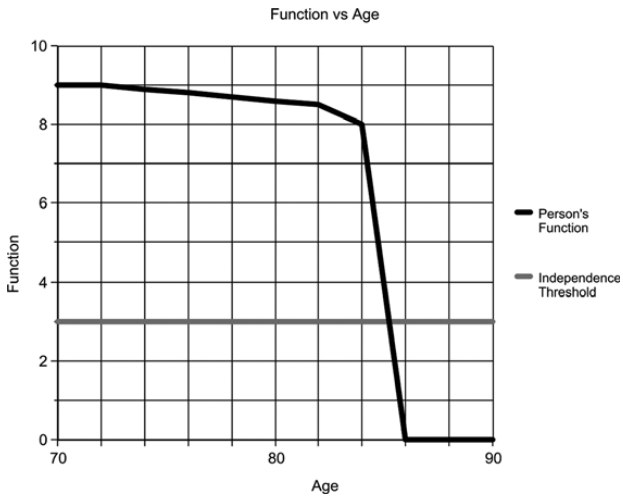

Figure 47.1 Elder with a high functional reserve who suffers a catastrophic illness.

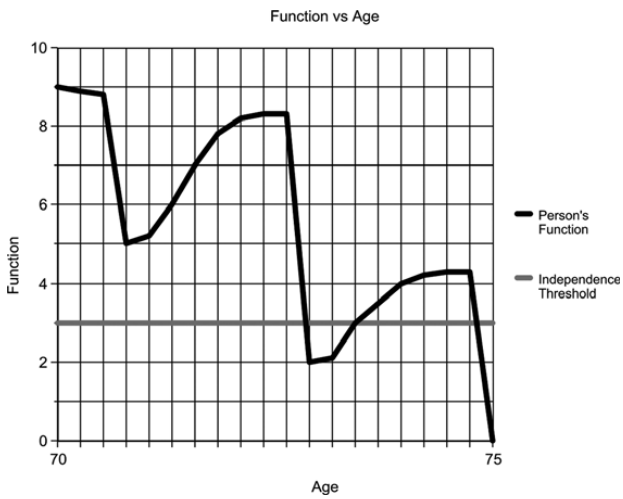

Figure 47.2 Person with a high functional reserve who suffers debilitating events but is able to recover through rehabilitation.

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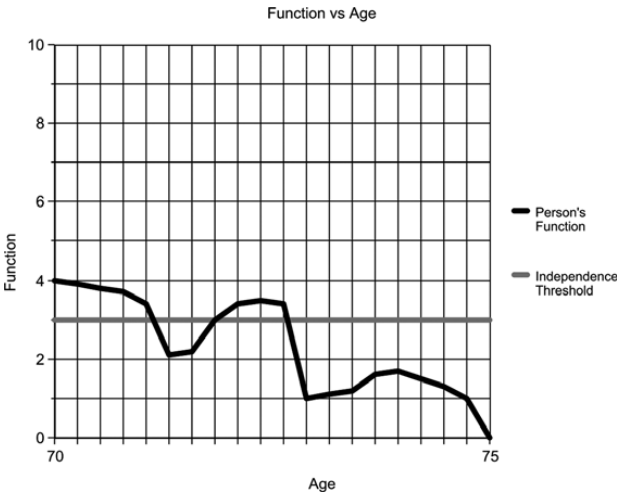

Figure 47.3 Elder with low functional reserve who declines into a state of dependence.

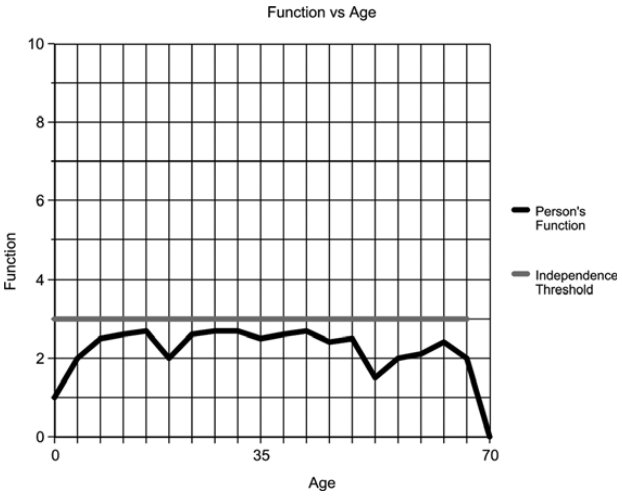

Figure 47.4 Person with a significant developmental disability who requires rehabilitation interventions early in life, then periodically during the aging process.

developmental or early-acquired disabilities, there will be more challenges in maintaining their function and the function of their caregivers in the face of aging.

Rehabilitation medicine physicians (physiatrists), geriatricians, and others caring for the elderly often find that there may be no perfect treatment for a disease or functional impairment associated with aging. Rather, the central focus is to find a compensatory mechanism to help restore function. A simple example would be a hearing aid for presbycusis, which does not completely restore hearing but aids the elder in day-to-day interaction. More serious impairments, such as spastic hemiparesis from a stroke, have no simple restorative device and will have profound effects on the person's function, psychological state, social role, and relationships. The rehabilitation of this individual involves a complex process that will require using a broader biopsychosocial model of medicine.

Impairment, Activity, and Participation

The World Health Organization provides a contextual framework to understand how a loss of a body function affects a person's ability to care for themselves and fulfill a role in society through the terms Impairment, Activity, and Participation (Table 47.1).[3] The terms Activity and Participation have replaced the older terms Disability and Handicap, respectively.

A person suffering from a hip fracture from a fall provides an excellent example of how to apply our understanding of the Impairment, Activity, and Participation model as a framework for the rehabilitation process. First, the fracture must be fixed and direct medical consequences of surgery addressed. Second, skilled physical and occupational therapy is employed to improve the activities of mobility and self-care. Lastly, consideration of the person's motivation, pre-fall and post-surgery functional status, home environment, and caregiver support will determine whether they may recover at home or in a short-term rehabilitation facility. Ideally, with successful rehabilitation, the patient may regain full participation in life and return to independent living.

The Functional Assessment

A physician or practitioner leading the rehabilitation efforts of an elder must first assess and optimize any medical problem that may act as a barrier to rehabilitation. For example, cognitive impairments from delirium, dementia, or depression may retard a patient's participation in mobility training and the re-learning of self-care skills. Poor cardiac or pulmonary status will reduce endurance and tolerance to therapy. Arthritic joint pain may limit range of joint motion and weight-bearing ability. Other complications of immobility, including pressure ulcers, venous thromboembolism, constipation, urinary incontinence, and disuse myopathy, need to be prevented or addressed, as they can limit progress toward functional independence. Once the geriatric medical evaluation has been completed, the rehabilitation practitioner can focus on

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specific components of the geriatric assessment related to function and rehabilitation potential.

Premorbid Functional Status

A person's functional status prior to the onset of an illness or impairment is one of the most important factors in determining rehabilitation potential.[4] If an elder suffers an injury causing a loss of function, rehabilitation can hope to improve the person's function close to their baseline premorbid functional status.

Table 47.1 Definitions of impairment, activity, and participation

Term	Definition	Example
Impairment	Loss of body part or function	Broken hip from a fall
Activity (Disability)	Loss of ability to perform an activity	Inability to walk and perform basic self-care
Participation (Handicap)	Loss of the ability to participate in a life situation	Unable to live independently at home

Current Functional Status

A person's functional status with a new impairment determines how much and what type of rehabilitation is necessary. There are many instruments to measure and communicate function, and in the past, different post-acute settings used their own measures. The Centers for Medicare and Medicaid Services (CMS) now mandates the use of the Functional Assessment Standardized Items (FASI)[5] across all post-acute care settings, allowing for better comparison of functional outcomes. This tool includes seven self-care and six mobility items (Table 47.2). The FASI is scored on a six-point scale (Table 47.3).

Intact communication and cognitive function are vital to learning new skills and regaining independence. There are many measures of cognitive function including the Montreal Cognitive Assessment, Mini-Mental State

Table 47.2 Functional Assessment Standardized Items

Self-care items:		
	Eating	The ability to use utensils to bring food to the mouth and swallow food once meal is placed before patient.
	Oral hygiene	The ability to use suitable items to clean teeth or use dentures.
	Toileting hygiene	The ability to maintain perineal hygiene, adjust clothes before and after voiding or having a bowel movement.
	Shower/bathe self	The ability to bathe self, including washing, rinsing and drying self. Does not include transferring in/out of tub/shower.
	Upper body dressing	The ability to dress and undress above the waist, including fasteners.
	Lower body dressing	The ability to dress and undress below the waist, including fasteners; does not include footwear.
	Putting on/taking off footwear	The ability to put on and take off socks and shoes or other footwear that is appropriate for safe mobility.
Mobility items:		
	Sit to lying	The ability to move from sitting on side of bed to lying flat on the bed.
	Lying to sitting on side of bed	The ability to move from lying on the back to sitting on the side of the bed with feet flat on the floor, and with no back support.
	Sit to stand	The ability to come to a standing position from sitting in a chair, wheelchair, or on the side of the bed.
	Chair/bed-to-chair transfer	The ability to safely transfer to and from a bed to a chair (or wheelchair).
	Toilet transfer	The ability to get on and off a toilet or commode.
	Walking	Once standing, the ability to walk at least 10 feet. Additional measures for walking 50 feet with two turns and walking 150 feet.
	Additional walking	For ambulators there are additional measures for distance walked, stairs, and ability to pick up objects.

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Table 47.3 FASI scoring system

Score	
6. Independent	Patient completes the activity by him/herself with no assistance.
5. Setup or cleanup assistance	Helper sets up or cleans up; patient completes activity. Helper assists only prior to or following the activity.
4. Supervision or touching assistance	Helper provides verbal cues and/or touching, steadying, or contact guard assistance as patient completes activity.
3. Partial/moderate assistance	Helper lifts, holds, or supports trunk or limbs, but provides *less than half* of the effort.
2. Substantial/maximal assistance	Helper does *more than half* of the effort.
1. Dependent	Helper does *all* of the effort. Patient does none of the effort to complete the activity. Or the assistance of 2 or more helpers is required for the patient to complete the activity.

Exam, and the Mini-Cog. The FASI uses the Brief Interview for Mental Status (BIMS) score for a gross assessment of cognitive function. The BIMS consists of three parts: repetition of three words, temporal orientation, and recall.[6]

The function of the bowel and bladder also need careful assessment, as retention or incontinence can interfere with therapy and lead to more dependence for care.

Living Environment and Equipment

A person's home living environment, including accessibility, is important to establish early in the rehabilitation process. Is the abode one- or two-story? If it is a two-story house, is there a possibility for a first-floor setup? How many stairs are there to get into the house? Are there handrails? These are just some of the questions that are necessary to determine the achievable functional goals that are needed to enable a person to return and thrive at home. For a person who is expected to be wheelchair level, plans for a ramp and measurements of door widths need to be done early in case construction or modifications are necessary. An elder's current stock of equipment and assistive devices, such as walkers, wheelchairs, commodes, and braces, needs to be assessed with the assistance of therapists for condition, fit, and appropriateness.

Social and Caregiver Support

The presence of a willing and able caregiver or supportive community of helpers is vital in the rehabilitation process, particularly for elders with major functional impairments. Often, there are limits to what can be achieved during the initial rehabilitation process, so caregivers must be identified early as part of the rehabilitation team, trained, and supported as they care for the elder through extended rehabilitation efforts at home. In the post-acute system, caregivers are important in the following ways: (1) a patient with a caregiver is more likely to be admitted to an inpatient facility for intensive rehabilitation, as the potential for home discharge is greater; (2) in the subacute rehabilitation setting, a person with a caregiver is more likely to return home than be relegated to long-term nursing home care.

Barriers to Rehabilitation and Functional Goals

Asking an elder's perception of why they think they have a functional problem may reveal underlying problems and barriers to the rehabilitation process. For example, asking a patient hospitalized for sepsis why they are unable to walk can reveal a new problem such as knee pain from an acute gout attack. Besides pain, other common barriers to assess are bowel and bladder function and impaired cognition due to delirium or depression.

Inquiring about a person's functional goals will assist with assessing motivation and with rehabilitation planning. An example would be asking a traumatic trans-tibial amputee their ultimate rehabilitation goal. If their goal is to be able to jog like they did before they lost their leg, a practitioner will plan for higher-level prosthetic components and intensive physical therapy for gait training. On the other hand, if the same patient feels that they are depressed and have no interest in walking, a referral to a psychologist and an amputee support group may be a priority.

The Rehabilitation Physical Exam

A comprehensive general geriatric physical exam should be completed; however, in the rehabilitation setting, more focus is placed on the neurologic and musculoskeletal aspects of the exam, as these two systems have the most effect on mobility, self-care skills, and cognitive function.

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Table 47.4 The rehabilitation team

Physical therapist	Specializes in improving a person's mobility, including balance and gait. May recommend assistive devices such as canes and walkers. May use manual therapy and administer physical modalities such as heat and therapeutic ultrasound.
Occupational therapist (OT)	Specializes in improving basic self-care skills (activities of daily living), such as bathing and dressing, and higher-level instrumental activities of daily living, including cooking and driving. May recommend home modifications and adaptive equipment such as reachers or commodes. OTs can manufacture functional splints and can improve function in persons with vision impairment or neglect using compensatory techniques.
Speech therapist	Focuses on evaluating and treating speech, voice, language, and cognitive impairments. Can evaluate and treat dysphagia.
Rehabilitation nurse	Monitors vitals, administers medications, performs wound care, manages bowel and bladder problems, assists with patient education and safety.
Recreational therapist	Specializes in the reintegration of the patient back into the community. Works on reducing stress and pain through relaxation techniques and diversion.
Neuropsychologist	Evaluates and treats cognitive, behavioral, and psychological impairments.
Orthotist/ prosthetist	Manufactures and fits assistive devices such as ankle foot orthoses or prosthetic limbs to be worn by the patient.
Social worker	Identifies and coordinates resources to help the elder receive necessary services and equipment.
Primary physician/ physiatrist	Evaluates the elder, manages medical issues, coordinates services, and orders equipment. A physiatrist is a physician with specialized training in rehabilitation medicine and the care of individuals with disabilities.

The Rehabilitation Team

The rehabilitation of an elderly person can be a complex process, requiring the expertise of multiple therapists and medical professionals. It is incumbent on a practitioner to understand the unique skills of each professional to be able to call on appropriate members to form an effective rehabilitation team. The patient and family are always the focus of our efforts and are considered active members of the team (Table 47.4).

Rehabilitation Settings

Multidisciplinary versus Interdisciplinary Rehabilitation

Rehabilitation of an elderly person can potentially succeed in several different settings, including the hospital, inpatient rehabilitation facility (IRF), skilled nursing facility (SNF), outpatient rehabilitation center, assisted living facility, or the home. In higher-level rehabilitation settings, including IRFs and some SNFs, the rehabilitation process is carefully coordinated and planned between therapy, medical, and social support services, making the experience an interdisciplinary effort. The group activity of this interdisciplinary team is synergistic, producing more than what the team could produce separately.[7] In the outpatient or home setting, multiple therapy and medical services are available but usually not closely coordinated, making the experience multidisciplinary rather than interdisciplinary.

Inpatient Rehabilitation Facilities

IRFs are suitable for patients with complex rehabilitation, nursing, *and* medical needs, such as a person with a spinal cord injury, amputation, burn, major multiple trauma, traumatic brain injury, or neurologic disorder such as stroke, multiple sclerosis, or Parkinson's disease. Elders who have suffered hip fractures can qualify for inpatient rehab if there is sufficient medical need to remain in a hospital setting. Patients must be able to participate in 3 hours of therapy a day, 5 days a week, in at least two disciplines of physical therapy (PT), occupational therapy (OT), or speech therapy (ST). Severely debilitated medical patients with complex medical problems such as renal failure, recent organ transplant, cancer, or left ventricular assist devices (LVAD) for heart failure may also qualify if they have sufficient therapy needs to justify intensive rehabilitation. The rehabilitation team is headed by a physician (most often a physiatrist) who formulates a detailed treatment plan, visits the patient at least three times a week to address medical and therapy issues, and leads weekly interdisciplinary team meetings that proactively address any barriers to rehabilitation. A typical length of stay is 14 days, with the expectation that the patient's goal is to return to the community, not to an SNF. Medicare Part

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A covers inpatient rehabilitation as part of the elder's inpatient hospitalization days, with an average cost to Medicare of $1,500 a day.[8]

Subacute Rehabilitation

Many nursing facilities have beds designated for skilled nursing and rehabilitation, along with long-term care beds for elders needing custodial care. SNFs are suitable for patients with more straightforward or less medically complex conditions such as hip fractures, elective joint replacements, or debility due to a common illness like pneumonia or urinary tract infection sepsis. In most cases, to qualify for Medicare-covered skilled services, a patient must have skilled nursing needs (IV antibiotics, enteral tube feeds, wound care) *or* rehabilitation needs in either PT, OT, or ST. Patients typically get 1–2.5 hours of therapy a day with no minimum participation requirement. A physician must visit the patient upon admission and at least once every 30 days; however, a patient undergoing rehab is often seen one or more times a week by a physician or advanced practice provider (physician's assistant or nurse practitioner). The typical cost of an SNF rehabilitation stay is $500 a day.[9] Medicare will cover 100% of an elder's stay at an SNF for the first 20 days and 80% of the cost thereafter for a total of 100 days, as long as there is a skilled nursing or therapy need. More often than not, a person will improve, plateau with therapy gains, and no longer qualify for Medicare-covered skilled nursing or therapy services before the 100-day mark. At that point, the elder returns to the community or is responsible for covering the cost of custodial care in the nursing home, unless the person has Medicaid or long-term care insurance coverage.

Home Health and Outpatient Rehabilitation

Intermittent multidisciplinary therapy and skilled nursing can be provided in the home or assisted living facility if the patient is homebound and has sufficient caregiver support. Each type of therapy or nursing service can visit the patient two to three times per week at home. Once the elder is not homebound, outpatient therapy can provide longer, more intensive therapy, often with equipment not found in the home. The cost to Medicare is in the range of $100–$200 per visit and is subject to Medicare payment caps.

Selecting the Rehabilitation Setting for the Elder

The studies that have attempted to compare rehabilitation outcomes for stroke,[10,11] hip fracture,[12,13] and joint replacement[14,15] patients in various settings (IRF, SNF, or home health [HH]) do not definitely favor one setting over another. However, IRFs tended to demonstrate better functional outcomes than SNFs, with more patients returning to the community and fewer patients being readmitted to the hospital.[16] However, the results are confounded by selection bias, lack of uniform functional measurements, and the fact that many patients will progress through multiple rehabilitation settings. In these studies and in practice, IRF patients have more complex medical and rehabilitation diagnoses, yet are able to tolerate 3 hours of therapy. SNF patients tend to be older and have cognitive deficits. HH patients are more likely to be younger, healthier, and have good home support.[11]

Fee-for-service Medicare expenditures on post-acute care rose to $58.6 billion a year in 2018,[8] partly because Medicare reimburses each rehabilitation setting separately each time an elder accesses their services. To restrict access to expensive post-acute care, Medicare places artificial barriers to access such as the 3-hour daily therapy rule for IRFs, 3-day qualifying stay for SNFs, and homebound status for HH. Recently, Medicare has implemented alternative payment models that integrate payment for post-acute rehabilitation care across an episode of care or a population of beneficiaries with the aim of providing quality care at less cost. Under these models, rehabilitation at home or in an SNF is preferred over a more costly IRF stay if quality metrics can be met and readmissions to the hospital are kept at a minimum.

Geriatric Assistive Devices

Practitioners often order assistive devices for an elderly person to enhance safety with mobility or independence with self-care. Each device has the potential to help, but its use comes at an increased financial and energy cost. Improper or ill-fitted equipment can also pose a safety hazard that promotes a fall. A practitioner must know the potential benefits and risks of a piece of equipment and seek the assistance of a therapist who can help properly fit the device and train the elder on proper use.

Canes provide an extra point of contact with the ground, adding stability and tactile information about the ground with walking (Figure 47.5). A simple hook cane is useful for improving walking balance and proprioception but can be unstable, as the force placed on the handle is not directly over the point of ground contact. A cane with an offset handle places the handle's center of gravity directly over the ground contact point, allowing

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Figure 47.5 Four-point and single-point cane with offset handles. Forearm crutch.

more stable weight-bearing. No more than 15% of body weight should be placed through a cane. The cane should be fitted so that the top of the handle comes to about the wrist crease or the elbow is flexed about 15–20 degrees when holding the cane.[17] To offload a painful lower-extremity joint, the cane should be held by the contralateral hand and advanced with the painful limb. Canes can come with foot attachments with three or four points of contact, increasing the cane's stability, but at the cost of more weight, less natural gait pattern, and potential difficulty with stairs.

Axillary crutches are rarely used in the geriatric population as they are difficult to handle, inherently unstable, and may cause axillary nerve compression if improperly used. Forearm or Lofstrand crutches have a cuff around the proximal forearm that allows hands to be free without dropping the crutch. They are easier to maneuver than axillary crutches and are often used for gait training after joint replacement surgery.[18]

Walkers provide a large base of support that can assist in walking stability. With the use of both upper extremities, a walker can help offload a lower extremity. The disadvantages of walkers are that they can be difficult to maneuver, especially with stairs, promote poor back posture, and decrease arm swing. Standard walkers with no wheels are very stable but require picking up to move forward, which slows down gait and requires significant upper body strength. They may be helpful for people after lower-extremity surgery with weight-bearing restrictions or for people with cerebellar ataxia. More commonly, elders use two-wheeled walkers or four-wheeled "rollator" type walkers (Figure 47.6). Two-wheeled walkers maintain a more normal gait pattern compared to standard walkers and can still be used for weight-bearing. Rollators may be too easily moved to prevent a fall and therefore cannot be used for significant weight-bearing. Rollators are well suited to help elders who need frequent rest breaks or use oxygen, as they often have fold-down seats or baskets to carry oxygen or other personal items.[19]

A manual wheelchair can be used by an elderly person if they are unable or unsafe to walk, or it can be used to help a caregiver transport a patient. A wheelchair needs to be carefully fitted to the patient by a therapist or wheelchair vendor to maximize its functional utility. Some considerations may include selecting: the proper seat height to allow propulsion with feet along with hands, elevating foot rests for comfort or edema control, detachable arm rest for slide board transfers, specialized seat cushion to prevent pressure ulcers, customized seat back to compensate for postural deformities, and lightweight design to reduce work to manually propel the chair or to lift the wheelchair for transport.[20]

Elders with severe weakness may require the assistance of a power-operated mobility device (Figure 47.7). A powered wheelchair's benefit of enhanced longer-range mobility at home and in the community must be balanced against the risk of worsening weakness, balance, and endurance from muscle disuse. Motorized systems are costly, potentially difficult to transport, and require extensive documentation for Medicare payment approval. Powered scooters can be a less costly alternative that may be lighter and easier to disassemble/transport, but have a wider turning radius and fewer seating customization possibilities as compared to powered wheelchairs. Scooters may be difficult to transfer into and cannot accommodate changes that come with progressive functional decline, such as those encountered in patients with multiple sclerosis or amyotrophic lateral sclerosis.

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Figure 47.6 Two-wheeled and four-wheeled walkers.

Figure 47.7 Motorized wheelchair and scooter.

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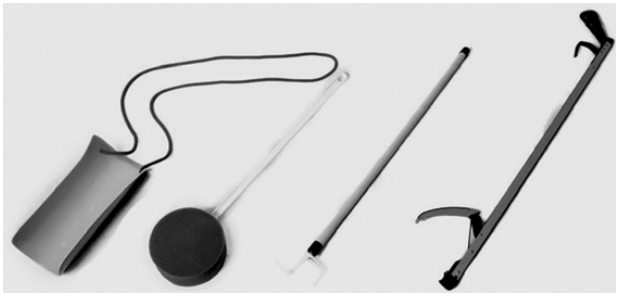

Figure 47.8 Sock aid, long-handle sponge, dressing hook, reacher.

All powered mobility requires intact cognitive faculties and "behind the wheel" testing and training to ensure safe use.[20]

An assortment of aids to help the elderly with self-care activities can be prescribed, usually with the assistance of an occupational therapist. These include simple devices such as mechanical "reachers" to pick up objects, dressing hooks to pull up pants, tub benches for safer bathing, and raised commodes to help with toilet transfers (Figure 47.8). Some centers are exploring ways of using robotics, brain–machine interfaces, regenerative medicine, and other technologies to restore movement and vital functions. Despite our technological advances, we are many years away from being able to replace the most valuable and versatile aid of all: a human caregiver.

Rehabilitation of Common Geriatric Problems

Stroke

With approximately 800,000 Americans experiencing a stroke each year, stroke has become the leading cause of long-term disability. It is a concern for elders, as the increasing prevalence of stroke is strongly correlated with advancing age. Approximately 11% of men and 13% of women over age 80 have been affected by stroke.[21]

While procedural and pharmaceutical interventions have made improvements in the treatment of acute stroke, a large portion of survivors are unfortunately left with moderate or severe disability. The most common impairments after a stroke include motor weakness, sensory deficits, and problems with speech, language, cognition, swallowing, and vision. Spontaneous neurological recovery mostly occurs within the first 3 months poststroke, with the most rapid functional recovery occurring in the first 30 days. Usually, there is very little functional motor recovery after 6 months poststroke, but swallowing, speech, and sensory dysfunction may gradually improve over a longer period of time.[22] Some poor prognostic signs include no recovery in the first 3–4 weeks after a stroke, flaccid paralysis at 4 weeks poststroke, severe neurological neglect, severe cognitive deficits, and advanced age.[23] Older stroke survivors may have a poorer stroke recovery prognosis given their increased likelihood of comorbid medical conditions and prior strokes.[24]

Traditional rehabilitation therapies attempt to enhance natural motor recovery and control using physical sensory and motor stimulation. Rehabilitation also includes task-oriented therapy where rehabilitation is focused on acquisition of skills for performance of meaningful and relevant tasks. Programs aim to be challenging, progressive, and optimally adapted to the patient's capabilities and environment, and invoke active participation to prevent learned disuse.[25] An example of this is constraint-induced movement therapy (CIMT), a technique in which a hemiparetic patient uses the weak limb while the strong, unaffected limb is constrained by wearing a mitt. CIMT with forced-use therapy has been shown to promote cortical reorganization and motor recovery.[26] A practiced form of gait therapy for hemiplegia is body weight-supported treadmill training (BWSTT), which involves the patient being suspended over a treadmill with the therapist assisting in leg movement through the gait cycle (Figure 47.9). BWSTT has shown superior effectiveness over traditional gait training, but requires specialized equipment and significant effort from a therapist moving the paretic leg.[27] Other interventions under study that may prove helpful in enhancing recovery include functional electrical stimulation,[28] use of neurostimulant and antidepressant medications to augment functional recovery,[29] virtual reality therapy,[30] and noninvasive brain stimulation using magnetic fields or direct transcranial current to activate dormant brain tissue.[31]

Spasticity is an upper motor neuron condition where muscles are involuntarily contracting because of damage of the central nervous system involved in movement. The associated stiffness or tightness may cause difficulty with performing activities of daily living (ADLs) or walking but may assist gait in some circumstances by preventing knee buckling. Therapy for spasticity is designed to reduce muscle tone, maintain or improve range of motion and mobility, increase strength and coordination, and improve comfort.[32]

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Figure 47.9 Body weight-supported treadmill training.

Therapy may include stretching and strengthening exercises, limb positioning, application of cold packs, and electrical stimulation. Oral centrally acting medications such as baclofen, tizanidine, and diazepam can be used to treat spasticity, but must be used with caution in the elderly, as they can cause sedation or confusion. Alternatives include botulinum toxin injections and phenol nerve blocks that reduce muscle tone without causing sedating side effects.

Orthotics are often prescribed to reduce or control spasticity while preserving range of motion and enhancing joint stability in paretic limbs.[33] A common upper-extremity orthosis fashioned by occupational therapists is the resting hand splint. This orthotic prevents contractures and reduces spasticity by keeping the wrist and fingers in relative extension, thereby breaking up the flexion synergy pattern. For a paretic or spastic lower extremity, an orthotist is commonly asked to manufacture ankle foot orthosis (AFO). AFOs come in many forms and have the potential to reduce foot drop by limiting plantar flexion in the swing phase of gait. In stance phase, an AFO limits dorsiflexion of the ankle, which in turn enhances knee extension and reduces knee buckling. Elongating the foot plate to prevent toes from curling under can interrupt plantar flexion synergism, reducing spasticity and the incidence of ankle contractures.

Pain after stroke commonly occurs in the hemiparetic shoulder and must be addressed for elder comfort and to allow progress with therapy. The cause of poststroke shoulder pain can be challenging to ascertain and requires a proper shoulder exam and at times radiographic imaging. The differential diagnosis includes glenohumeral subluxation due to muscle weakness, bursitis/tendonitis/rotator cuff tear from improper pulling or positioning of the arm, adhesive capsulitis due to spasticity and immobility, heterotopic ossification, and complex regional pain syndrome.[34] To reduce shoulder subluxation, it is important to support the arm while the patient is seated or standing using a vertical cuff sling or wheelchair lap tray. However, classic arm slings that promote adduction and internal rotation of the arm can increase the risk for developing adhesive capsulitis or frozen shoulder. Early hemiplegic shoulder rehabilitation goals include pain prevention using gentle range of motion, proper positioning, spasticity control, and caregiver education. However, once pain begins, a practitioner may need to consider physical modalities, massage, shoulder taping, topical and oral pain medications, neuromuscular electrical stimulation, or corticosteroid injections,[35] depending on the pain etiology.

Dysphagia is a common sequelae of a stroke, occurring in approximately 40% of stroke patients.[36] A speech and language pathologist (SLP) screens for swallowing dysfunction by identifying signs such as impaired cough or gag, drooling, coughing or choking with meals, pocketing of food, and dysphonia. High-risk patients will need to undergo a modified barium swallow or fiberoptic endoscopic evaluation of swallowing (FEES) to assess the severity of dysphagia and to determine fluid and meal consistency recommendations. Techniques taught by SLPs to reduce aspiration risk include elevating the bed to at least 30 degrees, turning the head to the paretic side, and performing a chin tuck when eating.

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Severe dysphagia may require alternative routes of nutrition intake, including consideration of a gastrostomy tube.[37]

Cognitive and communication impairments are influenced by the location of the stroke and are evaluated and treated by an SLP. Aphasias can be broadly categorized into expressive (Broca's), in which a patient has difficulty with speaking but has intact comprehension, or receptive (Wernicke's), in which comprehension is impaired but speech is intact. Patients with impaired comprehension are a challenge to rehabilitate given their limited ability to understand and follow commands. Another factor affecting cognitive performance may be depression, which affects approximately 31% of patients in the first 5 years after stroke.[38] Risk factors for poststroke depression include prior psychiatric history, significant impairment in ADLs, high severity of deficits, female gender, non-fluent aphasia, cognitive impairment, and lack of social support. Treatment of depression with an antidepressant medication, like a selective serotonin reuptake inhibitor (SSRI), has been shown to be beneficial in aiding in their poststroke recovery.[39]

Approximately 70% of patients discharging after acute stroke utilize post-acute care rehabilitation services, including SNFs, IRFs, home health-care agencies (HHCAs), and outpatient therapies. Most patients receive care in more than one of these settings. In the United States, the first rehabilitation setting is most commonly an SNF (32%), followed by IRF (22%) and HHCA (15%).[40] Stroke patients admitted to IRFs have been shown to have improved functional gains and reduced readmission rates as compared to other post-acute care settings; however, utilization is limited in large part because of increased system cost.[11,16]

Parkinson's Disease

Parkinson's disease is the most common movement disorder and the second most common neurodegenerative disorder (after Alzheimer's disease) encountered among geriatric rehabilitation patients. The diagnosis is based on the history and physical exam findings, which include bradykinesia and either resting tremor or rigidity. Nonmotor manifestations, such as psychosis, impaired cognition, sleep disturbances, and autonomic dysfunction, are also a significant cause of disability. The incidence of Parkinson's disease increases with age, affecting 1–2% of the population over 65 and over 4% of those over 85.[41]

Levodopa remains the mainstay of treatment. Despite optimal pharmacological treatment, functions such as gait, transfers, posture, balance, and speech can progressively deteriorate, leading to impaired self-care, mobility, stability, communication, and quality of life. Deep brain stimulation procedures may extend the period of functional stability but may not be an option for all patients or be entirely successful in restoring normal movement. Rehabilitation is an important supplement to pharmacological and surgical interventions for Parkinson's disease. Exercise is a vital part of motor learning, and evidence suggests that physical therapy works via exercise-dependent plasticity.[42] One of the most studied rehabilitation strategies in Parkinson's disease is the use of external sensory cues. This is based on the hypothesis that patients with Parkinson's disease have less central delivery of proprioceptive input but can compensate by using visual cues to activate alternative neural networks in the cerebellum to promote movement. Gait training with external rhythmic or intermittent auditory, visual, proprioceptive, or cognitive cueing has demonstrated short-term improvement in gait speed, step length, freezing, and balance in the Parkinson's patient; however, sustained improvement has not been demonstrated. More traditional physical therapy focusing on interventions to improve postural control, balance, and gait has shown immediate and short-term benefit in Parkinson's patients.[43] Physical therapy may help train patients with assistive devices, including the use of modified ski poles to walk in a pattern similar to cross-country skiing to improve gait velocity or the use of four-wheeled walkers, which can reduce freezing of gait, festination, and retropulsion (falling backwards).[44] Complementary exercises using Tai Chi, Qigong, and dance can also have both short- and long-term benefit and may improve both motor and nonmotor symptoms.[45] In Parkinson's disease, speech volume and articulation may be low or reduced, and dysphagia may be an issue, both of which can be addressed by a speech therapist. The best-studied treatment for hypokinetic dysarthria that has shown clinical effectiveness is the Lee Silverman Voice Treatment (LSVT®).[46] In essence, the patient is trained to "think loud and think shout" in order to increase the strength of the respiratory muscles to move air and to enhance vocal cord adduction to produce voice.

Hip Fracture

Hip fractures are common within the geriatric population, ranking as one of the top causes of hospitalization

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among the elderly.[47] Approximately 50% of patients sustaining a hip fracture are unable to regain an independent level of functioning, and 80% are using a walking aid 1 year after hip fracture.[48] The majority of hip fractures require surgery, and the goal is early repair (within 24–48 hours) and early mobilization both to optimize functional status and to prevent complications from immobility, such as pneumonia, muscle weakness, delirium, thromboembolic events, and pressure sores. Mobilizing patients on the day of surgery if possible or the day after is associated with improved functional mobility and better outcomes.[49] Of note is that ERAS (Enhanced Recovery After Surgery) programs that focus on earlier weight-bearing ambulation, optimization of pain control, nutrition and fluid management, and removal of devices that reduce mobility have been shown to decrease postop complication rates and improve the rate of discharging patients to home following hip fracture repair.[50] For hip fractures requiring replacement of the femoral head, it will be important to educate patients on appropriate hip precautions in order to avoid hip dislocation after surgery. For a patient who underwent a posterior approach total or hemi hip arthroplasty, this includes no hip flexion greater than 90 degrees, no hip adduction past neutral (i.e., no crossing the legs), and no internal rotation of the affected extremity. This can be difficult in patients with memory impairment, but studies show that the presence of mild or moderate dementia should not preclude inclusion in a rehabilitation program.[51,52] Establishing evidence-based strategies for improving mobility after hip fracture surgery remains challenging because of insufficient data and small sample sizes of many studies. However, one meta-analysis of Progressive Resistance Exercise (PRE) after hip fracture surgery showed improved mobility, ADLs, balance, and leg strength.[53] Another meta-analysis showed that balance training resulted in improved independence with ADLs, gait, and physical function.[54]

Rehabilitation of the elderly hip fracture patient also includes evaluating nutrition, with attention to vitamin D and protein deficiency. Both can have an impact on bone mass, falls, and recovery.[55] Checking vitamin D levels and addressing underlying osteoporosis is vital in the prevention of future fractures.

Lower-Extremity Amputation

In the elderly population, complications of diabetes and peripheral vascular disease account for most lower-extremity amputations. Mortality rates are high for vascular amputations, and appear to be related to serious comorbidities such as kidney and heart disease, dementia, and reduced preoperative functional status.[56] Preoperative mobility, independence in ADLs, ability to stand on one leg, fitness, and intact cognition are all predictors of good walking ability after lower-limb amputation.[57]

The primary goal of rehabilitation of the geriatric amputee is restoration of walking ability. An interdisciplinary rehabilitation team including a physiatrist, physical therapist, and prosthetist can help to address barriers to safe ambulation and prosthesis use. Key medical concerns addressed by the physiatrist include skin breakdown, phantom limb pain, residual limb pain, mental health issues (such as depression or adjustment disorder), contracture prevention, and musculoskeletal injuries related to biomechanical gait alterations.[58]

Gait training with a physical therapist is essential to improve the likelihood of safe ambulation with a prosthesis. Strengthening and endurance exercises are particularly important, as the increased energy expenditure of walking with a prosthetic leg can present a challenge for the elder. There is a 40% increase of energy expenditure for the unilateral below-knee amputee and a 100% increase for the unilateral above-knee amputee.[59]

Patients typically utilize a shrinker sock after amputation surgery to reduce limb edema and shape the residual limb for optimal prosthesis fitting. A rigid plastic limb protector may be utilized for lower-extremity amputees to protect fragile post-surgical skin and prevent contracture formation. The prosthetist can provide helpful patient education and is key in determining the most appropriate style of prosthesis and attachment system. Advanced prosthetic components now include on-board electronics to assist with force generation during exertional activities, stabilization to minimize falls risk, and negative pressure suction to aid prosthetic fitting.

Conclusion

Restoring or maintaining an elder's function and independence through rehabilitation can be one of the most challenging and rewarding aspects of a geriatric practice. Rehabilitation involves not just evaluating an elder's medical and functional issues, but also assessing their motivation, goals, caregiver support, and environment for recovery. More complex rehabilitation issues require a multidisciplinary team approach that may involve

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physical modalities, specialized medications, adaptive equipment, multiple therapies, and professionals specialized in rehabilitation. By understanding how to overcome the multiple impairments that can come with age, we can promote full participation in society and enhance an elder's quality of life.

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Chapter 48

Geriatric Sexuality

Lisa Granville

Introduction

Sexuality is an important part of health and quality of life at all ages and thus is an important area for health-care providers to address. With the aging of the baby boom generation, different attitudes and mores regarding sexuality are coming forward as a result of growing up in the "free love" era. In time, discussion of sexuality with older adults is anticipated to be more direct, open, and often initiated by well-informed patients. However, at this time a number of fallacies regarding sexuality later in life prevail. Many prefer to believe that sexuality in older adults simply does not exist. Brogan notes "there is a general societal belief that old people are, or should be, asexual and a false assumption exists that physical attractiveness depends on youth and beauty."[1] Misinformation and misperceptions about sexuality and older adults are held by both patients and clinicians. Failure to adequately address sexuality and diagnose and treat sexual problems can lead to depression and social withdrawal,[2] self-discontinuation of medications with adverse side effects to sexual satisfaction,[3] and increased risk for sexually transmitted infections including HIV/AIDS.[4]

Sexual Activity

The number of years of potential sexual activity in later life is increasing.[5] The demographic trends of Americans living longer, with increasing active life expectancy, and smaller families allows many older adults greater solitude and privacy to engage in sexual activity. Data on sexual activity of older Americans, available from three studies, reveals and reinforces some consistent trends. In 2009 the American Association of Retired Persons (AARP) surveyed 1,670 people 45 years and older.[6] The National Survey of Sexual Health and Behavior, conducted in 2009, included 14- to 94-year-olds, with 1,008 people over age 50.[8] From 2005 to 2006, the National Social Life, Health and Aging Project (NSHAP) surveyed 3,005 Americans 57–85 years old.[8] In all three studies, across all age groups, sexuality was more important to men than women. Men had more frequent sexual thoughts, feelings of sexual desire, and engagement in self-stimulation than women.[6,7,8] Adults with partners were much more likely than those without partners to engage in interpersonal sexual activities such as kissing, hugging, oral sex, and intercourse.[6,8,9] Adults with partners expressed more importance in having a satisfying sexual relationship, more frequent sexual intercourse, and more sexual satisfaction overall.[6,8,10]

Although they represent 5–10% of the older adult population, there is very little known about the sexuality of the lesbian, gay, bisexual, transgender, queer, intersex, asexual, and additional identities (LGBTQIA+) population. In 2011, the Institute of Medicine issued a report calling for more research on the LGBTQIA+ population with an increased focus on older adults, racial and ethnic subpopulations, and bisexual and transgender people.[11] Today's older adults grew up in an environment much less supportive of diversity. Examples include creation of an official diagnosis in 1952 listing homosexuality as a sociopathic personality disturbance and Senator McCarthy including gay men and lesbians on his blacklist. In 1973 the American Psychiatric Association removed homosexuality from the *Diagnostic and Statistical Manual of Mental Disorders*.[11] In many countries across the world, sexual orientation is still criminalized.[12] The Still Out, Still Aging MetLife Study conducted in 2010 surveyed 1,201 LGBTQIA+ people in the USA aged 45–64 years and found the extent of disclosure of sexual orientation and/or gender identity varied significantly. While 74% of gay men and 79% of lesbians were completely or mostly out, this applied to only 39% of transgender and 16% of bisexual people. Moreover, LGBTQIA+ respondents indicated that health-care providers are among the groups to whom they have not come out.[13] An analysis of NSHAP data on sexual-minority women, defined as women who have

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sex with women or both men and women, revealed all women placed similar importance of sexual activity, difficulty with lubrication, urinary incontinence, and other urinary symptoms.[14] In NSHAP, differences in sexual activity between sexual-minority and sexual-majority heterosexual women were found, with sexual-minority women having increased reports of receiving oral sex (42.5% vs. 21.2%).[14]

For people of all sexes, genders, and sexualities, there are both physical and emotional aspects to sexuality, and the desire for intimacy continues throughout life.[10,14] Physical closeness can be expressed in many ways, including holding hands, hugging, kissing, mutual stroking, masturbating, oral sex, and intercourse. Studies have shown that for older adults the current level of activity correlates with past sexual frequency, and most older adults desire more activity than what they have.[15] Lack of partners and lack of privacy are significant obstacles for sexual expression. Adults living in age-segregated environments, such as retirement communities, express more interest in sexual activity and engage in sexual activity more often than their cohorts who are not age-segregated.[9] Some older adults report that sex became more pleasurable and of greater importance with age.[10,16] Midlife women attribute positive changes in sexual function to higher self-confidence, increased self-knowledge, and better communication skills.[16]

In the National Survey of Behavior and Health, the incidence of vaginal intercourse declined from 51% of women aged 50 to 59 to 22% of women over 70 years, primarily as a result of loss of a male partner.[7] Avis examined the impact of age and gender on sexual function and found that older women reported a cessation of sexual relations due to death of a spouse (36%), illness of a spouse (20%), or a spouse's inability to perform sexually (18%). Only 10% of the older women reported a cessation of sexual activity due to their own illness, loss of interest, or inability to perform.[17] Szwabo noted that as roles change within a relationship, so can the sexual behaviors of the couple. If a woman has assumed the caregiver "nursing" role, it may make sexual feelings and expressions less intense, as the partner may be seen as a patient rather than a sexual partner.[18] Adults without partners appear to adjust their sexual expectations and priorities. In one study it was noted that most people reporting no importance of sex were found to have no current partner and no anticipation of a partner in their lifetime.[10]

NSHAP revealed that the decline in sexual activity with age is largely mitigated by partner availability. The prevalence of sexual activity declined with age from 73% among 57–64-year-olds to 26% among 75–85-year-olds. However, for those who still had a partner, sexual activity remained prevalent, with 65% of 57–64-year-olds and 54% of 75–85-year-olds having sexual activity at least two to three times per month. Among those who were sexually active, about half of both women and men reported at least one bothersome sexual problem, yet only 22% of women and 38% of men reported having discussed sex with a health-care provider since the age of 50 years.[8]

Barriers to Treatment

In an interview study of 45 adults aged 50 to 92 years old, patients expressed a preference to consult a general practitioner of similar age and gender to minimize embarrassment by discussing concerns with someone likely to have had similar experiences themselves.[19] Perceived providers' attitudes would limit interactions if perceptions existed that older people are or should be asexual or access to treatments involve age-based rationing. In this study, 97% said they would discuss sexuality if the provider brought it up and 80% stated a willingness to return for a designated sexual concerns appointment.

In a study of women diagnosed with vulvar or vaginal atrophy, 72% reported never discussing their symptoms with a health-care provider.[20] When gynecological consultations actively addressed sexuality in postmenopausal women, the diagnosis of sexual problems increased from 12% to 48%.[21] Although sexual problems are prevalent in postmenopausal women, in one study less than 1% of older women used medication to enhance sexual function.[6] Older women have many unattended concerns. Both providers and patients may mistakenly attribute sexual problems to "normal aging" and both may lack knowledge about services and resources. Providers should be mindful that intersectionally marginalized populations, such as the LGBTQIA+ community, may also have fears about open discussions of sexual activity. As with many potentially sensitive issues, it appears that patients are waiting for their health-care providers to raise the topic.

Sexual Response Cycle and Common Disorders

The traditional model of the human sexual response cycle has four phases described in a linear progression.[22] The first phase, desire, involves the brain and one's interest in or urge for sexual activity. The second phase, arousal,

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involves the vascular system and the body's response to stimulation. In males this is primarily recognized by penile erection, and in females by vaginal lubrication and genital engorgement. The peak of arousal is referred to as plateau. The third phase, orgasm, involves the spinal cord and perineal musculature. In this phase the body experiences involuntary contractions of the pelvic muscles and reproductive organs and males experience ejaculation of seminal fluid. In the fourth phase, resolution, the body recovers from orgasm with a physiological rest period. Currently, the sexual response cycle is understood to have greater variability, flexibility, and a more circular nature. In both sexes the relationship between desire and arousal is complex, with variable order and overlap; the motivations for sex are multiple.[22] In women, lack of desire is a common concern; initiating sexual activity has been shown to stimulate desire. The *Diagnostic and Statistical Manual of Mental Disorders*, 5th edition (DSM-5) has adjusted criteria for diagnosing sexual dysfunctions, merging desire and arousal disorders in females, and creating separate diagnostic classifications for males and females.[23]

In 1966 Masters and Johnson's landmark study of older sexuality noted that natural aging leads to a need for more time to engage in sexual activities. Advancing age is associated with delayed arousal and a greater need for genital stimulation; reduced penile rigidity and vaginal lubrication; loss of the sensation of ejaculatory inevitability; and increasing anorgasmia.[24]

Tables 48.1 and 48.2 outline sex-specific sexual response cycle markers, changes with aging, and common disorders for males and females.[5,8,22] Whereas females experience menopause and its impact on sexuality over a relatively short period of time, males' physiologic changes are thought to occur over a longer period of time with less awareness as change is occurring. The physiological changes with age alone are insufficient cause to cease sexual activity, and for some these changes are felt to enhance their sexual activity.

Misconceptions Regarding Sexually Transmitted Infections (STIs)

Given that STIs are primarily considered a health issue of young people, there is very limited information on this topic in the older population. Lacking research, it is unclear if STIs are increasing in the older population or if the increased numbers are merely a reflection of the rapidly expanding population in general. There are several issues that increase the potential for older adults to acquire STIs. Longer, more active living combined with increased rates of later-life divorce, remarriage, sexual relationships outside of marriage, and access to internet dating sites increase the number of new sexual partners.[25,26] Age-related changes in the immune system may increase susceptibility to HIV infection.[27] Postmenopausal people with vaginas are more susceptible to the transmission of HIV virus because of atrophic changes in the vaginal mucosa leading to microabrasions as a result of intercourse.[25,27] For older adults, negotiating safer sex may be unfamiliar and challenging, they lack knowledge to identify HIV/AIDS risk factors, and they are less likely to use condoms.[4,25,27] Public health promotion materials regarding STIs fail to adequately target older adults.[4,25] Health-care providers lack awareness of seniors' sexuality, thus they fail to engage in conversations about risks and are less likely to test for the virus.[4,25,28]

In the past, many national health agencies provided stratified STI and HIV data only up to age 45 to 49 years; globally, lack of HIV data on 50+-year-olds persists.[12,25] In 2016, 48% of the HIV population in America were 50+ years old; in 2017 people over age 50 accounted for 17% of new HIV/AIDS diagnoses.[29] It is estimated that by 2030, 70% of those with HIV will be 50 years or older.[30] In the 1980s the primary route of HIV transmission in older adults was contaminated blood, with the older adult having higher rates of medical procedures. Now with routine testing of the blood supply, sexual intercourse and needle sharing are the main sources of HIV infection.[29] In recognition of the increasing prevalence of HIV, the Centers for Disease Control issued a guideline September 2006 advocating "in all health-care settings, screening for HIV infection should be performed routinely for all patients aged 13–64 years. Older adults who are at increased risk, such as those with new sex partners, should also be screened."[31] The US Preventive Services Task Force April 2013 guideline, and June 2019 guideline update, concur with this screening as a Grade A recommendation, indicating there is high certainty that the net benefit is substantial.[32]

HIV in the Elderly Population

Misdiagnosis and delayed diagnosis of HIV in the older population occurs because the HIV symptoms can be similar to other conditions associated with aging: weakened immune system, weight loss, fatigue, swollen lymph nodes, skin rashes, respiratory problems, depression, and decreased cognition or physical abilities. Delay

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Table 48.1 Males

Sexual response cycle: males	Markers	Changes with aging	Disorders**
Desire: brain	Desire/urge for sexual activity	Testosterone decrease in 55+ may affect libido	Hypoactive Sexual Desire Disorder (affected by illness, performance anxiety, relationship problems) NSHAP data: * Lack of interest 28.2%; 28.5%; 24.2% Performance anxiety 25.1%; 28.9%; 29.3%
Arousal: vascular system	Penile erection Genital engorgement Testes Scrotum	Longer time for arousal (often need physical stimulation), erections less firm, sperm production declines	Erectile Disorder (most common male dysfunction); Sexual Arousal Disorder; Sexual Pain Disorder NSHAP data:* Difficulty achieving, maintaining erection 30.7%; 44.6%; 43.5% Sex not pleasurable 3.8%; 7.0%; 5.1% Intercourse pain 3.0%; 3.2%; 1.0%
Plateau: peak of arousal	Full penile erection Testicular elevation and swelling Pre-ejaculatory fluid		Premature Ejaculation NSHAP data:* Climax too quickly 29.5%; 28.1%; 21.3%
Orgasmic: spinal cord and perineal musculature	Involuntary rhythmic contractions of the pelvic muscles, reproductive organs Ejaculation of seminal fluid	Ejaculatory control improves, fewer contractions per orgasm, volume of ejaculate decreased	Orgasmic Disorder NSHAP data:* Inability to climax 16.2%; 22.7%; 33.2%
Resolution	Subjective sense of relaxation "Refractory period"	Physiologically extended refractory period	

* NSHAP respondents were asked about presence of a problem for "several months or more" during the previous 12 months; data is divided into three age groups, 57–64 years; 65–74 years; and 75–85 years, respectively.

** The disorders listed are based on DSM-IV-TR classification in use at the time of the NSHAP study data collection.

Table 48.2 Females

Sexual response cycle: females	Markers	Changes with aging	Disorders**
Desire: brain	Desire/urge for sexual activity	Unclear: theory of low estrogen causing decreased libido is being reconsidered	Hypoactive Sexual Desire Disorder (most common female dysfunction; affected by illness, performance anxiety, relationship problems) NSHAP data:* Lack of interest 44.2%; 38.4%; 49.3% Performance anxiety 10.4%; 12.5%; 9.9%
Arousal vascular system	Vaginal lubrication Clitoral erection Genital engorgement Vulva Vagina Uterus Breast changes	Less increase in breast size, reduced elasticity of vaginal walls, decreased vaginal lubrication, less muscle tension Often related to estrogen deficiency	Sexual Arousal Disorder; Sexual Pain Disorder NSHAP data:* Lubrication difficulty 35.9%; 43.2%; 43.6% Sex not pleasurable 24.0%; 22.0%; 24.9% Intercourse pain 17.8%; 18.6%; 11.8%
Plateau: peak of arousal	Vasocongestion of outer third of uterus, vagina Elevation of uterus		
Orgasmic: spinal cord and perineal musculature	Involuntary rhythmic contractions of the pelvic muscles, reproductive organs	Fewer contractions per orgasm, ability for multiple orgasms may decrease	Orgasmic Disorder NSHAP data:* Inability to climax 34.0%; 32.8%; 38.2%
Resolution	Subjective sense of relaxation	May have refractory period	

* NSHAP respondents were asked about presence of a problem for "several months or more" during the previous 12 months; data is divided into three age groups: 57–64 years; 65–74 years; and 75–85 years, respectively.

** The disorders listed are based on DSM-IV-TR classification in use at the time of the NSHAP study data collection.

in diagnosis is concerning because it is associated with an increased risk of both AIDS and death. Increasing age is also an independent predictor of AIDS and death among those with HIV.[25] HIV-infected elders are frequently isolated because of the dual stigma of being elderly and living with an STI. Both of these factors make it difficult for seniors to disclose their disease state to family and friends, thereby forfeiting the social support needed to assist with their psychosocial requirements as well as their treatment plans.[28]

Effective antiretroviral therapy (ART) for HIV has allowed many infected people to live to 50+ years and is making AIDS-defining illnesses increasingly rare in those with ART-suppressed HIV.[27] Initiating ART at 50+ years is associated with an overall mortality risk 32% higher than those initiating at age 25–49 years.[25] HIV-associated non-AIDS (HANA) conditions are increasing and are associated with advancing age and chronic inflammation. HANA include cardiovascular disease, infectious and noninfectious cancers, osteopenia/osteoporosis, liver disease, renal disease, and neurocognitive decline. There is an emerging consensus that HIV and/or its treatment affects the process of aging and/or development of disease; people with HIV experience increased morbidity and mortality.[27] In 2012 the National Institutes of Health identified HIV and aging as an area of critical need for research and outlined specific knowledge gaps.[27] In 2014, the Joint United Nations Programme on HIV/AIDS (UNAIDS) developed a comprehensive strategy to end AIDS by 2030. Known as 90–90–90, by 2020 it targets 90% of all people living with HIV to know their status, 90% to receive sustained antiretroviral treatment, and 90% of those treated to reach viral suppression. Unfortunately, the UNAIDS strategy does not focus on age groups beyond 49 years old.[33]

In older people with HIV, multimorbidity is becoming the norm, individuals are surviving long enough to experience HIV as a chronic disease, and increased survival results in social supports of family and friends falling away, thus increasing the challenge of coordinating their care.[34] Recognizing the importance of geriatric concepts to the aging HIV population, in 2010 a special meeting convened at the White House on HIV and Aging. This collaboration between the American Geriatrics Society and the American Academy of HIV Medicine focused on developing clinical treatment strategies, and in recognition of the rapidly evolving developments in this field, it created an online site where information is frequently updated (https://aahivm-education.org/hiv-age).[34]

Impact of Common Medications, Medical Conditions, and Surgical Procedures

Adverse drug reactions (ADRs) are a common, often overlooked cause of sexual dysfunction. The drug classes "nervous system" and "cardiovascular system" account for the most registered sexual ADRs.[35] Often when drug side effects are studied, direct questioning of sexual ADRs is not done; the incidence of sexual ADRs may be largely unknown. Selective serotonin reuptake inhibitors (SSRIs) initially reported <10% of patients with sexual dysfunction; with direct questioning by providers the percentage rose to 60–70%.[36] Recently the persistence of sexual ADRs after discontinuation of medication has been noted for both SSRIs and 5-alpha reductase inhibitors.[37] Termed post-SSRI sexual dysfunction and post-finasteride syndrome, in both conditions males experience loss of libido and sex drive, difficulty achieving erections, and genital paresthesia.[37]

A number of medical conditions contribute to sexual dysfunction and raise patient concerns regarding health consequences of sexual activity. Medical conditions and their treatments may negatively alter one's perception of body image, create preoccupation with concern of exacerbation of symptoms, reduce exercise tolerance, and limit flexibility and positions of comfort. Tables 48.3 and 48.4 outline patient advice for common medical conditions and common surgical procedures.[38,39,40,41,42]

Erectile Dysfunction (ED)

ED is the most common disorder of male sexual health. ED is defined as the recurrent inability to attain or maintain a penile erection sufficient for sexual performance. ED shares risk factors with cardiovascular disease and is now also recognized as an independent marker for increased risk for cardiovascular disease.[41] Management of ED provides an opportunity for cardiovascular risk reduction. The prevalence of ED increases with age. In the Massachusetts Male Aging Study of 1,290 males aged 40 to 70 years, the probability of severe ED tripled from 5.1% to 15%; the probability of moderate ED doubled from 17% to 34%; and the probability of mild ED remained constant at 17%.[43] Many medical conditions and their treatments contribute to the development of ED. Common etiologies include diabetes mellitus, hypertension, cardiac disease, chronic

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Table 48.3 Common medical conditions

Condition	Advice and information
Cardiovascular disease	• Male erectile dysfunction (ED) affects 44–65% with CAD, 80% with heart failure • ED is a marker for CVD, often preceding CVD event by 2–5 years
Arrhythmia with implantable cardioverter defibrillator	• Worry of causing an arrhythmia or shock may exist, especially among those who have experienced ICD shocks during sex • For some, the ICD will be seen as a "rescuer," and sexual interest and activity may increase
Myocardial infarction	• Abstinence often advised for 8–14 weeks, although there is limited data to support this practice • Duration of abstinence depends primarily on patient desire, general fitness, conditioning • Ability to climb 1–2 flights of stairs is generally considered adequate fitness for sexual activity
Angina	• Late morning activity after a full night's rest • Use of supine position to reduce exertion level • Creation of a relaxed atmosphere for sexual encounters
Heart failure exacerbation with pulmonary edema	• Abstain from sexual activity 2–3 weeks or until ability to climb 1–2 flights of stairs is restored
Hypertension	• No need to limit sexual activity • Be aware that antihypertensive medication and untreated hypertension lead to erectile dysfunction in males; effects in females not well studied
Stroke	• Sexual function may be impaired, desire is usually unaffected • If loss of desire is present, screen for depression • Physical stimulation can be focused on the unaffected side of the body • Pillows and headboards can be used for support
Emphysema and other causes of shortness of breath	• Use intervals of rest • Select positions requiring limited exertion • Use oxygen as needed
Arthritis	• Use general pain reduction approaches: exercise, rest, warm baths, analgesics prior to exertion • Try different sexual positions to minimize joint pain • Use time of day when pain and stiffness are least severe
Diabetes mellitus	• Male erectile dysfunction (ED) is 2–5 times more common than in general population; 49% of males 65+ with type 2 DM • For some males, establishing good control of diabetes reestablishes potency • If diabetes is already well controlled, ED is likely irreversible, consider ED management (PDE5 inhibitors effective)
Dementia	• Inappropriate sexual behaviors (ISB) lack universal definition, some are labeled inappropriate only because performed publicly • Lack of privacy, attitudes of family/caregivers have influence • ISB prevalence 1.8–25%; more common in hospital than community setting
Depression	• Sexual desire is affected • Treatment of depression restores sexual interest Note: Selective serotonin reuptake inhibitors (SSRIs) are associated with orgasmic dysfunction, decreased sexual desire, decreased arousal

kidney disease, depression, anxiety, and prostate cancer surgery and radiation.[22,44] Treatment options are varied and include lifestyle modification, medications, penile implants, and vacuum devices. Lifestyle modification is aimed at increasing exercise, weight loss to lower BMI below 30 kg per m,[2] and smoking cessation.[45] For most men, PDE5 inhibitors, such as sildenafil citrate, are the first-line treatment option for ED, including those men with diabetes mellitus, spinal cord injury, and use of antidepressants.[46] PDE5 inhibitors are considered equally efficacious, and in the presence of sexual stimulation they are 67–89% effective. Side effects of these medications include headache, flushing, and dyspepsia, and they should not be used by males taking nitrates.[46,47] Intracavernosal injection of vasoactive agents, such as alprostadil, are also effective. The most

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Table 48.4 Common surgical procedures

Condition	Advice and information
Hysterectomy	• Abstinence advised 6–8 weeks to allow adequate wound healing • Females sensitive to cervical and uterine contractions during orgasm may notice the loss of these sensations • Depression is common and may impair desire
Oophorectomy	• Effects of decreased androgen, estrogen, progesterone not well studied • Sexual frequency is sometimes increased with removed fear of pregnancy
Mastectomy	• Loss of desire occurs with embarrassment, perceived loss of femininity, fear of being unattractive • Periodic depression common for 1–2 years post-procedure • Rehabilitation programs encouraged for patients and partners to deal with physical/psychological concerns and enhanced communication
Prostatectomy	• Abstinence advised for 6 weeks to allow wound healing • Transurethral resection of prostate, used for benign prostatic hypertrophy, leads to retrograde ejaculation and erectile dysfunction • Complete prostatectomy, used for prostate cancer, leads to erectile dysfunction in 60% or more
Orchiectomy	• Psychological impact substantial; physical impairment of function limited • Counseling before and after surgery is highly recommended

common reported side effect is penile pain; some males are averse to needle use. Intracavernosal therapy has a success rate of 70–90%.[48] Intraurethral insertion of vasoactive agents is an alternative. This approach is termed medicated urethral system for erection (MUSE) and is successful 43–60% of the time.[48] Side effects include urethral irritation in patients and some partners. Vacuum pump with constrictive bands is a common alternative to medications; it is contraindicated in males with sickle cell anemia, blood dyscrasias, and those taking anticoagulants. This method often achieves an erection; however, patient satisfaction varies between 27% and 74%.[49] The vacuum device requires some skill and an understanding partner. Males receiving adequate training report better device satisfaction.[39] Penile implants are generally reserved for those in whom alternatives were unsuccessful. The operation requires destruction of the corpus cavernosus, thus eliminating any future pharmacologic treatments.[39]

Sexual Encounter Enhancements

Sex Toys

Health-care providers should develop a general awareness of the types of sex toys that are commonly used and related health-care considerations. There are several reasons why a patient may develop an interest in toys, including lack of a partner, reentering the dating scene, and interest in adding variety. The patient may look to the health-care provider for guidance in how to use toys, validation that toy use is an acceptable practice, and health risk education. In addition to these roles, the health-care provider may need to assist retrieval of misplaced or out-of-reach equipment.

The following guidelines facilitate the safe use of toys. When putting something in a body opening, be aware that sharp, breakable, and objects that are hard to hold on to can be problematic and are discouraged from use. One should listen to their body; pain is a good indicator to slow down, pull out, or explore other options. Toys should be cleansed thoroughly between use with soap and water and allowed to dry completely. When cleaning porous materials (silicone, latex) a little bleach can be added and should then be thoroughly rinsed to avoid chemical burns. Battery or electronically operated equipment should not be completely submerged. A condom can be used on many toys and will significantly facilitate cleanliness. Condoms should always be changed between partners and when moving from anus to vagina. Using a toy with only one individual, termed dedicated use, is another effective strategy for safer sex practices.

Lubricants

There are many different lubricants available. Lubricants are used with condoms, with toys, for masturbation, for anal sex, and frequently in postmenopausal females to counteract vaginal dryness. When selecting a lubricant there are three main considerations: the ingredients; the purpose; and reactions between the chemicals and the person, toys, and safer sex supplies. The three main

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types of lubricants are water based, silicone based, and oil based. Water-based lubricants are very popular because they are safe to use with latex condoms and rubber toys. Advantages include ease of cleaning, stain free unless color has been added, non-greasy feel, and reactivation with water or saliva. Silicone-based lubricants last a long time, don't dry out, and small amounts are very effective. Limitations include difficulty in cleaning, damage to silicone toys, and vaginal irritations and infections. Oil-based lubricants are most often used for male masturbation. Limitations include difficulty in cleaning, not recommended for vaginal use, and damage to rubber toys and latex condoms. Lubricants may have additives. Desensitizing agents, such as benzocaine, originally used in anal lubricants, are also useful for vaginal lubricants in people who experience vaginal, labial, clitoral, and/or cervical pain during vaginal penetration. Caution is advised that decreased sensation may increase injuries. Additives may also include flavors and scents. Plain lubricants taste mildly chemical and slightly bitter. Edible lubricants are water based and often contain glycerin. Patients should be advised that in warm, moist environments such as the vagina, sugar promotes yeast and bacterial infections. Lubricants also vary in consistency from light liquids to heavy gels. Heavier lubricants with ample slip are recommended for anal sex and may also be useful with chronic medical conditions (e.g., Sjogren's syndrome) associated with sticky vaginal and/or anal mucus. Patients are encouraged to test a small quantity of lubricant for adverse reactions, especially if using for vaginal intercourse.

Coital Positions

With advancing age, a number of factors may influence a patient's preference for coital positions. Considerations include limited exercise tolerance, pain aggravated by certain positions, and embarrassment related to medical devices (ostomy pouch) or procedures (mastectomy, orchiectomy). Penetration from the top (missionary and similar positions) facilitates intimacy including talking, hugging, kissing, eye contact, and close body contact. Touching by the person on top is often limited because of holding oneself up, and prolonged activity may be physically challenging. Partners may find this position uncomfortable if the person on top is heavy. Penetration from the bottom (riding and similar positions) facilitates intimacy for similar reasons. With the person on top in control of depth, speed, and rhythm of penetration, one's orgasm may be facilitated and pain with penetration can be minimized. This position works well for partners of different heights. Prolonged activity may be physically challenging for the person on top. The sitting/kneeling position may be a desired alternative for patients with abdominal bloating, discomfort, or medical devices. While some consider lack of eye contact impersonal, this may be an advantage for those embarrassed by altered body image. If this position is hard on the knees, use of a chair may facilitate comfort. The rear entry (doggie-style) position also facilitates touching, is easy on the muscles and requires less energy than those positions previously mentioned. This position facilitates deep penile penetration and has limited eye contact. The side-by-side (spoons) position requires the least amount of energy and is therefore useful for patients with limited mobility or stamina. This position facilitates touching, close body contact, and slower encounters. With increased time and less deep penile penetration, this position is especially useful for patients with premature ejaculation.

Patients of all ages have questions and concerns about sexuality. People in the LGBTQIA+ community, recently coming into their queer identity or coming out to others, may be developing new understanding about their sexuality. Many providers may not feel comfortable talking about sexual issues or even taking a sexual history, especially in their older patient population. Gaining a comfort level in this area comes with practice and with personal knowledge of the potential changes and concerns associated with sexuality in the aging population.

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Chapter 49

Aging in Adults with Intellectual Disabilities and Severe and Persistent Mental Illness*

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Introduction

Aging is a universal experience, and likewise the key factors to successful aging: avoiding disease, maintaining high cognitive and physical functioning, and maintaining engagement in life.[1] Successful aging can be less clearly defined for individuals with intellectual disability (ID), severe and persistent mental illness (SPMI), and autism spectrum disorder (ASD); however, many of these same strategies still apply.[2,3,4] The issue has become more important to address as the number of older adults with ID, SPMI, and ASD continues to increase.[4,5] As these segments of the population continue to grow and age, challenges to optimizing their care become more complex as they become more medically complex. Meanwhile, as these individuals are now more likely to live in the community and outside of institutions,[6] their family caregivers are also aging and facing limitations related to their own health and to the care that they can provide.[7]

In this chapter we will review the epidemiology, key medical comorbidities, therapeutic and community resources, and treatment strategies for individuals with ID, autism, and SPMI. We will also look at decision-making and the role of the interprofessional team. Throughout, we will discuss strategies that may reduce health disparities and potentially improve the individual's experience with care and their clinical outcomes.

Intellectual Disability

Definition

ID is defined as "a disability characterized by significant limitations in both intellectual functioning and in adaptive behavior, which covers many everyday social and practical skills," with an onset before the age of 18.[8] Intellectual functioning refers to general mental capacity (learning, reasoning, problem-solving). Adaptive behavior includes conceptual skills (i.e., language, literacy, managing money), social skills (i.e., interpersonal skills, social responsibility), and practical skills (i.e., activities of daily living, safety).[8] ID is sometimes described by degree of impairment (mild, moderate, severe, or profound) or by intelligence quotient (IQ)[8,9] (Table 49.1). It's important for the clinician to realize that these categorizations are imperfect measures and that the individual may have a preferred learning or communication style and is likely to perform at different levels based on the task or expectation at hand and supports provided (i.e., visual or verbal prompts).

ID is a subset of the broader category of developmental disability.[10] A developmental disability is defined as a severe, chronic disability resulting from mental and/or physical impairments that are likely to be lifelong, manifested before age 22, and resulting in functional limitations in three or more of the following areas: self-care, language, learning, mobility, self-direction, capacity for independent living, and economic self-sufficiency.[11] Examples of developmental disabilities include ASD, cerebral palsy, fetal alcohol syndrome, and spina bifida. Certain conditions are associated with both ASD and ID including Rett syndrome, Fragile X syndrome, tuberous sclerosis, Prader-Willi syndrome, Angelman syndrome, Velo-cardio-facial syndrome, and Down syndrome (DS).[12]

Etiology

ID can be due to genetic conditions, infection, metabolic abnormalities, nutritional deficiencies, or other external causes. DS, caused by a trisomy of chromosome 21, is the most frequently identified cause of ID.[13] Fragile X is the most common inherited form of ID, and fetal alcohol syndrome the most common acquired form.[14] While the cause of ID in many patients is either multifactorial or unknown, identifying the etiology, if possible, may help

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Table 49.1 Levels of functioning in individuals with intellectual disability

Intellectual functioning	Adaptive functioning	Communication
Mild ID	Unskilled job	Large vocabulary
IQ = 55–75	Can make familiar	Trouble with conversation
6th-grade equivalent	medical decisions	Some complex sentences
Moderate ID	Supported employment	Phrases and simple sentences
IQ = 40–50	Residential supervision	Concrete Q and A
2nd-grade equivalent	Support with decisions	
Severe ID	Ongoing support	Single- or two-word phrases
IQ = 25–35	Limited ability to participate in medical decision-making	One-step commands
Profound ID	Continuous support	Typically nonverbal
IQ < 20–25	Not able to make medical decisions	May gesture

the clinician develop plans of care best suited to the individual's needs and risk factors. Clinicians should also recognize that some individuals with common developmental disabilities, such as cerebral palsy or autism, may or may not have an ID.

Prevalence, Life Expectancy, and Common Comorbidities

In terms of prevalence, actual numbers are difficult to obtain because of variances in how the disability is defined, the criteria used for inclusion, and the age group being examined (i.e., children vs. adults).[11] It is estimated that 6.2 million people living in the United States have been diagnosed with intellectual or developmental disability.[15] Depending on definitions and prevalence estimates used, adults with ID are estimated to represent approximately 1–4% of the population and are frequently reported with adults with developmental disabilities (I/DD vs. ID alone).[16]

In general, the number of US adults over age 60 with I/DD is growing and is estimated to reach 1.2 million by 2030.[17] Globally, the prevalence is estimated to be approximately 10.37/1,000 population or about 1%, with higher rates in lower-income countries.[18] Although the average life expectancy is lower than that of the general population,[4,13] it continues to increase because of improved medical care and access to support services.[19] Currently, life expectancy for individuals with I/DD is 66 years, although it may be lower for individuals with certain genetic syndromes (i.e., DS) or those with significant intellectual and/or physical disabilities.[20]

Adults aging with I/DD face unique and complex challenges. Individuals with I/DD experience health disparities and are likely to have poorer health than their age-related peers.[16] In particular, I/DD are associated with higher rates of vision and hearing abnormalities, obesity, seizure disorder, skin conditions, dental caries and disease, gastrointestinal conditions, thyroid disease, osteoporosis, and mental health diagnoses.[13] Adults with I/DD often experience chronic health conditions earlier (e.g., osteoporosis, oral health disease, diabetes). Meanwhile, they also experience common age-related conditions, including cataracts, progressive hearing loss, and osteoarthritis.[4] Many adults with I/DD have sedentary lifestyles and consume unhealthy diets, which leads to cardiovascular disease[21] and obesity.[22] Older adults with I/DD are also at high risk of falls, which can lead to significant injuries.[22]

Dementia deserves special consideration. In general, aging adults with ID have a similar risk of dementia to the general population, but the risk is much higher and the onset earlier in individuals with DS.[23] The onset of dementia in adults with DS may be in the 40s and the average age is in the early 50s.[23] Screening tools, such as the NTG-Early Detection and Screening for Dementia tool, are in development to better identify early cognitive impairment and aid in diagnosis.[23,24]

Individuals with ID may have higher rates of mental health conditions, although estimates of prevalence vary widely, from 7–97%, because of a lack of uniform diagnostic criteria and a limited number of studies.[13,25] Depression and anxiety may be more common in older adults with ID compared with the general population.[26,27] Interestingly, the informal mental health diagnosis used in some studies was "challenging" or "problem" behaviors.[27,25]

In a study in the Netherlands that compared causes of death between individuals with ID and the general population, individuals with ID were more likely to die from respiratory disease than the general population.[28] Similar to the general population, other primary causes of death were cancer and cardiovascular disease.[28] Compared with other causes of ID, individuals with DS were more likely to die from respiratory disease and

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dementia and less likely to die from cancer and cardiovascular disease.[28]

Approach to Treatment and Healthy Aging

Social Determinants of Health

One must consider social determinants of health in the approach to healthy aging. Beyond the diagnosis or classification of the individual, social determinants of health impact patient outcomes and contribute to health disparities.[13,16] Conceptually, the relationship between social determinants of health and I/DD has been described as a cascade effect beginning with underlying comorbidities associated with the primary condition.[16] Adults with I/DD may then have limited access to quality health care because of funding constraints or the absence of care in their communities to meet their unique needs. The comorbid conditions may be related to or exacerbated by the specific underlying diagnosis of the individual, such as DS and dementia, along with polypharmacy and medication side effects and the living environment. Screening and detection of diseases in this population is more challenging because of communication barriers between the patient and clinician (e.g., ability to express pain or complete an audiology evaluation), lack of validated scales for the population (e.g., depression and anxiety screens), and difficulties with adherence to treatment (e.g., tolerating blood draws, swallowing medications, wearing hearing aids). Individuals with autism, with or without ID, are at higher risk for many chronic medical conditions, including higher rates of cardiovascular disease, that impact quality of life and are associated with a decreased life expectancy.[30]

Experience of Aging

Adults with I/DD experience the same challenges with aging as the typical population. Successful aging includes maintaining good physical and mental health, living a healthy lifestyle, maintaining social connections, participation in valued activities, and experiencing a high quality of life satisfaction. To successfully age, adults with I/DD require increased support because of complex factors that impact their physical, social, and emotional health. The primary care team can advocate for adults with I/DD to receive the supports and services necessary to facilitate successful aging. While there may be general treatment strategies that apply to this patient population, an individualized, patient-centered approach to care is required, given the heterogeneity that exists even among individuals with the same syndrome or condition. Below are strategies the primary health-care practitioner can utilize to support adults with I/DD in successful aging. Many apply to those with SPMI and ASD as well. Special considerations for those populations will be discussed later.

1. Maintain good physical health through screening and prevention.
   Unless condition-specific guidelines suggest otherwise, patients with I/DD should be offered the same options for preventive care as the general population. The clinician also needs to consider condition-specific guidance but must be cautious about diagnostic overshadowing.
2. Empower adults with I/DD in taking charge of their own health and participating in their own health care and decisions.
   Communication limitations or differences can make the process of communicating with the health-care provider difficult or ineffective, as adults with I/DD may have cognitive and/or speech and language deficits that make it difficult for them to communicate symptoms, concerns, or questions to the physician.[30] Communication may be further impacted by low levels of health literacy among adults with I/DD.[31] Often information is communicated to the health-care provider by caregivers who may be family members or paid staff. Sometimes the caregiver is in tune with the needs of the adult with I/DD, especially if they have a long history with the adult and can discern changes in behavior, mood, or cognitive ability that may signal the need for further investigation. In other cases, the caregiver may be new to the situation (i.e., a newly hired direct support personnel who has only been with the adult for a short time) and may not be as aware of potential issues affecting the adult with I/DD. Alternatively, the caregiver may not recognize important signs or symptoms as possible indicators of changes in physical or mental health, and so they may fail to report them. Box 49.1 highlights best practices to improve communication with individuals with I/DD.

   Adults with I/DD may lack the cognitive capacity to participate fully in decisions regarding their health and collaborate with the health-care provider to determine the best course of action, but the clinician should not assume this.[32] Providers need to understand who the patient's legal decision maker is, whether it is the patient, guardian, or power of

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Box 49.1 Best practice tips for practitioners

As a best practice, clinicians should:[9,41]

a. Establish rapport – remember to speak directly with the patient and not only the caregiver.
b. Choose appropriate language – give concrete instructions such as please take off your shoes vs. get ready for a foot exam. Use the term intellectual or cognitive disability and not mental retardation. If the patient's or caregiver's preference is unclear, use person-first language, which places the person before the disability. For example, an adult with Down syndrome vs. a Down syndrome adult.
c. Listen – listen to what the patient says and ask for the patient to repeat if necessary. Allow enough time for the patient to respond, as processing takes additional time for those with ID.
d. Communicate without words – use visual aids and act or demonstrate what you are going to do or what you are asking.
e. Explain clearly – inquire about preferred learning styles and give clear, brief instructions. Teach-back may help gauge understanding.
f. Identify who is the patient's legal decision maker while involving the patient to the extent possible in their own care. Is it the patient, power of attorney, or guardian? Is there a secondary decision maker designated?
g. Understand and verify the medical history.
h. Review the medication list.

attorney. As decision-making capacity can change because of a degenerative condition, acute circumstances, or illness, the clinician needs to readdress this over time and refer appropriately when a change in decision-making capacity is evident.

As with all patients, the individual with ID/D may lack the ability to understand the importance of consistently following recommendations to manage their own health. This could include the need for diet modification, increased physical activity, or medication management. In these cases, the practitioner should use a collaborative process with the patient and caregiver if appropriate to develop interventions that address the patient's goals and reflect their interests and values.[32] As with all conversations with patients and families/caregivers, it is important to be aware of health literacy so that the caregiver fully understands the options, can collaborate in a way that represents the desires of the adult with I/DD, and is able to carry out the plan.

Several strategies are effective in empowering adults with I/DD to participate in their health-care decisions. Patient passports have been used to increase communication between patient and provider and ensure continuity of care.[33,34] The passport includes important information about the patient's medical history but also their likes, preferences, and activities.[35,30] Examples include guided health assessments, such as the Comprehensive Health Assessment Program (CHAP), which improve health checks and maintain records of health actions or hospital passports.[36]

A multidisciplinary team developed and implemented a communication passport for adults with ID and autism spectrum disorder who were experiencing mental health problems and were admitted to an adult inpatient psychiatric unit.[37] The passport was useful in communicating relevant information, especially once patients were discharged back into the community.[37]

3. Consider where the individual lives.
The living situation may factor into treatment recommendations and available supports for the patient. Some adults with I/DD live in family homes and receive care from parents, siblings, or other family members. Other adults live in their own apartments with individualized support or in life-sharing programs where they live with individuals who are not members of their biological families. Others may live in small group homes with other individuals with I/DD, larger congregate settings, or long-term care settings and nursing homes supported by trained staff.[8]
4. Review medications.
Patients with I/DD are frequently on multiple medications by multiple prescribers, with an estimated 20–40% on at least five medications.[38] In those with ID or autism and dementia compared with those

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without dementia, individuals were more likely to be prescribed benzodiazepines (55% vs. 36%) and antipsychotics (50% vs. 39%) and more likely to be prescribed antipsychotics than those with dementia in the general population (50% vs. 25%).[39] Adults with I/DD can be supported to participate in their medication management. When appropriate, be sure the individual and the caregivers understand the purpose of the medication. Visuals, such as pictures or even simple videos, may be useful to show proper medication use.[40]

5. Manage pain.
Identifying the presence of pain, locating its source, and providing effective management especially for chronic pain is a complex process. The individual with ID may not be exhibiting behaviors typically associated with pain because of sensory processing dysfunction or high pain threshold. Likewise, those with increased sensitivity may be demonstrating signs of pain with typically benign stimuli (i.e., light, noise).[41] Contrary to the popular notion that adults with ID do not experience pain or have a higher threshold,[42] Walsh and colleagues (2011) identified the prevalence of chronic pain in adults with IDD at 15.5%.[43] Pain is often unreported by the individual,[44] undetected by caregivers, undiagnosed,[42] or believed to be part of the aging process,[45] which can lead to under-treatment.[46] Common causes for pain include arthritis, chronic or recurrent infections, painful physical conditions (such as gastroesophageal reflux), and musculoskeletal problems (i.e., spasticity).[46,43] Walsh and colleagues (2011) found statistically significant associations between the presence of chronic pain and physical disability, paralysis, or problems with mobility.[43] Caregivers reported that chronic pain manifests in self-injurious behavior, depression, anxiety, or behavioral problems[43] and impacts quality of life.[44]

Providers should be vigilant in identifying valid ways for adults with ID to report pain. Detection of pain is complicated by the presence of the cognitive deficit associated with ID as well as impaired communication. Pain is a subjective experience and is measured most often by self-report.[46] STOP-ID is a self-reporting tool with an online application that can be used on a tablet or laptop. Preliminary testing with 40 adults with DS indicated the tool might be useful in helping facilitate communication between the adult with ID and care provider about the presence and extent of pain, but further research is needed before this tool can be introduced into clinical practice.[46]

Nonpharmacologic interventions can be successful in reducing pain along with or as a substitute for pharmacologic interventions. Activities that may be effective for some patients include those that encourage the natural production of endorphins, such as participating in exercise, dance or physical activity, listening to music, and/or engaging in valued activities that bring comfort and relaxation.[47] Caregivers should be encouraged to integrate these types of activities into the daily routine as part of a comprehensive pain management protocol.

When pharmacologic interventions are used, the principles of pharmacotherapy are like that of the general aging population, but clinicians should have heightened awareness about medications that can cause sedation or affect cognition.

6. Maintain good mental health.
Estimates for mental health problems in adults with I/DD range from 10–39%,[48,49] and up to 50% of the population with I/DD may have comorbid psychiatric disorders.[50] Prevalence rates of depression and anxiety in adults with I/DD are difficult to determine, with a meta-analysis of the literature revealing that among adults with ASD, an estimated 27% have anxiety disorder and 23% have depression.[51] The aging experience includes many changes that can either exacerbate an existing mental health issue or result in a new mental health condition such as depression or anxiety.

Adults aging with I/DD also experience the same losses that are associated with all aging, such as loss of a job, home, friends, and family (because of relocation or death), and reduced skills and abilities due to physiological changes. Their experience may be further complicated by their history and life events, living situation, level of cognitive impairment, and limited coping skills.[52,53] Adults with I/DD also experience a variety of unique life changes that can result in feelings of stress, loss, or grief, such as changes in the primary paid caregiver due to staffing turnover or scheduling modifications, or in the living situation or housemates, such as due to a transition from a family home to a group home or community residence.[52] This can lead to anxiety, worry, or grief, which may manifest as physical symptoms,[54] breathlessness due to anxiety,[55]

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disruptions of sleep, changes in appetite, extreme fatigue, and somatic complaints unassociated with any specific physical illness. Emotional pain of grief can include anger, sadness, depression, lack of interest, and/or reluctance to participate in normal activities.[55]

Access to mental health care, accurate diagnosis, and appropriate use of psychotropic medications are of utmost importance in this population. It is critical that clinicians look for medical causes and other factors, such as grief, abuse, or loneliness, when asked to evaluate a patient presenting with challenging behaviors for mental health issues. Although commonly used screening tools are not validated in the population, an empiric trial with selective serotonin reuptake inhibitors (SSRIs) can be considered if clinicians suspect depression, anxiety, or OCD.[56] Medications should be discontinued if there is no clinical response at an appropriate dose after 6–8 weeks of treatment or if the side effect profile is unacceptable.[56]

Clinicians also need to set aside the myth that psychotherapy is not appropriate for adults with I/DD.[57] On the contrary, given its effectiveness with other populations,[58] adjustments need to be made to the psychotherapy approach to better fit the expressive and receptive language skills of this group.[57] According to Fletcher (2011), there are 10 practice modifications that clinicians can implement to make psychotherapy adaptable and helpful to individuals with I/DD:

(1) Language – use language that is congruent with the individual being served and free of analogies or obstructions.
(2) Frequency of sessions – sessions should be adjusted to brief, twice-per-week visits initially in order to establish therapeutic rapport and build relationship.
(3) Shorter sessions – should be limited to no more than 30 minutes to maximize attention span; adjustments for longer sessions can be made as needed.
(4) Duration of therapy – although short-term approaches can be considered, plan for a longer duration because developing the therapeutic relationship takes time, as does termination. The latter should be handled with the utmost care to prevent the individual from experiencing the termination as another significant loss in their life, especially if the clinician is regarded as an important figure in the patient's life.
(5) Utilize a more structured and directed approach – this approach fills silence gaps in the session and helps the individual not to interpret the silence as personal rejection.
(6) Communication with collaterals – to ensure everyone is on the same page, extend the network of care to include other team members and organizations involved in the patient's care.
(7) Modify complexity of interventions – approach the session on a step-by-step basis to foster understanding; provide a lot of repetition and maintain simplicity throughout.
(8) Therapist needs to be supportive – open and end sessions with supportive statements to help buffer and reframe patient's tendency toward feelings of inadequacy, self-defeat, and experiences of failure.
(9) Therapist needs to be flexible – adjust the therapeutic and theoretical approach (i.e., CBT, supportive therapy, etc.) based on the patient's ability, presenting problem, and individual need.
(10) Therapist needs to be part of a team approach – clinicians should be part of an interprofessional team to ensure discussion of conceptual issues with the patient; foster optimal treatment collaboration to advance patient's goal for independence and self-reliance.[57]

Individual and group psychotherapy and behavior therapy along with other modification strategies have been found to be helpful in treating depression and anxiety in impaired older adults.[58] Some preliminary evidence suggests mindfulness-based approaches may also be effective in improving the psychological well-being of individuals with I/DD.[59] Supporting this group to maintain good mental health requires clinicians to look at the whole person and beyond the disability for the source of suffering and distress, which is often associated with underlying depression, anxiety, comprehension challenges, and a myriad of other stressors.

7. Increase physical activity and encourage healthy eating.
   Adults with ID are less likely to maintain a healthy lifestyle that includes regular physical activity and healthy eating patterns.[60] At baseline, adults with ID have lower levels of cardiorespiratory fitness compared with the general population.[61,62] In a study that looked at individuals with ID and

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extremely low levels of fitness, even small improvements in fitness were associated with decreased mortality.[61]

Adults with ID have higher levels of obesity,[60,4] which increases the risk for chronic condition such as hypertension, diabetes, sleep disorders, and cardiac and respiratory diseases.[4] Several evidence-informed health promotion programs are available that focus on supporting adults with ID in living healthy lifestyles.[63–68] It is important to collaborate with caregivers to encourage lifestyle changes that will support healthy aging.

8. Thoughtfully approach dementia and end-of-life care. Dementia can be especially challenging to diagnose in individuals with ID.[7] While the onset of dementia is earlier in individuals with DS, clinicians should be cautious making the diagnosis in individuals younger than 40, as alternate diagnoses are much more likely. Even when considering a diagnosis of dementia in an individual with DS in their 40s or 50s, health-care providers need to evaluate the individual for other comorbidities, including hypothyroidism, sensory impairment, mood disorder, and sleep apnea. Although the quality of evidence is low, pharmacotherapy with antidementia medications has not shown to benefit individuals with DS.[56] Other psychotropic medications can be considered if behavioral strategies fail to manage associated symptoms and may be associated with improved quality of life for individuals with DS and caregivers.[56] Nonpharmacologic strategies can be helpful in managing symptoms such as behavior upset, fear, and anxiety, and in addressing challenges related to providing care. The strategies will change over time as the condition progresses.[69]
9. Foster an interprofessional team approach. Interprofessional team approaches are recommended to improve patient outcomes.[70] Interprofessional primary care teams have been shown to be beneficial to patients with complex needs[71,72] and chronic conditions.[73] While the specific impact on patient outcomes is as yet unclear,[74] interprofessional teams rate patient experience and improved patient health status as quality indicators to demonstrate the value of interprofessional team collaboration.[75] The interprofessional team works in partnership with the patient and caregiver to provide a patient-centered intervention that addresses multiple factors to optimize the health and well-being of adults with I/DD and their caregivers, including facilitating social connections and engagement in valued activities.

In addition to primary health-care providers, occupational, physical, and speech therapists, along with behavioral health providers, nurse care coordinators or case managers and social workers, can provide the supports and resources necessary to assist the adult with ID in aging successfully. Below is a brief description of each team member and potential contribution to the process.

Behavioral Health Providers (BHPs) play an integral part on the interprofessional team. They are often embedded in primary care to serve as Behavioral Health Consultants (BHCs) to improve detection, diagnosis, and treatment of psychological and substance use issues and provide appropriate crisis management services (i.e., management of suicide risks) as needed.[76] BHPs offer a wide range of targeted evidence-based interventions focused on maximizing patient self-management and skills development. They address behaviors associated with health risk and support the care team to improve pain management and pain-related outcomes using nonpharmacologic coping strategies.[77] When working with individuals with complex care needs, BHPs play an important role consulting with the primary care provider to optimize communication and facilitate patient and family engagement as well as help the care team formulate biopsychosocial treatment strategies.[76,77] BHPs can support the care team by assessing the patient's perception to treatment and possible barriers to health-care recommendations.[76] They may do so by addressing issues around stigma and culture-specific factors that may influence the health beliefs of the patient or caregivers about medication, behavior, and relationships with health-care providers. BHPs may also help facilitate debriefings in the aftermath of a loss or adverse outcome and assist in meeting with patients and families when complex medical decisions must be made.[78]

Together with social service colleagues, BHPs coordinate referrals to additional resources including to psychiatry and tertiary or higher levels of mental health or substance recovery support.[77] The focus on real-time collaboration, warm handoffs, and regular meetings among the care team reduces potential risks and enhances the ability to readily identify and address any changes in psychiatric symptoms, and

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assists patients in developing the skills needed to improve and maintain good health.

Occupational therapists develop and implement customized interventions to improve one's ability to perform daily activities. After performing a comprehensive assessment of the individual and the environment, the occupational therapy practitioner uses a variety of methods, including activity modification, environmental adaptation, and/or skills training, to enable the adult with I/DD to participate in desired or needed daily occupations.[40] Occupational therapy services are provided directly to an individual with ID, indirectly through consultation with caregivers, or as a combination of the two.[79]

Physical therapists define themselves as "movement experts."[80] They collaborate with patients to develop individual plans to improve movement, reduce pain, restore function, and prevent disability.[80] Physical therapists contribute to the care of aging adults with I/DD by evaluating movement dysfunction and developing an individualized plan to restore, maintain, or promote physical function. Physical therapists address movement complications associated with aging, such as fall risk[81] and pain management.[43]

Speech-language pathologists are experts in prevention, assessment, diagnosis, and treatment of speech, language, and communication disorders.[82] They also address cognitive-communication and swallowing disorders in children and adults. Examples of interventions speech-language pathologists provide individuals with I/DD include interventions to enhance oral communication[83] and intelligibility; [84] introduction and training in alternative communication methods; [85] and information and guidance on swallowing disorders.[86]

Social workers. Social work is defined as the professional activity of "helping people obtain tangible services; counseling and psychotherapy with individuals, families and groups; helping communities or groups provide or improve social and health services; and participating in legislative processes."[87] Social workers devise effective and realistic care plans to ensure safe and stable housing for patients. They may assist patients in finding employment and/or applying for disability benefits or navigating through the complexities of applying for and obtaining financial assistance. In collaboration with the care team, they process referrals to link patients or family to internal and external community agencies for needed resources. Social workers often act as advocates for patients to make sure the appropriate level of resources or services is received and assist the care team in addressing the psychosocial aspects of the impact of disability.

Care coordinators/case managers may come from various disciplines such as nursing or social work. They focus on reducing barriers, establishing linkages, and sharing information between and within agencies in the community. Perrin et al. (2018) suggested planned, proactive, and structured communication between all members of the care team is an important component that can reinforce disease management and prevention in this population.[88]

Severe and Persistent Mental Illness

Definition

Severe and persistent mental illness (SPMI) is a group of mental health disorders that presents in early adulthood and persists, significantly impacting psychosocial functioning.[89,90] Diagnoses typically classified as SPMI include schizophrenia and schizoaffective disorder, treatment refractory major depression, and bipolar disorder.[89,3]

Etiology

Genetic and biologic factors along with environmental factors are felt to contribute to SPMI.[89]

Prevalence, Life Expectancy, and Common Comorbidities

The number of older adults with SPMI is growing as the baby-boomer population ages and is estimated to account for 4–6% of the population.[91] Individuals with SPMI have decreased life expectancy, dying 10–20 years earlier than the general population.[3] The mortality gap in this population has increased over time, with leading causes of death being cardiovascular, respiratory, metabolic diseases, and cancer.[3,91] Suicide and unintentional injury are common, and tobacco use and obesity contribute to excessive mortality.[3,91] Unrecognized medical diagnoses also remain a significant factor.[91] Rates of substance abuse, high-risk sexual behavior, and HIV infection are increased in this group of patients.[89] One study found decreased survival was associated with higher age, Western background, years

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of cigarette smoking, symptoms of disorganization, and anticholinergic side effects of antipsychotics.[3]

Approach to Treatment and Healthy Aging

Important aspects of treatment include community-based multidisciplinary treatment, family support and intervention, identification of comorbid substance use, and health risk reduction.[89] When treating older adults with SPMI, consider the following three skills training programs that have been shown to be effective in randomized clinical trials: Helping People Experience Success (HOPES), Functional Adaptation Skills Training (FAST), and Cognitive-Behavioral Social Skills Training (CBSST).[91]

HOPES aims to enhance independent functioning and community tenure by integrating psychosocial skills and preventive health management. The Skills Training Program's unique curriculum is specifically tailored to older adults and includes age-specific adaptations of established techniques to support living longer, healthier, and more fulfilling lives. The comprehensive curriculum includes a range of topic areas, including: Making the Most of Leisure Time; Living Independently in the Community; Communicating Effectively; Making and Keeping Friends; Healthy Living; Making the Most of a Health Care Visit; and Using Medications Effectively.[91,92]

FAST is a group-based, 24-week program designed to enhance daily functioning, independent living, communication, and illness management skills. It targets six areas of daily functioning: medication, medication management, social skills, organization and planning, transportation, and financial management.[91,93]

CBSST combines aspects of CBT and social skills training (SST) within the framework of the biopsychosocial stress-vulnerability model of schizophrenia. CBSST teaches cognitive-behavioral coping techniques, social functioning skills, problem-solving, and compensatory and neurocognitive impairments.[91,93]

Recent innovations in digital tool applications, as well as the now prevalent use of telehealth, are also positioned to play a vital role in enhancing the quality of life of older adults and contributing to their independence. Pilot studies by Pratt et al. (2013) and Godleski et al. (2012) have demonstrated the potential feasibility and effectiveness of automated telehealth in improving illness management for those with SPMI and medical comorbidity.[94,95] These mobile health (mHealth) platforms and apps offer patients the opportunity to manage their own health care (i.e., reminders to take medications on schedule). In a study in which individuals with SPMI and co-occurring diabetes used an automated telehealth intervention, 67% experienced a mean decrease in fasting glucose from baseline as well as a significant decrease in health visits.[91,94] Participants also experienced significant improvements in their ability to manage depression and diastolic blood pressure. Additionally, the integration of some aspect of social media within a collaborative care model could further improve self-management and delivery of interventions. Older adults with SPMI are already turning to this platform for connection and to share collective experiences dealing with physical health and mental illness as well as to find peer support, advice, and mental health information resources.[91]

Autism

Definition

Autism was first formally recognized in the 1940s, and diagnostic criteria were established in the 1980s. Today we think of autism as a complex neurodevelopmental condition, and in 2013, the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-5) began using the term autism spectrum disorder (ASD), which encompasses prior diagnostic terminology including autistic disorder, Asperger's disorder, childhood disintegrative disorder, and pervasive development disorder-not otherwise specified (PDD-NOS).[5] ASD is currently defined as persistent deficits in social communication and social interaction across multiple contexts including social-emotional reciprocity, deficits in nonverbal communicative behaviors used for social interaction, and deficits in developing, maintaining, and understanding relationships.[10]

Etiology

The etiology of ASD is unclear and has been associated with both genetic and environmental factors (e.g., parental age, birth weight, infection).[96] Structural changes in the brain and behavioral differences between males and females have been demonstrated, with ASD being more common in males.[96]

Prevalence, Life Expectancy, and Common Comorbidities

The prevalence of autism is likely between 1% and 2%, although prevalence estimates are challenging because of

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changes in definition and the fact that ASD has only been formally diagnosed for around 40 years.[5,10] The numbers are particularly skewed and misleading for older adults, given changes in the diagnostic criteria and recognition of symptoms.

Individuals with autism experience decreased life expectancy and risk of premature death because of multifactorial and modifiable risk factors that are difficult to quantify.[5,97] Association of earlier death with more severe intellectual disability is suspected.[12] Epilepsy, sudden unexplained death, and injury remain important causes of death for this population.[12,29] People with autism experience increased rates of mortality when hospitalized, even when other factors are controlled for.[98]

Caregivers of children with autism report more difficulty finding a usual source of care for the individual, utilizing services, and acquiring adequate insurance coverage compared with children with other developmental disabilities and mental health conditions, and it is likely that this is exacerbated in adulthood.[99] Compared with the general population, adults with autism have reported lower general and chronic condition self-efficacy, higher odds of unmet physical and mental health needs, lower rates of tetanus vaccination and Pap smears, and greater use of the emergency department.[100]

Common medical comorbidities include epilepsy, cardiovascular and endocrine conditions, immune conditions, obesity, and sleep disorders. Feeding and nutritional deficiencies, which may be related to sensory issues, are common, as are other gastrointestinal problems, such as gastroesophageal reflux and constipation.[5,29] Depression, anxiety, and PTSD are more common in people with autism.[5] With age, the pattern of behaviors among people with autism may shift, with repetitive behaviors becoming less frequent.[12]

Approach to Treatment and Healthy Aging

It is important to understand the patient's personal strengths and challenges. Although ASD is considered to be a spectrum, the individual's strengths and challenges do not operate in a linear manner and instead can be thought of in terms of existing on multiple axes (e.g., spoken language, written communication, sensory sensitivity, emotional regulation).[5]

Some principles for best practices related to communication are the same as for ID, but there are some notable differences. Some self-advocates from the autism community prefer identity-first language (i.e., autistic adults) rather than person-first language (i.e., adults with autism).[5] Difficulties with communication are core to the diagnosis of ASD, and there can be significant discrepancies between receptive and expressive language among individuals in this population.[101] It's critical for the clinician to understand an individual's communication strengths, preferences, and needs. Barriers to health care cited by autistics include fear or anxiety, inadequate time to process and communicate with the provider, sensory issues, and cost concerns.[102]

As a best practice consider:[101]

a. Be literal and specific (Does your side hurt? vs. How do you feel?)
b. Nonverbal communication may be challenging. Do not force/expect a patient with ASD to make eye contact. "Stimming" (repetitive motions like hand flapping and pacing) is often a way for the individual to relieve stress. If unsure, ask the patient or caregiver what it means. (Is the visit getting too stressful or are we okay to continue?)
c. Allow necessary time for processing. Consider longer appointment times, regular follow-up visits, and encouraging patients to make a list of concerns.
d. Be aware of sensory issues. Natural light may be preferred to fluorescent. Encourage patients to bring or provide items that reduce noise or increase sensory stimulation, such as headphones or sensory objects like stress balls. Schedule the visit at a time when the office is quieter or less busy.
e. Offer clear, step-by-step instructions for follow-up care or testing and for how to communicate with office staff. Consider a check-in after the visit to check understanding.

As discussed earlier, facilitating a multidisciplinary approach to maximize the patient's status is important. Areas of focus that may be especially important in the patient population include:

a. Could the patient benefit from an assistive or augmentative communication (AAC) device?[5]
b. What is the role for nonpharmacologic approaches such as cognitive-behavioral therapy for anxiety or depression and exercise to help manage aggression and anxiety?[5]
c. As improved quality of life may be associated with a better social support system, how do you help the individual create that?[12]

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Overlap of ID, SPMI, and Autism

The *Diagnostic Manual–Intellectual Disability Manual* (DM–ID) was published first in 2007 and updated in 2017 with a goal of improving diagnosis of mental illness in those with I/DD using criteria based on observation of behaviors.[103] Prevalence of psychiatric disorders is likely higher in individuals with I/DD compared with the general population, but estimates vary greatly because of diagnostic uncertainty and limited research.[103] Individuals with I/DD, especially those with more severe impairments, may have receptive and expressive language differences that make it difficult for them to report their symptoms and feelings.[103] Diagnostic overshadowing by the clinician may attribute symptoms to the disability and not a mental health diagnosis.[103] Challenging behaviors, including self-injury, need to be carefully evaluated to determine if they are due to physical or psychiatric disorders.[103]

Person-centered care is critical when addressing the challenging needs of this very heterogeneous and not well-understood population.[90] For those with co-occurring diagnoses of I/DD and SPMI, the number of specialized professionals and facilities is limited, further complicating access to care and accentuating health disparities, especially in a time of crisis.[90] Care can be siloed since the agencies that provide care for individuals with ID and SPMI are frequently separate.[90]

Individuals in this group are at elevated risks for developing PTSD due in part to the disorder being frequently inadequately assessed,[104] underdiagnosed, misinterpreted, and untreated,[105] and understudied in this population.[106,107] Exposure to and experience of negative life events,[108] as well as the range of potentially traumatic experiences, further adds to the increased risk factors and vulnerability of this group.[105] Intellectual and communication deficits may interfere with and further complicate the accurate diagnosis of PTSD in this population.[109,110]

Given the high rates of comorbidity of PTSD with other mental health disorders (i.e., major affective disorders, dysthymia, substance abuse disorders, anxiety, or personality disorders), there is often a lack of understanding of the range of factors that influence effective assessment and treatment in this population. Although a number of well-established therapy treatments, including CBT, have been demonstrated as being effective in managing PTSD in the general population, there is a paucity of empirical studies demonstrating similar effectiveness for individuals with ASD, ID, and SPMI.

Most recently, Eye Movement Desensitization and Reprocessing (EMDR) therapy has emerged as feasible and possibly effective therapy for this population. This could be because EMDR does not require homework, is largely nonverbal, and does not involve graduated exposure to trauma triggers.[111] Even with this promising approach, there are still gaps in our understanding and knowledge of PTSD[109] and what successful treatment of PTSD looks like in this population.[111]

It is important that ongoing attention be provided on how to effectively manage the health-care and behavioral health needs of this population that is increasingly living in independent and semi-independent settings. Providers working with this population should ensure that assessment of trauma and related symptoms is a routine part of overall care,[111] even as behavioral equivalents to PTSD symptoms are being identified.[108] Asking questions about changes in mood and activities should also be considered and use of adaptive coping strategies should be encouraged among this population.

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Chapter 50

Home- and Community-Based Long-Term Care

Rebecca Elon and Fatima Sheikh

Introduction

The need for older adults to receive home- and community-based care, by either informal family help or support from an external organization, increases with age. The 2019 Profile of Older Americans[1] reported that the population aged 65 and older grew from 38.8 million in 2008 to 52.4 million in 2018, a 35% increase, and is projected to reach 94.7 million in 2060. With this rise in the older population, the demand for home- and community-based care is expected to continue to rise as well. According to the United States Department of Health and Human Services (US DHHS), in 2019 the percentage of older adults aged 85 or older who needed help with personal care was 21%, more than twice the percentage for adults 75–84 (8%) and five times the percentage of adults 64–74 (4%).[1] Another study found that between the ages of 85 and 89 years, more than half of older adults received a family member's help because of functional limitations. From 90 years onward, only a minority of individuals (24%) did not need some help from others.[2] The cohort of persons aged 85 and older is one of the fasting-growing age cohorts, projected to more than double from 6.5 million in 2018 to 14.4 million in 2040.[1]

Most of the support to stay in one's own home with dependencies in activities of daily living (ADLs) and instrumental activities of daily living (IADLs) comes from family or other informal caregivers. A 2000 survey found that 8.5% of the men and 10.4% of the women were identified as spousal caregivers. Spousal caregivers were significantly older than non-caregiver spouses.[3] In the USA, older women outnumbered older men at 29.1 million to 23.3 million, and a larger percentage of older men were married (69%) as compared to older women (47%). About 28% of older adults lived alone, but that percentage increased to 44% for women aged 75 and older.[1] Having a spouse or living with others increases the likelihood of being able to receive care in a home setting when functional impairment occurs. Conversely, not having a spouse, living alone, and being an older woman are risk factors for requiring residential community-based care (such as entry into an assisted living facility) or institutional long-term care (such as nursing facility care).

A National Academy of Medicine Report from 2016 found at least 17.7 million individuals in the United States (7.7% of the adult population aged 20 years or older) were caregivers of an older adult with a health or functional limitation.[4] Family caregiving, usually provided primarily by a spouse or daughter, occurs largely out of public view within the home and requires a significant commitment of time and effort on a daily basis. One study found that 65% of older adults with long-term care needs relied exclusively on family and friends.[3] The informal (unpaid) caregivers provide the majority of long-term care for the older adult population and are the backbone of home- and community-based long-term care. The informal caregivers are also central to the older adult's access to and receipt of health care, formal (paid) in-home personal care, and community-based services.[4] (See Chapter 60 for further discussion of the elderly, their families, and their caregivers.) This chapter will discuss the publicly and privately funded resources that are available within communities to assist the informal caregivers in their role and functionally impaired older adults without informal caregivers in living their daily lives.

Public Structures for Home- and Community-Based Care

In 1965, the US Congress passed the Medicare and Medicaid legislation as well as the Older Americans Act. Each of these laws provided some support for care in the older adult's home and community under specified circumstances. The Affordable Care Act of 2010 expanded options for states to rebalance their Medicaid programs by increasing the proportion of funds directed to community-based service, as opposed to institutional long-term care. Despite this governmental financial and programmatic support, chronic long-term care services

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are predominantly paid for as out-of-pocket, private-pay expenses, with or without the help of private long-term care insurance, unless the person is or until the person becomes impoverished and eligible for Medicaid. (For a discussion of long-term care insurance, see Chapter 62.)

The Older Americans Act

The Older Americans Act (OAA) supports a range of home- and community-based services such as meals-on-wheels and senior center group meals, in-home services, health promotion programs, chronic disease prevention and management programs, transportation, legal services, elder abuse prevention, and caregiver support.[5] The OAA established the Administration on Aging that is now part of the Administration for Community Living (ACL) within the US DHHS. The OAA provides funding to an aging services network that includes over 600 area agencies on aging, 56 state agencies, over 200 tribal organizations, two native Hawaiian organizations, and 20,000 local service providers. The programs are generally open to those 60 and older, focusing on those with greatest social or economic need.[5] In March 2020, the US Congress reauthorized the OAA with unanimous, bipartisan support, demonstrating the popularity and core nature of the programs it supports.[6] Clinicians or caregivers who wish to learn about the various services offered locally through this funding mechanism can contact their city or county Area Agency on Aging, Department on Aging, or Health Department. Many of these entities publish an annual booklet listing the various local public and private organizations providing home- and community-based services. They may also offer an in-home evaluation and referral service as the way to enter the formal care system and determine which local agencies may best meet the needs of the older adult and/or caregiver.

Home- and Community-Based Care under Medicare

Medicare Part A, generally known as hospital insurance, and Medicare Part B, generally known as medical insurance, both offer limited home care benefits.[7] The Medicare home health benefit is often utilized upon discharge home after an episode of hospitalization or after a short-term Medicare Part A stay in a skilled nursing facility to help ensure a safe transition and continuation of care. In order to qualify for this benefit, the beneficiary must be homebound, defined by Medicare as: (1) having trouble leaving the home without help such as use of a cane, wheelchair, walker, or crutches, needing special transportation, or needing help from another person because of an illness or injury; (2) being advised that leaving the home is not recommended because of an underlying condition; (3) not being able to leave the home because it requires a major effort. There must be a face-to-face visit with a physician (or certain other health-care professionals who work with the physician) in which the clinician certifies the need for home health services. Medicare will cover medically necessary part-time or intermittent skilled nursing care, physical therapy, speech and language therapy, or continuation of occupational therapy. Services may also include medical social services, a part-time or intermittent home health aide, durable medical equipment, and medical supplies for in-home use. The care must be provided by a Medicare-certified home health agency, which is paid under a prospective payment system. The average length of stay in Medicare-certified home health dropped 35%, from 106 days to 69 days, in 2000 when the transition from fee-for-service reimbursement to the prospective payment system occurred.[8] A subsequent analysis found a further drop in the average length of stay in Medicare-certified home health to 45 days.[9] Length of stay in home health was found to correlate with risk of hospitalization within 90 days of discharge from home health, with those who had at least 22 days of home health care and at least four skilled nursing visits during that time demonstrating lower hospital readmission rates compared to those with shorter lengths of stay or fewer skilled nursing visits.[10] The benefit is not intended to provide nonskilled services or ongoing services over the long term. The best way for a caregiver to know if the Medicare beneficiary qualifies for the Medicare home health benefit is to speak to the primary care clinician. If there is a new change in status that might require a short-term, part-time skilled home care intervention, the beneficiary might qualify, even in the absence of a recent hospital or nursing facility stay. The best way for a clinician to determine if their patient might be eligible for Medicare home health services is to call the Medicare-certified home health agency admissions office and discuss the case. Medicare does not pay for routine, ongoing, nonskilled personal care, like help with ADLs or routine medication administration. These would be defined by Medicare as noncovered custodial care needs.[7]

Medicare Part C, generally known as the Medicare Advantage Plan, offers the option to enroll in one of several different types of managed care organizations. The plans are offered by private companies, must follow

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Medicare rules, offer most of what is otherwise offered through Medicare Part A and B, and can offer additional services such as vision, dental, hearing, and wellness programs. Many of the plans also offer prescription drug coverage (Medicare Part D) as a basic benefit of the plan.[7] To determine what home care services are offered through a Medicare Advantage Plan, the caregiver or clinician would need to look at the plan's description of benefits summary, check the plan's website or Medicare Compare, or call the beneficiary services number.

Medicare Part A includes the hospice benefit that can provide additional in-home care for those beneficiaries who are terminally ill, certified to have a prognosis of 6 months or less, and agree to focus on care for comfort and symptom management rather than pursuing curative interventions. Hospice enrollees will receive in-home visits and assistance from nurses, nursing assistants, social workers, chaplains, volunteers, physicians and nurse practitioners, and other clinicians, when needed.[11] Drugs and durable medical equipment for pain relief and symptom management are included. Continuous hospice care can be provided at home up to 24 hours a day for a limited time when an enrollee is having a symptom crisis that the primary caregiver is not able to manage alone. Examples of when this level of care might be activated include: uncontrolled severe pain; trouble breathing; refractory nausea, vomiting, or diarrhea; change in consciousness; restlessness or agitation; seizures; or when the patient is actively dying. The hospice benefit also pays for up to 5 days of a respite stay in a hospice-related facility, if the usual caregiver needs a rest. More than one respite stay can be allowed, but the hospice program will allocate the resource based upon the patient and family needs. Medicare guidelines state that respite stays "can only be provided on an occasional basis."[11] Hospice programs also provide bereavement services to the caregiver after the death of the enrollee.

Home- and Community-Based Care under Medicaid

Medicaid is the US public health insurance program for people with low income. It currently serves about one in five Americans, including primarily children, pregnant women, parents, the disabled, and the elderly.[12] As of June 2019, all 50 states and the District of Columbia have at least one Medicaid program that provides assistance to older individuals living outside of nursing facilities, including in-home services, adult day care, adult foster care, and/or assisted living.[13] Historically, the Medicaid program was very heavily biased toward payment for institutional care for both younger and older adults with functional impairments, rather than community-based services. The US Supreme Court in *Olmstead v. L.C.* (1999) held that unjustified segregation of persons with disabilities within chronic care institutions constitutes discrimination in violation of the Americans with Disabilities Act.[14] The ruling directed all public entities to provide home- and community-based services to persons with disabilities, explaining that "institutional placement of persons who can handle and benefit from community settings perpetuates unwarranted assumptions that persons so isolated are incapable of or unworthy of participating in community life."[14] Although the case was brought by younger disabled persons, it was a landmark decision that benefited older adults with functional impairments as well. The 2010 Affordable Care Act provided states with financial incentives to further rebalance their Medicaid long-term care services, to increase the proportion that is community-based.[15]

Since Medicaid funds come from both the federal government and individual states, the rules come from two sources. Although the federal government sets certain standards, the states are given flexibility on what services they provide, especially for home- and community-based care. Medicaid State Plans (also known as regular Medicaid) in most but not all states will pay for home care in the form of personal care services or personal attendant services. Medicaid waivers will pay for home care and other related in-home support services for those beneficiaries who meet criteria for nursing home admission but prefer to live at home or in other community-based residential settings. The content of the waiver services and their availability will vary state to state.[13]

The various home care services offered by Medicaid programs can include: (1) home health care provided by health-care professionals such as doctors, nurses, and rehabilitation specialists; (2) personal care at home for assistance in performing ADLs or IADLs; (3) homemaker services for assistance with household chores; (4) family and caregiver support for training and respite; (5) home and environmental accessibility modifications such as wheelchair ramps, stair lifts, walk-in bathtubs, etc.; (6) personal emergency response systems; (7) transportation assistance; (8) hospice care; and (9) adult day care providing supervision in a community-based setting, meals, personal care, and social activities, as well as respite for a caregiver who needs to work or attend to their own personal needs. In many states, Medicaid will pay certain family caregivers to provide care to the Medicaid

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beneficiary. Rules determining what services are offered, who can be paid, and under what circumstances vary state to state.[13]

Home- and Community-Based Care with Both Medicare and Medicaid

Individuals who have both Medicare and Medicaid are often referred to as the "dually eligible" within insurance parlance. There are many insurance products specifically designed to address this population called "special needs programs," structured as managed care products offered through private health insurance companies. They typically offer an expanded portfolio of home- and community-based options, in order to provide appropriate care and reduce the cost of care, since the dually eligible population is typically composed of many vulnerable adults with high utilization of emergency and acute care services.[16]

The Programs of All-Inclusive Care for the Elderly, or PACE, are among the most service-intensive, community-based programs for the dually eligible.[17] This model of home- and community-based care began with an effort by members of the Chinatown-North Beach community in San Francisco to better meet the needs of its frail elders through a program called On Lok.[17] The model's goal is to help keep frail elders safely in their own homes through social, rehabilitative, and medical services provided by an interdisciplinary team, with the medical day care center serving as the mission control site. After small-scale replication projects were conducted in various other cities, the PACE model became an officially recognized Medicare and Medicaid provider type. To qualify, the older adult must meet their state's criteria for nursing home admission. In 2020 there were 135 PACE programs operating 272 PACE centers in 31 states serving over 54,000 participants.[18] Some PACE participants without Medicaid pay privately to enroll, but private-pay revenue within PACE remains a small percentage overall.

Home- and Community-Based Care for Special Populations

Older Veterans

The US Department of Veterans Affairs is a cabinet-level federal agency administering numerous benefits for veterans, including disability compensation, vocational rehabilitation and employment, education, housing, home loans, insurance, health care, and burial. Veterans Administration Health Care (VAHC) is open to all veterans discharged with other than a dishonorable discharge but is provided based upon eight levels of eligibility criteria. The highest priority level is reserved for those with service-connected disabilities. The lowest priority level is for those with higher income and no service-connected disability.[19] Veterans who are enrolled in VAHC have access to the Home and Community-Based Care (HCBC) as part of the standard medical benefit if they meet the clinical need criteria and if the service is available in their geographic locale.[20] HCBC can include: home-based primary medical care with house calls from a doctor, nurse practitioner, or physician assistant; skilled home care with a registered nurse and other team members; care management; social services; rehabilitation therapies; psychology services; nutritional consultations; pharmacy services; adult day care; homemaker services; home health aides; remote medical monitoring; hospice and palliative medicine; and respite care. HCBC is a comprehensive benefit for those veterans who qualify.[20]

Native American and Alaska Native Elders

The Indian Health Service (IHS) is a division within the US DHHS providing direct care and public health services within 36 states to the 573 federally recognized American Indian and Alaska Native tribes. Members of these tribes and their descendants are eligible for services through the IHS that serves approximately 2.56 million of the nation's estimated 5.2 million American Indians and Alaska Natives. Many tribes operate their own health systems independent of the IHS. Although the Veterans Administration in recent years has coordinated with the IHS to provide home- and community-based services to older American Indian Veterans, such services within the IHS in general for all tribal elders tend to be underdeveloped.[21] Of note, as of 2010 American Indians and Alaska Natives have an average life expectancy that is 5.5 years less than the rest of the US population.[22]

Older Adults with Alzheimer's Disease and Related Dementias

The Alzheimer's Association is a national organization with state chapters focused on several clinical areas including maximizing quality care and caregiver support, as well as accelerating Alzheimer's disease research and advocacy at the local, state, and federal levels. Offerings

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vary from state to state and between locales, but may include driving safety referrals, 24/7 helplines, caregiver support groups, educational programs, care consultations, safety assessment services, and referrals to appropriate community resources.[23]

Adult day care programs can provide social engagement, meals, activities, and ADL support (such as bathing or showering and incontinence care) for those older adults with dementia who still live at home, but whose caregiver may need to work during the day or need respite from caregiving to attend to other activities. Most day care programs provide transportation to and from the center. Medical adult day care programs have nursing services on-site for medication management and clinical monitoring. Most programs require private payment arrangements but may be funded through long-term care insurance or Medicaid. Approximately half of all adult day care participants have dementia diagnoses, but most centers also serve older adults with functional impairments from stroke, Parkinson's disease, and other causes.

Older Adults with Mental Illness and Substance Use Disorders

According to the National Council on Aging, two thirds of older adults with mental health and substance misuse problems do not receive the treatment they need. These untreated conditions among older adults are associated with poor health outcomes, higher health-care utilization, increased complexity of the course and prognosis of many illnesses, increased disability and impairment, compromised quality of life, increased caregiver stress, increased mortality, and higher risk of suicide.[24]

The Preadmission Screening and Resident Review (PASARR) process outlined in federal regulation is intended to keep those persons with serious mental illness and intellectual disabilities out of the institutional long-term care system and in community settings when appropriate and possible. PASARR screening occurs prior to admission to nursing facilities. A Level I screen is a preliminary assessment to determine whether serious mental illness or intellectual disability is present. If the Level I screen is positive, then an in-depth Level II screen is conducted to determine the individual's needs and the most appropriate setting, and to develop a set of recommendations for services to inform the individual care plan.[25]

Community-based residential treatment centers with staff who are trained in caring for those with chronic mental illness are available in some locales. Day treatment mental health facilities, also known as psychiatric day hospitals or partial hospitalization programs, can provide intensive community-based treatment for those with psychiatric disorders that require more active treatment than can be provided safely by an outpatient clinic but do not rise to the level of requiring the 24-hour around-the-clock care of the inpatient psychiatric hospital.[26] The day treatment mental health-care teams often consist of: on-site psychiatrists for assessment, therapy, and medication management; nurses for medication administration, education, and care planning; psychologists for neuropsychological testing and group or individual therapy; social workers for counseling, group sessions, needs assessment, and resource referrals; and activity professionals for therapeutic engagement. The clients participate in the program during daytime hours and return to their home or other residential site for the night. The day treatment mental health program provides continuity of care for those patients recently discharged from an inpatient psychiatric unit. It can stabilize patients with frequent hospitalizations who are at risk for ongoing recurrent hospitalization. Older adults with major depression, bipolar disorder, anxiety disorder, paranoid disorder, schizophrenia, early dementia with mood disorder or other psychiatric symptoms, and other conditions such as Parkinson's disease, stroke, or seizure disorders complicated by psychiatric symptoms may benefit from the day treatment mental health center services.[26] In 2018, there were 350 mental health/psychiatric day treatment programs in the USA.[27]

Older adults, caregivers, and clinicians should contact their local Department on Aging or similar agency to understand the local resources for older adults with mental health and substance use disorders. In general, mental health resources are underdeveloped in many locales. (For additional information, see Chapters 20 and 21.)

Vulnerable Elders Who Are Mistreated at Home

All team members providing services within the home setting must be observant for signs and symptoms of elder mistreatment. Mistreatment can include physical, sexual, and psychological abuse; neglect by caregivers or self-neglect; and financial exploitation. Caregivers who are overwhelmed are more likely to be involved in abusive behaviors toward vulnerable elders.

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Providing appropriate in-home assistance is an important aspect of abuse prevention and intervention. Adult Protective Services (APS) is a social services program provided by state and local governments serving older adults who need assistance because of elder mistreatment. In all states, APS is charged with receiving and responding to reports of adult maltreatment and working closely with clients and a wide variety of allied professionals to maximize client safety and independence.[28] State laws regarding the obligation to report suspected elder mistreatment vary. Home- and community-based service providers should be familiar with reporting requirements for their states and know how to make referrals to the local APS agency. (For more detailed information on elder mistreatment, see Chapter 54.)

Community-Based Residential Options

US Department of Housing and Urban Development (HUD) Housing for Older Adults

Surveys of older adults repeatedly confirm their desire to stay in their own homes and receive any needed care at home as they age in place. Significant housing insecurity exists for many older adults because of changes in their financial status with retirement or illness, threatening their ability to remain in their homes. In 2017 there were 24 million homeowners and 7 million renters aged 65 years and older. In 2016 the older homeowners had median home equity of $143,500 and median net wealth of $319,200. By comparison, the median net wealth of age-comparable renters was only $6,700.[29] Although the Social Security and Supplemental Security Income (SSI) programs have reduced poverty rates among older people, in 2016 half of all people on Medicare had annual incomes of less than $26,200 per person.[30] In 2017 the threshold for poverty under the official federal poverty measure was $11,765 in annual income for an individual aged 65 years and older. Using this measure, 4.7 million older adults in the USA in 2017 lived in poverty – 9.2% of the 65-plus age cohort. Using the Supplemental Poverty Measure that takes into account geographic variations in cost of living as well as factors such as medical expenses that can contribute to elder impoverishment, 7.2 million older adults in the USA lived in poverty – 14.1% of the 65-plus age cohort. Poverty rates among people aged 65 years and older increase with age and are higher for women, African Americans, Hispanics, and people in poor health.[30]

The 1937 US Housing Act was enacted to provide federal housing assistance during the Great Depression. In 1959, the Housing Act was amended with Section 202 expanding affordable housing for older adults by providing direct federal loans and capital advances to nonprofit entities to build housing for low-income seniors. Congregate meals, housekeeping services, transportation, and care management services are offered in many HUD senior housing locations, but most of them require the resident to be independent in ADLs. In 2016 there were 8,000 Section 202 HUD-sponsored senior communities.[29] Applications for HUD senior housing can start by contacting either the local housing authority or the administrator of the building of interest to the individual. Most sites have long waiting lists.[31] In 1974, Section 8 of the Housing Act was amended to provide vouchers for assistance in paying rent. The individual or family must have income less than 50% of the median income for their region and pay 30–40% of their adjusted income for their housing in a HUD-approved building, the federal government paying the remainder. There are typically long waiting lists to obtain Section 8 vouchers.[31]

Naturally Occurring Retirement Communities (NORCs), Villages, and Continuing Care Retirement Communities (CCRCs)

NORCs

NORCs are neighborhoods or apartment buildings in which a large portion of the residents have reached retirement age. When the neighbors organize a Supportive Service Program (often with the help of a social service agency), the older neighbors can gain access to social and health-related services with the goal of staying at home and aging in place. NORCs are sometimes referred to as Continuing Care Retirement Communities (CCRCs) without walls. They form partnerships with local governmental agencies, public safety agencies, civic, religious, and cultural institutions, community service providers, various community stakeholders, philanthropies, and corporations. New York State has the most developed regulatory structure for defining and supporting NORCs via public–private partnerships.[32]

Villages

The Beacon Hill Village in Boston enrolled its first members in 2002 and is the original model for aging-in-place villages.[33] Villages are geographically defined, self-governing entities funded by membership fees and

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donations. They focus on life enrichment, community connection, and providing and arranging services for their members with the goal of supporting aging in one's own home. The Village movement is considered a creative consumer response to the challenges of aging in place. Participants in this model tend to be wealthier and healthier than the average age-matched cohort. It is estimated there are currently over 400 Villages in the USA.[34]

CCRCs

The CCRC model of long-term care is available to wealthier older adults who may wish to stay within the same community through different phases of the aging process. Residents typically start out living in an independent apartment. In-home services to support the independent apartment dwellers are generally more readily available than they might be in the general community. On-site health clinics, bank branches, beauty salons, educational and social programs, religious services, physical fitness programs including pools, personal trainers and workout rooms, restaurants, and other amenities are generally part of the model. CCRCs also have on-site assisted living units and skilled nursing facilities to take care of their members throughout the aging process when increasing care needs arise, necessitating care at a higher level than can be made available in the independent apartment.

The 2019 average initial entry fee was $329,000 but can top $1 million in some communities. The average monthly fees run from $2,000 to $4,000. There are several different types of contractual financial models within CCRCs. Type A, or a life care contract, is generally associated with the most expensive initial fees, but then provides a full range of services over time without increasing fees as the level of care increases. Type B has lower initial costs and a more limited portfolio of services but would be associated with higher fees over time as service needs increase. Type C is a fee-for-service contract, Type D a rental contract, and Type E an equity contract. There are currently approximately 2,000 CCRCs in the USA.[35]

Residential Long-Term Care Settings

When the care needs of an older adult exceed the capacity of their family members or informal caregivers to provide such care at home, or when such caregivers are lacking, admission into a residential long-term care facility may be necessary. Residential care refers to settings that provide room and board with additional services such as personal care and social and health-care support.

Table 50.1 Differences in assisted living facilities (ALFs) and skilled nursing facilities (SNFs)

	ALFs	SNFs
Provides room and board	+	+
Provides meals/dining service[a]	+	+
Apartment-like accommodation[b]	+	-
Shared rooms[c]	+/-	+
Mostly private pay[d]	+	-
Provides 24-hour skilled nursing care	-	+
Social model	+	+/-
Medical/nursing/rehab model	-	+
Smaller facilities on average <25 beds	+	-
Larger facilities on average >100 beds	-	+
Heavily regulated by federal and state govt	-	+
Regulated by states only	+	-

[a] – ALFs generally provide standard meals; SNFs can provide specialty diets (altered consistency, diabetic, lower sodium, renal, etc.)

[b] – ALFs accommodations vary from studio to full apartment with kitchen and bath

[c] – ALFs generally have private rooms/apartments but some with shared room for lower cost; older SNFs had mostly 2 beds, or sometimes 3 or 4 beds per room on long-term care units; newer SNFs had more private rooms, especially on post-acute units

[d] – Some ALFs accept Medicaid for limited number of beds; SNFs generally have post-acute care paid for by Medicare or private insurance and long-term care that has some private-pay residents (mostly spending down to Medicaid), but predominantly Medicaid in most environments

Historically, residential long-term care has been divided into community-based sites (such as assisted living facilities or board and care homes) and institutional long-term care (such as nursing facilities or state mental hospitals). In recent times the sharp line between community and institutional entities has been blurring because of overlap in some of the traditional concepts. Table 50.1 provides a comparison of community-based residential long-term care versus institutional long-term care. (Also see Chapter 51 for additional discussion of the distinction between assisted living and nursing facility care.)

Assisted Living Facilities/Communities (ALFs)

ALFs serve individuals who need help with ADLs but do not need 24-hour skilled nursing care over an extended period of time. They offer a mix of companionship, privacy, independence, and security in a home-like setting. The average size of an ALF is 33 beds. Small communities

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with 4 to 10 beds make up 46% of ALFs, medium communities with 11 to 25 beds make up 15%, large communities with 20 to 100 beds make up 32%, and extra-large with over 100 beds make up 8%. There are 28,900 ALFs in the USA with close to 1 million beds. Accommodations vary between one- or two-bedroom suites with full kitchens and bathrooms to smaller studio-type spaces. Services generally include: 24-hour supervision and assistance; meals and dining services; housekeeping and maintenance; laundry and linen service; personal care and ADL support; medication management and administration; exercise, health, and wellness programs; activity programs with recreational outings and spiritual activities; coordination with outside service providers; and transportation. Some ALFs focus on specific populations such as those with Alzheimer's disease and related dementias. Other facilities, usually the smaller to medium-sized facilities, may focus on caring for adults with intellectual or developmental disabilities.[36]

Smaller and medium-sized facilities/communities may go by several other names including: group homes, board and care homes, personal care homes, adult family care homes, residential care homes, or adult foster care homes.[36] The median cost for assisted living residential care was just over $4,000 per month in the USA in 2019. The smaller homes are generally less expensive but they may have fewer programmatic elements. The cost for care in smaller homes ranged from $1,500 to $4,500 per month in 2019 in the USA. Since much of the cost relates to the real estate costs, in some high-cost locations ALF care may exceed $10,000 per month.[37] Most of ALF care is private pay, with or without long-term care insurance. Medicaid support of ALF care through the waiver programs varies state to state.[36]

Medical Care in Home- and Community-Based Settings

Medical care is generally not the primary focus within home- and community-based settings; however, having a strong medical presence within the care team is likely to facilitate the best possible service experience and outcomes. In his article on care of the aging patient, Dr. Robert Kane outlined in detail the physician's role within the maze of residential options in home- and community-based services, including: (1) skillful assessment and medical management considering the patient's goals, life expectancy, and comorbidities; (2) clear and ongoing communication with the patient, family, and other care providers; (3) advocacy for patients and families in getting their wishes and needs addressed; and (4) active management of care transitions with adequate transfer of salient information and determining who will assume responsibility for the ongoing primary care in different settings.[38] A recent systematic review of nine studies (with n = 46,156 total patients) evaluating home-based primary care versus usual care outcomes for homebound elders reported fewer hospitalizations, hospital bed days of care, emergency department visits, and long-term care admissions.[39]

Older adults typically continue with the primary care medical practice they have attended over time when they become homebound or enter a community-based residential care setting. Cognitive or functional impairment or high-risk chronic health conditions may prevent an older adult from visiting the primary care office-based setting. When the patient is no longer able to leave the home or facility for an office visit, or if the primary care office is no longer able to respond to the needs of the team and caregiver as well as of the patient, the older adult may choose to switch to a medical practice that will make house calls or on-site visits to the residential care setting. There are increasing numbers of such community-based medical practices in the USA. In 2019 there were 1,100 direct primary care practices in the USA in which the patient pays for care directly, rather than going through insurance company payments. Of these, 68% stated they offered house calls, with eight of the practices being completely mobile without office-based care.[39]

Medical house calls can be performed by physicians, physician assistants, and nurse practitioners as part of continuity of care within an office-based practice, through hospital-based programs, standalone practices, or special programs like Veterans Administration or academic medical centers. House-call providers often collaborate with other agencies or organizations for home health care and other services, such as the delivery of meals at home through the local "meals-on-wheels" program.

A homebound status of the patient is not required for house calls, which is different than home health services provided under a Medicare-certified agency that requires homebound status to render care at home. House-call medical encounters are reimbursable by Medicare, Medicare Advantage plans, Medicaid, and most private insurers when the provider supports the medical necessity of the visit with their medical documentation.[40]

House calls can be part of patient-centric integrated care models like the Veterans Administration Home-Based Primary Care (HBPC) programs, which provide

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regular care to those older adults who are homebound. Medical care in the home aims to assist older adults in maintaining their physical and psychological well-being and function. The focus of care is primary, secondary, and tertiary prevention, with quality management of chronic medical conditions to slow progression and prevent acute exacerbations. The overarching goal is to help keep older adults functioning in their home environment, support their caregivers, and prevent unnecessary emergency department visits and acute hospital admissions. Most HBPC programs have interdisciplinary teams integrated into the model of care that may include nurses, social workers, pharmacists, medical technicians, rehabilitation team members, and case managers. House calls offer community-based primary care and palliative care, which is often tailored to the preferences of the older adult, as it is rendered in their own home environment and can improve patient satisfaction. A house-call visit provides an opportunity for assessment of the home environment and its safety, and allows observation and assessment of other unique areas of care that may not be readily apparent during an office visit such as functional status at home, caregiver situation, medications, availability of food, and overall insight into the living conditions of the older adults that may directly or indirectly affect their health and well-being. Such home assessments can lead to interventions focused on improving home environment and safety, medication adherence, nutritional status, and other clinical issues.[40]

Hospital-at-Home Programs

Hospital-at-home programs are an innovative care model to deliver acute hospital-level care to the older adults at their homes. Older adults who are identified by a physician as candidates for hospital care during the emergency department or office visit may qualify to receive hospital-at-home services through a specialized program. Instead of inpatient admission, they would be transferred home to receive extended nursing care and daily physician visits in the home setting. Diagnostic studies (e.g., X-rays, ultrasounds, electrocardiograms, and echocardiograms), medical treatments (e.g., oxygen, intravenous antibiotics, and fluids), and specialized skilled services (respiratory, nursing, pharmacy services) can be provided as part of these programs. Hospital-at-home programs have been demonstrated to decrease costs of medical care and yield better patient outcomes compared to inpatient care in the selected participants. This model has not been widely adopted in the USA because of multiple barriers including referring physician concerns about patient safety, potential associated legal risk, and lack of reimbursement by insurers.[41]

Home- and Community-Based Care in the Time of COVID-19

When the COVID-19 pandemic emerged in the USA in early 2020, nursing facilities and other congregate living settings were hit hard with high infection rates among staff and residents and associated high case-fatality rates. The impact of the facility-based outbreaks, along with the subsequent ban on visitation in facilities, had a great impact on home- and community-based services as well. Many hospitalized older adults who would have been sent to nursing facilities automatically upon hospital discharge for post-acute convalescence and rehabilitation decided to go directly home instead to avoid COVID-19 exposure. When confronted with the ban on visitation, many families decided to take their loved ones back home, rather than suffer the consequences of separation. The exodus from and lack of entry into congregate settings during the time of COVID-19 resulted in a much heavier demand for home health services, beyond what many agencies were able to staff. In the early days of the pandemic, hospitals attempted to discharge as many patients as possible, to prepare for the anticipated COVID-19 surge. This created an increase in patients requiring home health follow-up.[42] Many home health direct care staff stayed home because they had to care for their children whose schools were closed. Some decided to stay home when shelter-in-place orders were issued, rather than expose themselves and their families to potential COVID-19 infection. Adult day care centers were closed for business. Many medical offices and clinics closed during the early months of the pandemic surge. Although telehealth visits were often made available, many homebound elders lacked the technology or knowledge to participate.[43] Many homebound older adults and family caregivers found their usual support structures vanish during this time. Recognizing that alternatives were needed, the Centers for Medicare and Medicaid Services (CMS) released a toolkit to increase access to alternatives to care in congregate settings. This included expedited changes in waiver services, self-direction programs, and the ability to legally hire responsible relatives. It has been proposed that increased self-direction of home- and community-based services may be a lingering positive effect once the COVID-19 pandemic has retreated.[44] (For further discussion of COVID-19, see Chapter 51.)

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Financial and Workforce Issues in Home- and Community-Based Care

Insurance programs, including long-term care insurance policies, do not provide long-term, 24-hours-a-day, 7-days-a-week, in-home care on an ongoing basis for personal care and ADL support. Such care may be available through a private-pay home care agency, but the hourly cost for 24-hour services is prohibitively expensive for the vast majority of older adults or their families. The cost of in-home senior care will depend on the number of hours the designated worker spends with the client, as well as the kind of service and supplies that are required. Every state has its own median cost of care and charges associated with in-home personal care. The national average for in-home personal care was approximately $4,000 per month with a median hourly rate of $20 in a 2015 survey.[45] Even those who can afford to pay privately may find that the availability of such service is extremely limited. The workforce issues that have limited the entry into and the retention of the in-home and community-based workforce have been studied extensively. The direct care workforce is often poorly trained, poorly compensated, disrespected, and restricted in their duties, with little opportunity for improving their education and skills for advancement in the workforce.[46] Yet the demand for such services is set to increase dramatically with the aging demographics. In his book, *Who Will Care for Us?*, Paul Osterman makes a compelling argument that improving the jobs and circumstances of direct care workers is both desirable and feasible and in fact, imperative, in order to improve the care and outcomes and reduce health-care costs. It is also a social justice issue, since most low-wage direct care workers are part of a job market that perpetuates entrenched and corrosive inequality.[46] Addressing workforce issues in long-term care is more than an academic public policy concern, since, barring premature death, most of us have the need for home- and community-based long-term care services for our families or ourselves looming in our collective futures.

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Chapter 51

Institutional Post-Acute and Long-Term Care Medicine

Rebecca Elon and Fatima Sheikh

Introduction

The modern era of medical care in nursing facilities in the United States can be traced to an outbreak of Salmonella food poisoning in a Baltimore, Maryland nursing home in the summer of 1970.[1] After enjoying a meal of shrimp and deviled eggs, 95 of the 144 residents of Gould's Convalesarium became ill with diarrhea.[2] Thirty-six resident deaths were linked to this epidemic. Almost half of the facility staff also developed a diarrheal illness, but all survived. At the time, this was thought to be the most serious single incident of Salmonella poisoning in the USA. There were delays in recognizing the outbreak situation, making the diagnosis of Salmonella, and reporting it to the health department. The meal mentioned above, although temporally related and highly suspected as the single source of contaminated food, could not be proven conclusively as causative because of the delays.[3] The state health department and state medical society conducted investigations into the situation. Medicare officials also made an investigation that was followed by a congressional investigation, reports, and public hearings.[4]

At the time, there was no federal requirement that all nursing homes employ a medical director. The convalesarium had a "principal physician" as required by state regulation but his role was described as "obscure."[4] He did not have any specified duties or responsibilities, nor did he receive any compensation for the titular role. He served as attending physician for 11 of the 144 facility residents. When deposed about the outbreak, the principal physician stated that a sharp increase in cases of diarrhea was not alarming, since diarrhea is not uncommon in the elderly.[2] There were more than 40 doctors who served as attending physicians at the facility. When the attending physicians were called about a diarrheal illness in their patients, the nursing facility staff testified they did not report the large number of other residents who were ill, nor did any of the physicians inquire if any other residents had diarrhea. The state medical society investigation concluded that since Salmonella was a reportable illness to the state health department, each attending physician who ultimately made the diagnosis or became aware of Salmonella in their patients was obligated to report it. Since none of them did so, they were in violation of state law. Since there was "nothing malicious" in the failure to report, however, no sanctions were recommended. The conclusion was that there was nothing contrary to usual medical practice in the physicians' actions and that no single physician could technically be held responsible for the totality of the health care in the home.[4] The state health department special investigative panel concluded that nursing home regulations and oversight were insufficient and ineffective. The secretary of the Maryland Department of Health and Mental Hygiene (DHMH) reported estimates that 99% of the cases of Salmonella in the state were never reported. The US Surgeon General testified that the number of Salmonella cases in the USA annually was estimated to be over 2 million, but that nationally only 20,000 were reported. Both officials recommended physician and nursing home staff education about infection prevention, control, and outbreak response.[3,4]

In 1975 the US Senate Special Committee on Aging conducted an investigation into the overall poor conditions in US nursing homes, highlighting the Salmonella outbreak in its report. One of their supporting papers, entitled "Doctors in nursing homes: The shunned responsibility," declared:

> The Baltimore epidemic, therefore, brought to light several very important consequences stemming from the absence of the physician in nursing homes. . ..The need for physicians to exercise greater responsibility for the one million patients in US nursing homes is abundantly clear. . ..Clearly infectious disease can be controlled only if physicians take a more active interest in the care of nursing home patients . . ..Until doctors take a greater interest, the litany of nursing home abuses will continue, the majority of America's nursing homes will be substandard and the quality of patient care will be unacceptable.[4]

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The Committee issued the following set of recommendations for improving medical participation in nursing homes:

1. US schools of medicine should place greater emphasis on geriatrics in their general curricula;
2. Continuing education programs in geriatrics should be established for physicians;
3. Funds should be provided to help establish departments of geriatrics in schools of medicine;
4. US nursing homes should be required to have either a medical director or an organized medical staff. Smaller facilities could have medical directors serving part-time. The medical director should bear overall legal responsibility for the care of patients in the facility;
5. Funds should be provided to train medical corpsmen discharged from the armed services with the intent that they could serve in nursing homes under the physician's direction, assuming some of the duties of the physician in caring for nursing home patients;
6. A program should be enacted providing training in geriatrics for physician's assistants and nurse practitioners to work in nursing homes;
7. A program should be enacted providing funds to schools of nursing to establish training programs for nursing home personnel;
8. Comprehensive health planning agencies should give preference to nursing homes so they can be built near hospitals and physicians' offices to reduce the distance doctors have to travel to visit patients;
9. Physicians should be required to view bodies of patients who have died in nursing homes and death certificates should be required to be signed within 24 hours of death;
10. Physicians should make greater efforts to report infectious diseases. State statutes should provide appropriate fines and penalties for failure to do so;
11. Physicians should exercise greater control of medications for nursing home patients. Only emergency medications should be prescribed over the telephone. Medications so prescribed should be ratified by the physician within 24 hours. Thirty-day stop orders for medication and treatment orders should be enforced;
12. Physicians should be required to see patients at least once every 30 days under the Medicare and Medicaid nursing home programs.[4]

Many of these recommendations were enacted over the following decades. Medical participation in US nursing homes has improved dramatically since 1970, though there is still great variability in the quality of medical practice and medical direction among US nursing facilities. This chapter will discuss the basics of nursing home medical direction and medical care in US nursing homes, beginning with a description of the infection control issues faced by US nursing facilities during the COVID-19 pandemic.

COVID-19 in US Nursing Homes in 2020

On January 21, 2020 the US Centers for Disease Control and Prevention (CDC) and the Washington State Department of Health announced the first diagnosed case of COVID-19 in the USA resulting from infection with the SARS-CoV-2 virus in a man who had returned from visiting his family in Wuhan, China.[5] The first nursing facility outbreak of COVID-19 was announced on February 28, 2020 in Washington State after a nursing facility resident who was sent to the hospital with a respiratory illness was found to be SARS-CoV-2 positive. The first COVID-19-related death in the USA on February 29, 2020 was a nursing facility resident from the facility with the initial outbreak (facility A).[6,7]

The local health department along with the CDC launched an investigation and intervention at facility A that included quarantining of exposed persons, isolation of known cases, contact tracing, and on-site enhancement of infection prevention and control measures. When the index case was first identified, at least 45 residents and staff dispersed across facility A had symptoms of a respiratory illness. On March 5, 2020 the World Health Organization declared COVID-19 an international pandemic. By March 18, 2020, 167 persons associated with facility A were SARS-CoV-2 positive, including 101 residents (8 of whom were asymptomatic), 50 health-care personnel, and 16 visitors to the facility. The hospitalization rate for the positive residents was 55% with a case fatality rate of 34%. The average age of those who were COVID-positive was 83 years for the residents, 62.5 for the visitors, and 43.5 for the facility personnel. No staff from facility A with COVID-19 died of the disease. By March 18, 2020, 30 nursing facilities and assisted living facilities in the same county had COVID-19 diagnosed in their staff and residents.[7] By late March at least 146 other nursing homes across the country had confirmed cases of COVID-19.[8]

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Facility A was a Center for Medicare and Medicaid Services (CMS) 5-Star facility and initially did not appear to have any baseline quality problems that would have placed it at greater risk than other facilities for a COVID-19 outbreak.[6,7] Issues identified in the facility that contributed to the facility and regional outbreak, however, included: (1) delayed recognition, reporting, and response to a respiratory outbreak (regardless of etiology); (2) staff working at more than one facility; (3) no paid sick leave for staff, creating an incentive to work while ill; (4) residents being transferred to other facilities despite the respiratory outbreak; (5) difficulty diagnosing COVID-19 on the basis of symptoms alone; (6) initial limited availability of testing supplies and early recommendations restricting testing to symptomatic persons who had traveled to China or were exposed to a positive case; (7) unfamiliarity with recommendations for proper use of personal protective equipment (PPE); and (8) shortages of PPE.[7] As the death toll in facility A rose to 45 and regulators conducted their focused infection control survey of the facility, deficiency citations resulted in civil monetary penalties of $610,000 being levied against the facility. The fines were contested by the ownership of the facility, and an administrative law judge dismissed most of the deficiency citations, citing lack of adequate evidence of deficient practice from the surveyors' report. The government appealed the administrative law judge's ruling, with the next round of hearings having been scheduled for 2021.[8]

On March 5, 2020 the first outbreak of COVID-19 was reported in a Washington State senior community comprising independent and assisted living units when two residents were hospitalized and diagnosed with COVID-19.[9] The community comprised 45 independent living (IL) and 38 assisted living (AL) residents along with 62 staff members. On March 5, 2020, 3 of 80 residents (3.8%) and 2 of 62 staff (3.2%) tested positive for SARS-CoV-2. All were asymptomatic at the time of testing. On March 6, a number of infection control measures were put into place, including: (1) all the residents were isolated to their rooms; (2) no communal meals or activities were allowed; (3) no visitors were allowed into the facility; (4) all staff were screened for symptoms upon reporting to work each day, and not allowed to work if febrile or exhibiting symptoms; and (5) enhanced hygiene was instituted with numerous hand sanitizer stations placed around the facility, and housekeeping staff increased the cleaning and disinfecting of high-touch surfaces.[9] All residents and staff were retested 1 week later. One additional asymptomatic resident tested positive. The dramatic difference in the rate of infection, hospitalization, and deaths between the nursing facility and the IL/AL senior community was thought to be due to several factors including: (1) earlier recognition, reporting, and intervention; (2) greater ability for social distancing since the residents had private apartments in the IL/AL site; and (3) less contact with health-care personnel due to being healthier at baseline than the nursing facility cohort.[9]

As of August 31, 2020, there were 169,601 deaths from COVID-19 reported in the USA, and 71,485 (42%) had occurred in residents of nursing facilities and other residential care facilities.[10] Persons 85 years and older comprised 31% of all COVID-19 deaths, 75 to 84 years old 26%, 65 to 74 years old 21%, and 55 to 64 years old 20%, with those younger than 35 years old comprising 0.8% of all COVID-19 deaths.[10] Approximately 2.1 million Americans were living in nursing facilities and other congregate residential care settings, comprising approximately 0.6% of the total US population. By August 31, 2020, 3.4% of the entire US residential long-term care population had died of COVID-19. By the end of October 2020 CMS reported a total of 268,707 confirmed cases of COVID-19 in US nursing facilities, with an additional 150,195 suspected cases.[11]

With the crisis in the nursing and residential long-term care facilities, articles in the lay and professional press discussed what or who was responsible for the failure to protect the nation's most vulnerable members from the pandemic. Early public policy approaches were questioned and criticized.[12,13] Nursing facilities were accused of providing substandard care. Criminal charges of "causing or permitting serious bodily injury or neglect of an elder," with possible prison time if convicted, were brought against a superintendent and medical director of a facility where 80 people died of COVID-19.[14] Calls for enhanced enforcement of federal regulations were ubiquitous, causing CMS in March 2020 to suspend all routine nursing home inspection surveys and conduct focused infection control surveys nationwide in all 15,000 Medicare and Medicaid certified nursing facilities.[15] Advocates for nursing home reform called for heightened regulatory toughness and higher fines against facilities, but some acknowledged that not all outbreaks could be avoided, even with adequate baseline infection control practices in place. Industry and professional groups cited chronic core issues, such as the difficulty in recruiting and retaining adequate high-quality staff, that left facilities vulnerable when crisis hit. Staffing issues were exacerbated by the pandemic when staff became ill and could not work, required quarantining because of

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exposure, left the field because of fear of contracting COVID-19, or had to stay home when their children's schools closed. Initially there was an inability to get adequate testing in the facilities, and there were shortages of PPE. The role of the asymptomatic staff members in bringing the virus into the facility was not recognized in the early weeks. All of these issues created a perfect storm of pandemic devastation. Mark Parkinson, the former governor of Kansas and president of the American Health Care Association (AHCA – the major US nursing home industry group), stated, "Our profession faces its greatest challenge in history. . ..This isn't like the usual fight we have in [Washington] D.C. over a 2% increase or cut or over some crazy regulation. Instead, this is a battle for the lives of our residents, our staff and the very survival of our sector."[8]

Institutional Long-Term Care Infection Prevention and Control in the Pre-COVID-19 Era

Infection prevention and control has been an ongoing area of concern in nursing facilities since the 1970s outbreak of Salmonella in the Baltimore nursing facility. For decades, federal regulations have required infection control programs within Medicare and Medicaid certified nursing facilities.[16] Most state regulations also require infection control programs in their licensed facilities. Some states require a specific educational program for the nursing facility infection prevention and control practitioner (IPCP), as well as designating a half-time infection control position for facilities with fewer than 200 beds and a full-time position for 200 or more beds.[17] Despite the regulatory mandates, infections and outbreaks in nursing facilities have remained a prevalent threat to residents and staff over the years.[18]

Prior to the COVID-19 pandemic, respiratory outbreaks accounted for almost half of all infectious disease outbreaks in nursing facilities, with influenza A being the most commonly reported causative agent. Influenza B, parainfluenza, respiratory syncytial virus, chlamydia pneumoniae, and Legionella also cause facility respiratory outbreaks, but are less commonly seen.[18] Since annual seasonal outbreaks of influenza in facilities are common and expected, the prevention and intervention procedures in most facilities are typically well rehearsed. Vaccination of residents and staff against influenza is an annual early autumn campaign in all facilities. Vaccination rates of residents of greater than 80% of the population correlate with a decreased risk of facility outbreak, though even successful vaccination campaigns do not totally eliminate the risk because of viral antigenic shifts and/or diminished responsiveness in frail elderly persons.[18] The 2020–2021 high-dose quadrivalent, recombinant, and adjuvanted influenza vaccines all create a stronger antibody response in older adults, but the risk of hospitalization and death with their use remains to be established in randomized controlled trials.[19] A study of the older high-dose trivalent vaccine in 823 US nursing facilities demonstrated lower rates of hospitalization due to respiratory illness in those receiving high-dose rather than standard-dose vaccines.[20]

Obtaining consent for annual influenza vaccination at the time of admission to the facility has boosted rates of vaccination, but the facility cannot force residents to take the vaccine. Rates of resident acceptance of the vaccine are much lower than 80% in many facilities. One study found that vaccinating health-care workers was an important factor in decreasing influenza mortality in the facility residents.[21] Many hospitals, health systems, and nursing facilities in the USA over recent years have made consent to take the influenza vaccine a condition of employment, with exceptions only for those with valid medical contraindications. This has increased staff rates of vaccination to over 90% in many facilities. Staff who don't take the influenza vaccine have been required to wear a mask while in contact with residents.

As seen in both the Salmonella and COVID-19 examples, early recognition, intervention, and reporting are key to successful control of an infectious outbreak in an institutional facility. A study of the impact of required training for IPCPs demonstrated that those who had taken a standardized 2.5-day course were able to detect outbreak situations much sooner than those who had not received the education.[22] The role of the IPCP (usually a nurse) is essential to early recognition of facility outbreaks. The standard of care requires that line listings be kept on an ongoing, routine basis by the IPCP for the common nursing facility infections, including respiratory, gastrointestinal, urinary, and skin infections. Historically, the IPCP would attend the facility's "morning report" or "end-of-day sign-out" to hear from the unit managers their reports of any infections or other notable occurrences from the prior shift or day. With the advent of electronic health records, the IPCP can also get data on resident temperatures, symptoms, and lab studies collected from resident electronic records. The IPCP would also keep in close communication with the human resources scheduling office to hear about any infectious

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symptoms associated with employee callouts. Separate line listings are generally kept for facility residents and staff. Daily review of the line listings would reveal any unusual numbers of events, such as residents or staff with respiratory, gastrointestinal, or other symptoms.

A line listing is a standardized approach to collecting and organizing data. Each row represents one person or case of disease. Each column represents information about one characteristic, such as name, unit or room location, date of onset, various symptoms, lab results, interventions, etc. Separate line listings would be kept for each category of infection; for example, one line listing for respiratory cases, another for gastrointestinal cases. Sample line listings are available on the CDC website.[23] When the IPCP notes an increase in cases, the administrator, director of nursing, and medical director should be notified, in order to develop a response.

In most states, even one laboratory-confirmed case of influenza in a facility is reportable to the local health department. Even in the absence of laboratory confirmation, in general, two or more cases of influenza-like illness on a specific unit would also be reportable to the health department, even outside of the normal influenza season. The health department epidemiologist and IPCP generally help guide the facility's infection control response, which should include: (1) advising on specific disease testing approaches; (2) keeping residents and staff on their own unit rather than traveling throughout the facility; (3) confining residents to their rooms when necessary; (4) canceling group dining and activities; (5) reviewing isolation procedures and necessary PPE for active cases and exposed individuals; (6) enhancing environment cleaning; (7) requiring that any visitors or staff who are ill refrain from entering the facility; (8) urging those who initially refused vaccination to take the influenza vaccine; (9) having the health department medical officer and facility medical director discuss when and how to order prophylactic anti-influenza antiviral medication for the residents and staff to limit the outbreak; (10) increasing the monitoring of staff's compliance with PPE and handwashing recommendations in addition to ongoing staff education about infection control policies and procedures; and (11) deciding when the facility needs to be closed to new admissions because of the outbreak and when admissions can be resumed after the outbreak is brought under control.

As of February 2020, prior to the recognition of COVID-19 in US nursing facilities, the Office of the Inspector General of the US Department of Health and Human Services reported that State Survey Agencies had cited 6,600 nursing facilities (approximately 43% of all CMS participating facilities) over the prior year for infection prevention and control program deficiencies, including lacking a plan of correction in place for the deficiencies.[24] The report on its post-COVID-19 infection control facility surveys is expected in 2021.

The Role of the Facility Medical Director

The Salmonella outbreak in the Baltimore nursing facility in 1970 illustrated the need for physician management of the well-being of the entire facility population, rather than the typical medical focus by the attending physician on the clinical care of the individual patient. The medical director needs to be the physician to the facility rather than, or in addition to, being a clinician for individual residents. In 1967, the Maryland State Commission on Aging appointed a Commission to conduct a survey on nursing home care in the state. The Commission noted that "Care and morale seemed better in those homes that had an interested physician, one who took the time to advise the administration on medical matters and helped the director of nursing with patient care problems." The Commission recommended that nursing home physicians band together and provide a "united professional voice regarding problems in medical management of patients in chronic disease facilities." Such a group began meeting in Maryland and drew up standards for medical practice in nursing homes that was subsequently approved by the state medical society.[1]

In 1971, the US Department of Health, Education and Welfare (HEW) asked the American Medical Association (AMA) to sponsor seminars on the role of the nursing facility medical director. The AMA, in collaboration with other professional and industry organizations, conducted 10 seminars around the country, one in each HEW region. There were over 1,500 attendees, including 558 physicians. In 1973, the AMA House of Delegates approved "Guidelines for a medical director in a long-term care facility" that outlined 15 duties and tasks for the medical director to "help ensure the adequacy and appropriateness of the medical care provided to patients" in nursing facilities. In 1974, HEW gave the AMA another grant to work with state medical societies to provide education on nursing facility medical direction at local levels. By 1976, 3,700 health professionals, including 1,200 physicians, had attended the seminars in 17 states.[1]

In 1977, the American Health Care Medical Directors Association (AHCMDA) was organized following

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a seminar for medical directors sponsored by the Georgia Health Care Association, the AMA, and Georgia nursing facility medical directors, led by William Dodd, MD, who served as the founding president of the AHCMDA. In 1980, the organization changed its name to the American Medical Directors Association (AMDA).[25] In 2014, AMDA changed its name to AMDA – The Society for Post-Acute and Long-Term Care Medicine. The Society's national organization headquarters are located in Columbia, Maryland, with state chapters in all 50 states.[26]

AMDA – The Society for Post-Acute and Long-Term Care Medicine – has become the premier source of management and leadership education and advocacy for nursing facility medical directors. The Society offers a core curriculum in nursing facility medical direction that focuses on the major roles, functions, and tasks expected of medical directors to successfully perform their duties. It introduces the medical director to the complexities of US regulatory compliance, which is a major concern for all US nursing facilities.[26] This curriculum was the evolution and expansion of the Medical Director Training Program developed by Dr. James Pattee and Dr. Tom Altemeier at the University of Minnesota in the 1980s.[27] The Society's journal, *JAMDA – The Journal of Post-Acute and Long-Term Care Medicine*, publishes research related to post-acute and long-term care management and practice.[28] The Society also has an annual meeting, webinars, and numerous online resources to support the nursing facility medical director, in addition to public policy advocacy for improving care in nursing facilities.

The American Board of Post-Acute and Long-Term Care Medicine administers a certification program for nursing facility medical directors.[29] Medical directors who have completed the AMDA core curriculum and other relevant clinical continuing education credits, have obtained experience in a facility setting, and are in good professional standing may apply for certification. The Certified Medical Director (CMD) Credential indicates the physician has taken the time to obtain the baseline training, knowledge, and skills to be able to perform the role of medical director in a capable and high-quality fashion. Over 4,500 physicians had received the CMD Credential as of 2020.

The major issues requiring current nursing facility medical directors involvement include: infection prevention and control; antibiotic stewardship; appropriate prescribing (especially with regard to psychoactive medications); safety of residents (such as falls prevention programs); improving transitions of care between various settings; improving clinical capacity of the staff to help reduce unnecessary hospitalizations and readmissions to hospital; understanding of regulatory requirements and risk management strategies; knowledge of quality indicators with state and national benchmarks and how medical care impacts these indicators; awareness of the components of the CMS 5-Star rating system for nursing facilities and how to help the facility improve its care and ratings; credentialing of clinicians and promoting quality medical services; and fostering positive relationships with the local hospitals and service agencies. All of these issues require effective communication skills by the medical director. The medical director needs to be in contact with the attending physicians and other practitioners on a regular basis, both informally and formally through medical staff meetings, and not just when problems arise. Medical directors must be comfortable and confident in their own clinical expertise and ability to review the work of their peers and provide constructive feedback.

There are more issues in most nursing facilities than a part-time medical director can address in a comprehensive fashion. It is therefore necessary for the medical director to work with the administrator and director of nursing to focus on the key issues for the facility, which will likely vary over time. Medical directors should also make a realistic assessment of how much time it will take for them to perform the role in an attentive fashion and negotiate a reasonable stipend for their administrative time. Since clinical medical malpractice insurance does not cover administrative work, medical directors should receive written confirmation from the facility that their activities are covered by the facility's insurer for their administrative role or obtain such coverage on their own.

The COVID-19 pandemic brought into further focus the need for capable and involved nursing facility medical directors who could help the facility and the medical staff keep up with the quickly evolving recommendations from CMS, CDC, the various state and local health departments, and local jurisdictions.[30] The COVID-19 pandemic demonstrated the need for rapid team learning and successful implementation of new and evolving directives. The successful companies and facilities in responding to the challenges of the pandemic were those that became effective "learning organizations."[31] The medical director during COVID-19 had a heightened and essential role to play in communication with the clinicians and serving as the liaison between the medical staff,

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the facility administration, nursing staff, local hospitals, and health department. Many medical directors performed admirably during the pandemic, but large variability in nursing facility medical director participation and knowledge remains. The consequences of inadequate nursing facility medical direction were illustrated most profoundly during the time of COVID-19.

Medical Care in US Nursing Facilities

The Structure of Medical Practice in Nursing Facilities

The structure of medical care delivery in US nursing facilities has been in evolution over the past decades. In the 1970s, as illustrated in the Baltimore Salmonella case, nursing facilities generally welcomed as many physicians as possible to serve as the nursing facility attending physician for their patients/residents. The facilities believed that the individual physicians would encourage their own patients to be admitted to the facility if they served as an attending physician there, thereby increasing referrals and admissions. This model also provided continuity of care for the patient when their long-term outpatient physician managed their nursing home medical care. This model of medical participation is called the "open medical staff model." As medical care of the nursing facility resident/patient became more complex, it was necessary to transition to attending physicians who had enough time to devote to their nursing facility practice, and not just stop by in their spare time after rounds at the hospital and after office hours were completed. Most nursing facility admissions currently come from hospitals, where full-time hospitalists are increasingly the ones caring for the patients during acute hospitalization. Discharge planning nurses or social workers are now the ones discussing with patients and families which nursing facilities would be the best to choose from for post-acute care at hospital discharge. The primary care, outpatient, office-based attending physicians are no longer the ones in charge of discharge planning for their patients. Their patients often are assigned to the care of a nursing home physician who is focused exclusively on this site of practice. This model is known as a "closed medical staff model."

The dual role of most US nursing facilities includes both residential, long-term care (LTC) and short-term, post-acute care (PAC). Those who live in the LTC setting are generally referred to as "residents" rather than "patients," since the goal is to try to provide a more home-like atmosphere and a less institutional or "medicalized" approach. In reality, however, the LTC residents benefit greatly from physicians, nurse practitioners (NPs), and physician assistants (PAs) who provide care that is respectful of the setting as the person's home and are knowledgeable in geriatric medicine.

The frequency of visits in the LTC setting has been set in federal regulation dating back to the original Medicare and Medicaid legislation in 1965. The initial physician visit is required by federal regulation to be made within the first 30 days after admission, with visits every 30 days for the first 90 days and every 60 days thereafter. Although the physician is required to make the initial visit, a nonphysician practitioner is allowed to make a "first visit" if medically necessary prior to the formal initial physician visit. Most facilities have policies that require that the first medical visit be made within 24 to 72 hours after admission, to promote safe and successful transitions of care. Federal regulation allows the NP or PA to make every other of the mandated visits. Therefore, the physician would only be required under federal regulation to visit the nursing facility resident once every 120 days, if the NP or PA was making the interim visit. Many states and nursing facilities have requirements for more frequent physician participation.

Since post-acute care nursing facility admissions generally come from hospitals, the patients are often in need of ongoing close medical supervision and monitoring. Facility policies typically require that the first post-acute medical visits be made within the first 24 to 48 hours after admission. Since the post-acute nursing facility admission is for medical and rehabilitative purposes, the people admitted to the post-acute service are generally referred to as "patients." These patients need to be seen as often as medically necessary, typically at least weekly for the first 30 days, or more often if there are acute issues that arise and need more intensive medical management for treatment in place, to prevent rehospitalization.

Physician groups that serve only nursing facilities have increased in number over the past decade. Some of these organizations are national in scope and gain access to a "market" by purchasing practices of doctors with large nursing facility caseloads, or by creating preferential arrangements with the nursing facility company. These national physician organizations have created significant disruption in traditional models of nursing home medical care in some locales.

In the mid-1980s, a Medicare demonstration project to try to improve the medical management of long-term care nursing facility residents was funded by the Health Care Financing Administration (HCFA – currently known as

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CMS). In the EverCare model, NPs who were trained in geriatric medicine were placed in nursing facilities under capitated financing. They were in the facility much more frequently than the typical nursing facility attending physician and focused on preventive care, establishing advance directives, effective chronic disease management, early detection and intervention for intercurrent illness, and limiting unnecessary care. The EverCare NP had the ability to authorize skilled nursing services when indicated without having to begin with a three-night hospitalization. The model was shown to result in lower mortality rates for participants, fewer avoidable hospitalizations, and cost savings.[32] After the demonstration project ended, EverCare became an official managed care provider type, known as the Medicare Institutional Special Needs Program or ISNP. The original EverCare program is now an Optum ISNP through the insurance company United Health Care. Many other organizations have subsequently developed their own ISNPs, including some nursing facility companies, since the potential for financial reward, if well managed, is significant.

Post-acute programs within nursing facilities provide rehabilitative care, with the goal of returning the person back to their prehospital level of health and function. If the patient is unable to achieve a level of function allowing a return home, transitioning to the LTC nursing home program may be necessary. The goal of the LTC program is to provide a home-like environment, with the support of the interdisciplinary team, to help the person achieve their highest practicable level of function and well-being. Nursing facilities also provide respite care, which is a short-term admission of a person requiring support of activities of daily living, in order to give the usual home-based caregiver some days of rest, free of their caregiving duties. Respite care of hospice patients is part of the hospice Medicare benefit and is often provided in nursing facilities. Non-hospice respite care is also available, usually under a private-pay arrangement.

End-of-life care also increasingly occurs in nursing facilities, either with the support of the hospice team when the LTC resident enrolls in the formal hospice benefit or through a palliative care program without formal enrollment in the hospice benefit. Medicare beneficiaries cannot enroll in the formal hospice benefit while they are on the Medicare Part A skilled nursing benefit for PAC, since they are each exclusive, standalone benefits. The rehabilitative goals of PAC and the end-of-life symptom management goals of hospice care are considered mutually exclusive. Post-acute patients can transition to palliative care goals while still on the Medicare Part A skilled nursing facility benefit if they continue to have skilled nursing needs that qualify them for the benefit.

Liberalized regulatory acceptance of and insurance payment for telemedicine visits in nursing facilities during the COVID-19 pandemic allowed clinicians to continue to care for their nursing facility residents and patients remotely. Although telemedicine facilitated interaction and continuity during crisis, it degrades the importance of the physical presence of the medical team in the life of the nursing facility, limits the information gathered from the physical exam, requires additional nursing time when staff are already experiencing shortages, and creates new burdens of documentation. The most useful contribution of telemedicine in the post-COVID-19 era may be for access to medical specialist consultations, which have often been difficult to obtain because of the inability of the specialists to make room in their schedules for nursing facility rounds and limited access to transportation to get nursing home residents to the physician offices.

The Content of Medical Nursing Facility Medical Practice

Patients are admitted to hospital for evaluation and/or treatment when their symptoms or diagnoses require a higher level of care than can be provided in the outpatient setting. Residents or patients are admitted to the nursing facility when their functional dependencies require a higher level of care than can be provided in the home or community setting. With the expansion of home- and community-based services, an important question at the time of nursing facility admission and during follow-up visits is whether there is potential for the person to return to a less restrictive community setting.

Important aspects of assessment and treatment of the nursing facility resident or patient include: evaluation of mental status, including being able to distinguish and address delirium and dementia; evaluation of decision-making capacity and discussing advance directives and goals of care with either the resident/patient or responsible party; understanding which preventive interventions are important and appropriate based upon the person's age, functional status, and prognosis; knowing how to assess the medication regimens for efficacy and potential adverse drug effects and undertake gradual dose reductions or pharmacological debridement when appropriate; knowing how to work with the clinical team to implement guidelines and protocols to manage chronic illness effectively; and early recognition of changes in status with the ability to intervene at the facility rather than use the hospital emergency

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department and hospital admission excessively. An extremely important aspect of clinical management in the nursing facility population is the ability to help residents/patients and their families make the transition from curative expectations and interventions to palliative goals of care when the clinical situation and prognosis warrant the change in focus. Ignoring this aspect of medical care can result in recurrent hospitalizations with limited to no benefit to the patient and frustration for all involved. Trust that is built on open and honest communication over time is the most important component of this clinical skill.

This textbook, *Reichel's Care of the Elderly*, contains a wealth of clinical information that is relevant to the care of those who reside in post-acute and long-term care settings. Mastery of this information will place the clinician in good stead to be able to respond to the medical issues that arise. The American Geriatrics Society, which was the sponsor of the original editions of this textbook,[1,25] is the premier professional organization in the USA dedicated to research, education, and advocacy on clinical and policy issues that impact older adults and frail elders. Their website,[33] journal,[34] and review syllabus[35] are also rich sites of information to guide the clinician caring for those in post-acute and long-term care settings.

A career as a nursing facility medical director and/or attending physician can be tremendously satisfying and rewarding, serving the most frail and vulnerable members of society. When nursing facility residents, patients, their families, and the facility staff encounter a medical director or attending physician who has the knowledge and interest, spends the time to do a thorough, conscientious job, and communicates effectively, their gratitude can be overwhelming. The old-fashioned values of clinical medicine – of taking the time to educate, discuss, negotiate, set high personal and facility standards, and make important connections with patients, families, and staff – can be cultivated in this often neglected practice venue. These values make a huge difference in the morale and moral fabric of the institution, which in turn cannot help but improve outcomes, both measurable and unmeasurable. While physicians may work diligently to implement nursing home reform and renewal, they may find the process produces a personal reform and renewal as well.

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Chapter 52

Hospital-Based Care

Maureen Dale, David H. Lynch, and Debra L. Bynum

Movements in Geriatric Inpatient Care

Institute for Healthcare Improvement Age-Friendly Health Systems

In 2017, The John A. Hartford Foundation and the Institute for Healthcare Improvement (IHI) partnered with the American Hospital Association and the Catholic Health Association of the United States to disseminate the Age-Friendly Health Systems initiative.[1] The group's mission was to set standards of care for health systems so that they would "keep healthy older adults healthy, be proactive in addressing potential health needs, prevent avoidable harms, improve care of those with serious illness and at the end of life, and support family caregivers throughout."[2] The group identified five health systems to pioneer their approach, and focused their approach on the "4Ms" of good geriatric care: what matters to the patient, medication, mobility, and mentation. By late 2020, nearly 200 hospitals and practices had been recognized as "committed to care excellence" by reporting data on the hospital's implementation of the 4Ms and the number of patients reached, and another 481 hospitals and practices are participating in the movement but have not yet reported data.[1] While not exclusively focused on the inpatient world, this movement has potential to positively impact and standardize inpatient care of older adults to reduce and prevent hazards of hospitalization like falls and delirium, and to emphasize person-centered care for acutely ill older adults.[3]

Dementia-Friendly Hospitals

Hospital admissions can be frightening and particularly deliriogenic to patients with dementia, who are already at increased risk of experiencing hazards of hospitalization. In the same spirit of the Age-Friendly Health Systems movement, many hospitals and health-care systems understand the importance of developing dementia-friendly hospitals to better serve these patients. Hospital systems have focused on a variety of interventions, including modifying the physical environment (see later) and training programs for staff in dementia care. These training programs often emphasize communication strategies, support of the dignity and security of patients, attention to pain, fear, and anxiety, and management of agitation and other dementia-related behaviors.[4,5,6] Hospital systems like University of North Carolina at Chapel Hill (UNC) Health Care have used a combination of online modules and in-person training sessions to train all clinical and nonclinical staff members, from physicians and nurses to the environmental services and nutrition services staff, to achieve a comprehensive approach to dementia-friendly care in the hospital.[7]

Acute Care of the Elderly Unit

Benjamin Franklin first coined the phrase "An ounce of prevention is worth a pound of cure" when referring to fire safety in the 1700s; however, it is equally applicable today to the inpatient care of older adults. Optimal care of hospitalized older adults is complex and multifaceted. As discussed in other sections, older adults hospitalized for an acute illness are at high risk of experiencing adverse outcomes including delirium, falls, pressure ulcers, and iatrogenic complications, as well as functional and cognitive decline.[8] Units that focus on Acute Care for Elders (ACE) have been designed specifically to attend to the prevention of these adverse outcomes.[9] The ACE unit model consists of several key components: (1) patient-centered care, guided by comprehensive geriatric assessments; (2) prevention and management of geriatric syndromes using nurse-driven care plans; (3) comprehensive care transition planning; and (4) regular review of medical care focused on preventing incident geriatric syndromes and iatrogenesis.[10] Other key components of the ACE model include interprofessional team meetings, presence of a geriatrician or geriatric nurse practitioner on team rounds, and modifications to the physical

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environment that promote safe mobility and cognitive stimulation.[11] The first randomized controlled trial of an ACE unit was published in 1995 by Landefeld et al., and demonstrated the benefits of this novel approach.[12] A systemic review in 2012 compared the effectiveness of acute geriatric unit care with that of usual care. It found that ACE unit care was associated with fewer falls, less delirium, less functional decline at discharge, shorter length of stay, fewer discharges to nursing home, lower cost, and more discharges to home. No differences were found in functional decline, mortality, or hospital readmissions.[13] Since the introduction of ACE units in the 1990s, this model of care has been adopted in many hospital systems around the world. Some studies in the interim have shown mixed results regarding patient outcomes. These studies have highlighted that implementing this model of care is complex; it requires buy-in from all stakeholders and it takes time to develop highly functional teams. The most successful units have focused heavily on the principles highlighted above and have worked hard to improve consistently by gradually implementing practices that have been shown to be effective in improving the acute care of the elderly.[10]

Hospital-at-Home

In recent years, there has been a significant increase in the capacity of health systems throughout North America, Europe, and other parts of the world to provide health care to patients in their homes.[14] The driving force behind this expansion is often a desire to decrease costs while simultaneously improving the quality of care that is being delivered. Additionally, as discussed in other sections, the aging population is increasing pressures on secondary health services, and this has led to growing interest in interventions that can prevent or shorten hospitalizations.[15] While extending health care to meet people in their homes is certainly not a novel idea, providing hospital-level care at home was not explored in a meaningful way until the early 2000s.[16] Hospital-at-home programs are extremely heterogeneous but can largely be divided into two broad categories: admission-avoidance hospital-at-home and early-discharge hospital-at-home. In 2016 and 2017, two Cochrane Reviews summarized the evidence in these areas. The first review included 16 randomized controlled trials of admission-avoidance hospital-at-home programs, with a total of 1,814 participants. They found no difference in mortality at 6 months of follow-up. There was a slight decrease in the likelihood of living in residential care; patient satisfaction with health care received was increased, but there was insufficient evidence to assess caregiver satisfaction; and total costs of the initial episode of care may be cheaper, but this is complicated by unmeasured caregiver costs and differences between health systems. Overall, they concluded that admission-avoidance hospital-at-home may be an effective alternative to inpatient care in certain patient populations, but it is difficult to extrapolate to entire patient populations, as randomized studies have been small with a narrow focus.[16] The second review included 32 trials involving early-discharge hospital-at-home programs. It found that there was consistently no difference in mortality across studies. There were mixed results across different patient populations with respect to readmissions, patient satisfaction, and risk of institutional living. Finally, it is uncertain whether hospital-at-home has an effect on cost.[17] One major limitation of the current evidence base is that many studies have focused on specific patient populations such as patients with chronic obstructive pulmonary disease (COPD). This makes it difficult to apply evidence to all patients. Furthermore, the effectiveness of hospital care at home may be reduced in certain patient populations if it does not include best practices on certain specialized units such as stroke units or ACE units.

Geriatric-Friendly Hospital Spaces

With an increased understanding that health systems and hospitals need to be age-friendly, there has been an increased interest in age-friendly design of hospital spaces. The design principles are guided by an understanding that older adults often have visual, hearing, and sensory impairments that impact their relationships to unfamiliar spaces. Adapting spaces to meet the needs of older adults can make these spaces safer and enable older adults to function more independently within them. Geriatric-friendly spaces can offer orientation cues, facilitate mobility, and help prevent hospital complications like falls, delirium, and deconditioning.

Lighting is important in age-friendly design: using natural light is helpful in delirium prevention, and efforts to minimize glare on walls and floors can help reduce falls and disorientation as well. Night lights in and around bathrooms and near doorways can also help decrease the risk of falls at night.

Warm colors are easier for older adults to see, and use of contrasting colors can help call attention to doors as well as the demarcation between walls and floors. Furniture in patient rooms should have nonslip fabrics

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and also be in contrasting colors to the walls and floors. Rooms should also have easily visible clocks and calendars to help facilitate orientation.

Nonslip matte floors are important, and avoiding bold or busy patterns can help minimize visual perceptual challenges for older adults, particularly older adults with dementia. Hallways should have ample signage that is simple, color-coded, and with large print to help with wayfinding.[18,19]

Inpatient Geriatric Assessment

Evaluations of older adults' medical problems, social situation, functional status, cognition, and care goals during hospitalization help guide comprehensive care plans during admissions to the hospital. Older adults can have significant changes in their cognition and function during acute illnesses of any kind, and understanding their prehospitalization abilities is instructive. Studies have demonstrated that performance of these comprehensive assessments during hospitalization increases the likelihood that older adults will be alive and in their own homes at follow-up.[20]

Functional Assessment

A functional assessment should outline a patient's ability to perform activities of daily living and instrumental activities of daily living prior to their admission to the hospital. It should also outline how independent they were in the performance of these tasks. This sets a helpful benchmark for therapy services staff members during hospitalization, and can help guide discharge planning: if a patient has had a significant change in their functional status, they may not be able to discharge to home without the addition of services or a caregiver, or may need a short stay in a skilled nursing facility for rehabilitation. Decreased functional status at hospital discharge is a strong predictor of 30-day readmission to the hospital, and for patients readmitted from skilled nursing facilities, this carries with it a significantly increased mortality rate.[21,22]

Cognitive Assessment

Cognitive assessments during hospitalization should first assess whether the patient is delirious. This can be done simply by performing the Confusion Assessment Method (CAM), but also requires an understanding from family or caregivers of what the patient's baseline cognitive status is.[23] Additional testing with traditional screening tools like the Mini-Cog or more in-depth testing with the Montreal Cognitive Assessment (MoCA) or St. Louis University Mental Status (SLUMS), if warranted, can take place if the patient is not delirious, and is particularly helpful in determining how to deliver discharge counseling on medication changes and anticipatory guidance regarding symptoms. It can also be helpful to formally assess and document cognition for future providers, as patients often are discharged to a skilled nursing facility under the care of providers who do not know them.[24] Cognitive impairment is another predictor of rehospitalization, and therefore is important to recognize during a hospital stay.[21]

Advance Care Planning

The medical team needs to be aware of any preceding advance directives, including a health-care power of attorney and any do not resuscitate (DNR) or medical orders for scope of treatment (MOST) forms, ideally at the time of admission. But it is also important to continue conversations about serious illness even during admission, to ensure that a patient's care goals are being respected. As many older adults have chronic diseases that result in frequent exacerbations, like congestive heart failure and COPD, it is vital to continue discussions of care goals, including wishes surrounding rehospitalization and ICU care, to prevent unwanted escalations of care and to ensure that patients and their caregivers understand the diagnosis and prognosis.[25] Any new discussions during hospitalization should be well documented and passed on to the post-acute care team and primary care physician for the patient, to ensure these conversations can be continued and built upon in the outpatient setting.[24]

Frailty and Its Implications

Frailty is a syndrome that leads to a state of increased vulnerability to adverse outcomes from minor stressors. There are several scales and instruments used to measure frailty in research, which primarily look at components of fatigue, resistance, gait speed, weight loss, and illness burden, but none of these have become an implemented standard in inpatient care. Many of the components of the comprehensive geriatric assessment, however, can help providers identify frailty syndrome in older adult inpatients. This is crucial, as it is a predictor of mortality, readmission, and new nursing home placement at the time of discharge.[26] Awareness of frailty can again help inform discharge planning and advance care planning discussions.

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Prevention of Common Hospital-Acquired Conditions (HACs) in the Elderly

HACs such as pressure injuries, aspiration pneumonia, malnutrition, falls, venous thromboembolism, and sleep disturbances are unfortunately commonly seen poor outcomes associated with hospitalizations in elderly patients. Underlying all of these hazards of hospitalization is delirium – delirium is in itself an HAC that is associated with a higher risk of mortality and morbidity and, in addition, increases a patient's risk for developing every other HAC.[27]

Prevention of Pressure Injuries

Pressure injuries are seen in 12–16% of hospitalized patients and have been identified as a core Patient Safety Indicator with the Centers for Medicare and Medicaid Services (CMS) as part of their Hospital-Acquired Condition Reduction Program (HACRP). It is important that clinicians understand the morbidity and mortality associated with pressure injuries and have the ability to assess the risk for and stage of pressure injuries in older adults entering the hospital. Clinicians should do a complete skin exam on older adults upon admission, taking care to closely examine areas such as the back and buttocks, bony protuberances, and delicate skin under medical devices such as tubing and braces. Any pressure injury should be described in detail in the health record, with a description of the diagnosis or type of pressure injury, stage, location, length, and presence of tunneling or erythema or granulation tissue. If possible, photographs of the pressure injury should be taken and uploaded to the patient's health record. Pressure injury staging is based upon depth. Stage 1 pressure injuries are identified by non-blanchable erythema, stage 2 by partial-thickness tissue destruction, stage 3 by full-thickness tissue destruction, and stage 4 by full-thickness tissue destruction with visible bone, tendon, or muscle. It is important to accurately assess and document if a pressure injury is full thickness but unstageable due to overlying exudate or if there is a suspected deep-tissue pressure injury with intact skin, making it unstageable. For patient care and for the importance of appropriate reimbursement with the HACRP through CMS, it is critically important to document fully the presence and assessment of any pressure injury that is "present on admission."

There are multiple scales to assess a patient's risk for developing a pressure injury while in the hospital. A Cochrane Database Review demonstrated limited evidence that any of these scales performed any better than clinical judgment in predicting a patient's risk. Risk is increased in patients who are immobile, have dermatitis or other reasons to have skin that is not intact, have poor perfusion or poor nutritional status, are on steroid medications, have medical devices that are in constant contact with skin, have had weight loss and areas that are under more pressure with bony prominences, or have areas that are exposed to moisture such as those associated with incontinence. Most important is that an assessment is done.[28]

There are multiple other strategies aimed at reducing the development of pressure injuries, including creams, nutritional support, positioning, turning every 2 hours, and mattress supports – none of these have much evidence to support or refute their use. The American College of Physicians (ACP), based upon a systematic review of the evidence, developed three overarching recommendations aimed at preventing the development of pressure injuries in hospitalized patients. First, clinicians should perform a risk assessment, which could be clinical judgment or any number of available scales, to identify patients who are at high risk of developing pressure injuries. Second, clinicians should choose advanced static mattresses or advanced static overlays for high-risk patients (strong recommendation, moderate-quality evidence). Finally, the ACP recommended against using alternating-air mattresses or overlays, as there is no evidence that they are better, they are costly, and there is some evidence that they cause sleep disturbance and discomfort for patients.[29]

Prevention of Pressure Injuries – Key Points

1. Assess for and document any pressure injuries that are present on admission (including photographs that are included in the patient's health record).
2. Assess risk for pressure injury (either by clinical judgment or available scale).
3. While commonly done, interventions such as barrier creams and repositioning every 2 hours do not have data to support their use.
4. Clinicians should choose advanced static mattresses or overlays for high-risk patients. Alternating-air mattresses and overlays have not been shown to be better, and given their higher risk and potential for patient discomfort, it is recommended that clinicians do *not* use these devices.

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Prevention of Aspiration

Hospitalized older adults often have multiple risk factors for aspiration pneumonia including dysphagia, neurological diseases such as parkinsonism and strokes, dementia, and impaired consciousness due to delirium. Patients with swallowing disorders may benefit from a full speech and swallow evaluation. Evidence suggests that oral feeding is better than enteral tube feeding. There is some evidence to suggest that a mechanical soft diet with thickened liquids has lower risk than a pureed diet with thin liquids in high-risk patients. Patients should eat sitting up and should never be supine while eating or drinking. Additional strategies involve careful hand assistance, ensuring the patient's chin is down and head turned to one side, encouraging small volumes with multiple swallows, and coughing after each swallow. Good oral hygiene with mechanical cleaning may also decrease the risk of developing pneumonia. The use of oral care with chlorhexidine is controversial given potential increased mortality associated with toxic effects of this agent if aspirated.[30]

Prevention of Aspiration Pneumonia – Key Points

1. Enteral tube feeding does not decrease the risk for aspiration.
2. Mechanical soft diets with thickened liquids may be safer than pureed diets with thin liquid in older patients with dysphagia.
3. Good oral hygiene and careful oral feeding with small bites, chin-down positioning, and sitting upright with coughing after swallows may decrease the risk of aspiration.

Malnutrition in the Hospital Setting

Malnutrition is common in older adults who are hospitalized and can be exacerbated by common practices such as keeping patients "Nothing Per Oral (NPO)" for prolonged periods for potential procedures, placing patients on restrictive diets for diabetes or heart failure, or not encouraging patients to get out of bed to eat. There is evidence that an individualized nutritional treatment during hospitalization with an assessment by a dietician in the hospital and three at-home visits post-discharge was able to improve nutritional status and reduce mortality in older adults.[31]

Malnutrition in the Hospital Setting – Key Points

1. Avoid restrictive diets in older patients.
2. Avoid prolonged periods of NPO.
3. Encourage patients to be out of bed for all meals.
4. Strongly consider consult to dietician for older adults at risk for malnutrition.

Sleep Disturbances in Older Hospitalized Patients

Disruptions to sleep are common in the hospital, and evidence suggests they have a negative impact on older patients. Barriers to sleep include medical care interruptions, anxiety and underlying insomnia, and environmental factors such as hospital noise and light. In general, pharmacological interventions are of limited value, and many have significant risks. Melatonin, given at a dose of 1–3 mg 30 minutes prior to bedtime, has been shown in some studies to improve sleep quality and duration in hospitalized patients. Medications such as benzodiazepines or anticholinergics are not recommended in older adults, as the risk for falls and delirium is unacceptably high. Other agents such as Zolpidem are also associated with negative side effects and delirium in older patients. Trazodone, while commonly used as a safer alternative to benzodiazepines, is also associated with an increased risk for delirium and falls in hospitalized older adults. The risks and benefits of using an agent such as Trazodone need to be carefully assessed.

Nonpharmacological interventions such as relaxation techniques, sleep hygiene programs, and daily bright light therapy may be of benefit and are recommended given their low risk for harm. In addition, noise reduction, reduction of nighttime interruptions, and sleep education can all be of benefit.[32]

Improving Sleep in the Hospital Setting – Key Points

1. Avoid unnecessary medical care interventions such as phlebotomy and vital signs at night and in the early morning.
2. Decrease light and noise in the evening and overnight.
3. Improve mobility and bright light during the day.
4. Low-dose melatonin may be of benefit and is low risk.
5. Avoid benzodiazepines and anticholinergic agents given the risk of delirium.

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Reducing the Risk of Venous-Thromboembolic (VTE) Events

Patients over the age of 60 are at increased risk for VTE events during hospitalizations, especially those who are immobile, critically ill, have residual weakness from a prior stroke, or have underlying cancer. Care must be taken to individually assess each patient for their risk of thrombotic events while balancing their risk for bleeding. Many of the same risk factors for VTE (older age, cancer, critical illness) are also associated with an increased risk of bleeding with anticoagulation. In general, most older adults hospitalized with an acute illness will be considered at moderate or high risk for VTE, given their age and any associated comorbidities. If there is not a clearly increased risk for bleeding (recent intracerebral hemorrhage, active gastrointestinal ulcer, recent bleeding, platelets less than 50,000), then strong consideration should be given to pharmacological primary prophylaxis with low-molecular-weight heparin (LMWH) or unfractionated heparin (UH) for patients with a creatinine clearance of less than 30 mL/min). For patients with a high risk of bleeding in addition to a moderate or high risk of VTE, mechanical prophylaxis with pneumatic compression devices is recommended over nothing. In older adults, care must be taken to prevent leg ischemia in patients with peripheral vascular disease, and careful examination of the underlying skin should be done frequently to prevent any skin breakdown.

While there is some evidence that mechanical prophylaxis has some benefit, pharmacological primary prophylaxis is preferred and there is no benefit to combining mechanical and pharmacological interventions in medical patients.[33] Finally, while VTE is common in patients who are bedridden, there is not clear evidence that pharmacological prophylaxis during a hospitalization for an acute illness reduces their risk for pulmonary embolism or mortality. Quality indicators at the health-care systems level will lead most of these patients to receive primary prophylaxis during their hospital stay, but it is important to understand that this may not impact their overall survival or longer-term outcomes in the frail elderly who are unable to be out of bed.[34]

Duration of prophylaxis is not clear. There is some evidence that extended VTE pharmacological prophylaxis with some of the newer agents such as direct factor Xa inhibitors for 30 days after discharge may be associated with less risk of VTE. But this must be balanced with other studies demonstrating mixed benefit with other agents such as enoxaparin, rivaroxaban, and apixaban and increased risk of bleeding with extended duration.[35]

Prevention of VTE – Key Points

1. Older patients are at higher risk for both VTE and bleeding, and care must be taken to assess and balance the risk for each.
2. For patients at high risk of VTE, strong consideration should be given to the use of LMWH as primary prevention.
3. There is limited evidence for mechanical devices in the prevention of VTE. They are possibly better than nothing, but must be balanced with patient sleep and comfort.
4. Consideration should be given to continued pharmacological prophylaxis for up to 30 days after hospitalization in patients who are at high risk for VTE.
5. There is no evidence that the use of pharmacological prophylaxis improves overall outcomes in patients who are chronically bedridden.

Reduction of Injurious Falls

Falls are common in hospitalized older adults and are associated with increased morbidity, mortality, and costs. At the same time, falls are difficult to actually prevent in the hospital setting. In 2008 the CMS listed injurious falls of hospitalized patients as a "never event" – events that are of high cost and high volume, lead to a higher reimbursed diagnosis-related group (DRG) when designated as a secondary diagnosis, and are "reasonably" preventable through evidence-based interventions. Falls in the hospital resulting in hip fractures are one of the core targets of the HACRP, and the rate of these events is used as a Patient Safety Indicator (PSI) that allows the CMS to adjust payments to hospitals based upon performance. Unfortunately, falls are more common in frail older adults and may be difficult to prevent. Overly aggressive measures to reduce falls in the elderly such as restraints and strict bedrest are not appropriate, and commonly used devices such as bed alarms are not effective.[36] What can be done are common-sense measures that include reducing lines and catheters, avoiding medications associated with delirium and carefully monitoring the use of medications like diuretics and antihypertensives that may be associated with orthostasis, assisting patients to be mobile and out of bed, avoiding the use of chemical and physical restraints, and most

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importantly, taking measures to reduce the development of delirium.

Reducing Falls in the Hospital Setting – Key Points

1. There is no evidence that bed alarms work, but they are commonly used.
2. Avoid strict bedrest and restraints (including raising all four bedrails, which can increase the risk of patient injury).
3. Untether – avoid urinary catheters and unnecessary intravenous lines, mechanical devices, telemetry, and oxygen tubing.
4. Carefully review medications daily.
5. Improve mobility during the day; focus on reducing delirium.

Prevention of Delirium

Delirium occurs in over 20% of all hospitalized older adults and is associated with increased costs, higher morbidities and length of stay, and increased mortality.[37] A controlled clinical trial of delirium prevention in 2017 demonstrated that older hospitalized patients who developed delirium had a significantly increased 90-day all-cause mortality (24% vs. 6%) compared to those who did not. In addition, older adults who developed delirium were more likely to have been restrained with physical restraints or by the use of urinary catheters (33% vs. 16%), were more likely to have had common HACs such as falls and pressure ulcers (37% vs. 12%), and were more likely to have experienced "noxious insults" such as sleep deprivation, acute malnutrition, dehydration, and aspiration pneumonia during their hospital stays.[38] Importantly, a landmark study of delirium prevention in 1999 demonstrated that delirium could be prevented with a multicomponent intervention including a focus on daytime mobility and lights, human interaction, careful medication reviews, avoidance of sleep disturbance, and freeing patients from restraints including unnecessary lines and catheters.[39]

Prevention of Delirium – Key Points

1. Delirium is associated with increased morbidity and mortality.
2. Delirium is tightly tied to all of the other negative HACs; preventing delirium has potential for preventing falls, aspiration, immobility and VTE, and pressure ulcers.
3. Delirium can be prevented with a targeted and multicomponent approach to care.

Transitions of Care

Older adults receive medical care in many distinct locations, which include but are not limited to home, independent living facilities, assisted living facilities, skilled nursing facilities, and hospital. A transition in care occurs when patients move from one location to another. Transitions in care represent a vulnerable time for all patients, but for older adults the chance of experiencing an adverse event due to an unintended error is magnified.[40] Poorly implemented care transitions can lead to patient and family dissatisfaction, avoidable rehospitalizations, medication errors, missed follow-up appointments, and provision of care that is not in line with patient goals or preferences.[40] Understanding these risks and taking proactive measures to prevent harm can greatly improve patient outcomes. A comprehensive approach to safe transitions in care should include bidirectional communication between clinical professionals, preparation of patients and family members for upcoming transitions, education of health professionals on the risks associated with care transitions, and regular evaluation of transitions to and from each distinct location.[41] Ideally, this comprehensive approach will minimize avoidable errors, improve patient satisfaction, and promote goal-concordant patient-centered care.

Ideal Discharge Summaries for Older Adults

Discharge summaries should equip the post-acute care team to continue the care plan safely after hospitalization and help anticipate barriers to care and follow-up needs.[42,43] In older adults, a discharge summary should address not just the primary reason for hospitalization, but should also acknowledge the broader chronic issues that may hinder the care plan in the post-acute setting. The discharge summary should include a reconciled medication list, including details of why medications were stopped or started. It should include details of functional changes that occurred during hospitalization to ensure the patient has proper support, equipment, and follow-up in post-acute care. It should also include a cognitive assessment that indicates whether a patient had delirium during hospitalization. Ideally, the discharge summary should also include any testing that identifies possible underlying cognitive impairment or

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dementia that needs to be assessed in the post-acute care setting.[24,44] Finally, the discharge summary should include any advance care planning discussions that occurred during hospitalization – too often we have important conversations with patients regarding their goals of care but fail to document them, causing discontinuity of these discussions after discharge.[45]

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Chapter 53

Introduction to Palliative Care

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Introduction

Over the last century, the United States has seen a cultural shift in the way people experience illness and care at the end of life. Tremendous technologic advances in health care have increased life expectancy and ushered in a new era of caring for patients with increasingly complex acute and chronic illnesses. Significant growth in the cost of caring for seriously ill patients is recognized as unsustainable, while at the same time patients report the current disease-focused care model to be inadequate in meeting their needs.[1] In recent decades, the specialty of palliative care has emerged to better address the individual needs of the sickest patients, while at the same time working to orient the health-care system in delivering the highest-quality and most cost-effective care.

Defining Palliative Care

The World Health Organization defines palliative care as:

> An approach that improves the quality of life of patients and their families facing the problem associated with life-threatening illness, through the prevention and relief of suffering by means of early identification and impeccable assessment and treatment of pain and other problems, physical, psychosocial and spiritual.[2]

Across care settings, palliative care is delivered to all patients with serious illness, regardless of age, prognosis, or diagnosis. Attention is paid to coordination of care, physical pain and symptom management, psychosocial determinants of health, and optimizing care at the end of life. Palliative care clinicians are trained in facilitating communication, advance care planning, discussion of patient and family goals of care, quality improvement efforts, and integrating cultural aspects of care.[3] As palliative care is holistic in nature, it is optimally delivered by a team made up of palliative care physicians, nurses, social workers, chaplains, and members of other disciplines depending on individual patient needs.

Although the specialty of palliative care maintains its roots in the hospice movement, a distinction must be made between palliative care and hospice. Whereas palliative care can be integrated into the care of any patient with serious illness at any stage of their disease process, hospice care is typically provided during the last 6 months of the patient's life, when the decision to forgo curative treatment has been determined to be appropriate. Palliative care can be provided alongside curative or disease-directed therapy to maintain quality of life and patient-centered care. This is often achieved in collaboration among members of the patient's medical team consisting of primary care providers (PCPs), subspecialty providers, and palliative medicine interdisciplinary teams.

Palliative Care Emerges as the Standard in Quality Care for the Seriously Ill

There has been compelling evidence to suggest that palliative care interventions are associated with improved outcomes in areas such as symptom control, quality of life, mood, and less aggressive treatment at the end of life.[4,5,6,7] Palliative care consultation for seriously ill hospitalized patients is associated with a reduction in overall health-care costs,[8,9,10,11,12,13,14] decreased ICU length of stay,[12,13,14] and improved patient, family, and provider satisfaction and perception of patient care outcomes.[6,10,15,16]

There is also a growing recognition of the importance of these services to be delivered early in the disease course and across the spectrum of ambulatory and community care settings. The landmark Temel et al. study (2010) showed early integration of palliative care alongside standard oncologic care in the ambulatory setting was associated with improved mood and quality of life, and most notably, longer median survival. Later secondary cost analysis of the Temel data did not show that the added cost of palliative care services was associated with increased overall cost.[17,18]

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Specialist versus Generalist Palliative Care

As the need for improvement in care for those coping with serious illness has been recognized, the demand for palliative care specialists and programming has grown significantly. Although the availability of hospital-based palliative care consultation teams and community palliative providers has become more prevalent,[19] gaps in funding and workforce resources present barriers to meeting the complex needs of patients.[20,21,22] Furthermore, it has been suggested that this reliance on specialist care can, in some cases, add another layer of complexity and expense to the care of the seriously ill.[23]

Some essential aspects of the delivery of palliative care can be successfully provided by the patient's PCP or specialists. Since this relationship is often developed over the course of years spent caring for the patient, an opportunity exists to draw on this therapeutic relationship in such activities as advance care planning and discussion of patient and family goals. If the needs of the patient begin to take on greater complexity, such as in instances of refractory pain and symptom management, or with management of truly complex decision-making or conflict, then the palliative care specialist can work in collaboration with the generalist provider to address these issues. With this collaborative model, needs of a greater number of patients can be met, while supporting the essential role of both generalist and specialist palliative care providers.

It is important to recognize the importance of ongoing professional education to help foster competency in the provision of generalist palliative care. Programs such as Education in Palliative and End-of-Life Care (EPEC), Oncotalk, and End-of-Life Nursing Education Consortium (ELNEC)[24,25,26] were developed to teach and reinforce basic skills in pain and symptom assessment and management, goals-of-care discussion, advance care planning, and care for the imminently dying patient. References such as the Serious Illness Conversation Guide[27] and the Family Goal Setting Conference Pocket Guide[28] have also been made widely available to help support best practice in communication and goal-setting. Increasing access to educational resources such as these, coupled with the continued growth of access to palliative care specialists, will best serve to further advance access to quality palliative care.

Navigating Communication at the End of Life

Successful communication with patients and families about issues surrounding serious illness and end-of-life care is an essential skill for all health-care providers. However, research has consistently shown that clinicians feel inadequately prepared in the provision of end-of-life care. This is particularly true surrounding the delivery of bad news and discussion of death and dying.[29,30,31] It is important to note, however, that patients and families often expect to be well informed about their diagnosis, and to act as active participants in collaborative decision-making with their health-care provider.[32,33] An important step in establishing a relationship with patients can be to determine how much and what type of information they would like to receive by asking them about their preferences.

Communication with patients and families who are facing serious or life-threatening illness can be challenging, but can be achieved using a systematic approach that encourages active listening, clear language surrounding medical information, and discussion of patient- and family-centered goals (see Table 53.1). When discussing medical information with patients or surrogates, it is important to first establish their current understanding of the illness. This can help to clarify misperceptions and inform the direction of the conversation. Once the clinician has a good understanding of the patient perspective, the current medical status and options can be reviewed with careful attention to avoid medical jargon and overly technical information. Information about prognosis can be offered in terms of an expected range of hours to days, days to weeks, weeks to months, etc. While uncertainty should be acknowledged, prognostication provides the patient and family the opportunity to set their goals and expectations according to their own values and preferences.

As the patient and family take time to assimilate information, take a moment to respond to expressed emotion or allow for brief silence. At this point in the conversation, information about the patient's worries, hopes, and willingness for tradeoffs can be elicited. After questions are answered and concerns addressed, relevant options and plans can be discussed. It can be helpful to ask permission to offer specific recommendations for interventions that are most closely aligned with the patient-stated goals or philosophy. For example, the clinician may make recommendations regarding resuscitation, artificial nutrition/hydration, returning to the

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Table 53.1 The family meeting or goal-setting conference: approach to successful communication

1. **Preparation:**
   - Review chart: know treatment course, prognosis, treatment options
     - Coordinate medical opinions among consultant physicians
     - Decide what tests/treatments are medically appropriate
   - Clarify goals for the meeting
   - Decide who you want to be present from the medical team
2. **Establish proper setting:**
   Private, comfortable; everyone sitting in a circle
3. **Introductions/goals/relationship:**
   - Allow everyone to state name and relationship to patient
   - State meeting goals; ask family to state their goals
   - Build relationship; ask nonmedical question about patient: *Can you tell me something about your father?*
4. **Family understanding of condition:**
   - *Tell me your understanding of the current medical condition*
   - For patients with a chronic illness, ask for a description of changes over the past weeks/months (activity, eating, sleep, mood): *How have things been going the past 3 months?*
5. **Medical review/summary:**
   - Summarize "big picture" in a few sentences – use "dying" if appropriate
   - Avoid jargon or organ-by-organ medical review
6. **Silence/reactions:**
   - Respond to emotional reactions
   - Prepare for common reactions: acceptance, conflict/denial, grief/despair; respond empathetically to conflict/denial (see item 10 below)
7. **Present broad care options/set goals:**
   - Provide prognostic data using a range
   - Present goal-oriented options (e.g., prolong life, improve function, return home, dignified death)
   - Make a recommendation based on knowledge/experience
   - Ask: *What is important in the time you have left?*
8. **Translate goals into care plan:**
   - Review current and planned interventions – make recommendations to continue or stop based on goals
   - Discuss DNR, hospice/home care, artificial nutrition/hydration, future hospitalizations
   - Summarize decisions being made
9. **Document and discuss:**
   - Write a note: who was present, what decisions were made, follow-up plan
   - Team debriefing
10. **Managing conflict:**
    - Listen and make empathetic statements: *This must be very hard*
    - Determine source of conflict: guilt, grief, culture, family dysfunction, trust in medical team, etc.
    - Clarify misperceptions; explore values behind decisions
    - Set time-limited goals with specific benchmarks (e.g., improved cognition, oxygenation, mobility, etc.)

Information adapted from "Family Goal Setting Conference Pocket Card" and used with permission from the Medical College of Wisconsin.

hospital, hospice care, or continuation of specific disease-directed therapies. Some clinicians may fear that making specific guidance can limit the patient's autonomy, but grounding these recommendations in what you have come to understand both personally and clinically about the individual patient can help to address these concerns. Providing clear guidance in decision-making also helps to reduce the burden of surrogate decision-making and allows patients and families to participate in truly informed decisions. After providing this assessment, the clinician should check in with the patient or surrogate to see how the recommended plan feels to them.

At the close of the conversation, a summary of decisions made and subsequent plans for next steps and follow-up should be discussed. It is also important to review and debrief with participating health-care providers; this will help to encourage continuity and cohesion within the interprofessional team.

Even as this approach to communication is designed for the semiformal discussion of specific goals of care, the principles can be applied in difficult conversations throughout the relationship with the patient. Skill in responding empathetically, in delivering prognostic information, and in using clear language surrounding medical information should be practiced and applied to daily patient interactions.

Symptom Management

Patients often have a significant symptom burden at the end of life; pain, nausea, delirium, and dyspnea are amongst the most common. There are many factors that complicate effective symptom management, including older age, polypharmacy, impaired cognition, and end-organ failure.[34] Frequently, patients have untreated symptoms in the last days to hours of life, with up to 90% of patients experiencing pain during the last week of life.[35] Symptom control is important in all stages of illness, but must become a primary focus at the end of life.[36] The following is a brief review of assessment and treatment of common symptoms at the end of life.

Pain Assessment

Assessment of pain is critical at the end of life. This varies based on whether the patient is responsive at the time of assessment or not. If able to communicate, the initial evaluation of any patient's pain needs to take into account the current pain symptoms as well as any prior history of pain. Manifestations of chronic or acute pain in elderly, seriously ill patients are often complex and multifactorial.

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For example, age-related osteoarthritis may often obscure complaints of new bony or joint pain that may be from metastatic malignancy. A thorough pain assessment must include, via patient self-report if able, a description of the location, quality, onset, and exacerbating and relieving factors. Most mistakes in diagnosing and effectively treating pain in severely ill patients come from neglecting some aspect of the pain assessment.[37] The Agency for Health Care Policy and Research (AHCPR) guidelines describe pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage."[38] Thus, one important aspect of effective pain control in seriously ill patients is to believe the patient or family member's report of pain.

The quality of a patient's pain can commonly be described in terms such as sharp, stabbing, throbbing, shooting, or tingling. This assessment helps in the determination of the type of pain the patient experiences. Nociceptive pain, which is derived from pain receptors, is either visceral or somatic. Visceral pain is often described as deep, aching, and colicky, is poorly localized, and often is referred to cutaneous sites, which may be tender to palpation. In cancer patients, this pain often results from stretching of viscera by tumor growth. Somatic pain is often reported as constant, aching, or gnawing. Neuropathic pain affects peripheral nerves, spinal cord, or the central nervous system (CNS).[39] It is often described as burning, aching, or shooting in quality and can be persistent or spontaneous.[40]

Assessment of pain should include, when possible, some objective measure of severity. Various scales exist to document pain, such as the Visual Analog Scale (VAS) or the numerical pain distress scale. An important aspect of severity of pain and degree of relief is to assess what patient goals and expectations are. Because many elderly adults underreport pain, engaging with the patient about what to expect from therapy is critical to the success of treatment.[41]

In nonverbal patients, the assessment of pain has added complexity. Observation of patient behaviors is recommended in nonverbal, seriously ill patients.[42] Behaviors that are commonly considered pain-related include facial grimacing, moaning, or rubbing a body part. Some behaviors are less obvious. These include agitation, restlessness, irritability, or confusion. Various assessment tools are available to guide assessment of pain in elderly patients, such as the pain assessment in advanced dementia scale (PAIN-AD) or the checklist of nonverbal pain indicators (CNPI). As a patient becomes nonverbal during the dying process, previous pain should be assumed still present and must continue to be effectively treated.[43]

Management

Although older patients are often at higher risk of drug-related adverse effects, appropriate treatment of pain in seriously ill patients is crucial to improve quality of life. Undertreatment of pain in elderly patients is associated with significant adverse outcomes, including functional impairment, falls, anxiety and depression, and greater health-care costs.[44,45]

Opioid and nonopioid adjuvants should be the cornerstone of treatment in all severe, persistent pain. For most patients with cancer-related or other severe pain, the use of opioids is necessary and the benefits vastly outweigh the risks. Often, elderly patients' pain is undertreated because of concern about use of opioids.[45,46] Selection of opioids should take into account end-organ function, availability, cost, ease of use, and the patient's past experiences. A combination of short- and long-acting opioids is crucial to successfully treating patients with persistent pain. Often, the use of immediate-release opioids is a first step in pain control, especially in previously opioid-naïve patients.

There are various age-related changes associated with the effectiveness and pharmacodynamics drug properties, including changes in metabolism, distribution, and excretion.[44] Decreases in hepatic and renal function are often present in healthy elderly patients, let alone in the seriously ill elderly cohort.[47] Decreased metabolism and excretion can result from poor end-organ function, and should be taken into account when dosing opioids. Ultimately, very effective and safe pain relief can be achieved with individualized monitoring and titration. In the absence of obstruction or problems with absorption, an alert elderly patient can tolerate oral medications. There is no change in oral bioavailability in elderly patients.

There is increased recognition of the devastation that the opioid epidemic has caused, with data demonstrating an almost fourfold increase in overdose deaths between the years of 1999 and 2008.[48] Physicians are increasingly uncomfortable using and prescribing opioids because of this trend.[49] This creates concern surrounding access and the ability to control pain in patients with serious illness. Safe, effective pain management in patients with end-stage illness often must include opioids.

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Increased recognition and training is needed to ensure appropriate dosing and access to opioids for dying patients, when indicated.

Risk assessment surrounding opioid use disorder is both clinically feasible and relatively straightforward in verbal patients.[50] Several screening tools exist to help predict risk of development of opioid use disorder, if this is applicable to selected patients with sufficient prognosis. Documentation of risk and ongoing methods to lower risk when possible is helpful, but patients who are dying and in need of opioids should have access to ensure suffering does not occur toward the end of life.[51,52]

Considerations for Selection of Opioids

Morphine is the most commonly prescribed opioid because of cost and availability.[46] There are long-acting and immediate-release oral tablets, elixirs, suppositories, and intravenous or subcutaneous dosing options. Elderly patients with impaired renal or hepatic function should be monitored closely. In patients with renal failure, the metabolites of morphine may accumulate and ultimately cause neurotoxicity.[47] Oxycodone, a semisynthetic opioid similar in bioavailability to morphine, is available in oral preparations. Oxycodone is hepatically metabolized and renally excreted as well.[53] Hydromorphone (Dilaudid) is a semisynthetic derivative of morphine that is available as oral tablets, liquid, suppository, and parenteral formulations. The metabolite of hydromorphone is renally excreted and associated with neurotoxicity at increasing doses as well.[54] Fentanyl is a synthetic opioid and estimated to be 80 times more potent than morphine. In patients with true opioid allergies, fentanyl is a safe alternative because it is completely synthetic. Because of its high lipid solubility, fentanyl is available as a transdermal patch. This is a safe alternative in elderly patients who cannot swallow, but should never be used first-line in opioid-naïve patients. Fever increases the rate of absorption of transdermal fentanyl.[55,56] Intravenous fentanyl is also available and is a safe alternative in patients with compromised renal function.[56] Both ketamine and methadone can be safely used in elderly individuals, but because of their variable half-life, unpredictable side-effect profile, and high risk of accumulation without careful monitoring and titration, these medications should be managed by pain and palliative medicine specialists.

Understanding equianalgesic conversions is the cornerstone to safely starting, maintaining, or converting patients with serious illness on any opioids. (See Table 53.2 for equianalgesic conversions.)

Table 53.2 Equianalgesic opioid conversion ratios

Drug	PO/PR (mg)	Subcutaneous/IV (mg)
Morphine	30	10
Oxycodone	20	n/a
Hydrocodone	20	n/a
Hydromorphone	7.5	1.5
Oxymorphone	10	1
Fentanyl	n/a	0.1 (100 mcg)

Dosing and Titration

Opioid therapy must be selected on an individual basis, taking into account both medication of choice and route of administration. Intravenous and subcutaneous dosing is often required at the end of life for various reasons. Patient-controlled analgesia (PCA) pump use is often beneficial in elderly patients with severe pain. Indications for PCA pump include: unable to tolerate oral route, escalating pain that needs to be managed rapidly, severe incident pain, and dose-limiting side effects with other routes of administration. Contraindications to the use of a PCA pump are primarily centered on concern for delirium or patient ability to participate in their own care.

Titration of opioids is often required in patients nearing the end of life. Escalation of both long-acting and immediate-release formulations may be necessary to maintain adequate pain relief. The immediate-release formulation should be between 10% and 20% of the total daily dose of the long-acting opioid. Increases in opioids, via all routes of administration, can be done safely every 24 hours. To effectively control pain, the minimum increase should be 25% of the total daily dose and can be as much as a 100% increase, depending on the frequency of the use of the breakthrough opioid and the severity and circumstances of the pain syndrome. The dose should be increased until adequate pain control is achieved or intolerable side effects occur.

Although there is no specific maximum dose of any one opioid, if pain is no longer controlled on a specific regimen or dose titration yields treatment-limiting toxicities, opioid rotation may be necessary. This helps achieve a favorable balance between analgesia and side effects.[57,58] The principle of incomplete cross-tolerance is important to the successful rotation from

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one opioid to another. Cross-tolerance occurs when continued use of one opioid leads to tolerance of another substance with similar pharmacologic properties. Incomplete cross-tolerance occurs with opioids, and thus dose decreases are needed. The percentage of decrease required is variable; the amount of pain the patient is currently experiencing, end-organ function, and other comorbidities may force a greater or lesser percentage decrease. The usual range of dose decrease is between 25% and 50%.[59]

Adjuvant Analgesics

In patients with serious illness, many nonopioid medications are useful as adjuvants in the treatment of severe pain. Adjuvant analgesics may be used alone or in combination with opioids. These include anticonvulsants, antidepressants, corticosteroids, and topical anesthetics. Neuropathic pain is often best treated with nonopioid medications. Current guidelines recommend tricyclic antidepressants (TCAs) or dual reuptake inhibitors of both serotonin and norepinephrine as the first-line treatment, along with calcium channel binding agents (gabapentin and pregabalin) for neuropathic pain.[60] Side effects of TCAs primarily limit their use in elderly patients. Common side effects include urinary retention, constipation, and delirium. As with most drugs in elderly patients, starting at a low dose with a slow titration is helpful to monitor and avoid serious side effects or drug–drug interactions.

Corticosteroids are useful in various types of pain syndromes. They are the standard of care in malignant spinal cord compression. In addition, bony pain from malignant metastasis and inflammatory pain from invasion of abdominal viscera also respond well to the anti-inflammatory effects of corticosteroids. Dexamethasone is commonly the preferred agent because of its duration of action as well as its limited effects on blood glucose levels.[46] Evidence of the benefit of steroids in cancer patients is not high quality, but corticosteroids have a lower side-effect profile than nonsteroidal anti-inflammatory drugs (NSAIDs) in elderly terminal cancer patients and can be useful in the management of nausea and vomiting as well.[61]

Cannabis is increasingly an adjuvant to pain and other symptom control that elderly patients utilize.[100] Medicinal cannabis dates back in documented history to the world's oldest pharmacopoeia, a written summary of what was known about herbal medicine through the late sixteenth century.[62] There are over 100 known phytocannabinoids, the most well recognized being Δ9 tetrahydrocannabinol (THC) and cannabidiol (CBD). The common forms of plant-based cannabis for use include leaf that can be smoked or vaporized, oral tincture, pill, oil concentrate, or food products with THC or CBD incorporated that can all be ingested orally. Data surrounding use for cancer and noncancer pain is mixed, but the popular appeal of cannabis continues to drive its use medically.[63]

Additionally, interventional procedures to manage pain are, at times, very valid options in patients at the end of life. Specifically in cancer patients, nerve blocks (celiac plexus, superior hypogastric, lumbar) are effective in relieving severe visceral and neuropathic pain from malignancy.[64]

Nonpharmacologic Approaches to Pain Management

Although there is not a large body of evidence, many nonpharmacologic, complementary therapies can provide pain relief in elderly patients nearing the end of life. Various sources recommend trials of acupuncture for pain as well as other cancer-related symptoms owing to the minimal side effects of treatment. Although no high-quality data is available, a 2011 meta-analysis showed no significant side effects of acupuncture, but they were unable to recommend acupuncture for adults with cancer pain because of a lack of evidence of benefit.[65] Additional complementary approaches to pain management include Reiki massage, meditation, therapeutic massage, and physical therapy, as well as art and music therapy. Many of these are often employed through hospice programs. All of these can be used alone or in combination with pharmacotherapy.

Nausea and Vomiting

Mechanisms

Nausea and vomiting are common symptoms during the end of life. These cause significant physical and psychological distress for both patients and their families. Understanding the pathophysiology of nausea and vomiting is important in selecting the most beneficial therapy as well as avoiding polypharmacy. Various pathways mediate nausea and vomiting. Table 53.3 shows common pathways stimulated in nausea and vomiting. These pathways provide input to the vomit center in the brain, resulting in either nausea or vomiting when a specific, minimum threshold is met.[66]

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Table 53.3 Common pathways stimulated in nausea and vomiting[66,69]

Pathway	Input	Neuroreceptor
Vestibular system	Motion, labyrinth disorders	Acetylcholine, histamine
Chemoreceptor trigger zone	Drugs, metabolic disturbances, bacterial toxins	Dopamine, serotonin, neurokinin
Peripheral pathways	Mechanical stretch, gastrointestinal mucosal injury, local toxins and drugs	Mechanoreceptors and chemoreceptors (serotonin) in gastrointestinal tract and viscera
Cortex	Anxiety, sensory input, increased intracranial pressure, meningeal irritation	Acetylcholine, histamine, serotonin

Assessment

A thorough history and physical exam is the first step in determining cause and subsequent treatment for nausea and vomiting. Once the most likely cause is established, determination of the specific mechanism and neuroreceptors involved will aid in targeted therapy and avoiding polypharmacy, a common complication in elderly patients. Various mnemonics exist to help guide a targeted physical exam and understanding of the likely cause. See Table 53.4 for one specific mnemonic useful in nausea and vomiting.

Management

Once a determination of the likely cause of nausea and vomiting is deduced, a mechanism-based treatment strategy is effective. Using the most potent antagonist to the indicated neurotransmitter receptor has been effective in up to 80% of cases of nausea and vomiting at the end of life.[67] Although a mechanism-based treatment strategy is very effective, there is some evidence that starting with an empiric regimen consisting of a dopamine antagonist is also successful. See Table 53.5 for common antiemetics, sites of action, and starting doses.

Generally, the best method of treatment involves choosing one antiemetic with the desired receptor site of action and increasing that medication until a maximum dose is achieved or intolerable side effects occur. If a second antiemetic is needed, choosing one with a different site of action is more effective with less risk of adverse events.[66]

Table 53.4 Mnemonic useful for nausea and vomiting[66,69]

V	Vertigo, vestibular
O	Obstruction of gut
M	Motility dysfunction within gut
I	Inflammation of gut
T	Toxins, drugs
I	Intracranial – increased intracranial pressure
N	Nerves – anxiety, depression
G	Gums, mouth, oropharynx – thrush, mucositis, etc.

Corticosteroids are often effective in refractory nausea and vomiting. They have an unknown mechanism of action, but are thought to be effective via a decrease in gut edema. Although rarely necessary, refractory nausea and vomiting may require palliative sedation. The use of propofol has been investigated for its known anti-serotonergic activity.[69]

Dyspnea

Dyspnea, the subjective sensation of breathlessness, is a frightening symptom at the end of life. It can be either acute or chronic and is associated with many end-stage pulmonary, cardiac, renal, and malignant diseases. Greater than 90% of patients with chronic lung disease suffer from dyspnea in the last year of life.[70]

The perception of dyspnea should be considered analogous to the perception of pain in patients with end-stage illness: these are subjective findings. Clearly, both pain and dyspnea can cause tremendous suffering and should be aggressively managed. The pathophysiology behind dyspnea is complex, with many various pathways. Increases in PaCO2 or decreases in PaO2 or pH can stimulate either central or peripheral chemoreceptors. Additionally, activation of mechanical receptors in either the chest wall or respiratory musculature may stimulate breathlessness. Some patients may feel breathless with a normal arterial blood gas, while others may feel comfortable with marked abnormalities on blood gas.[71]

Management

Asking patients to rate the severity or distress of their dyspnea is suggested to assess both its impact and allow a baseline value to evaluate effectiveness of therapy.[70] This assessment should be routinely documented in medical records to guide management and interdisciplinary care.

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Table 53.5 Common antiemetics, sites of action, and starting doses

Drug	Brand name	Receptor site of action	Dose/route
Metoclopramide	Reglan	Dopamine and serotonin (only at high doses)	5–20 mg PO or IV before meals and at bedtime
Haloperidol	Haldol	Dopamine	0.5–4 mg PO, SQ, IV q4–6 hours
Prochlorperazine	Compazine	Dopamine	5–10 mg PO, IV q6 hours (also available in suppository form)
Chlorpromazine	Thorazine	Dopamine	10–25 mg PO, IV q6 hours (also available in suppository form)
Promethazine	Phenergan	Histamine, acetylcholine, dopamine	12.5–25 mg PO, IV q6 hours (also available in suppository form)
Diphenhydramine	Benadryl	Histamine	25–50 mg PO, IV q6 hours
Ondansetron*	Zofran	Serotonin	4–8 mg PO, IV q4–8 hours
Aprepitant	Emend	Neurokinin	125 mg PO x1, then 80 mg PO daily x 2–3 days
Olanzapine	Zyprexa	Dopamine, serotonin	2.5–5 mg PO daily
Scopolamine	Transderm scop	Acetylcholine	1.5 mg transdermal patch q3 days
Meclizine	Antivert	Acetylcholine	12.5–50 mg PO q8 hours

* one example of medication in this class

Therapeutic interventions should be based on underlying etiology whenever possible. Potentially reversible causes of dyspnea include pulmonary edema, bronchospasm, severe anxiety, and infection. Pharmacologic (bronchodilators, steroids, antibiotics), interventional (thoracentesis, chest tube placement), and nonpharmacological (fan, relaxation therapy, music) options should be explored.[70]

Supplemental oxygen is the standard of care for patients who are hypoxemic; however, minimal literature exists on its benefits in patients who do not have hypoxemia. Given its ease of use, minimal side effects, and rapid onset of action, a trial of supplemental oxygen should be used in any patient with subjective complaints of breathlessness.

At the end of life, the use of opioids, benzodiazepines, and steroids is still the cornerstone of treatment. Often patients are not in a hospital setting or may no longer want invasive procedures nearing the end of life. Morphine is often prescribed orally, intravenously, or nebulized. A Cochrane Review comparing nebulized morphine to placebo revealed no evidence to support its use in the treatment of breathlessness.[71,72] However, both oral and intravenous morphine, or other opioids, can be very effective in treating dyspnea at the end of life. Although trials have shown variable results, the dosage and intervals have been called into question in the trials that showed little effect.[70] Additionally, multiple studies have shown that use of palliative morphine has no effect on survival time.[73]

Benzodiazepines have been shown to reduce dyspnea in patients. These are believed to work via reductions in the perception of breathlessness and should be considered when there appears to be a large anxiety component.[74] However, these medications may increase drowsiness or exacerbate delirium. Thus, they should be used only when dyspnea is not controlled with opioids and other less psychoactive options.[75]

Care for the Imminently Dying

Many primary care clinicians will participate in the care of a patient who is imminently dying. Unfortunately, few providers report having received adequate training on end-of-life care and struggle with recognizing the dying process, treating symptoms, and advising family.[76] Patients and families all desire a "good death," often defined as "free from avoidable distress and suffering for patients, families, and caregivers," although there may be differences in how we get there.[77]

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Most Americans want to die at home, but this has not been the trend over the last century. In 2017, for the first time since the twentieth century, home surpassed the hospital as the most common place of death, with 30% of Americans dying at home, 29% in the hospital, 20% in a nursing home, and 8% at a hospice facility. Younger people, females, and racial and ethnic minorities are less likely to die at home.[78]

Hospice services are designed to provide end-of-life symptom management and guidance for families but have been historically underutilized. Only 48% of all Medicare beneficiaries who died in 2017 accessed hospice services. Unfortunately, patients are often referred late in their disease, with a median length of stay of 24 days, which can lead to suboptimal utilization of resources.[79] Racial minorities are less likely to access hospice services, with African Americans making up 8% of all hospice patients despite representing almost 13% of the US population.[79] Criteria for admission to hospice require a physician to certify a patient may have a prognosis of 6 months or less if the disease runs its natural course. Further disease-specific criteria are beyond the scope of this chapter but can be found at the National Hospice and Palliative Care website (www.nhpco.org).

Hospice offers four levels of service. Routine home care is the most common, provided in home or nursing facility by an interdisciplinary team. Continuous home care offers continuous nursing care in the home for a short period of time during a crisis period. Inpatient respite care is designed for short-term custodial care to relieve caregivers. General inpatient care offers higher-level, acute symptom management in an inpatient setting.[80] All service levels are provided by an interprofessional team, with services tailored to the needs of the patient and family.

The multidisciplinary team includes a physician who directs parts of the patient care. The patient can choose to have their PCP as the attending physician, and any treatment provided by the PCP related to the terminal illness can be billed directly to Medicare B using the CPT/EM code and a GV modifier. Only a provider not employed by the hospice can bill Medicare directly. Alternatively, the hospice physician/medical director may serve as the attending physician managing the direct care and billing through the hospice, and keep the PCP informed and part of the dialogue. Administrative and supervisory duties of the medical director/hospice physician are included in the hospice payment rate. All patient care not related to the terminal diagnosis continues to be billed to Medicare using appropriate billing codes.[81]

Of those dying in hospitals, end-of-life care is often less than adequate. Of families of patients dying in the hospital, many felt the patient and family had unmet emotional needs.[82] A meta-synthesis identified respectful, compassionate, expert care as well as good communication to be priorities for patients and families.[83]

Because many people still die in the hospital, it is important for physicians to be comfortable managing their care. There are several physiologic changes that occur in the last hours or days of life that need to be recognized. Weakness and fatigue gradually increase until the patient is bedbound. Patients often eat less and may stop drinking. This can be very distressing for families who are concerned their loved one is "starving" or is thirsty. Multiple studies have shown parenteral or enteral feedings do not alleviate symptoms or extend life for patients in the dying process.[84,85] Studies have shown patients experience very little hunger, and it is thought the resulting ketosis and dehydration may create a sense of well-being. Providing guidance and education to loved ones about a patient's lack of hunger or thirst is often very effective in decreasing caregiver distress. Families should be instructed to provide good oral and nasal hygiene, which can provide the patient comfort and minimize thirst.[86]

It is important to recognize respiratory changes common at the end of life to appropriately educate family and to control symptoms. Tidal volumes diminish; often there are apneic periods and increased accessory muscle use; Cheyne-Stokes respirations usually develop. As the patient becomes less conscious and the gag reflex diminishes, respiratory secretions often accumulate in the back of the throat, leading to gurgling respirations. This "death rattle" is estimated to occur in 25–90% of dying patients and predicts most will die within 2–3 days.[87] It is important to reassure families that this is not distressing to the patient. Nonpharmacologic interventions such as discontinuation of parental fluids and repositioning to ensure postural drainage can be effective. Aggressive suctioning is not recommended, as it is ineffective and can be uncomfortable. Antimuscarinics such as scopolamine, glycopyrrolate, or hyoscyamine have been the mainstay of anecdotal treatment despite having a poor evidence base. There is emerging evidence that they may be more effective if given prophylactically rather than waiting for the symptom to occur.[87,88] Side effects including dry mouth, urinary retention, and delirium should be taken into consideration.

Terminal delirium occurs in 28–83% of patients at the end of life and may be hypoactive, hyperactive, or

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mixed.[89] It is important to look for potentially reversible causes such as constipation, uncontrolled pain, and other medications. Antipsychotics have been the mainstay of treatment; however, recent studies have questioned their effectiveness. A 2018 Cochrane Review found low-quality evidences for drug therapy to treat delirium.[90] The National Institute for Health and Care Excellence (NICE) specifically excluded end-of-life delirium, leaving even less guidance.[91] Haloperidol has remained the drug of choice despite the limited evidence, and benzodiazepines can be considered for sedation and those with Parkinson's or Lewy Body dementia.[91]

Pain should be managed aggressively with opioids at the end of life, utilizing the tools and guidelines described above. Concerns about opioids hastening death have been refuted in the past by using the ethical principle of double effect. This principle states that an action with potential bad effects (respiratory sedation) is ethically permissible if the good effect (pain relief)is the intention.[92] Furthermore, emerging evidence supports the fact that appropriate and aggressive pain management with opioids at the end of life does not cause respiratory depression or affect survival.[93] A small number of patients may have symptoms that are not well controlled with maximal medical interventions. In these cases, patients may require controlled palliative sedation.[94]

There is good-quality evidence emerging that there is a beneficial relationship between overall patient well-being and religious commitment or practice. Many physicians still think it is inappropriate for them to discuss spirituality with their patients, feeling it is out of their range of expertise or is too intrusive. Studies have shown patients with life-threatening illnesses use religion to sustain hope and help them cope.[95] Spiritual well-being has also been shown to enhance quality of life, improve coping with depression and anxiety, and assist with positive reframing.[96] Support of this is well within the scope of a clinician's practice and is critical to providing comprehensive care at the end of life. Several screening tools exist and are simple to complete, such as Stenhauser's "Are you at peace?" or Mako's "Do you have spiritual pain?"[97] Addressing spiritual concerns can ensure appropriate referrals to chaplaincy services when indicated.

Anticipatory guidance is crucial to helping families with the dying process. It is difficult to predict exactly when a person may die, and the clinician should avoid giving specific numbers. It is more appropriate to give ranges such as hours or hours to days. Basic information on the signs of death is appropriate to be relayed to family. Experts recommend reviewing the process, the signs of death, and the death notification arrangements with caregivers to reduce stress at the time of death.[98]

Normal grief reactions may vary widely. When grief is prolonged, very intense, or interferes with physical or emotional well-being, it is considered complicated. One study showed risk factors for complicated grief included low education level, loss of partner or child, history of or current depression, and pre-loss grief symptoms.[99] Consultation with someone skilled in bereavement care may be necessary. As a general resource, local hospice organizations provide bereavement counseling and support. If complicated issues with grief and bereavement are suspected, a referral to a hospice grief program is likely helpful.

As medical technology continues to both prolong life expectancy and alter the dying process, skilled palliative care continues to be a growing necessity for elderly patients and their families. Investments in education about communication and symptom management can drastically improve the dying process for patients, family, and providers.

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Chapter 54

The Mistreatment of Older Adults

Seyed Parham Khalili and Laura Mosqueda

The Breadth and Depth of Elder Abuse

Elder mistreatment is an insidious and underreported form of abuse frequently missed by health-care professionals and even friends and family of the affected older adult. For research purposes, the US National Academy of Sciences (NAS) defined elder mistreatment as an intended or unintended act that causes harm or serious risk of harm to an older adult by a caregiver or other person who stands in a *trust relationship*.[1] For clinical purposes, the definition may be broader, without concerns for the nature of the relationship. Globally it is estimated that over one in eight older adults experiences at least one form of elder mistreatment across his/her lifetime, including psychological and emotional mistreatment (11.6%), financial mistreatment (6.8%), neglect (4.2%), physical mistreatment (2.6%), and sexual mistreatment (0.9%).[2]

Epidemiologic patterns in the United States differ somewhat both across the country and in socioeconomic, racial, and ethnic group comparisons. Domestically there is growing consensus that at least among community-dwelling older adults the past-year prevalence of elder mistreatment is approximately 10%, with the caveat that sampling and methodological limitations may mean this figure underestimates the phenomenon, particularly among the "old old" and adults with cognitive impairment or dementia.[3] Granular findings from the National Elder Mistreatment Study suggest that among community-dwelling adults, psychological and emotional mistreatment are most common (lifetime prevalence of 21.7%), followed by physical mistreatment (lifetime prevalence of 12.0%), and sexual mistreatment (lifetime prevalence of 7.0%), though data is limited to estimates of past-year prevalence for neglect and financial mistreatment (5.1% and 5.2%, respectively), and findings are sparse for poly-victimization (past-year prevalence 1.7%).[4,5]

With respect to older adults in post-acute care, assisted-living, and long-term care settings, there is a paucity of published epidemiological studies. A redacted dataset on Canadian nursing homes suggested that as many as one third of reported cases of mistreatment occurred in the long-term care setting.[6] US studies analyzing unverified anonymous reports from a cohort of US facilities staff suggest that elder mistreatment in this setting may be significantly underreported.[7,8] There remains much to be understood about the nature of elder mistreatment in these increasingly utilized care settings, from personal to systemic causes.

Studies of elder mistreatment across racial and ethnic groups highlight additional aspects of heterogeneity across communities. Data from the National Social Life, Health and Aging Project, the first nationally representative probability sample of community-dwelling adults aged 57–85 years across the USA, has been used to estimate the prevalence of mistreatment. This dataset, which oversampled Latinos, African Americans, men, and individuals aged 75–84, found that relative to non-Hispanic Whites, Latino respondents faced lower odds of emotional mistreatment, while African American respondents appeared to face higher odds of financial mistreatment.[9] Other studies focusing on specific geographies provide a more nuanced picture. Work by DeLiema and colleagues exploring the prevalence of mistreatment among Hispanic/Latino adults aged 66 years and older in low-income communities in the Los Angeles area noted that 40.4% of adults reported some form of abuse (nearly 25% reported psychological abuse, 10.7% physical abuse, 9% sexual abuse, 16.7% financial exploitation, and 11.7% neglect).[10] In a study of elder abuse service utilization in New York City, Hispanic/Latino older adults were less likely to report and seek help, particularly when the perpetrator was a child or grandchild.[11] A study of African American older adults living near Pittsburgh, Pennsylvania suggested higher rates of mistreatment compared to non-African Americans, particularly for financial exploitation (23.0% vs. 8.4%) and psychological mistreatment (24.4% vs. 13.2%).[12] Among a Chicago-area study of Chinese-American

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older adults, 15.8% of respondents reported at least one form of mistreatment, exacerbated by worse and worsening health, and attenuated by larger family size.[13] Clearly, there is much more to learn about the interface between ethnicity and culture in elder mistreatment, and clinicians should be alert to our implicit biases while remaining alert to the possibility of abuse.

The clinical and social impact of elder mistreatment is another area of active research. In terms of health-care utilization, mistreatment nearly doubles the rate of hospitalization even after adjustment for comorbidities, and may lead to premature institutionalization, which can both affect quality of life and magnify health-care costs.[14,15] While institutionalization is usually associated with multimorbidity and cognitive impairment, elder mistreatment has been found to independently increase risk for multiple individual disease processes, overall disability, and premature death, even after adjusting for demographics and chronic conditions.[16,17] Longitudinal follow-up assessments of outcomes associated with mistreatment in the National Elder Mistreatment Study have highlighted an increased risk for mental health pathology including depression, generalized anxiety disorder, and post-traumatic stress disorder by as much as 200–700% for certain types of abuse, which is further exacerbated by factors such as low socioeconomic status and poor self-rated health, and is potentially attenuated by protective factors including strong post-mistreatment social support.[15,18–20] The seriousness of the mistreatment from the perspective of victims is another area of inquiry, with recent work suggesting that the severity of the acts in cases of emotional mistreatment are mitigated by the age and functional status of the victim, frequency of abuse, and closeness of the relationship to the perpetrator.[21]

The psychological impact of elder mistreatment goes beyond the older adult by affecting family and other members of the victim's social network. A nationally representative telephone survey of adults found that nearly 30% of respondents knew an older adult who had experienced mistreatment, and nearly two thirds of the respondents, who often then assumed a helping role, reported personal distress themselves.[22] From an individual-, family- and systems-level perspective, elder mistreatment can be devastating in the short and long term, and it remains essential that health-care providers and other professionals remain vigilant and proactive during clinical encounters.

Risk Factors for Older Adults and Involved Persons

Case

Mrs. T comes into your office for follow-up on her hypertension, atrial fibrillation, and diabetes (the HbA1C from 2 days prior was 8.9; her office blood pressure is 178/82 and heart rate is 80 beats per minute). She is on enalapril/hydrochlorothiazide, apixaban, and metformin. As her primary health-care provider, you have known her for 8 years, and in the past few months her previously well-controlled blood pressure and diabetes have been difficult to control. Today she has bruises on her left cheek just in front of her ear, and on the right upper shoulder/base of the neck. You ask her how she got the bruises and she says, "I fell when I got up to go to the bathroom in the middle of the night. You know me . . . I bruise so easily! Next time I'll pay more attention and make sure I put a light on." You ask a follow-up question: "Mrs. T, I'm concerned because it's unusual to get a bruise on your head and neck from a fall such as this. I know there's been a lot of stress at home. Is anyone hurting you?" She breaks down in tears, explaining that her son moved in about 6 months ago and has been taking her money, first with her hesitant permission and now with impunity. More recently he has been physically threatening and 2 days ago, when he was inebriated, he grabbed her shoulder and hit her on the side of her face.

There are numerous older adult-, perpetrator-, and system-level factors that increase risk for elder mistreatment. With respect to community-dwelling older adults, a number of associated medical and psychosocial characteristics are commonly cited in the literature, including but not limited to multimorbidity, physical impairment and frailty, cognitive impairment and dementia, formal mental health diagnoses and psychological stressors, low social support and functional dependency, low socioeconomic status, and potentially cultural norms that may impede reporting of abuse.[23,24] Gender may play a role, as several studies have revealed differences where female sex may be associated with a higher chance of emotional mistreatment, and in one study physical mistreatment against men had more "pathological" characteristics described as perpetrator interactions with law enforcement, psychological problems, substance use, and chronic unemployment.[25,26]

Elder financial fraud and scams constitute a larger pool of financial crimes against older adults, affecting approximately 1 in 18 cognitively intact community-dwelling older adults, with mounting evidence that risk

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for financial exploitation increases in the presence of cognitive impairment. A 2015 study by Han et al. of community-dwelling adults without dementia found that while cognitively intact older adults could be susceptible to scams, risk increases significantly in the presence of even mild cognitive impairment.[27] These types of insidious financial crimes ultimately increase morbidity, mortality, and health-care utilization, though whether this constitutes elder mistreatment based on the NAS definition specifically is contingent on whether the older adult has a relationship to the individual based on trust.[28] Nonetheless, there may be significant overlap in risk factors and vulnerabilities that predispose certain older adults to financial exploitation, whether by a trusted person in their life or a stranger. Contextualized within the concept of Routine Activity Theory, which posits that crimes occur in the presence of a "motivated offender and suitable target" and in the absence of protective individuals, a study by DeLiema examined neuropsychological profiles of victims of fraud and financial elder mistreatment; both cohorts of older adults performed similarly and poorly on tests of cognitive function and financial decision-making.[29] Another study using a sample of case referrals to the Los Angeles County Elder Abuse Forensic Center (LACEAFC) compared the neuropsychological profiles of the mistreated older adults with a cohort of community-dwelling control subjects, finding that the former group performed more poorly on the Mini-Mental Status Exam (MMSE), calculations, and tests of executive functioning, all domains that are associated with financial capacity.[30] Similarly, the aforementioned study by Han et al. noted that compromised performance in the specific domains of episodic memory and perceptual speed were particularly notable among cognitively impaired older adults at heightened risk for exploitation.[27] Future research exploring these and other findings related to vulnerabilities toward financial exploitation may shed additional light on particular scenarios, risk and protective factors, and interventions.

Growing research on another piece of this puzzle, older adults' social networks, provides additional insight and raises more questions. Some researchers have found that older adults with less dense core networks including fewer kin are more likely to experience polyvictimization, particularly when the most common perpetrators are children and other relatives embedded within the same social network.[31,32] Other work has suggested that larger social networks are not always more protective; however, as the degree of social support within the network matters for financial mistreatment (and possibly other forms of mistreatment), expansion of the social network may then be protective provided that the new relationships are meaningful in terms of actual support.[33] Relatedly, help-seeking behaviors have become another area of inquiry. It may be that some older adults are persuaded to seek help within and outside their social network when the mistreatment becomes severe enough, when there is more than one type of mistreatment, when the older adult is more independent, when the perpetrator is less embedded within an overlapping social network, and/or at the point where the older adult fears for their physical safety.[34,35]

While much of the discussion regarding elder mistreatment assessment revolves around assessment of the older adult experiencing the abuse, there is growing interest in identifying strategies for assessing the (alleged) perpetrator as well. Prior findings in studies of perpetrators of mistreatment have suggested that psychiatric illness, substance use, unemployment, a history of arrest or legal issues, smaller social networks, higher perceived caregiving burden, and co-residence with the older adult may amplify risk of abuse.[23,25] A latent class analysis of adult protective service cases in Illinois by DeLiema and colleagues identified four specific categories of perpetrators, labeled "caregiver," "temperamental," "dependent caregiver," and "dangerous."[36] The "dangerous" individuals were more likely to be more aggressive, financially dependent, and irresponsible, with higher rates of substance use. Similarly, in another study of elder mistreatment cases, one in four perpetrators used substances or had a substance use disorder, and these perpetrators were mostly men not involved with caregiving who generally committed more than one type of mistreatment simultaneously.[37] In yet another study, the cluster of substance use and mental health diagnoses has been associated with as much as one third of cases of emotional and two thirds of cases of physical abuse, and was associated with greater histories of mistreatment and more severe emotional problems among victims.[25] These findings increasingly illustrate a "perfect storm" of individual characteristics, interpersonal dynamics, external social and economic stressors, and other variables that can jointly amplify the risk for mistreatment.

Inquiring about Mistreatment and Examining the Older Adult

Case

Mrs. P is a 76-year-old woman with moderately advanced Parkinson's disease, poor vision, and diabetes.

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Her 51-year-old son lives with her because he was recently laid off and agreed to take on the role of caregiver, as she needs an increasing amount of help with instrumental activities of daily living and activities of daily living. She had several falls in a 6-month period and was noted to have bruises in many locations on her trunk and face. She was seen several times by her primary care doctor who accepted the idea that people with Parkinson's disease are likely to fall and never questioned her about the falls or her injuries. At the time of her hip fracture, it was learned that her son had been shoving her in anger and occasionally punching her if she asked for help at an inopportune time. This had been going on for the past 8 months. She never told her doctor because "he never asked me."

Given the often-hidden nature of this phenomenon, inquiring about elder mistreatment risk factors and occurrence should be a routine part of clinical assessments of older adults in any care setting, even in the absence of overt physical signs of injury. How the questions are asked may be as important as the questions themselves. An empathic and nonjudgmental tone in a private setting can help establish a safe space for the patient to divulge often painful and traumatic aspects of their lives. Approaching the scenario using open-ended questions and neutral language may also help clinicians guard against the intrusion of unwarranted assumptions and premature closure of the differential diagnosis in cases where mistreatment is less obvious. Of note, there has been research examining the ability of older adults to report elder mistreatment, and in a recent study it was found that the majority of older adults could do so even in the presence of comorbid cognitive impairment, further supporting the notion that clinicians should avoid making assumptions and should ask all patients about possible elder mistreatment.[38]

While screening older adults for elder mistreatment is an important first step for clinicians and other professionals across care settings, the evidence for the use of specific tools is mixed. Despite the fact that there are a multitude of screening instruments and several have demonstrated relatively good internal consistently and usefulness for early recognition, no individual tool was singled out as clearly superior in a recent systematic review, specifically with respect to real-world health outcomes.[39] Similarly, an evidence report and systematic review for the US Preventive Services Task Force included only one screening tool for elder mistreatment and found that the instrument in question demonstrated low sensitivity (46%) and specificity (73%) for detecting physical or emotional mistreatment.[40] Moreover, another recent study examining practitioners' views when using one of five validated screening instruments (Vulnerability to Abuse Screening Scale [VASS], Elder Abuse Suspicion Index [EASI], Elder Assessment Instrument [EAI], Caregiver Abuse Screen [CASE], and the Brief Abuse Screen for the Elderly [BASE]) exposed significant issues including problematic terminology and question phrasing, a lack of recognition of cultural factors, and limited utility in situations involving a victim with cognitive impairment.[41] With respect to screening the alleged perpetrators, a recently published instrument by Conrad and Conrad, called the Abuser Risk Measure, provides another potential tool for frontline providers.[42] Both the full 21-item and abbreviated 9-item (screening) version were predictive of physical and emotional mistreatment and suggestive for risk of financial mistreatment. Clinicians should consider the use of a screening instrument for the older adult and/or the other involved individuals, along with careful history-taking (including integration of collateral information whenever possible), a detailed physical examination, and judicious use of diagnostic tests. While we wait for the development of validated, pragmatic screening tools in the clinical setting, the clinician may ask probing questions such as: Are you afraid of anyone? Is anyone hurting you? Is anyone using your money without your permission?

Elder mistreatment might be suspected and uncovered in a number of clinical and nonclinical settings. The emergency department is one such environment that has received growing attention, considering that elder mistreatment is estimated to be diagnosed in as little as 0.013% of emergency department visits in the USA.[43] Providers in the emergency department and other care settings can remain vigilant regarding the possibility of elder mistreatment during assessments of patients alone and during interactions with family members, companions, guardians, and caregivers.[44] As detailed by Rosen et al., particular observations that may raise concern could include conflicting accounts of events, frequent interruptions by the caregiver, signs that the caregiver appears either overwhelmed or uninterested/uninformed, signs that the older adult is fearful of or hostile toward the caregiver, or signs of substance use by the caregiver or older adult.[45] Additional findings during the interview that further increase suspicion may include unexplained injuries or a history of frequent injuries, frequent emergency department visits for similar events, delays in care-seeking, and fragmented care, including often more than one health-care source, missed

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appointments, and poor adherence to care plans.[45] Specific questions that may shed light on the scenario can include assessing the older adult's functional status and physical abilities, directly asking about each type of abuse, and screening for mood disorders and substance use by the older adult and the caregiver/other involved person(s).[46]

Examiners can then advance the diagnostic process by not only including a thorough general physical assessment, but also maintaining a critical eye toward injury patterns that may be the result of mistreatment. While a more extensive description is beyond the scope of this section, visible signs and symptoms might include burns and bruising in atypical locations or suggestive of immersion-related injury, cigarette burns or other pattern injuries that indicate object use (such as belts, clothes hangers, or wrist restraints), injuries in different stages of healing, traumatic alopecia and other scalp injuries, ophthalmologic injuries, sexually transmitted infections and focal trauma, poor hygiene, malnutrition and dehydration, and pressure injuries.[45,46] Nonaccidental bruising in this context has been found to be more likely to be located on proximal upper extremities (especially the lateral arm), proximal lower extremities, and posterior torso, face, head, and neck, often with larger individual bruises (greater than 5 cm) and multiple bruises.[45,47] Just as with younger patients, scenarios suggestive of possible sexual mistreatment warrant an assessment by a trained sexual forensic examiner.[45] In all cases timely, accurate, and complete written documentation of findings is essential. Finally, examiners must also maintain a differential diagnosis for a multitude of confounders related to physiologic aging, signs of chronic and advanced disease, and medication-related side effects or adverse events, as the differential diagnoses for mistreatment may be broad.

Laboratory testing, imaging, and other diagnostic testing may also assist with the assessment. Few if any individual findings are pathognomonic for abuse, so suggestive physical findings and supplemental laboratory and radiologic findings must be analyzed in the larger context of history and presentation. Imaging that suggests abnormalities such as injuries in various stages of healing, high-energy fractures incongruent with reported mechanisms of injury (and the older adult's physical capabilities), or other concerning findings should be discussed with the radiologist for collaborative assessment.[45] Laboratory assessment may improve detection of toxicity related to substance use or inappropriate medication use, subtherapeutic medication levels suggesting poor adherence, signs of dehydration, metabolic abnormalities associated with malnutrition, muscle breakdown, and other potential consequences of elder mistreatment.[47,48] Communication with other providers and consolidation of prior medical records might help establish the chronicity of findings as well as clarify confounders for the patient in question.

One particularly challenging and common diagnostic predicament includes older adults with cognitive impairment and/or dementia. As noted earlier, older adults with cognitive impairment are at significantly elevated risk for mistreatment and this may be underreported, despite the fact that many of these individuals can indeed articulate the mistreatment to a third party. The following case illustrates some dementia-related aspects of the older adult–caregiver dynamic.

Case

Mr. J is an 86-year-old man with mild-to-moderate Alzheimer's disease. As it became unsafe for him to continue living independently, he was welcomed into the home of his son and daughter-in-law and their three children, aged 14–19 years. He had always been a fastidious person, but after the first few months of the move he was often disheveled and unconcerned about his poor hygiene. When the family encouraged him to bathe, he would get angry and yell at them. They yelled back. When they tried to help him get undressed and put on clean clothes, he would resist their efforts and sometimes hit them. They hit him back.

This is a scenario that many providers have seen and understand. Despite our empathy and appreciation for what the family is trying to do with the best of intentions, this is still elder abuse. As we have learned from colleagues in domestic violence, it can be easy to blame the victim and thus miss our opportunity to protect and intervene. The fact that an older adult may be "difficult" or "hard to take care of" or "demanding" does not excuse an abusive act. It does, however, allow us to understand the dynamics and provide appropriate support for both the elder and the family. Asking direct questions of both the family and the older adult at an early stage of this scenario could have allowed a clinician to identify the growing stress and anger and potentially provided an opportunity to intervene sooner.

Interventions, Systems, and Reporting Mechanisms

Prevention of elder mistreatment is an area of widespread interest, though data on specific strategies has

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been mixed across several systematic reviews. A 2019 systematic review by Rosen et al. examined 115 elder mistreatment programs and noted that few studies used a high-quality design or included an acute care hospital (13%), and the majority of the interventions were educational (53%), with fewer numbers of studies examining multidisciplinary teams (21%), therapy or counseling (15%), or legal services/support (8%).[49] An earlier Cochrane systematic review of programs for preventing or reducing elder mistreatment in community-based and institutional settings unfortunately noted multiple similar limitations, including a high risk of bias, which ultimately limited the strength of the evidence for any one intervention.[50] While some programs appeared to improve detection of mistreatment in institutions, educational interventions did not substantially and reliably improve knowledge among providers and caregivers; in addition, programs aimed at supporting victims of mistreatment seemed to improve reporting rates, but the effect on actually reducing mistreatment remained unclear.[50] Another systematic review examining interventions aimed at professionals, older adults, and caregivers found evidence of effectiveness for reducing use of restraints by long-term paid caregivers, but not other types of interventions.[51] Finally, a systematic review of community-based programs only identified high-quality evidence for two studies of psychological interventions, with only one of the two studies (START program) potentially lowering the risk of mistreatment; one of four additional studies examined multidisciplinary interventions but showed lower-quality evidence of a reduction in abuse risk.[52]

Given the limitations to this existing data on prevention and intervention programs, there has been a call for more research exploring not only models that encapsulate the conceptual and observed psychosocial complexities of elder mistreatment, but also a practical framework for translating such findings into action. One recent contribution to this literature is the Abuse Intervention Model, which leverages insight from interpersonal, sociocultural, and multisystem theories to posit a three-domain model including the vulnerable older adult, trusted other, and context of the interaction(s).[53] This model provides a shared language for professionals, older adults, and caregivers and can be applied to scenarios commonly encountered in real-world settings, helping categorize level of risk and identify potential strategies for mitigation of harm.[53] Another recently published approach by Du Mont et al. describes key components of a Social Inclusion Framework for hospital-based interventions with 12 specific determinants to be reviewed for each individual (in order of importance): history of trauma/abuse, communication needs, disability, health status, mental capacity, social support, culture, language, sexuality, religion, gender identity, and socioeconomic status.[54]

There is ongoing interest in translating the aforementioned frameworks into hospital- and other health-care-setting-based strategies for early identification and intervention. The hospital experience may include pre-arrival interactions with emergency medical services (EMS) providers, the emergency department assessment, the inpatient course on a medical or surgical service, and even the immediate post-hospitalization period, whether in a post-acute care facility or home. Upon arrival, EMS providers may be privy to valuable information about the older adult's built and lived environment, including signs of hoarding, food scarcity, vermin infestations, temperature extremes, and nonfunctioning utilities.[45] EMS providers may frequently encounter these and other scenarios that raise suspicion, but they may also face obstacles when assessing and reporting these findings, potentially because of a lack of specific training in this area, time constraints, and obstacles to reaching, communicating with, or hearing back from other professionals in the emergency department. Proposed strategies may include use of photographs on home assessments, training programs, or use of a hotline for reporting, for example.[55] A recent publication by Cannell et al. evaluated a specific screening tool for use by EMS professionals called the Detection of Elder Mistreatment Through Emergency Care Technician (DETECT), which appeared to statistically significantly improve the relative risk of mistreatment detection (RR 3.03) without compromising the validation rate among cases reviewed by adult protective services.[56]

Once in the emergency department, vulnerable older adults may encounter a variety of professionals whose assessments can be critical for timely identification and intervention in cases of mistreatment. In addition to careful history-taking and medical examinations, integration of insights from all members of the multidisciplinary team is crucial. This includes anyone who had contact with the patient and alleged perpetrator: nurses, technicians, social workers, patient escorts, and others who may spend significant facetime with the parties and provide a unique perspective. Clinicians' primary interventional goals during an assessment include acutely treating and medically stabilizing the patient, simultaneously ensuring the patient's safety, and reporting suspicion to appropriate parties,

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which include adult protective services and, rarely, law enforcement.[45] Unlike child protective services, adult protective services formally initiates investigations after discharge from the hospital, and part of the assessment process in the emergency department includes deciding if the older adult should be formally admitted for safety and workup, which additional factors need to be reviewed for safe discharge, whether mandated reporting applies (as it varies by state), and whether there are issues of health-care decision-making or guardianship that need to be addressed.[45]

Whether hospitalized because of a general clinical diagnosis or because of concerns for safety and need for additional elder mistreatment evaluation, the admission provides a valuable opportunity for a nuanced assessment in a controlled setting where the older adult may be evaluated without the influence of the potential perpetrator. There is growing interest in multidisciplinary approaches in this setting. As described by Rosen and colleagues, one such approach, called the Vulnerable Elder Protection Team (VEPT), is an emergency department-based service that can be activated by any provider, leading to evaluation by an elder mistreatment-trained emergency medicine physician and social worker and subsequent care coordination and collaboration with inpatient providers for admitted older adults.[57] While data is lacking on elder mistreatment-specific hospital programs that might prevent or stop abuse, approaches such as the VEPT model are promising. Further work exploring continuity of care beyond the admission, including primary care physicians and other providers involved in the care of affected older adults, may also help introduce additional strategies for elder mistreatment recognition and risk mitigation beyond the transient hospitalization period.

The clinic and home environments represent additional settings where hospital-based assessments can be continued and/or new evaluations may be initiated. As telemedicine visits become more common, these offer another window into the home situation. Privileged with a meaningful longitudinal relationship with the older adult (and their caregivers, family, and other involved persons), primary care providers are usually well positioned to fulfill a unique and valuable role in unearthing and addressing mistreatment. Informally, this may mean that a patient's physical, emotional, and cognitive health is understood more deeply, allowing for sometimes subtle changes to be recognized earlier. This patient–clinician relationship may also allow greater appreciation of interpersonal dynamics in the older adult's relationships, and might be conducive to a higher likelihood of disclosure of abuse. Unfortunately, findings have been limited, and mixed, in terms of the effectiveness of formal, protocol-based approaches for reducing mistreatment in this setting. A randomized controlled trial of a program called START (STrAtegies for RelaTives), which consisted of manual-based coping strategies and was aimed at family caregivers, appeared to show reductions in caregiver anxiety and depression but not abusive behavior.[58] Clinicians and other health-care professionals need to draw upon the insights gleaned from their comprehensive assessment to tailor interventions to the patient, their caregivers, and the lived environment. A complicated but not uncommon scenario is described below, illustrating several of these points and how a multidisciplinary approach can leverage the team's collective strengths to develop a safe and sustainable action plan for the older adult and his/her caregivers.

Case

Mrs. U's three adult children (two daughters, one son) had been noticing her memory problems for about a year. They had escalating concerns and brought her to the office for an assessment. In the past few months, she had: two minor car accidents; gotten lost when walking in her neighborhood; and put a paper plate on top of the gas stove to heat a meal, which started a kitchen fire that was quickly extinguished by her visiting son. A thorough examination by an interdisciplinary team resulted in a diagnosis of probable Alzheimer's disease. At the time of the family conference the psychologist and physician assistant explained the diagnosis to the family and discussed prognosis and the need for more assistance. Concerns regarding her behaviors, which included agitation, especially when trying to convince/help her to get into clean clothes, and a volatile temper, were discussed. The family explained that they had discussed some of these issues and had an idea: one of the daughters volunteered to move in with mom. She didn't work, was unmarried, and had no children. Further inquiry about this 42-year-old daughter revealed that she had bipolar disorder, which had been adequately treated for a few months. She had a long history of cyclical problems related to going on and off her medications. Knowing that the equation of a daughter with poorly controlled bipolar illness plus a mother with dementia and agitation could add up to an abusive situation, the team explained that it would be best to work on an alternate plan. The social worker provided counseling and assistance with the alternatives, and a high-risk-for-abuse situation was avoided.

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Moving forward, innovative strategies are increasingly important not only because the cohort of older adults is rapidly growing in number and more individuals will wish to remain at home to "age in place," but also considering the rapid and sweeping changes ushered in by the coronavirus pandemic. Authors Makaroun, Bachrach, and Rosland outline several considerations and potential opportunities for clinicians and policymakers. They identify risk factors including increased stressors and financial vulnerability for older adults and their caregivers, reduced and/or fragmented caregiving, changes in the nature and intensity of cohabitation in the setting of possible concurrent increases in behaviors such as substance use, and potential limitations to technological uptake for older adults (though this may be an opportunity as well).[59] Adapting to these changes will be yet another challenge for older adults, their caregivers, and health professionals.

Finally, an understanding of reporting mechanisms for elder mistreatment and how a systems-based perspective can facilitate evaluation and treatment is essential. Surprisingly, in a 2004 analysis of adult protective services agencies, as little as 1.4% of reports were made by physicians.[60] Part of the issue may lie with confusion regarding reporting requirements and processes that vary geographically across the USA and differ from the more widely understood protocol for reporting child abuse. As of 2020, clinicians are mandated elder mistreatment reporters in most states according to the US Department of Justice and the American Bar Association, with the latter organization's website providing a helpful outline of state-specific guidelines detailing who must report, when the report should be made, how to report, and salient additional resources.[61] In most states, a report to adult protective services (for community-dwelling older adults) or to the Long-Term Care Ombudsman (for older adults who live in a licensed facility) is required if the clinician has a reasonable suspicion of abuse or neglect. It is not necessary to have made a definitive diagnosis. Simultaneously, clinicians should notify other involved providers, their institution's social work and case management teams, and other key personnel in administration. Moreover, clinicians need to consider notifying local law enforcement in scenarios involving patient safety and the occurrence of a crime, which is a rare event.

Ultimately the affected older adult is best served when a multidisciplinary team can be convened to examine the issue comprehensively and facilitate a safe and actionable plan for intervention. Nonclinical concerned individuals, including the affected older adult, friends, family, and others, can also seek help through several mechanisms. Situations that represent immediate and life-threatening danger warrant activation of emergency services through 911. Other concerning scenarios may be addressed through the federal Administration for Community Living (ACL) and Administration on Aging (AoA) Eldercare Locator, a free service available online and through a toll-free hotline (1-800-677-1116).[62] The National Center on Elder Abuse is another resource for reliable information.

Conclusion

Astute health-care professionals have the opportunity to prevent, detect, and intervene in cases of elder mistreatment. However, this can only be done if the possibility of elder mistreatment is unfailingly on the differential diagnosis in many circumstances. The older adult who has frequent falls, injuries, a sudden change in behavior or demeanor, who starts missing appointments, or who is not taking medications as expected may in fact be experiencing mistreatment. While we do not want to accuse unfairly, it is important that we ask questions when a suspicious injury or event occurs and then assess whether the response from the older adult and/or the other involved persons is appropriate and believable. Our patients may have no one else who will look, ask, and listen with compassion. Health-care professionals have the knowledge, skill, and resources needed to not only safeguard an older adult's health and well-being but also possibly even save their life.

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Chapter 55

Driving and the Older Adult

Joanne G. Schwartzberg, David B. Carr, and Annie C. Harmon

Introduction

Driving is an essential and highly valued instrumental activity of daily living (IADL) that becomes increasingly difficult to safely maintain with age-related medical conditions. In 2017, nearly 44 million licensed drivers were over age 65 in the United States and approximately 250,000 were injured in motor vehicle accidents.[1] Health-care providers can play an important role in helping older adults make decisions about driving; they are uniquely positioned to (1) identify and modify risk factors associated with on-road safety, (2) offer rehabilitation strategies to improve safety and extend driving life, (3) report unsafe driving when appropriate, and (4) combine clinical information with resources related to driving to support safe continued community for older patients. Clinicians face myriad challenges in assessing older patients' medical fitness to drive, including multiple comorbidities, acute and/or chronic medical conditions such as dementia, modifiable and/or irreversible conditions, and polypharmacy. Clinicians may also be reluctant or unsure of how to address driving issues because of the potential impact on the relationship with the patient and legal/ethical concerns. These choices can be especially difficult in states that mandate health-care providers report drivers who are medically unfit to drive, adding further stress and uncertainty for providers. However, assessment and intervention are important to prevent injury and the potential loss of driving privileges, the latter of which may have a negative impact on quality of life.

This chapter will describe the functional abilities necessary to be a safe driver at any age; acute and chronic changes and medical risk factors for driving impairment; clinical tools to assess or gauge current driving performance; opportunities to intervene or refer patients flagged for impairments; resources to support patients transitioning from driver to nondriver; and finally, ethical and legal concerns for clinicians advising patients on driving.

Older Adults and Driving

Older adults rely on personal vehicles for 90% of their transportation.[2] As a group, older adults are generally safe drivers. They wear seatbelts more frequently, drive when conditions are safest, and exhibit fewer aggressive or high-risk driving behaviors compared to other age groups.[1] Older adults are remaining behind the wheel longer than ever, both by choice and necessity. Nearly 80% of older adults live in auto-dependent areas, meaning rural or suburban areas that require driving more frequently and farther distances,[2] making driving a crucial tool for continued independence. The average 70-year-old driver has approximately 11 years left to drive a car.[3]

Even though many older persons self-restrict their driving to compensate for age-related changes and disease,[4,5] crash rates per mile traveled start increasing for drivers between ages 70 and 74, increase markedly after age 80, and are highest among drivers 85 and older.[6] Increased crash rates per mile traveled may be inflated since older driver mileage is more frequently city driving, which typically has higher crash rates than freeway driving.[7] These differences can be taken into account by risk-exposure density methods.[8] In addition, high-mileage drivers regardless of age will have lower crash risks. It appears to be only the low-mileage drivers (<2,000 miles per year) that are at increased risk per miles driven.[9] This suggests that older adults who drive infrequently (e.g., only for medical or household needs) are not necessarily lowering their crash risk.

Unfortunately, when motor vehicle crashes do occur, there is an excess risk of injury and death in older adults compared to middle-aged drivers.[6] In 2017, over 7,000 older adults were killed in motor vehicle crashes, and over 250,000 more were injured.[1] In other words, crashes kill 20 older adults every day, with an additional 700 older adults injured in motor vehicle crashes. Potential underlying reasons may include older adults' fragility, limited physiologic reserves, side effects from medications, greater

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susceptibility to impact based on the types of crashes (broadside collisions from unprotected left-hand turns), or driving older vehicles that are less crashworthy.

The risks of driving must be weighed against individual rights and rewards associated with driving. Referred to as the "ultimate IADL,"[10] driving is one of the most valued activities among older adults.[11] Driving offers convenient transportation and supports continued independence, especially important given the dearth of alternative transportation options in most of the USA. Unfortunately, many older adults reach a point where they reduce or stop driving for safety or comfort. Transitioning from "driver" to "former driver" negatively affects individuals' physical, mental, and social health. Because of these potential consequences, it is crucial to ground advice in medical reasoning based on patients' current function rather than chronological age.

Understanding the functional demands of driving can help focus clinical assessment and decision-making. The demands of driving generally involve three main functional areas: vision, cognition, and motor function:

- Vision – visual acuity, contrast sensitivity, field of view to observe roadway environment, including other vehicles, pedestrians, road signs, lanes, etc.
- Cognition – processing visual information, shifting attention, reasoning and prioritizing information, memory, language, judgment, and decision-making
- Motor function – range of motion, strength, coordination with the ability to carry out decisions, vehicular control

Impairment in any of these increases on-road risk, regardless of patient age. Clinicians should follow up with a more in-depth clinical assessment to gauge individual risk and the appropriate necessary next steps.

Clinical Assessment

The first step of a medical assessment for driving ability is a targeted history. Be alert for functional changes, medical conditions, medications, or symptoms that may impair vision, cognition, or motor skills required for driving. The second step is to ask about driving history and current driving behaviors to identify exposure and relevant driving events that can signal impairment. Third, complete a targeted physical and cognitive examination to evaluate current functional abilities. With this information, appropriate next steps can be identified based on patients' individual needs.

Step 1: Targeted history of patients' functional changes and medical risk factors (i.e., acute and chronic medical conditions, medications, or symptoms) that may impair driving skills.

Functional Changes

Functional abilities essential for driving can decline because of normal physiologic changes of aging, increased comorbid illness, or both.[12] Impairments in vision, neuromuscular strength and speed, and cognition have been linked to crash risk for older adults. Changes to vision occur as a normal part of aging, and are also caused by common age-related diseases, such as cataracts, glaucoma, and maculopathy. Any condition that impairs visual sensory, visual processing speed, and visuospatial abilities may increase crash risk.[13,14]

Cognitive domains such as language, attention, memory, visuospatial skills, and executive function are related to driving and typically assessed in clinical settings.[15] Changes that reduce or limit functional range of motion, flexibility, strength, and endurance influence driving safety as well. Neuromuscular factors such as reduced neck rotation, impaired coordination or muscle strength, slowed brake reaction time, and/or decrements in coordination are concerns in regard to vehicle control. Fall history has been associated with higher risk of motor vehicle collision.[16]

Medical Risk Factors

Many medical conditions are often reviewed by national organizations (Table 55.1) with updated recommendations[17,18] and have been associated with impaired driving performance or increased crash risk.[19] Medical illnesses and treatments that may be linked to driving difficulty in self-report, case-control, and retrospective population studies include a history of falls, coronary artery disease, stroke or transient ischemic attack, kidney disease, and – in women – arthritis.[20–22] Since the odds ratios for crash prediction from a single medical condition are small, some national organizations have developed pathways or protocols to assist clinicians with determining the functional impact of medical conditions on driving ability. The American Medical Association created an advisory panel that developed the Physician's Guide to Assessing and Counseling Older Drivers, first released in 2003 and updated by the American Geriatrics Society and National Highway Traffic Safety Administration

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Table 55.1 Medical risk factors for driving impairment

Acute or unstable medical events: acute myocardial infarction, angina, hypoglycemia, stroke or transient ischemic attack, traumatic brain injury, syncope, vertigo, seizure, surgery, delirium, sleep apnea, hypersomnolence, falls, gait change
Chronic medical conditions:
Vision – field cuts, cataracts, glaucoma, macular degeneration, hypertensive or diabetic retinopathy, retinitis pigmentosa
Cardiovascular disease – unstable angina, arrhythmia, valvular disease, congestive heart failure, orthostasis
Neurologic disease – seizures, cognitive impairment/dementia, Parkinson's disease, multiple sclerosis, peripheral neuropathy, stroke, brain injury
Psychiatric disease – depression, anxiety, psychosis, alcohol or substance abuse
Musculoskeletal disability – arthritis, foot abnormalities, previous fractures, cervical disease, amputation, restricted range of motion
Respiratory disease – obstructive sleep apnea, chronic obstructive pulmonary disease, asthma, disease requiring oxygen supplementation on a daily basis
Metabolic disease – diabetes, renal failure, thyroid disease, hypoglycemia

Many medical conditions can impair a patient's fitness to drive by causing functional decrements in vision, motor control, and cognition.

Table 55.2 Common medications associated with crash risk

Hypnotics
Muscle relaxants
Antidepressants
Benzodiazepines
Parkinson's medications
Opioids
Sedating antihistamines
Antiepileptic drugs (AEDs)

Any medications with psychoactive, cardiovascular, neurologic, or potentially sedating agents can impair driving.

(NHTSA) as the Clinician's Guide to Assessing and Counseling Older Drivers in 2019.[23] These recommendations are usually based on clinical consensus, clinical experience, expert medical opinion, or best practices, since evidence may be limited. Individuals with multiple comorbidities and polypharmacy make evaluation extremely challenging.

Progressive neurodegenerative illnesses such as Alzheimer's disease and related dementias (ADRD) and Parkinson's disease have been linked to impaired driving performance and greater crash risk because of their adverse impact on cognitive skills, including memory, visual-spatial skills, attention, reaction time, processing speed, and executive function.[24,25] Therefore, for patients with progressive neurodegenerative diseases, the question of driving cessation is not if, but when. The consensus among safety experts is that persons with moderate to severe dementia should stop driving.[26] Two evidence-based reviews in the literature propose algorithms based on the recent evidence, which may be useful to the clinician in managing and assessing older adults with dementia who would like to continue to drive.[27,28] These approaches include rating dementia severity, assessing functional status, obtaining a driving history, and focused physical examination and referral when appropriate. Additional driving simulator or on-the-road assessments are likely to be needed.[29]

Medications

Use of certain medications is also linked to increased crash risk, including benzodiazepines, opioid analgesics, alcohol, muscle relaxants, and sedating antihistamines (Table 55.2).[30] Depression and the use of antidepressant medications may also impair driving performance in older populations,[31] although the underlying illness may be the actual cause. Overall, any medication whose direct or side effects alter vision, awareness, balance, muscle strength, or coordination increases driving risk. A careful review of patients' medication lists can identify medications that can safely be deprescribed or replaced with similar options that do not have the impairing side effects.

Step 2: Driving history interview to collect relevant information about patient's driving behaviors and significant events.

A general driving history interview gives patients and caregivers the opportunity to express concerns or details that may be overlooked or withheld during the visit. Questions for the patient (Table 55.3) should include driving frequency, recent changes, and awareness of others' reactions to their driving. Caregivers' answers to similar questions (Table 55.4) offer an outside view for comparison. Providers may wish to provide these questions or use the

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Table 55.3 Patient driving history questions

1. Do you now drive a car?
2. How many days did you drive this past week?
3. Have you noticed any change in your driving habits in the past year? Please check all that apply.
Do not drive at night
Do not drive on freeways
Do not drive in rain/snow, bad weather
Do not drive during rush hour
Do not drive as far
Only drive when I absolutely must
Prefer for others to drive
Have cut back because of being too sick/tired
4. In the past year, have you had any of the following events? Please check all that apply.
Accidents
Fender-benders
Near-misses
Tickets
Discussions/warnings
5. Have you ever forgotten where you were going?
6. Do others honk at you or act irritated?
7. Have you recently gotten lost while driving in a familiar place?
8. Have others said they are worried about your driving, criticized you, or refused to ride with you?

Asking patients about their recent driving provides valuable context to guide discussions and identify any concerning behaviors or events.

Table 55.4 Caregiver driving history questions

1. Does the patient drive?
2. Have you noticed any unsafe driving?
3. Do you feel uncomfortable riding with the patient?
4. Has the patient gotten lost?
5. Does the patient rely on a co-pilot?
6. Does the patient rely on passengers?
7. Do others worry about the patient's driving?
8. Does the patient forget where they are going?
9. Do others have to drive defensively?
10. Have others refused to ride with the patient?
11. Has the patient changed their driving habits?
12. In the past year, has the patient had any of the following? Please check all that apply.
Accidents
Fender-benders
Near-misses
Tickets
Discussions/warnings

Caregivers, often included in discussions about older patients' driving safety, can provide a useful second perspective on patients' driving.

Clinician's Guide to Assessing and Counseling Older Drivers questionnaire "Am I a Safe Driver?" as a written handout for self-evaluation prior to initiating an assessment.[23] There are additional questionnaires available that are recommended by expert consensus such as those available online from the Hartford Guide and the Alzheimer's Association (see Table 55.6). Some investigators have theorized that impairments in other higher-order IADLs that tap into executive function (e.g., finances, cooking) may be reasonable proxies for unsafe driving. There is some literature to support this notion.[32,33] However, it should be noted that, with the exception of the Driving Behavior Questionnaire,[34] few of these questionnaires have been validated in the literature as an effective method of identifying unsafe drivers. Driving history "red flags" that deserve further evaluation and follow-up[18] are listed in Table 55.5.

Table 55.5 Driving history red flags

Infrequent trips/low exposure
Recent (past 1–2 years) at-fault motor vehicle crashes
Gets lost in familiar places
Relies heavily on passenger "co-pilot" to instruct or provide essential information
Stopping without reason in roadway
Moving violations or warnings from police
Slow driving or complaints about speed/honks of other vehicles
Expressions of concern from family, caregivers, professionals
Increased fear or stress associated with driving

Patients who exhibit certain driving behaviors or events are cause for immediate concern, and require further examination to evaluate fitness to drive.

Step 3: Targeted physical examination to evaluate patient's visual, cognitive, and motor function.

In addition to the general physical exam maneuvers needed to investigate any positive history items found above, the Clinician's Guide to Assessing and Counseling Older Drivers recommends special items recorded on their Clinical Assessment of Driving-Related Skills/CADReS to evaluate vision, motor, and cognitive function.[23] Far visual acuity should be assessed with the traditional Snellen E chart, and visual fields can be checked by

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confrontation testing. If a Snellen chart is not available, a Rosenbaum pocket chart can be obtained or downloaded to a smartphone for checking near visual acuity. Motor function is gauged through assessments of walking, range of motion, and strength. The rapid-pace walk (RPW) is based on the time it takes the patient to walk a 10-foot path, turn around, and return, with a score of 10 seconds or longer scored as abnormal. Range of motion testing using normative tables and/or a goniometer and manual motor strength testing (using a 0–5 scale) are also assessed. The Timed Get Up and Go test is frequently done in Annual Wellness Visits, but little empirical research has been published about its association with driving outcomes. Findings are mixed, depending on the sample setting and driving outcome measured.[35]

Cognitive screening is performed in part using the Clock Drawing Test (CDT),[36] where the patient is verbally instructed to draw the face of a clock and to place the hands at 10 minutes past 11 using a blank sheet of paper and a pencil. Memory, visual-spatial skills, attention, and executive skills are some of the cognitive domains that are tapped during the CDT. More than two errors on the CDT is associated with unsafe driving behaviors based on driving simulation performance. The Trails-Making B (TMT-B) test is also recommended, with a time for performance over 180 seconds considered abnormal and meriting intervention. Patients are asked to connect dots in a path in sequence that alternates between numbers and letters, such as "1-A-2-B." Poor performance has been prospectively linked to crash risk in studies of older adults with dementia[37] and older adults during license renewal.[38] A recent review also noted that there was a strong association with TMT-B and road test performance, validating the 3 or 3 rule (180 second cutoff or three errors).[39] The TMT-B form, along with the CADReS and other tests, scoring sheets, and instructions, is available in the Clinician's Guide for Assessing and Counseling Older Drivers, which can be downloaded from the American Geriatrics Society website.[23] It should be understood that most clinicians would not determine fitness to drive based on brief psychometric screens but use that information to consider further referrals.

Next Steps: Education, Training, Referrals, and Resources

Driver (Patient) Education and Training

Driving self-assessment and education programs are widely available and useful as a prevention strategy to raise awareness and educate drivers on their current level of driving risk. The Driving Decisions Workbook is a self-assessment instrument designed to include medical content, and it has been studied against road test outcomes.[40] Health-care clinicians can recommend driving refresher courses, driving self-assessment, MyMobility Plan from the Centers for Disease Control and Prevention (CDC), and self-education programs that include the Automobile Association of America's (AAA's) Senior Driver program and the AARP Driver Safety Program (Table 55.2). A feature of the AAA program is the Roadwise Review,[41] a CD-ROM or online computer-based home assessment program that includes assessment of leg strength and general mobility, head/neck flexibility, high- and low-contrast visual acuity, working memory, visualization of missing information, visual search, and Useful Field of View (UFOV). AAA also offers a program to review medications that have the potential to impair driving.[42] All of these programs could benefit by further validation to determine their efficacy to reduce crash risk.

Referrals

Clinicians should try to minimize or compensate for the presence of impairments, so that the patient may continue to drive safely. Discontinuing sedating medications, identifying and treating obstructive sleep apnea, or improving muscle strength with physical therapy are potential interventions that could improve driving skills. Additional clinicians who may assist the primary care clinician in assessment of driving skills include ophthalmologists, neurologists, psychiatrists, neuropsychologists, and occupational therapists. If the patient's deficits cannot be medically corrected and do not have further potential for improvement with medical intervention, referral to a driver rehabilitation specialist (DRS) may be necessary.

A DRS is often an occupational therapist who undergoes additional training in driver rehabilitation. A DRS should be able to do either driving simulation and/or on-road performance-based testing to specifically determine the patient's level of driving safety. In addition, the DRS may be able to suggest adaptive equipment (e.g., spinner knob, enlarged side-view mirrors, hand controls, left-foot accelerator) or training techniques (e.g., enhanced visual search in patients with visual field cuts). A list of certified driver rehabilitation specialists can be obtained from the Association for Driver Rehabilitation Specialists or the American Occupational Therapy Association (AOTA).

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Patients and health-care providers need to be aware, however, that DRS services are often not covered by health insurance other than state Workers Compensation and Vocational Rehabilitation programs.

Also, DRS specialists are often located in urban areas, which may impede referral from rural locations. A local driving school referral or driver education specialist may not be the equivalent of a medical DRS specialist, but may be helpful in the absence of these resources.

Health-care clinicians may be called upon to document and verify the presence of impairment to help obtain access and financial support for adaptive equipment, driving rehabilitation, or restricted driving privileges. Table 55.6 lists resources for driving interventions and referrals.

Table 55.6 Older driver resources

1. *Consider driving (function) in the context of your patient with medical illness(es)*
   a. Clinicians Guide to Assessing and Counseling Older Drivers (4th edition) geriatricscareonline.org/ProductAbstract/clinicians-guide-to-assessing-and-counseling-older-drivers-4th-edition/B047
   b. CMA Fitness to Drive www.cma.ca/En/Pages/drivers-guide.aspx
   c. Austroads Fitness to Drive austroads.com.au/__data/assets/pdf_file/0022/104197/AP-G56-17_Assessing_fitness_to_drive_2016_amended_Aug2017.pdf
2. *For challenging cases, consider referring to a driving rehabilitation specialist*
   a. American Occupational Therapy Association www.aota.org/Practice/Productive-Aging/Driving/driving-specialists-directory-search.aspx
   b. Association of Driver Rehabilitation Specialists: ADED aded.net
3. *State Licensing and Reporting Guidelines*
   a. Know the laws in your state www.iihs.org/topics/older-drivers#driver-license-renewal
4. *Consider online web resources and/or office handouts*
   a. We Need to Talk and At the Crossroads www.thehartford.com/mature-market-excellence/publications-on-aging
   b. Alzheimer's Association: Dementia and Driving Resource Center www.alz.org/care/alzheimers-dementia-and-driving.asp
5. *Self-Help Assessment or Education*
   a. CDC MyMobility Plan www.cdc.gov/motorvehiclesafety/older_adult_drivers/mymobility/index.html
   b. AAA SeniorDriving Products seniordriving.aaa.com
   c. DriveSharp from Posit Science www.drivesharp.com/aaaf/index
   d. AARP Traffic Safety Course www.aarpdriversafety.org
   e. ADEPT Lifelong Driver www.adeptdriver.com/products/lifelong-driver
6. *Transportation Alternatives*
   a. Social Workers or Local Area on Aging acl.gov/help

Resources on older drivers are available for clinicians to learn more about assessing patient fitness to drive, as well as supporting older patients' mobility planning and transitioning from driver to nondriver.

Driving Cessation

If the patient must stop driving entirely – a circumstance termed driving retirement or cessation – clinicians should try to limit the adverse consequences for the patient if loss of the ability to drive cannot be prevented. Similar to work retirement, helping the patient to plan in advance by discussing the eventual need for driving cessation can minimize negative effects and facilitate a smooth transition. The multiple adverse consequences of driving cessation for older adults are well documented and include depression, dependency, caregiver strain, social withdrawal, increased risk of entry into long-term care facilities, and restricted mobility.[43]

The health-care clinician's recommendation for driving retirement should emphasize concern for the patient's safety and the safety of others as the primary reason for driving retirement. Even so, many patients are understandably upset or angry upon receiving the recommendation, and in the case of cognitive impairment, some may lack the insight necessary to understand the consequences. In addition to allowing adequate time for discussion, it may be helpful to reinforce the recommendation by asking the patient to repeat back to you the reasons for driving retirement, to provide a prescription on which "Do Not Drive" is written, and to help the patient create a plan for alternative transportation. Some patients may also benefit from identifying peer driver behaviors they consider to be unsafe, and using those examples to set their own threshold for when they would consider themselves unsafe to drive. Identifying a trusted friend or family member whose opinion is honored by the impaired driver may also help to support the recommendation. Keep in mind that a spouse who depends on the patient for transportation may find it difficult to support a recommendation for driving retirement. If the clinician does not have the time or expertise to address this important aspect, then referral to a social worker or gerontological care manager should be considered.

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Clinicians need to take steps when they believe a patient is medically unfit to drive. In mandatory reporting states, reporting guidelines should be followed without prejudice. Providers in other states must rely on their clinical judgment, state statutes, and preferably legal advice to determine if or when to report. A follow-up letter documenting the recommendation for driving retirement should be sent to the patient and – if the patient consents – to involved family members. A copy should also be kept in the chart for documentation.

Clinicians should attempt to evaluate their patients for whom driving is no longer possible soon after driving retirement, both to monitor for compliance with the recommendation to stop driving and to check for signs and symptoms of depression and anxiety. Extra care should be taken to assure that travel can be arranged for those who may have difficulty obtaining food, medications, and medical office visits. Finding alternative means of transportation is difficult for older adults in both rural and metropolitan settings that lack well-developed systems of mass transportation that can accommodate cognitively and/or physically frail older adults. The burden of meeting transportation needs will likely fall mostly on family, friends, and neighbors, some of whom may also suffer from undiagnosed driving impairment. Increased use of on-demand mobility such as Lyft, Uber, and Curb may be helpful to provide cost-effective rides in certain situations. GoGoGrandparent allows older adults to use Uber or Lyft without use of a cell phone, and rides are monitored. Social agency and volunteer organization referrals for meeting transportation needs are an important part of the patient care plan for driving retirement, often beginning with the Area Agency on Aging. For patients who lack capacity and insight, it is essential for the appointed guardian or caregiver to help the patient comply with the recommendation to stop driving. Many strategies have been employed, from placing reminder signage on doors to removing the vehicle altogether.

Legal and Ethical Concerns

The American Medical Association's (AMA's) Code of Medical Ethics Opinion 8.2 Regarding Impaired Drivers and Their Physicians[44] lists many of the issues faced by health-care clinicians who find themselves caring for older adult drivers. In particular, clinicians often find themselves in an ethical conflict between the standard of patient confidentiality and the duty to protect public safety. Many primary care clinicians may also be reluctant to report impaired drivers to their local driver's licensing authority for fear of jeopardizing their relationship with the patient. This concern must be weighed against public safety and state requirements for reporting unsafe drivers.

It is essential for clinicians to know and comply with their state's reporting laws and to document all activities in the patient's chart. All conversations and efforts to communicate with the patient and caregiver, recommendations, referrals for further testing, direct observations, counseling, formal assessment, medical interventions, patient education handouts, and referral reports related to your recommendations regarding driving should be clearly documented, and copies should be kept in the patient's chart for future reference and possible protection in the event of third-party litigation. Policies in clinics regarding referral to the state should include consideration of obtaining local legal advice. At a minimum, clinicians should document their fitness-to-drive discussions with the patient and, if the patient lacks decision-making capacity, a family member. The Insurance Institute of Highway Safety (IIHS[45]) keeps a website updated on the policies and laws of individual states in regard to older driver licensing (Table 55.6).

State laws for reporting potentially medically unsafe drivers can vary widely in terms of what conditions require mandatory reporting, the process for reporting or assessing a flagged driver, and protections for good-faith reporters. State motor vehicle websites are the best resources for information. However, the health-care clinician cannot ultimately suspend or remove a patient's right to drive. It is important to keep in mind that only the state Department of Motor Vehicles has the authority to perform a legal action regarding licensure.

Conclusions

There will be more older adult drivers over the next few decades, the majority of whom will be safe based on overall crash statistics. For the health-care clinician, fitness-to-drive evaluations capture the classic problem of the needs and priorities of the individual versus the needs and priorities of society. The clinician must help to negotiate a delicate balance between the two, which requires evidence-based assessment tools and steps in the evaluation and management process. By focusing on medical conditions, diagnoses, and/or medications that can impact vision, cognition, or motor control, clinicians can identify patients who may be at higher risk of impaired driving, regardless of age.

In addition to patient health information and asking about current driving behaviors, performance on paper-

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and-pencil clinical screens can gauge cognition, visual scanning, and motor function to inform next steps. Depending on the individual, the appropriate action may be no change to current driving behaviors, referrals for further testing or rehabilitation to improve on-road safety, reducing driving based on individual impairment and needs, or ceasing driving altogether. Regardless of outcome, beginning an ongoing conversation about driving and preparing for a potential nondriving future can normalize and ease older adults' transition from driver to former driver with minimal loss of community mobility.

In summary, the key points of this chapter are:

- Clinicians should consider driving as a key activity of daily living task and, similar to falls, realize that they can play an important role in the assessment and management of personal and public safety.
- There will be more older adult drivers in the coming years, some of whom will be medically impaired.
- Clinicians should focus on any visual, cognitive, and motor function deficits from any cause (including diseases, age-related decrements, illness, excessive sleepiness, and/or medication side effects) when making recommendations.
- There are evidence-based tools, including brief in-office screens, to help clinicians assess risk of driving impairment and identify appropriate next steps, including counseling, driving remediation, or retirement.
- Conversations about driving will ideally begin prior to concerns, and may be guided by self-assessments or driver education programs available in-person and online.
- There are other health professionals who may be able to assist in driving assessment and/or remediation including driving rehabilitation specialists for skills-based evaluation.
- Support and assistance preparing for and transitioning to former driver is pivotal, and may benefit from including social workers/gerontological care managers to assist with alternative transportation.

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Chapter 56

Integrative Medicine

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Introduction

Americans, including older adults, are increasingly using complementary and alternative medicine (CAM): therapeutic modalities, practices, and products that either supplement or substitute for conventional approaches and are not conventionally used or taught in mainstream Western medicine.[1–3] These modalities can be broadly grouped under the categories of mind–body therapies, manual/body-based therapies, biologically based therapies, and alternative medical systems (e.g., Ayurvedic medicine, homeopathy, naturopathic medicine, and traditional Chinese medicine), and include such practices as meditation, yoga, musculoskeletal manipulation, herbal medicine, acupuncture, nutrition, and energetic healing.[3–5] Increased use of CAM has paralleled burgeoning research showing safety and efficacy of many of these therapies.[5,6] Though mainstream medical education and health care have lagged in uptake of CAM education and practices, the public has forged ahead in combining CAM use with conventional care, often without communicating about this with their conventional care providers.[1,7] With upward trends in CAM use among all subgroups of aging populations, including the large cohort of aging baby boomers, the demand for CAM-savvy health-care providers is urgent and long overdue.[8]

Fortunately, increasing numbers of conventionally trained health-care providers and institutions are recognizing the value of becoming familiar with CAM philosophies, techniques, and practitioners, both for enhancing patient communication and for improving clinical practice. Many practitioners are taking steps toward postdoctoral education in functional or integrative medicine, allowing them to more knowledgeably integrate CAM into their practice.[9,10] Concurrently, the workforce of providers who are trained in CAM-oriented practices and philosophies is increasing – for example, licensed acupuncturists, naturopathic physicians, and chiropractic physicians – and many of their patients are older adults.

The terms "integrative medicine" or "integrative health care" refer to the selective incorporation of CAM diagnostic and healing approaches into mainstream health practices and systems.[5,9] Ideally, integrative medicine includes a recognition of the strengths of biomedicine as well as the benefits of holistic and natural healing approaches, bringing these together in individualized, patient-centered care that strengthens the patient's self-healing capabilities.[9] Integration of CAM approaches in geriatrics can benefit their well-being and health outcomes, throughout the continuum of care.

This chapter begins with a brief overview of the demographics of CAM use in the US aging population and describes consumers' rationales for use of CAM therapies, then reviews CAM modalities and uses for common conditions impacting health and function in the geriatric population. The chapter concludes with a discussion of roles that integrating CAM can play in enhancing health care of older adults, the challenges of integration, and the steps that conventional practitioners can take to successfully integrate such approaches into their practices.

CAM Use among Older People

In 2007, a national survey of CAM use confirmed the growing popularity of CAM modalities, with approximately 40% of all adults reporting use of one or more types of CAM in the last 12 months.[4] Subgroups of high CAM users included those with higher education and income, those with greater numbers of chronic conditions, and cultural subgroups such as Native Americans and Native Hawaiians. Older people made up a substantial proportion of CAM users, with 41% of those aged 60–69, and 32% of those aged 70–84, reporting

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CAM use in the last 12 months.[4] The more recent 2012 survey showed similar results, with little change in CAM use for older adults.[3] The most recent 2017 national survey, which focused on the use of specific CAM practices not targeted in other large national surveys, found increases in CAM use from 2012 to 2017 for yoga (from 9.5% to 14.3%), meditation (from 4.1% to 14.2%), and chiropractic (from 9.1% to 10.3%).[11] Although yoga was most popular among young adults (aged 18–44), 12.2% of mid-aged adults (aged 45–64) and 6.7% of those 65 and older practiced yoga. Meditation, while most popular with mid-aged adults, was almost equally popular with older adults (15.9% vs. 13.4%, respectively). Chiropractic was used almost equally by young, mid-age, and older adults (9.9% vs. 11.4% vs. 9.5%). CAM use will likely continue rising in parallel with the aging of the baby boomer cohort, fueled by CAM's growing popularity and availability in the USA.

Why Are Consumers Using CAM?

Growing global consciousness and information access have brought heightened awareness and acceptance of other systems of health care as well as increased knowledge of CAM options. A primary motive for CAM use is philosophical, and includes congruence with holistic beliefs, attraction to natural/organic products and treatments, recognition of the value of preventive care, and motivation to treat the source of illness rather than merely alleviating symptoms. For example, in one national survey, those who agreed with the statement that "the health of my body, mind and spirit are related, and whoever cares for my health should take that into account" were more likely to use CAM (46%) than those who did not endorse this item (33%).[12] Population subgroups whose health beliefs and philosophies differ from those of mainstream medicine, whether or not they are cultural minorities, have long been major users of traditional or alternative therapies.[13,14]

Common reasons given for utilizing CAM therapies are the desire to prevent illness or maximize well-being. For example, there is a growing popularity of mind–body approaches such as meditation and yoga for managing stress. Another common rationale is to provide a nonpharmaceutical or nonsurgical alternative for particular conditions. For example, a patient may choose to prevent the development or progression of type 2 diabetes by modifying diet, adding a mindful exercise regimen, and managing stress. An American Association of Retired Persons (AARP) survey of CAM use by people aged 50 and older, in which 53% reported CAM use, found that for CAM users, 66% used CAM to treat a specific condition, 65% for overall wellness, 45% to complement conventional medicine, and 42% to prevent illness.[15]

Those who seek to recover from life-threatening diseases are often highly motivated to use CAM treatments, usually as complements to conventional care. For example, the 2012 National Health Interview Survey (NHIS) found that 33% of patients with a history of cancer (median age 66 years) reported CAM use in the past 12 months, with the most commonly used CAM modality being herbal supplements (35.8%), followed by chiropractic or osteopathic manipulation (25.4%), massage (14.1%), yoga/tai chi/qigong (7.6%), mantra/mindfulness/spiritual meditation (6.9%), and smaller percentages of other CAM modalities.[16] Those who suffer from chronic diseases, including those with symptoms that are either poorly understood, treated with only partial success by conventional medicine, or whose treatments include unwanted side effects, often investigate CAM treatments. Additionally, high CAM users include those wishing to enhance the likelihood of success of a particular health-care intervention. For example, patients undergoing surgery may choose to incorporate certain CAM therapies, such as herbs, hypnosis, visualization, homeopathic remedies, or energetic therapies.

Finally, use of CAM may be motivated by the desire to have control over health matters, for example seeking home remedies or over-the-counter solutions. Lower costs may also be a motivating factor. One study found that those who use mainly alternative forms of health care may do so because they distrust conventional providers, settings, systems of care, or treatments, and place a high value on their inner life and experiences, as well as control over their own health.[12] These values and attitudes are supported by many alternative care providers and are in alignment with the philosophies undergirding their treatments. Providers of integrative medicine, to varying degrees, are also supportive of this philosophical perspective.

Patient–Provider Disconnect about CAM in Conventional Health Care

One striking finding in research on use of complementary modalities in the USA is that 95% of patients who use alternative care also use conventional care, often for the same condition, and even more surprisingly, the vast majority of patients – up to 70% – do not tell their conventional practitioners about their use of alternative

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modalities.[1,7,16] Reasons given for such nondisclosure include the belief that physicians would not understand or be knowledgeable about the therapy; that they are not asked; fear of disrespect or disapproval; and their belief that the physician is uninterested.[1] An analysis of 2012 NHIS data, from adults who had a primary care physician and had also used CAM in the last 12 months, found that 42.3% did not disclose the use of their most used CAM modality.[7] Highest nondisclosure was for those using yoga and meditation (64.7% and 64%, respectively), with lower nondisclosure for users of acupuncture (35.5%) and herbal medicine and/or supplements (24.9%). In most cases, nondisclosure was due to physicians' failure to ask about CAM use (46.2%). This lack of communication may inhibit coordination of care, as well as result in increased medical errors, noncompliance, possible duplication of services, greater health-care costs, and undocumented outcomes.

Integrative medicine offers a patient-centered approach to care that recognizes the need to understand and respect the patient's beliefs and values, thereby enhancing trust, cooperation in care, and skillful utilization of the powerful role that beliefs play in illness and health.[17] Continuity and quality of care is enhanced when health professionals can communicate knowledgeably and coordinate effectively with patients and other members of the patient's health-care team, who may include naturopathic doctors, chiropractic doctors, acupuncturists, massage therapists, or homeopaths. CAM education as well as an integrative health-care perspective, whether through an individual provider or a health-care system, could facilitate such communication.

Overview of CAM and Use for Specific Conditions

The following is a brief overview of CAM therapies in use in the USA, with examples of those shown to have therapeutic value in the care of older people. Texts and in-depth reviews of CAM therapies and research provide much greater detail.[10,18–21]

Mind–Body Therapies

Mind–body therapies incorporate an understanding of the interaction of cognitive and emotional processes with the body organ systems, and the underlying psychobiological mechanisms by which communication occurs, including immune and neurotransmitter substances. Examples of mind–body therapies are biofeedback, hypnosis, guided imagery, mindfulness meditation, and various forms of mindful exercise.

Biofeedback

Through training in biofeedback and a subset, neurofeedback, patients can learn to modify their own vital functions (such as breathing, skin temperature, heart rate, or even electroencephalogram [EEG] brain waves), thus preventing, controlling, and treating a range of syndromes, including back and neck pain, pelvic floor dysfunction, difficulty swallowing, migraine and tension headaches, asthma, stress-related symptoms, hypertension, and palpitations related to arrhythmias.[22–26]

Hypnosis and Guided Imagery

Hypnosis uses the power of suggestion to induce trance-like states that access deep, often unconscious, levels of the mind to effect positive behavioral change. It has been used effectively to treat acute and chronic pain, sleep disorders, anxiety, phobias, symptoms of asthma, irritable bowel syndrome, and other conditions, and may be a safe alternative to pharmaceutical medicine.[27,28] Guided imagery involves auditory suggestions, either given by another individual or through a recording device, for relaxation and stress management. For example, imagining being on a beach or another favorite, safe place can facilitate a peaceful state of mind. Guided imagery is often incorporated into hypnotic inductions. Two important uses of hypnosis and guided imagery in older adults are for surgical anesthesia (either as a substitute or supplement for conventional anesthesia) and for pain control.[29]

Mindfulness Meditation

Meditation has been used and taught since its roots in ancient India and is widely popular in the USA. In mindfulness meditation, the practitioner usually focuses on an image, a sound, or simply breathing as an anchor to being in the present moment. They are taught to observe whenever the mind becomes distracted by thoughts of past or future, gently let go of the distracting thought, and bring the attention back to the present moment. Training in mindfulness can help manage stress, enhance emotional regulation, and improve outcomes for those with chronic pain conditions, as well as enhance immune function and decrease anxiety and hypertension. Mindfulness-training programs can be adapted for older groups or individuals and can easily be taught online or via telephone.[30–37]

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Mindful Exercises

Mindful exercises include such practices as yoga, tai chi, qigong, Alexander Technique, Pilates, and Feldenkrais, some thousands of years old and others developed only recently. These practices are adaptable for use by older people in varying states of health and can be used to improve mental and physical function. For example, tai chi has been found to improve sleep and reduce pain in people suffering from osteoarthritis, and enhance balance, reduce falls, and improve symptoms of Parkinson's disease.[38–42] Yoga has been adapted for older people with varying degrees of fitness, including wheelchair-bound elders, and has been shown to improve risk factors for heart disease, diabetes, side effects of cancer, and musculoskeletal pain.[19,21,43–45]

Manual/Body-Based Therapies

Manual and body-based therapies range from traditional to contemporary techniques, usually performed by a skilled practitioner, emphasizing physical touch, manual manipulation of tissues or energetic systems, and often involving movement. Examples are chiropractic (described under Alternative Healing Systems), massage, and other forms of bodywork (such as reflexology, functional integration, structural integration, and kinesiology), as well as various touch therapies.[10,18–21]

Massage and Bodywork

Massage and bodywork involve many subcategories and techniques and can easily be individualized based on clients' needs and preferences. Massage can promote relaxation, relieve muscle pain and headaches, and alleviate a range of stress-related conditions, lessening the need for pharmacological therapies.[46–55] Massage can reduce swelling and increase lymphatic circulation, thus alleviating chronic inflammatory conditions, facilitating the removal of toxins from the body, and enhancing recovery from illness, and has been used effectively in hospitals, nursing homes, and hospice settings.[48,56–65]

Touch Therapies

Touch therapies are energy-based healing systems that include laying on of hands, Reiki, and therapeutic touch. In laying on of hands, an ancient art found in various spiritual traditions, the practitioner directs healing energy, purported to come from a universal force, to the patient, or to the site of illness. Reiki, which traces its origins from Tibet, is one form of this therapy. Therapeutic touch, another variation, was developed by Dolores Krieger, PhD, RN, and Dora Kunz, a healer, and is now being used in US hospitals, particularly by the nursing profession. Therapeutic touch may be useful in reducing pain and anxiety and promoting healing and can be particularly applicable to those needing gentle loving care.[19,66–70]

Biologically Based Therapies

Biologically based therapies encompass a diverse array of approaches including dietary therapies, herbal medicines, and dietary supplements, and they comprise the most popular CAM category used in the USA.[3,4] Although many of these are utilized by conventional medicine, they are much more central to the care plans of CAM practitioners such as naturopathic physicians, chiropractic physicians, and homeopathic practitioners, as well as integrative medicine providers, such as physicians trained in functional medicine.[10,18] Examples of biologically based therapies include the following.

Dietary Therapies

Conventional and alternative medicine agree on the beneficial effects of a diet high in fresh fruits and vegetables and the protective effects of phytochemicals, but beyond that, there are many variations in diet espoused both within conventional medicine and within CAM.[71–73] For example, Ayurvedic medicine practitioners make dietary recommendations based on one's body constitutional type, called prakriti, and traditional Chinese medicine practitioners prescribe diets based on both body constitution and illness characteristics.[19,74] CAM therapies and integrative health care are generally allied with the natural foods movement, which emphasizes the importance of whole, unprocessed, unrefined, organic foods, and the elimination of food products containing pesticides, antibiotics, hormones, refined sugars, and food additives (e.g., preservatives, dyes, and artificial flavors) – substances that may play a role in decreased immune function, increased food allergies, increased chemical sensitivities, and other disorders.[75,76] Individualized evidence-based nutritional prescriptions that take into account the health status and preferences of the older person are essential, and can be helpful in preventing disease, slowing disease progression, restoring function, and controlling pain.[10,18]

A variety of nutritional approaches has been recommended for preventing and treating illness and promoting healing, with varying levels of evidence-based research for their effectiveness. Data from the 2017–18

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National Health and Nutrition Examination Survey (NHANES) reported the prevalence of hypertension as 54.5% for adults aged 40–59 and 74.5% for adults aged 60 and over.[77] Hypertension increases the risk for heart disease and stroke, two of the leading causes of death in the USA.[78] The DASH diet, an acronym for "Dietary Approaches to Stop Hypertension," emphasizes a dietary pattern high in fruits, vegetables, whole grains, and legumes, and low in both saturated and total fat, allowing limited amounts of low-fat dairy and lean meats. The DASH diet has been shown in large randomized controlled trials to effectively lower blood pressure in hypertensive, prehypertensive, and normotensive adults.[79,80] The DASH diet, combined with weight management, was also shown in a randomized controlled trial (RCT), the ENCORE trial, to significantly improve executive functioning, memory, and learning in hypertensive overweight adults compared to the control diet.[81]

Heart disease, the leading cause of death in the USA, claims the lives of over 650,000 US adults each year and has been linked for decades to diets high in saturated fat, trans fat, and cholesterol (found only in animal foods).[78,81] In the Lifestyle Heart Trial, published in the *Lancet* in 1990, Ornish and colleagues randomized participants to usual care or an experimental, comprehensive lifestyle modification program including a low-fat vegetarian diet emphasizing whole-plant foods, smoking cessation, stress management, and moderate exercise.[82] After 1 year, over 80% of participants in the experimental lifestyle change group demonstrated regression of coronary artery stenosis by coronary artery angiography, compared to control participants, who on average demonstrated progression of stenotic lesions.[82] Currently, another RCT by Ornish and colleagues is underway investigating comprehensive lifestyle medicine consisting of a whole-food, low-fat, vegan diet (21 meals per week supplied), nutritional supplements (multivitamin, fish oil, curcumin, vitamin C, vitamin B12, CoQ10, lion's mane, probiotic, and magnesium), exercise, stress management, and group support, in the treatment of early Alzheimer's disease (NCT04606420).

Cancer is the second leading cause of death in the USA, claiming the lives of approximately 600,000 Americans every year, with 1,701,315 new cancer cases diagnosed in 2017.[83] The highest rates for new cancer cases include cancers of the female breast, prostate, lung, colon, and rectum.[83] Similar to the Lifestyle Heart Trial described above, a comprehensive diet and lifestyle change program including a whole-food-vegan diet, nutritional supplements (soy protein, fish oil, vitamin E, selenium, and vitamin C), exercise, stress management, and group support was also shown to be effective for men with biopsy-proven, early-stage, low-risk, prostate cancer in an RCT.[84] Participants randomized to the lifestyle intervention were also found after 5 years of follow-up to have increased telomere length and decreased telomerase activity (both markers of aging, disease, and premature morbidity) compared to external controls.[85]

Over the past century, various dietary approaches to cancer have been advocated by research scientists, healthcare practitioners, and laypeople.[86] There is substantial observational data on nutrition and cancer in the scientific literature and certainly no shortage of websites, videos, and books filled with testimonials of various dietary approaches for treating cancer, including the BHP (Bill Henderson Protocol), Budwig Diet, Gerson Diet, Hoxsey Herbal Therapy, ketogenic diet, and various metabolic approaches.[86] It is beyond the scope of this chapter to summarize these dietary approaches, and further reading is recommended for practitioners who wish to become familiar with alternative approaches, the rationale behind those recommendations, and, importantly, what nutrient and energy deficiencies may result from adherence to various dietary protocols.[86] Zick, Snyder, and Abrams reviewed both negative and positive aspects of popular diets used by cancer patients, including a clinically useful table of potential nutrient deficiencies associated with five of the most popular dietary patterns (ketogenic, macrobiotic, paleolithic, vegan, and alkaline), along with nutritional counseling tips to address these concerns with patients.[87] Leading organizations like the American Institute for Cancer Research publish evidence-based guidelines on nutrition and cancer prevention and survival and can be consulted for evidence-based recommendations.[88]

There are many ways to classify various dietary approaches: broadly speaking, dietary patterns can be divided into four main categories; (1) omnivorous – includes plant-based foods and animal products (meat, eggs, cheese, and dairy); (2) pesco-vegetarian – includes plant-based foods plus fish and seafood; (3) vegetarian – includes plant-based foods plus eggs, cheese, and dairy; and (4) vegan – includes plant-based foods only and requires vitamin B12 supplementation. Beyond this, there are numerous variations that may emphasize inclusion or elimination of specific foods, food groups, or encourage specific dietary patterns (e.g., Mediterranean, DASH, MIND, paleo, keto, autoimmune-paleo, etc.). In regard to the top two leading causes of death, heart disease and cancer, evidence from a 2011 meta-analysis

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of seven prospective cohort studies totaling 124,706 participants, comparing vegetarian and nonvegetarian diets, found that vegetarians had significantly lower mortality from ischemic heart disease (29%) and 18% lower incidence of cancer.[89] Health-care practitioners are additionally encouraged to become familiar with popular fad diets to facilitate open communication with patients and to kept abreast of the peer-reviewed scientific nutrition literature.

Fasting is a time-tested ritual, used for centuries by a variety of cultures for both religious and healing purposes, such as to purge the body of endogenous and exogenous toxic substances, support immune function, and increase spiritual awareness and self-control. More recently, intermittent fasting (e.g., 6-hour eating window, 18-hour fast over 24 hours) has gained attention in the medical community and in popular culture as a growing body of evidence suggests it may confer neuroprotective and anti-aging effects while supporting metabolic health, stress resistance, and weight control.[90] Extreme fasts, which may involve drinking only pure water, may not be safe for people with underlying medical conditions and should only be done under medical supervision. Modified juice fasts include fresh organic vegetable juices and sometimes fruit juices.

Herbal Medicines and Nutraceuticals

Using herbs and other plants as remedies can be traced back to prehistoric times; they are still the predominant form of medicine for 80% of the world's population. Often gathered from surrounding environments and prepared by knowledgeable healers and laypeople, these medicines have long been used as tonics for preventing illness and as remedies for most functional disorders known to humankind (e.g., dyspepsia, respiratory disorders, menstrual disorders, anxiety, and depression), as well as for more serious organic disorders such as cancer. Until the late nineteenth century, physicians prescribed herbal preparations extensively, but with the growth of manufactured drugs, the medical profession's knowledge and use of herbs declined and practically ceased in the USA.

Meanwhile, since the 1970s there has been a major resurgence of interest and use of herbals and a growing consumer awareness of the roles played by various nutraceuticals in health and illness. Popular interest is reflected in market trends, with the dietary supplement industry projected to reach 56 billion dollars annually by 2024.[91] Fresh and dried herbs and a wide variety of nutraceuticals are available in health food stores, and many nutraceutical products can be purchased over the counter in pharmacies and online. CAM practitioners and integrative care providers, as well as many pharmacists, have been trained to advise on consumption of these products, including their interactions with pharmaceuticals, but conventional medical curricula continue to lag behind in education and training regarding uses and drug interactions.[92] Although effective use of many herbal preparations has been established by tradition, increasingly, randomized controlled clinical trials are showing efficacy of certain herbal preparations in specific conditions. For example, research has established the efficacy of St. John's Wort for mild to moderate depression.[93] With the widespread use of herbs and supplements for self-care among the elderly, it is essential that care providers facilitate good, open communication with their patients about their use.[94] As noted above, the majority of older persons do not communicate about CAM use with their providers.[15] Moreover, in one study, 80% of hospitalized patients reported use of a dietary supplement, with 52% reporting use of nonvitamin and nonmineral dietary supplements, while inquiry by providers about dietary supplement use was documented only 20% of the time.[95] It is essential that care providers incorporate knowledge about herbals and nutraceuticals into their continuing medical education to avoid harm and optimize care. There are excellent online sources to consult, such as the Natural Medicines Comprehensive Database, to stay abreast of the ever-changing science.[96]

Aromatherapy – Use of Essential Oils

Plant essences, including essential oils, have been used therapeutically for thousands of years in numerous cultures. "Aromatherapy," a term coined in 1937 for this ancient practice by the French chemist Rene Maurice Gattefosse, has been used for the treatment of such conditions as immune deficiency, bacterial and viral infections, and skin disorders, and as a tool for stress management.[19] The oils transmit their healing properties not only by inhalation but also by absorption through the skin, exerting much of their effect through their pharmacological properties and small molecular size. Aromatic molecules interacting with cells of the nasal mucosa transmit signals to the limbic system through which they connect with the parts of the brain controlling heart rate, blood pressure, breathing, memory, and hormone balance.[97] Research has shown that inhalation of particular essential oils can have either a calming or stimulatory effect on brain waves.[98] Aromatherapy, often combined with massage, has been used for pain,

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dementia symptoms, and anxiety in older patients in hospitals, nursing homes, and hospice.[99,100]

Alternative Healing Systems

Traditional Chinese Medicine

Acupuncture and acupressure are part of a complete system of healing developed in China over 5,000 years ago. Variations in philosophy and techniques have developed both within China and in other East Asian cultures such as Korea and Japan. In this system, health is believed to be dependent on the balanced flow of chi or qi, the vital life energy, throughout the body, and illness is due to a disturbance of chi. Acupuncture treatment balances the chi by inserting needles at points on the body where the chi flows through one of 12 channels or meridians. Diagnosis involves inspection (visual assessment of the patient, particularly the spirit, form, and bearing), feeling the pulse, observing the tongue and eyes, and questioning the patient about physical and social environment. Acupuncture treatment can be useful for pain management (e.g., back pain, knee pain, and migraine) and treating cancer-related symptoms (including nausea and vomiting), substance abuse, depression, dementia symptoms, and stroke.[19,101–104]

Homeopathic Medicine

Samuel Hahnemann, a German physician disheartened with the medical practice of the early 1800s, formally tested the ancient healing principle *similia similibus curentur* – "like is cured by like" – and subsequently established it as the basis of a system of medicine.[105] Central to homeopathic therapeutics is the infinitesimal dose, the smallest dose necessary to produce a healing response. Through experimenting with lower and lower doses of drugs in efforts to minimize side effects, Hahnemann developed a technique called potentization, in which the original substance is repeatedly diluted and successed (shaken vigorously after each dilution) to produce a medicinal substance diluted in many cases beyond Avogadro's number ($6.23 \times 10–23$), the point at which there is unlikely to be a single original molecule left.

Homeopathic treatment involves selection of a homeopathic preparation that produces symptoms in a healthy person similar to that of the patient's complete symptom picture. Even for acute conditions, prescribing takes into account the individualized response to illness. Particularly in chronic conditions, it may incorporate the person's entire symptomatology not only with regard to the present complaint, but over the course of a lifetime, a process termed constitutional prescribing. Although the mechanism by which the similar remedy acts is unknown, homeopathic theory maintains that the energetic imprint of information is somehow transmitted to the organism, stimulating innate healing capacities. Symptoms are viewed as the organism's expression of its life energy, and care is taken not to suppress them, but to use them to guide healing.

Homeopathy may be a useful and gentle healing modality for a wide range of acute and chronic ailments found in older people, including respiratory infections, allergies, insomnia, gastric upsets, fatigue, prolonged grief, anxiety, depression, and palliative care, especially when applied by skilled practitioners.[106–108]

Naturopathic Medicine

In 1885, Dr. John Scheel, a German homeopath practicing in New York, coined the term "naturopathy" to describe the natural healing methods such as diet, herbs, and hydrotherapy developed by German healers like Father Sebastian Kneipp.[109] The American School of Naturopathy was founded in New York City by Benedict Lust in 1900, and the naturopathic profession grew rapidly during the first quarter of the twentieth century.[109] A resurgence of interest in naturopathic medicine occurred in the 1970s, leading to tremendous growth and maturation of the naturopathic profession in North America over the ensuing decades.[110] Today the naturopathic profession is regulated in 23 of 50 US states and five Canadian provinces, where naturopathic doctors are required to have graduated from one of six accredited 4-year post-baccalaureate residential naturopathic medical schools and to have passed the NPLEX (Naturopathic Physician Licensing Examination) in order to obtain a license to practice naturopathic medicine.[111,112] Approximately 6,000 licensed naturopathic doctors are currently practicing in the USA.[112]

Naturopathic medicine is a distinct primary health-care profession that emphasizes prevention and the self-healing capability of the individual. Naturopathic physicians focus on identifying and removing obstacles to healing and treating the underlying causes of illness rather than merely eliminating symptoms. Naturopathic medicine is guided by six principles: Do No Harm, Identify And Treat The Causes, Treat The Whole Person, Focus On Prevention, Physician As Teacher, and The Healing Power Of Nature.[109] Naturopathic treatment is further guided by a therapeutic order: this can be visualized as a pyramid, where establishing the conditions for optimal health is at

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the base, and surgery is at the top; emphasis is placed on using the least force necessary to address the underlying causes of illness and resolve symptoms.[109]

Naturopathic physicians strive to use the gentlest healing methods possible, educating their patients and encouraging the patient's responsibility. The healing methods emphasize treating the whole person, including physical, mental, emotional, genetic, environmental, social, and spiritual factors. Therapeutic modalities include nutritional and botanical medicine, homeopathy, hydrotherapy, physical medicine, behavior change, pharmaceuticals, and minor surgery.[18,19] A 2019 scoping review of naturopathic medicine as a whole-system multimodality intervention, which included 33 published studies (n = 9859), concluded that naturopathic medicine is effective for treating a wide range of chronic conditions, including depression, anxiety, cardiovascular disease, type 2 diabetes, and musculoskeletal pain.[113] Interviews with older people who utilized naturopathic doctors found that patients sought naturopathic medical care because it was aligned with their values, including an emphasis on prevention, self-care, and healthy aging.[114]

Chiropractic Medicine

Chiropractic, founded in 1895 in Iowa by D. D. Palmer, a self-educated healer, is based on the understanding that structural distortions can cause functional abnormalities.[19] Vertebral subluxation of the spine is an important structural distortion that disturbs body function primarily through neurologic pathways. Chiropractic adjustment is a specific and definitive system for correcting vertebral subluxation, harmonizing neuronal function and stimulating the body's innate healing potential, focusing primarily on manual adjustment or manipulation of the spine. Chiropractic is the third largest independent health profession in the Western world, following allopathic medicine and dentistry, with more than 70,000 licensed practitioners in the USA trained in 4-year post-baccalaureate programs and seeing over 30 million patients per year.[19] Patients visit chiropractic physicians particularly for the prevention and treatment of neuromusculoskeletal conditions of low back pain, neck pain, and headaches.[19,115–117]

Prevalence of chiropractic use among Medicare beneficiaries aged 70 and older ranges from 4.1% to 5.4%; in younger beneficiaries it ranges from 6% to 7%.[115] Research comparing outcomes between users of chiropractic and users of medical care for treatment of uncomplicated back care suggests that chiropractic care may provide a protective effect on decline in function and self-rated health, as well as providing higher satisfaction with follow-up care.[116]

Spiritual Healing Practices

Spirituality is a powerful component of the healing process and is particularly important in the care of older persons.[118–121] Faith healing is one longstanding spiritual tradition, and often combines prayer and laying on of hands.[122,123] Shamanism, one of the oldest healing traditions known to humans, involves individuals trained to work with natural and spiritual energies, so as to diagnose illnesses, perform healing rituals, and communicate with the spirit world.[19,124] Recently in the USA, there has been a revival of the study of shamanic rituals and journeying for those needing spiritual healing.[124]

Rationales for Integrating CAM Therapies into the Care of Older People

There are several important reasons described below to consider incorporating the use of complementary therapies into the care of older individuals and patient populations.

Expanded Options for Patient Care

An integrative clinical practice provides an expanded array of health-care options when compared with conventional care. Although therapies such as prescription drugs may effectively address a particular condition, not every individual will respond well to a particular protocol. As well, many older people prefer and do equally well with nonpharmaceutical options. For example, for mild to moderate depression, an integrative practitioner might suggest aerobic exercise, nutrition, acupuncture, and/or botanicals as an alternative or complement to an antidepressant.[125–127] Management of hypertension might include a combination of diet modification, a mind–body therapy (e.g., hypnosis, biofeedback, tai chi, or mindfulness), and medication.[47,79,80,128] Patient choice and motivation are central in an integrative model of care.

Enhanced Patient and Provider Communication and Satisfaction

CAM practices, while varied, in general share a holistic perspective on healing, one that emphasizes an individualized approach to diagnosis and a patient-centered approach to treatment. Although many health practitioners spend ample

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time with patients and provide a multifactorial assessment, it is often the disease, rather than the person, that guides the approach to treatment. CAM and integrative care providers generally spend more time getting to know the patient's individual needs, values, and desires, as well as motivations. Providing a patient-centered approach to diagnosis and treatment may improve both patient and caregiver satisfaction.

Decreasing Dependency on and Overuse of Pharmacological Therapies

Increased use – and misuse – of pharmaceuticals is a significant motivator for integrating CAM with conventional medicine, particularly in caring for older persons. With the proliferation of medical specialties, each with its own cadre of medicines, polypharmacy and adverse drug interactions have become the rule rather than the exception, particularly for older patients with multimorbidity.[129–131] Substituting a CAM treatment for a pharmaceutical may be equivalent or perhaps do less harm. For example, one systematic review that examined multiple head-to-head trials of complementary therapies (e.g., acupuncture, St. John's wort, and exercise) versus antidepressants for major depressive disorder found no differences between most treatment groups in terms of response and remission.[132] In some instances, substituting nonpharmacologic therapies may reduce potential negative side effects, such as iatrogenic effects of multiple medications and the potential for drug dependency, while maintaining positive health outcomes.[125,126]

Enhancing Health-Care Outcomes

Judicious combining of conventional and complementary treatments often produces better outcomes than conventional therapies alone, particularly when outcomes include reduction of negative side effects of treatment. Such synergies may offer a variety of benefits: for example, adding massage, hypnosis, or guided imagery during surgical procedures may not only decrease anxiety, but also decrease reliance on medications, therefore lowering procedural risks due to anesthesia, and enhancing positive health outcomes.[29,133,134]

Added Emphasis on Disease Prevention, Wellness, and Self-Care

CAM and integrative health care's therapeutic emphasis on prevention, wellness, and self-care is attractive to many older people and may lead to better health habits and enhanced self-efficacy. For example, dietary management and mindful exercise – part of the regimens of many CAM systems – require self-determination, learning of new skills, and changing habitual patterns of behavior. The philosophy of many alternative systems of healing encourages continual personal growth and development through self-education (e.g., reading books and articles, listening to recordings and podcasts, attending workshops and seminars) that emphasize stress management and optimizing well-being through self-care.

Approaches to Education, Training, and Practice in Integrative Health Care

For the conventional practitioner, becoming educated about integrative health care may begin with purchasing a textbook, or taking an introductory course, to enhance providers' comfort in talking with patients about their CAM use. Becoming familiar with, and perhaps networking with, local CAM providers is a second step toward integration. Later, the practitioner may be inspired to acquire specific knowledge and skills about one or more complementary and alternative modalities to enhance self-care and add to their practice toolkit. There are many opportunities available for online and in-person learning and certifications. Depending on interest and motivation, the provider may investigate the variety of possible models of integrative health-care delivery, from solo provider, to referral networks with CAM providers, to more complex models such as multidisciplinary or interdisciplinary team practices. Joining state and national organizations can further enhance knowledge, skills, motivation, and capabilities for adapting to the culture of integrative health care.

The Challenges and Promise of Integrative Medicine in the Care of the Elderly

With considerable interest on the part of health-care consumers and many practitioners, and CAM's perceived and documented benefits, CAM integration with mainstream medicine is occurring steadily, albeit slowly. Barriers include limited third-party insurance coverage, limited CAM education in health professions curricula, and the slow dissemination of clinical outcomes about complementary and alternative therapies.

For the conventional practitioner who treats older people, the most compelling rationale for moving toward integrative health care may be their patients' growing use

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and demand for CAM and integrative health care. Since many patients are not discussing CAM with their providers, it becomes a matter of professional responsibility to become more conversant about CAM options. That includes staying up to date with the burgeoning medical literature on CAM efficacy and comparative effectiveness with conventional medicine. In many cases, it is simply "good medicine" for older patients – particularly when use of a CAM therapy promotes self-care, preventive self-maintenance, lower health-care costs, or reduction in inappropriate use of pharmaceutical medicine.

Finally, joining or leading an integrative medicine team may bring with it the personal and professional satisfaction that many providers of integrative health care enjoy, as they care for their patients in a patient-centered, humanistic atmosphere that emphasizes healing of mind, body, and spirit while simultaneously enhancing care.

An integrative approach to caring for the elderly throughout the continuum of care could be a win-win-win solution – for the older patient, the care provider, and the health-care system.

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Chapter 57

Retirement
A Contemporary Perspective

Kimberly A. Skarupski

Retirement means different things to different people. Retirement also looks different depending on the complex interplay among diverse factors, including: physical, cognitive, and emotional health and social well-being; various sociodemographic factors such as age, sex, race and ethnicity, education, household income, and marital status; and still other factors such as number of living children and grandchildren, living arrangement, geographic region, religion, and others. Some patients may be enthusiastically counting the days until they are able to retire from their vocation ("I can't wait to retire!"). Some may proclaim that they will never quit working ("I'm never going to retire!"), and yet others cannot retire ("I can't afford to retire!"). Regardless of how people think or feel about retirement, departure from full-time employment is a pivotal life event.

Perspectives of Retirement

Retirement is personal. A health-care provider who has a general understanding of her/his patient's life history, who has built a trusting relationship with the patient, and who engages the patient in meaningful dialogue may begin to understand how the patient is managing the myriad complexities of the retirement transition. An awareness of various retirement theoretical approaches and perspectives may help frame a conversation.

The life-course perspective views retirement from the individual's immediate social context as well as their sociocultural milieu or generational cohort.[1] Modernization theory posits that as a culture becomes more modern, the social value of older adults declines commensurately. Furthermore, modernization theory also observes that geographically dispersed families add to retirement location decisions, social support, and caregiving challenges.[2] Activity theory states that people who are more active and have more role engagement are more satisfied with life.[3] On the contrary, disengagement theory argues that retirement is a natural and functional process whereby older adults and society mutually separate from each other, both purposefully withdrawing.[4] Continuity theory observes that older adults desire to maintain their lifelong habits and levels of activity and role engagement.[5] Optimization with compensation theory uses a cost-benefit approach, positing that older adults strive to maximize resources and minimize losses; for example, downsizing housing and traveling more.[6] Similarly, socioemotional selectivity theory focuses on shifting attention to fewer, more intimate social networks, and away from larger, less intimate social networks.[7] Role theory stresses the transitions from various roles in life; for example, similarities and differences from the employee role to the retiree role.[8]

Retirement Disrupted

The world's population is rapidly aging. According to the US Census Bureau, by 2030, the proportion of adults in the United States aged 65 and older will be 21% compared to 16% in 2018,[9] and the life expectancy of adults aged 65+ is also increasing. An average American woman at age 65 is expected to live another 20.5 years and a man aged 65 another 18 years.[10] Age 65 was designated, somewhat arbitrarily, as the eligibility age for Social Security and Medicare welfare benefits in the Social Security Act of 1935 and subsequent amendment in 1965. Thus, age 65 was historically seen as the age when one retires from full-time employment. However, based on data from the Organisation for Economic Co-operation and Development (OECD), the average ages of retirement in the USA in 2018 were 67.9 for men and 66.5 for women.[11] The US Census Bureau, 2016 American Community Survey estimated that among those aged 65 and older, 21.9% of men and 14.2% of women were in the labor force. For those aged 65 to 74, 29.8% of men and 21.8% of women were in the labor force. Among those aged 75 to 84, 11.2% of men and 6.2% of women were in the labor force and only 3.7% of men

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and 1.5% of women aged 85 and older were still in the labor force.[12]

The twentieth-century concept of retirement as an abrupt departure from paid employment is largely being replaced in the twenty-first century by a more fluid transitioning process. Indeed, older adults are disrupting stereotypical concepts of retirement by choosing "when," personalizing "how," and crafting "what" retirement will look like for them. Historically, retirement might have been recognized by celebrating one's 65th birthday and/or hosting a retirement party where the guest of honor would be presented with the symbolic "gold watch" for a "lifetime" of service to the employer. Retirement jokes and gifts would typically include or refer to an increased prevalence or usage of: rocking chairs; knitting; golfing; fishing; medications; gray hair; aches and pains; and wrinkles. However, with today's increased life expectancy, age 65 is rarely considered to be "old." In fact, the chronological age labels of "young-old" (ages 65–74), "middle-old" (75–84), and "oldest old" (85+) may no longer be relevant, as contemporary society's older adults are generally healthier, more active, and have more education and more disposable income than previous generations.

With notable exceptions (e.g., military, airlines, employers with fewer than 20 employees), the Age Discrimination in Employment Act of 1967[13] prohibits employers from discharging any individual because of age. As such, many older adults decide *when* they want to retire and may continue working well past age 65. However, older employees may be involuntarily separated from employment for other reasons. Corporate layoffs may disproportionately affect older adults. In analyses of 13,988 newly retired respondents in 2014, 55% reported being forced or partially forced to retire.[14] Whether an older adult is thinking about retiring, has already voluntarily retired, or has been forcibly retired, retirement is complex.

One of the more immediate complex issues older adults face upon retirement from full-time employment is *how* to manage the retirement transition period. Retirement transitions may include bridge jobs, phased retirement, and labor market reentry.[15] Bridge jobs refer to the transition jobs between retirement from full-time career employment and ultimate withdrawal from the labor force. Bridge jobs may be out of financial necessity (they have to) or for quality-of-life reasons (they want to). Phased retirement is the gradual or partial retirement that may include a reduction in work hours, wages, overall earnings, or pension receipt. Labor market reentry or "unretirement" refers to a retiree returning to the workforce, which is not uncommon.

In their 2014 "Work in Retirement" report, Merrill Lynch surveyed 7,078 respondents aged 25+, including a nationally representative sample of: 720 from the Silent Generation (ages 69–89); 1,781 Baby Boomers (ages 50–68); 517 Generation Xers (ages 38–49); and 485 Millennials (ages 25–37).[16] In their analyses, they found that over 7 in 10 pre-retirees reported that they wanted to work in retirement. The authors surmise that it will become increasingly unusual for retirees not to work. They also characterized four types of working retirees, debunking the myth that those who work after retirement do so only out of necessity. The authors identified: (1) Driven Achievers (15%) – the "workaholics"; (2) Caring Contributors (33%) – those who aspire to give back to the community and may work for a nonprofit, or may be an unpaid volunteer; (3) Life Balancers (24%) – those who seek to maintain friendships and social connections and who seek work that is fun and not stressful; and (4) Earnest Earners (28%) – those who need the income and who may not be as satisfied with work, or who may have many frustrations and regrets. Furthermore, they found that nearly three out of five retirees launch into a new line of work, and working retirees are three times more likely than pre-retirees to be entrepreneurs.

Similarly, in their analyses of 10 waves (1992–2010) of data from the Health and Retirement Study (HRS), Cahill and colleagues (2015) studied the prevalence of retirement transitions.[15] They found that the traditional permanent exit retirement model was the exception rather than the rule. Among the HRS core sample (aged 51–56 in 1992), more than half (57% of men and 54% of women) took a bridge job; 14% of men and 8% of women phased down their full-time career job hours by 20% or more; and 13% reentered the labor market (about two thirds of the reentry jobs were part-time). Taken together, recent data refutes the rocking-chair retirement stereotype; that is, most older adults who retire from full-time employment continue to work in some capacity.

Contextual Factors Associated with Retirement

There are a wide variety of contextual factors that are associated with retirement decisions and transitions, including a broad array of health, sociodemographic, and other factors. Regardless of the type of contextual factor, in order to support an older adult's retirement transition and experience, the health-care professional

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and patient must have a relationship built on trust that facilitates open communication.

One undisputed fact of both work performance and health in retirement is that chronological age alone is not a good predictor. Although some patients may stop working because of their health, many older adults' chronic diseases and health conditions are being managed better than in the past.[17] The National Council on Aging (2017) reports that 85% of adults aged 65+ have at least one chronic health condition, while 68% have at least two conditions. The 10 most common chronic conditions among adults aged 65+ are hypertension (58%), high cholesterol (47%), arthritis (31%), ischemic heart disease (29%), diabetes (27%), chronic kidney disease (18%), heart failure (14%), depression (14%), Alzheimer's disease and dementia (11%), and chronic obstructive pulmonary disease (11%). In addition, one in four adults experience some mental disorder, including depression and anxiety disorders and dementia. In addition to the projected increases in dementia, substance abuse problems are also estimated to increase.[18]

The evidence on how retirement affects physical health is equivocal. In their systematic review of 22 longitudinal studies, Van der Heide and colleagues (2013) found contradictory evidence for the effect of retirement on perceived general health and physical health; some studies showed health improvement after retirement, some showed decline, some showed no effect, and one showed an unclear effect. The disparate findings likely result from study design issues, including how physical health was measured (i.e., subjectively reported vs. objective data), when physical health was measured (i.e., controlling for health status pre-retirement compared to post-retirement), the type of employment (i.e., blue collar vs. white collar), the type of retirement (i.e., voluntary vs. involuntary), and length of study follow-up in retirement. However, Van der Heide and colleagues did find strong evidence for retirement having a beneficial impact on mental health.[19]

In general, analyses of self-report data tend to show improved mental well-being and health in retirement, perhaps more so for those with higher socioeconomic status (SES) compared with their lower-SES counterparts.[20–23] There is also objective evidence for improved mental health in retirement. In their analyses of objective data from 7,138 retirees in Finland from 1995 to 2004, Oksanen et al. found that antidepressant medication use decreased across the 9 years spanning the transition to retirement. In fact, after controlling for the secular trend in rising antidepressant prescriptions, the authors found that the prevalence of antidepressant use decreased by one fourth from before retirement to after retirement.[24]

There is a general sense of specific factors that influence well-being in retirement. In their presentation of 20 years of research, Wang and Hesketh (2012) summarize and provide a comprehensive profile of five categories of factors that have positive impacts on people's physical, psychological, and fiscal well-being in retirement: (1) individual attributes, (2) pre-retirement job-related factors, (3) family-related factors, (4) retirement transition-related factors, and (5) post-retirement activities. For example, the individual attributes that have a positive effect on *physical* well-being in retirement include pre-retirement health status, healthy behaviors and habits, and financial status. The pre-retirement job-related factor that has the strongest negative impact on physical well-being in retirement is job-related physical demands. For retirement transition-related factors, health insurance coverage in retirement has a positive impact on physical well-being, but health insurance cost in retirement has a negative impact on physical well-being in retirement. The post-retirement activity that has a positive impact on physical well-being is bridge employment.[25]

Wang and Hesketh also enumerate numerous factors within the five categories that are associated with *psychological* well-being in retirement. The factors that positively impact psychological well-being in retirement include (1) individual attributes – financial status and physical health; (2) family attributes – married (vs. single/widowed) and marital quality; (3) retirement transition-related attributes – voluntary retirement, retirement planning, retiring to do other things, and retiring to receive financial incentives; and (4) post-retirement activities – bridge employment, volunteer work, and leisure activities. Factors that negatively impact psychological well-being within the five categories include (1) individual attributes – physical health decline; (2) pre-retirement job-related factors – work stress, job demands, job challenges, job dissatisfaction, unemployment before retirement, stronger work role identity; (3) family-related factors – spouse working status (working vs. not working); more dependents; losing a partner during retirement transition; (4) retirement transition-related factors – retiring earlier than expected; retiring for health-care reasons; and (5) post-retirement activities – anxiety associated with social activities.[25]

In addition to physical and cognitive health, emotional health and social well-being are important contexts in retirement. For many older adults, their job or career

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may be or may have been central to their identity; thus, transitioning out of full-time employment, either voluntarily or involuntarily, may represent a challenge at a personal identity level. One's lifetime vocation is considered a "master status," the primary identifying characteristic of a person that shapes their social identity. After withdrawing from full-time employment – voluntarily or involuntarily – how will your patients see themselves or introduce themselves to one another? Past-tense statements such as: "I used to be a teacher" or "I am a retired physician" or "I was in construction" communicate social status, work ethic, and a sense of pride and belonging. Having a sense of purpose or meaning in life and perceived locus of control is critical. This core life philosophy is illustrated in Okinawa, where there is no equivalent word for retirement. Instead, they talk about *ikigai*, which means "why I wake up in the morning."

How older adults prepare for and experience retirement is also influenced by their social reference groups. Reference groups include our significant others, family, friends, work colleagues, and social media. As your patients are consciously or subconsciously preparing for retirement, they may be engaging in "anticipatory socialization" and mulling questions such as: who will I be after I retire; what will I do; will my life have any purpose or meaning; with whom will I associate or identify? These questions are part of an expected psychological adjustment to retirement, regardless of whether the transition was voluntary or involuntary. In 1976, Robert Atchley[26] characterized four phases of adjustment: preretirement; the honeymoon stage; disenchantment; and reorientation. Each phase brings new challenges to a person's identity, their values, and their purpose.

A wide range of sociodemographic factors are also important components to the retirement experience of your patients. Factors such as age, sex, race/ethnicity, education, income, and marital status impact the decisions patients will make as they transition into and through retirement.

According to data from the 2016 American Community Survey (ACS),[12] the number of people aged 65 and older in the USA was estimated at 49.2 million. More than half (58%) of them were aged 65–74, 29% were aged 75–84, and 13% were aged 85 or older. Women outnumber men in older age, particularly in the oldest age category (age 85+), where there are nearly two females for every male. More than three quarters (77.3%) of older adults were White, 8% were Hispanic or Latino, 8.9% were Black or African American, and 4.2% were Asian alone. The remainder were two or more races (0.9%), American Indian and Alaska Native alone (0.5%), Native Hawaiian and Other Pacific Islander alone (0.1%), or some other race alone (0.1%). Among those aged 65 and older, 13% of both men and women had less than a high school education, 27% of men and 35% of women were high school graduates, 25% of men and 26% of women had some college or an associate's degree, and 32% of men and 22% of women had a bachelor's degree or higher. Approximately 78% of all householders aged 65 and older were homeowners; 62% of Blacks and 81% of Whites owned their homes. The median earnings of full-time, year-round workers aged 65 and older were $56,850 for men and $41,200 for women. Social Security was the most common form of income received in the prior 12 months for those aged 65 and older; 89.9% reported receiving Social Security in the prior 12 months, 48.8% reported income from retirement, and 37.2% reported income from earnings. The poverty rate is significantly lower in the older population compared to the total population. Whereas 13% of males and 15% of females in the total population are in poverty, 7% of males and 11% of females aged 65 and older are living in poverty. The poverty rate for older Asians is higher (13%) compared to their other aging, race, and ethnic counterparts and compared to those in the total population (12%), and even higher (18%) among Asians aged 85 and older. Most older adults had been married at some point in their lives; only 6% of both older men and women had never been married. Women were more likely to be widowed than men with increasing age; 2 out of 10 women aged 65 to 74 were widowed, 4 out of 10 women at aged 75 to 84 were widowed, and 7 out of 10 women (72%) aged 85 and older were widowed, compared to 35% of men aged 85 and older.

Other Contextual Factors and Trends

Caregiving for a spouse or another dependent is also a likely scenario for nearly one fifth of your patients. It is well known that family and unpaid caregivers provide the majority of care for older adults.[27,28] According to the 2015 Bureau of Labor Statistics, it was estimated that during 2013–14, 16.1% of the US civilian noninstitutionalized population aged 15 and older provided unpaid care to someone aged 65 or older.[29] Furthermore, the 2016 ACS data also showed that 30% of male and 25% of female grandparents aged 65 to 74 provided care to their coresident grandchildren. For those aged 85 and older,

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13% of male and 10% of female grandparents reported providing care to their coresident grandchildren. In fact, at each age category, more males than females reported providing care for their coresident grandchildren.[12] Caregiving and caregiving burden affect employee wellness and productivity[30,31] and is likely a strong consideration in retirement planning and health status in retirement.

Social support, resources, and networks are critical for well-being. The absence of social relationships, specifically social isolation, may be particularly harmful for older adults as they withdraw from full-time employment. In their analyses of the 2011 data from the National Health and Aging Trends Study, Cudjoe and colleagues constructed a social isolation typology, determining that 24% (7.7 million) of the community-dwelling older adults were socially isolated and 4% (1.3 million) were characterized as severely socially isolated.[32] There is a vast amount of literature documenting the absence of social networks and social isolation as risk factors for negative health outcomes. Various virtual social platforms and communication tools may help maintain social connections for older patients.

Finally, technology has changed and will continue to change the way people work. Work-from-home options have become more commonplace, particularly evident during the recent global COVID-19 pandemic. It is unclear how the increasing prevalence of corporate America's remote working policies has affected or will affect older employees and their retirement transition plans. Furthermore, we also do not know how the increased accessibility, acceptability, and utilization of technology for work will provide more options for older employees to stay in the marketplace.[33]

As we seek to discover, diagnose, treat, cure, and prevent diseases and chronic conditions in older age, we should be mindful of the major milestones and pivotal events in the lives of patients, one of which is retirement. Karpen (2017) suggests that proposed changes in vocabulary from "retirement" to alternatives such as reinvention, revitalization, renewal, revision, reinterpretation, renovation, retooling, redistribution, and redeployment may more accurately reflect older adults' strong preferences toward proactive engagement.[34] Undoubtedly, retirement as a concept will continue to evolve quickly, and retirees will continue to disrupt. Health care and health-care providers must be equally nimble and adaptable in our efforts to add more years to life, and more life to years.

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Chapter 58

Cultural Competence

Gwen Yeo

Our Ethnogeriatric Imperative

Effective geriatric care includes consideration of the varied backgrounds older patients bring to the clinical encounter. While it is important to recognize that all patients and all providers have cultural backgrounds that affect clinical interactions, the wide diversity of cultures among older patients' providers are likely to see increases in the complexity of geriatric care. All patients have cultural beliefs and identities that need to be respected; however, most discussions of cultural competence in ethnogeriatric care relate to the populations defined as ethnic or racial minorities. Based on the projected growth of the populations of ethnically and racially diverse older adults in the United States, the heterogeneity of cultural backgrounds among the patients American geriatric practitioners care for will increase dramatically, producing an "ethnogeriatric imperative." As illustrated in Figure 58.1, elders in populations categorized as ethnic minorities are expected to reach 45% of all older Americans by 2060, with the largest growth occurring among Hispanic and Asian populations.[1]

These categories, however, drastically underrepresent the cultural diversity that geriatric providers see, because within each population category, including the non-Hispanic White majority, are older adults and families from dozens of different countries of origin and distinct religious and regional subcultures. Adding to the complexity are individual differences in levels of acculturation to the mainstream culture. If older Americans were born in the USA, acculturation levels are influenced by whether they have lived predominantly in cohesive, culturally specific communities, such as American Indian reservations; if they are not native born, acculturation depends also on the age at which they immigrated. The large number of older adults who have immigrated at older ages to be with adult children are less likely to speak English or to be familiar with norms of the US society, especially the health-care system.[2]

Disparities

Quality of care. Much of the discussion of effective health care for ethnic and racial populations centers on disparities in health status and health care, or the degree to which their care is different from the majority population. The National Healthcare Quality and Disparities Report mandated by Congress has been published annually for 16 years and focuses on over 250 measures of quality and access to health care among various subsets of populations in the USA, including the racial and ethnic minorities. The 2018 Report confirms that disparities resulting in poorer health status and poorer health care exist for elders from many minority backgrounds. The report indicated that although the majority of the measures have been staying the same or improving over time, "Blacks, American Indians and Alaska Natives (AI/ANs), and Native Hawaiians/Pacific Islanders (NHPIs) received worse care than whites for about 40% of quality measures" ([3], p. 4).

Some specific examples relating to older adults from the Report include: relatively fewer Asian home health patients report they are treated with courtesy and respect than those from non-Hispanic White backgrounds; those from American Indian/Alaska Native backgrounds were more likely to say their health-care providers did not explain things in the way they could understand and did not spend enough time with them.[4]

The influential report *Unequal Treatment* indicated: female Medicare recipients reporting ever being screened for osteoporosis were significantly lower among Hispanics, Asians, American Indians/Alaska Natives, and Blacks; pneumococcal vaccine rates were lower among Hispanics, Asians, and Blacks; colorectal cancer screening was lower among Blacks and Asians; lower-extremity amputations were higher among Blacks; and pressure sores in nursing homes were more prevalent among American Indians/Alaska Natives, Hispanics, and Blacks.[5]

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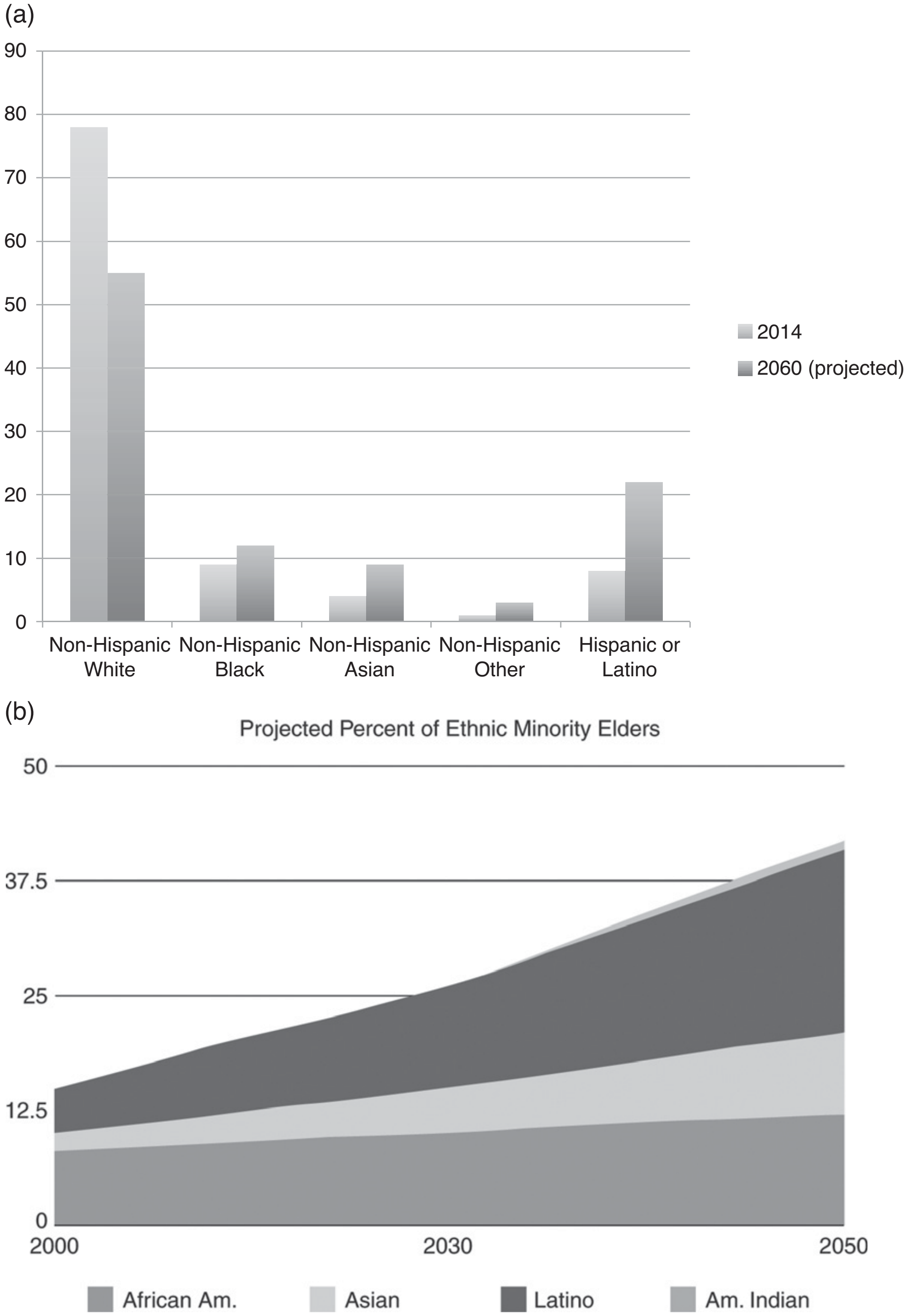

Figure 58.1 a. Percent of US population aged 65 and over by race and Hispanic origin 2014 and projected 2060. (A black-and-white version of this figure will appear in some formats. For the color version, please refer to the plate section.) b. Projected percent of ethnic-minority elders.

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Prevalence of disease. Another part of the disparities discussion is the differential prevalence or incidence of specific conditions among different populations of older adults. A few of those conditions include those discussed next.

Diabetes. One the most serious inequalities in health conditions that produces major consequences for older adults is related to diabetes. According to the National Diabetes Status Report, 2020,[6] compared to the rates in non-Hispanic Whites (7.5%), the age-adjusted prevalence of diagnosed diabetes among American Indians/Alaska Natives (14.7%) and Mexican Americans (14.4%) is almost twice as high. Among Asian Americans, Asian Indians have especially high rates (12.6%), while Chinese Americans have the lowest observed rates (5.6%). Among Hispanics (who can be of any race), those with Cuban backgrounds have lower rates (6.5%) than non-Hispanic Whites, while rates of Puerto Rican (12.4%) and Mexican Americans (14.4%) are very high. Non-Hispanic Black Americans also have high rates (11.7%). Men have higher rates than women in all the populations mentioned in the Report except Black and American Indian/Alaska Natives. In those populations with high rates, older adults are at increased risk of heart disease, diabetic retinopathy, kidney disease, and need for dialysis.[6] The Report found Blacks and Hispanics were more likely to experience end-stage renal disease due to diabetes.[4]

Heart disease. The leading cause of death among older adults in the majority population and all of the minority populations in 2017 was heart disease (except non-Hispanic Asian American women, where it was the second leading cause).[7] The age-adjusted death rates for that year indicate that Black/African Americans had higher death rates for heart disease (208 per 100,000) than Whites (169); Hispanics had lower rates (114) than Whites; and Asian/Pacific Islanders had much lower rates (85).[8]

Among African Americans, several important associations with heart failure, a common condition among older adults, have been identified. Higher incidence of hypertension and with it a predisposition for increased left ventricular mass are possible causative factors. Other associations include: lower rates of ideal cardiovascular health indicators, such as nonsmoking, physical activity, and lower body mass index; social determinants of health such as poverty, crime, and segregation; and a higher burden of oxidative stress. A diet high in preserved foods (even frozen and canned healthy foods) can increase fibroblast growth factor-23, which increases hypertension and heart failure.[9] In general, the same evidence-based treatment guidelines that apply to White patients with heart failure also apply to African Americans. However, the combination of hydralazine and isosorbide dinitrate has been advised specifically (but not exclusively) for African Americans.[10]

Dementia. Alzheimer's disease and other types of dementia seem to be unequally distributed by racial and ethnic groups among their older victims. Unfortunately, there is no nationwide data on dementia rates, but a systematic review of the literature published since 1985 found 114 articles with data on prevalence or incidence of dementia among racial or ethnic populations in specific communities in the USA.[11] While comparisons between study populations are difficult because of differences in methodologies such as measurements of dementia and ages of the samples, a wide range in the size of burdens of dementia among various populations was found. The majority of the studies were among African Americans; the percentage of older adults with dementia in those studies that reported results for ages 65 and over among African Americans ranged from 7.2% to 20.9%. Individual studies of prevalence in other populations ranged from a low of 6.3% among Japanese Americans to a high of 12.9% among Caribbean Hispanics.[11]

An interesting analysis of subpopulations among Hispanics showed the highest rates of both prevalence and incidence among all racial and ethnic populations were in the Caribbean Hispanics in New York City, and one of the lowest rates in both measures was among Mexican Americans in the Sacramento area in California. There were intermediate rates among Cuban women in Miami and Puerto Rican veterans.[11] This evidence of the great variation in the burden of dementia among the heterogenous populations designated as Hispanic makes it clear that to be meaningful, data needs to be disaggregated between the subpopulations rather than published for "Hispanics" as if it represents a homogeneous community.

For a more comprehensive list of disparities in rates of disease among older adults in diverse ethnic and racial populations, see the article "How will the U.S. health care system meet the challenge of the ethnogeriatric imperative?"[12]

While there are many more examples and numerous reasons for disparities, miscommunication, lack of cultural understanding, and lack of trust in cross-cultural clinical encounters are frequent, root causes of poor health outcomes.[13] So, how are geriatric clinicians to deal with the complexity of cultural expectations, health beliefs, health

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practices, and preferences with which they are confronted, knowing that there may be negative consequences if there are culturally based misunderstandings? An important place to begin is the conscious development of both individual and organizational cultural competence.[5]

Organizational Cultural Competence

The idea of cultural competence in health care is usually traced back to the work of Cross and colleagues in the late 1980s.[14] See Box 58.1 for a version of her definition.

For physicians and other providers to be culturally competent, they need to practice in, and be supported by, a culturally competent environment. The Office of Minority Health has developed an important set of 15 Culturally and Linguistically Appropriate Standards (CLAS) for clinics, hospitals, and health-care systems to advance health equity,[13] some of which are guidelines, and others mandates (see Box 58.2).

The first standard is a general statement of the goal of the standards for health equity. Standards 2, 3, and 4 are important statements of major considerations needed to achieve the goal – commitment of the leadership of the organization, diversity of the workforce, and education for everyone in the system on commonly used languages in the community.

Standards 5 through 8 have been interpreted as mandates for health-care organizations based on Title VI of the 1964 Civil Rights Act and subsequent court decisions that equate language access with discrimination in national origin[13] (see Box 58.3). The CLAS Standards require that free-of-cost, timely language access (interpreting and translation services) be offered to all limited-English-language patients; that everyone be informed that services are available; that providers of services be trained and competent; and that materials and signage be translated.

Standards 9 through 15 specify the methods for implementing prior standards, including using goals, demographic information, and assessments in the organization, and using assessments, collaboration, and information on the progress in implementing the Standards in the community. The website "Think Cultural Health" at the Office of Minority Health includes information on CLAS standards and suggestions for implementation (see www.ThinkCulturalHealth.hhs.gov).

In geriatric care, it is even more crucial for health-care organizations to provide adequate language access. Since immigrant older adults are the most likely to have limited English proficiency (LEP), having trained interpreters available is critical to patient-centered geriatric care. Using trained interpreters versus ad hoc interpreters (e.g., friends or family) has been found to improve patient care by accomplishing the following: decreasing communication errors, increasing patient comprehension, improving clinical outcomes, increasing patient satisfaction, reducing errors of potential consequence, reducing hospital length of stay, and reducing readmission rates.[15–17] Even if younger members of families are available to interpret for their elders, they may not have the vocabulary in one or both languages to communicate medical issues adequately, and they may have their own ideas about the elder's health condition so that perspectives of the elder may not be available to the clinician (which is crucial in assessing pain, possible elder abuse, or other symptoms the elder might want to keep private from family members). It is especially critical not to use children as interpreters even though they may have the best knowledge of English of any family members. Not only are they less likely to have adequate vocabulary, but the responsibility can be traumatic for them. When trained onsite interpreters are not available, it is extremely important for organizations to provide telephonic or video interpreting. (For discussion of provider skills needed in working with interpreters, see the section on Skills below.)

In addition to the very important CLAS standards, another important issue for organizational cultural competence in geriatrics includes making available cultural guides for providers to access in cases of cultural questions or misunderstandings. Cross-cultural interactions in health care often include contradictory expectations or judgments about best management decisions when the Western biomedical model collides with long-held cultural health beliefs.[18] Having a guide or consultant from the patient's cultural background who also understands the US health-care system is an important resource for clinicians to understand the older patient's

Box 58.1

Cultural competence is a set of congruent behaviors, attitudes, and policies that come together in a system, in an agency, or among professionals that enables effective work in cross-cultural situations. (Based on a definition by Cross et al.[14])

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Box 58.2 National Standards for Culturally and Linguistically Appropriate Services (CLAS) in health and health care

The National CLAS Standards are intended to advance health equity, improve quality, and help eliminate health-care disparities by establishing a blueprint for health and health-care organizations to:

Principal Standard

1. Provide effective, equitable, understandable, and respectful quality care and services that are responsive to diverse cultural health beliefs and practices, preferred languages, health literacy, and other communication needs.

Governance, Leadership, and Workforce

2. Advance and sustain organizational governance and leadership that promotes CLAS and health equity through policy, practices, and allocated resources.
3. Recruit, promote, and support a culturally and linguistically diverse governance, leadership, and workforce that are responsive to the population in the service area.
4. Educate and train governance, leadership, and workforce in culturally and linguistically appropriate policies and practices on an ongoing basis.

Communication and Language Assistance

5. Offer language assistance to individuals who have limited English proficiency and/or other communication needs, at no cost to them, to facilitate timely access to all health care and services.
6. Inform all individuals of the availability of language assistance services clearly and in their preferred language, verbally and in writing.
7. Ensure the competence of individuals providing language assistance, recognizing that the use of untrained individuals and/or minors as interpreters should be avoided.
8. Provide easy-to-understand print and multimedia materials and signage in the languages commonly used by the populations in the service area.

Engagement, Continuous Improvement, and Accountability

9. Establish culturally and linguistically appropriate goals, policies, and management accountability, and infuse them throughout the organization's planning and operations.
10. Conduct ongoing assessments of the organization's CLAS-related activities and integrate CLAS-related measures into measurement and continuous quality improvement activities.
11. Collect and maintain accurate and reliable demographic data to monitor and evaluate the impact of CLAS on health equity and outcomes and to inform service delivery.
12. Conduct regular assessments of community health assets and needs and use the results to plan and implement services that respond to the cultural and linguistic diversity of populations in the service area.
13. Partner with the community to design, implement, and evaluate policies, practices, and services to ensure cultural and linguistic appropriateness.
14. Create conflict and grievance resolution processes that are culturally and linguistically appropriate to identify, prevent, and resolve conflicts or complaints.
15. Communicate the organization's progress in implementing and sustaining CLAS to all stakeholders, constituents, and the general public.

perspective. These cultural guides with cross-cultural understanding could be from faith communities or hospital pastoral care departments, interpreters, nurses, or other clinicians from the patients' background, patient navigators, *promatores*, or community health representatives.

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Box 58.3 Title VI, Civil Rights Act of 1964

No person in the United States shall, on ground of race, color or national origin, be excluded from participation in, be denied the benefits of, or be subjected to discrimination under any program or activity receiving Federal financial assistance.

Provider Cultural Competence

There is growing evidence that clinician–patient interaction in cross-cultural encounters impacts patient adherence, clinical decision-making, patient satisfaction, health outcomes, and the overall quality of care.[5,19] So how do geriatric clinicians develop the competence to provide effective care for older patients from cultural backgrounds with which they are not familiar? Ideally there would be cultural competency training in health professions schools, but a survey of 2,047 medical residents in their final year of residency from a variety of primary and specialty disciplines indicated that, while 96% felt it was important to consider the patient's culture when providing care, many reported receiving little or no training in cultural competency skills while they were in residencies.[20] The percentage who had little or no training in specific cultural competency skills included: determining how to address patients from different cultures (50%), assessing patients' understanding of their illness (36%), identifying mistrust (56%), negotiating treatment plans (33%), identifying relevant cultural (48%) and religious beliefs (50%), understanding decision-making roles (52%), and working with interpreters (35%).[20]

Aspects of provider cultural competence can be considered from the framework of attitudes, knowledge, and skills.

Attitudes: cultural humility and unconscious bias. The geriatric clinician's journey to becoming culturally competent needs to begin with a broad base of cultural humility. It is impossible to be an expert in the hundreds of cultures and subcultures that might be represented among one's patients, so it is important to let the patients become the teachers and the clinician the learner in culturally related issues. It is always important to ask older patients their own perspective, even if the providers are "sure" they know. According to Tervalon and Murray-Garcia, cultural humility incorporates a commitment to self-evaluation and self-critique, and to redressing the power imbalances in the patient–physician dynamic.[21] The self-evaluation and self-critique referred to includes reflecting on one's own background and what conscious or unconscious biases might lead to assumptions about individuals from different cultures that could affect clinical interactions. Shulman and others showed that physicians made different clinical decisions for patients of different races and/or genders even when they presented with the same clinical symptoms. These authors suggest that different decisions might result from unconscious assumptions held by physicians.[22] Unconscious or implicit clinician bias is increasingly being studied in relation to clinical decision-making and to patients' satisfaction and their perceptions of clinicians.[23] For example, Green et al. found clinicians' implicit bias was related to their decisions about thrombolysis;[24] Oliver et al. found that, while physicians had implicit and explicit biases, those biases did not predict decisions about total knee replacements,[25] and Blair and colleagues found that clinicians with greater implicit bias were rated lower in patient-centered care by their Black patients.[26]

Northwestern Medicine cardiologist Clyde Yancy advises, "We should all check our assumptions at the door and not use stereotypes to define disease. Differences in heart failure that we have traditionally ascribed to race need to be revisited. Race as a biological construct is a non sequitur. Persistent discussions that imply race only allow us to introduce bias and halt our progress."[9]

Knowledge: cultural health beliefs, cohort experiences, and epidemiology. Developing cultural humility and being aware of one's biases, however, does not relieve the provider of the need to learn as much as possible about the older patient's native culture to use as background for the encounter without making the assumption that that particular elder adheres to any of that culture's specific values and health beliefs. This tension between what is traditionally considered "culturally competent" care, where the provider makes an effort to recognize the elder's cultural needs and preferences, and what is usually considered patient-centered care, where the elder is treated as a unique individual, is one of the most difficult challenges in effective ethnogeriatric care. In their three-volume series, *Doorway Thoughts: Cross-Cultural Health Care for Older Adults*, the Ethnogeriatrics Committee of the American Geriatrics Society (AGS) emphasized the importance of incorporating cultural information in

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working with diverse elders, based on the assumption that clinicians need to be somewhat familiar with the cultural background of elders before they open the door to the encounter.[27–29] This does not preclude individualizing the interaction, however. One of the AGS editors of the *Doorway Thoughts* series teaches clinicians to check out the cultural information with each older patient with questions reflecting possible culturally specific preferences, such as the following in working with an older Chinese patient: "Some people have found it helpful to balance their diets between foods that are considered cold or *yin* and those that are considered hot or *yang*. What beliefs do you have about balancing your diet?"

Cultural health beliefs and values. In an early classic description of clinical cultural competence, geriatrician Risa Lavizzo-Mourey and colleague identified knowledge of "population-specific health-related cultural values" as the first component.[30] These cultural values and beliefs include unique definitions of diseases common to some cultures that are not familiar in the biomedical model used by most American clinicians. Examples geriatric providers might encounter could be the concept of "*susto*," or fright, among some traditional older Mexican Americas who may believe *susto* causes a variety of symptoms; "high blood" in some traditional Southern African American families, referring in most cases to high blood pressure believed to be caused by too much blood; or the traditional experience of depression in many parts of Chinese society as physical rather than emotional, so that depressed elders might report pain, dizziness, or fatigue rather than feeling sad.[31]

Other manifestations of health-related cultural values that would be helpful for geriatricians practicing in cross-cultural care to know include culturally specific attitudes about diagnosis and treatment. Examples include the heavy stigma associated with mental illness and dementia among some traditional families from Korean, Vietnamese, and other Asian backgrounds that makes it less likely an older adult would have cognitive symptoms evaluated;[32] the belief that if an elder is told they have cancer, they will give up, which may lead many adult children from Middle Eastern or Filipino backgrounds to urgently request the physician not to tell their older parent the diagnosis; the reticence of some families from Mexican American backgrounds to use hospice for elders because of the hope for a miracle cure; and the preference of some older American Indians to have healing ceremonies in their tribal home communities rather than Western pharmaceutical treatment. The belief from classical traditional Chinese medicine that health is a matter of balance of the elements, such as "hot" (yang) or "cold" (yin), has influenced similar beliefs in many other Asian countries so that if an older patient describes their condition as "hot," they may not be referring to their physical temperature; they then may prefer herbal medicine or food choices to restore balance over Western medicine. Background knowledge of health beliefs and values among populations in the community the geriatric clinician sees is a crucial step to cultural competence, but it is imperative that the clinician never assume a particular older patient has those beliefs or values.

Cohort experiences. Another important component of the knowledge base for culturally competent geriatricians is knowing significant historical experiences that elders from specific ethnic backgrounds are likely to have had. Experiences that influence their trust in American health care, such as the common knowledge among African Americans of the Tuskegee Experiment in which African American men in a research study were not treated for their syphilis, are extremely important to understand. The discrimination African American, American Indian, Latino, Filipino, Chinese, and Japanese communities experienced may present a barrier to trusting cross-cultural health-care relationships. Other examples that may have affected elders' health or their attitude toward health care are the forced internment of Japanese Americans on the West Coast during World War II, and American Indians' forced attendance in boarding schools where they were punished for speaking their native language and forced to cut their hair and dress and behave like mainstream American children. Periods and circumstances of immigration are also important parts of background knowledge about a patient population that can be used in taking the health and social history of an older immigrant. For example, understanding the chaotic circumstances of the sudden evacuation of the first wave of Vietnamese immigrants at the end of the Vietnam War, or the difficult life in refugee camps and the dangerous voyages encountered by later waves of Vietnamese immigrants, forms the basis for targeted questions that help providers establish rapport and understand health-related experiences of older Vietnamese Americans.[33]

Epidemiology of disease risks. A second component of cultural competence knowledge identified by Lavizzo-Mourey and MacKenzie is knowledge of special risks of diseases and conditions populations face. Knowing what conditions are prevalent in older adults from specific

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backgrounds can make clinicians more aware of needed assessments and preventive health recommendations for particular patients. As discussed in the Disparities section above, there are special health risks that are over- or underrepresented in specific populations, which are important parts of the ethnogeriatric knowledge base.

Skills: Eliciting Explanatory Models, Showing Respect, Assessment, and Working with Interpreters

Eliciting explanatory models. Understanding older patients' perception of their conditions (their explanatory models) can help clinicians make recommendations that are consistent with patients' views and are more likely to increase their adherence to clinical recommendations. A widely recommended strategy for eliciting their explanatory models is to use questions similar to those developed by Kleinman and colleagues[34] (see Box 58.4).

Then the question becomes, what should the provider do with the information that was elicited? Various models to incorporate patients' perspectives into recommended management of geriatric conditions have been suggested, such as the LEARN model[35] in Box 58.5, but they all have in common the importance of negotiating an agreeable plan that allows clinicians to provide their best evidence-based care while recognizing and incorporating if possible their older patients' culturally influenced knowledge of their own health: the epitome of patient-centered geriatric care.

Showing culturally appropriate respect. One way a geriatric clinician can help to establish an immediate relationship with an older patient from a different cultural background is to greet the elder in a culturally appropriate way. How would one know whether to shake hands, bow, or look the elder in the eye – all of which differ culturally? This is where a cultural guide from the elder's background can be very helpful to give a short lesson in greeting etiquette. In general, touching, especially across genders, is not considered appropriate in many Middle Eastern and Asian populations, especially among Muslims. In other cultures, such as many Latino cultures, shaking hands and gentle touching are expected and considered reassuring. Sustained eye contact can be interpreted as confrontational or disrespectful among some cultures such as some American Indian traditions or in some parts of Asia, so providers may find older patients looking down during an encounter. If unsure about appropriate cultural greetings, asking the older

Box 58.4 Questions to elicit patients' explanatory models

What do you think caused your problem?
Why do you think it started when it did?
What do you think your sickness does to your body? How does it work?
How severe is your sickness?
Will it have a short or long course?
What kind of treatment do you think you should receive?
What are the most important results you hope to receive from this treatment?
What are the chief problems your sickness has caused for you?
What do you fear most about your sickness?

Note: There are different published versions of these "Kleinman Questions," some that include up to 12 questions. These nine are those from the original 1978 article.[34]

Box 58.5 The LEARN model of cross-cultural communication

- *Listen* with sympathy and understanding to the patient's perception of the problem
- *Explain* your perceptions of the problem
- *Acknowledge* and discuss the differences and similarities
- *Recommend* treatment
- *Negotiate* agreement

(Berlin E, Fowkes WA. A teaching framework for cross-cultural health care. *Western Journal of Medicine*. 1983; 139:934–938.)

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patient to provide a lesson would be acceptable. Older patients can also be helpful in instructing providers about their desired form of address and the name they prefer. In general, it is usually considered most respectful to use "Mr." or "Mrs." and the family name until instructed otherwise; in some cases, however, it may not be clear which name is the appropriate family name, so asking the elder is always safest. A particularly important part of showing appropriate respect is always to greet the older patient first before other family members, even if they don't speak English. Because older adults are held in much higher esteem in most other cultures than in the United States, both the older patient and other family members will expect deference to the elder.

Respect can also be conveyed by being careful not to use disrespectful movements or gestures. Showing the sole of one's shoe to someone is very insulting in many Middle Eastern cultures, and there are many hand gestures that are offensive in other cultures, such as several used by many Americans to express "OK" or "Come here, please." Again, being careful to follow cultural guidelines regarding touching is important.

Assessment. In addition to language issues, cross-cultural geriatric assessments need to take into consideration other issues such as appropriate respect, relevant health histories in the context of elders' cohort experience, asking permission to examine parts of the body, being aware of cultural taboos that prohibit touching some areas, and using linguistically and culturally validated formal assessment measures.[36] If cognitive status is assessed, there are many translations of the most common measures; for example, the Montreal Cognitive Assessment (MoCA) has been translated into close to 100 different languages, more than 21 of which have been validated,[37] and there are numerous other original measures validated to be accurate in specific populations. Similarly, it is important to use culturally appropriate measures to assess depression. Mui and colleagues found that some of the items in the Geriatric Depression Scale (GDS) were not appropriate for the six different ethnic populations of Asian elders they studied and made suggestions for modifications.[38]

Many elders use herbal and other remedies common in their countries of origin, and in most cases they do not volunteer that information to their physicians unless asked. It is important, then, to explore their use, especially in cases where there is a potential interaction with prescribed medications, as in the case of diabetes drugs. (For more information on ethnogeriatric assessment, see https://geriatrics.stanford.edu/culturemed/overview/assessment.html.)

Working with interpreters. As discussed earlier, it is vital for clinicians to insist on using trained interpreters with limited-English-proficient elders rather than family members, especially children. Skills in working appropriately with interpreters include: having them sit slightly behind patients so the patient faces the clinician; speaking in short phrases using lay terminology; recognizing that interpreters are obligated to interpret everything that is said so that they cannot be engaged in a side conversation; and not asking interpreters to perform tasks outside of their role, such as independently obtaining consent for a procedure. See an excellent example of appropriate clinical skills in using interpreters in the video developed by the Cross-Cultural Health Care Program, *Communicating Effectively through an Interpreter*.[39]

Conclusion

In our increasingly diverse society, to care for our rapidly growing older population appropriately it is essential to promote the conscious development of both individual and organizational cultural competence. Accounting for the cultural background and identities of older patients is a crucial step toward improving patient–provider communication, reducing health disparities, and improving health outcomes.

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Chapter 59

Health Literacy

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What Is Health Literacy?

Definitions of health literacy have significantly evolved over the past two decades. One of the most widely cited definitions is from the US Department of Health and Human Services (HHS) Office of Disease Prevention and Health Promotion (ODPHP). The HHS' longstanding former definition of health literacy was "the capacity to obtain, process, and understand basic health information and services needed to make appropriate health decisions."[1] This organization oversees the Healthy People Initiative, and every decade they update a new set of 10-year national objectives to improve health for all Americans. In the Healthy People 2030 initiative, they proposed a new definition: "Health literacy occurs when a society provides accurate health information and services that people can easily find, understand, and use to inform their decisions and actions."[2] This proposed definition appropriately shifts some of the responsibility to society to provide accurate health information. Additionally, Healthy People 2020 recognizes the increasingly important role that technology has in providing information.

This transition to shared responsibility in health literacy between information seekers and information providers is further elucidated by the Calgary Charter on Health Literacy. This charter was written by a multidisciplinary, international team with the hopes of generating a document that would guide health literacy theory and adult education.[3] It is defined as follows:

- Health literacy allows the public and personnel working in all health-related contexts to find, understand, evaluate, communicate, and use information.
- Health literacy is the use of a wide range of skills that improve the ability of people to act on information in order to live healthier lives.
- These skills include reading, writing, listening, speaking, numeracy, and critical analysis, as well as communication and interaction skills.[3]

Prevalence of Low Health Literacy

The 2003 National Assessment of Adult Literacy (NAAL) provided the first ever and only large-scale national assessment designed specifically to measure "adults' ability to use literacy skills to read and understand health-related information."[4] At the time, NAAL estimated that 80 million people in the United States had limited health literacy.[5] NAAL surveyed more than 19,000 adults aged 16 and older, concluding that more than one third (36%) of participants scored in the lowest two categories, "basic" and "below basic" health literacy skills. People scoring in these two categories may have trouble with skills such as reading and understanding the instructions on a prescription label or filling out an insurance form.[6] They also found that limited health literacy is higher in some populations, such as in older adults, minorities, those with lower incomes, and those with less than a high school education.[5]

Prevalence of Low Health Literacy in Older Adults

While the data from NAAL focused on all adults 18 years and older, data suggests health literacy disproportionally affects adults 65 years and older. National literacy surveys suggest more than 70% of older adults in North America lack the health literacy skills necessary to successfully interface with the health-care system.[7] NAAL determined that 59% of older adults score within the two lowest health literacy ranges, basic (30%) and below basic (29%).[8,9] Among this group, older adults reported difficulty using health-related documents (80%), using printed materials (71%), and understanding numbers and calculation (68%). Older adults with the following risk factors are at greater risk for low literacy: male gender, minority status, poor mental health, greater disabilities, limitations in activities of daily living (ADLs), lower income and education, and older age (>85). In addition, older adults are less likely to adopt utilization

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of health-related technology, perceiving this will not improve quality of life,[10] and many lack access to technology resources.[9] Despite this reality, health-care providers tend to overestimate health literacy skills, which may adversely affect transitions of care and outpatient disease management.[11]

The association between low health literacy and aging may be attributed, in part, to age-related sensory changes such as auditory and visual impairments, as well as to normal, mild cognitive decline and dementia.[12] It is estimated that 94% of older adults can expect to experience loss in either hearing, vision, taste, or smell, and two thirds of adults will experience loss in two or more of those senses.[13] According to the National Institutes of Health (2017), approximately 25% of adults between 65 and 75 years old have hearing difficulties, and half of adults over 75 have some form of hearing loss.[14] Most hearing loss is sensorineural, making it especially difficult to hear high-pitched voices. This reduced ability to discriminate high-pitched sounds can be misunderstood by providers and lead to shouting, whereas speaking in a lower pitch without increasing volume would be more effective for communicating. Also, simple cerumen (wax) buildup can also cause conductive hearing loss, which can muffle voices. Unfortunately, hearing loss can be associated with social stigma, which may affect older adults' comfort levels with addressing their hearing loss with care providers.[13] Additionally, even when hearing impairments are identified, accessing needed auditory assistive devices may not be possible because of a lack of insurance coverage and financial constraints.

Studies have demonstrated that age- or disease-related vision impairments interfere with access to adequate health-related information.[15] Even subtle impairments can influence the ability to understand and appropriately adhere to medications.[16] Approximately 20% of older adults have issues with vision.[13] Visual changes may be due to changes in visual acuity, but may also be the result of other conditions such as diabetic retinopathy, glaucoma, or cataracts.[13] Although there exists an association between vision impairments and insufficient access to health information, older adults may not disclose vision impairments in the absence of routine screening because of fear of stigma or loss of independence such as loss of their driver's license.[15] This under-recognition could delay appropriate assessment and management of vision impairments, thereby compromising access to health information, further threatening independence and causing safety issues.

In addition to sensory impairments, age-related cognitive decline throughout adulthood can also affect health literacy.[17,18] A causal relationship between health literacy and cognitive health has been reported,[19] and one study found that even after controlling for sociodemographic factors and disease-related factors, cognitive function was the strongest predictor of health literacy status in both men and women.[20] With age, cognitive abilities referred to as "fluid," such as verbal fluency, working memory, and reasoning, undergo mild decline with aging even in the absence of neurodegenerative conditions. These cognitive abilities are integral to health literacy, and mild changes can affect decision-making abilities and overall ability to self-manage, such as when to seek out medical care or how to follow recommended treatment plans.[18,20–22] This is in contrast to cognitive abilities termed "crystalized," which include general knowledge and vocabulary, and remain stable with age.[7]

Furthermore, the incidence of dementia increases with age, and people with dementia are susceptible to low health literacy that results in poor advance care planning and poor health outcomes.[23] Yet, early cognitive decline is often not recognized or diagnosed.[23] Over time, dementia results in functional decline and increased dependence on caregivers who might also be older and have issues with health literacy.[24] As such, it is important to recognize that while older adults may struggle with low health literacy themselves, caregiver health literacy may be equally as important to patient health literacy in health outcomes for older adults.[24] One study suggested caregivers had an average age of 67 and multiple comorbidities. One third of them had limited health literacy, further complicating their abilities to provide oversight or assume responsibility of management of chronic conditions.[24]

Impacts of Low Health Literacy on Older Adults

Many characteristics of some older adults put them at increased risk for poor health literacy and thereby poor health outcomes. Common characteristics of older adults with low health literacy include: poor decision-making ability, cognitive impairments, and inadequate social support.[8] Social isolation and loneliness have also been associated with low health literacy.[25] Low health literacy can lead to suboptimal engagement in health promotion behaviors, poor self-care, physical impairments, more chronic conditions, poorer overall physical health,

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and increased mortality risk.[8] Older adults are particularly at risk for poor health outcomes given an increase in medical comorbidities, multiple physicians, and polypharmacy.[26] This, in addition to low health literacy, can result in difficulty self-managing these multiple chronic conditions and coordinating care across various providers and settings that may not be age-friendly. Poor self-management is also compounded by overall health status and level of disability.[8] It has been suggested that management of certain conditions such as asthma,[27] obesity,[28] congestive heart failure,[11] and diabetes[28,29] may be disproportionately impacted.[24,20] Hydration-related medical conditions secondary to overhydration or dehydration also increase with age, and are more likely in patients with low literacy.[30]

Low health literacy also impacts older adults' health behaviors and physical function, including their use of preventive services and adherence to medication management.[8,19,31–33] Specific to medications, low health literacy is associated with reduced knowledge of medication names and purposes and incorrect interpretation of labels and directions included with prescribed and over-the-counter medications.[19] Community-dwelling older Americans with lower health literacy are also up to 2.5 times more likely to experience a clinically meaningful decline in physical function and increased difficulty with activities of daily living (ADLs) – regardless of preexisting factors such as cognitive impairment, education, or race. Beyond physical decline and limited mobility, low health literacy is also associated with worse overall health status and physical fitness, pre-frailty and frailty,[28] increased disability and pain, increased prevalence and severity of certain conditions, reduced quality of life, poorer disease outcomes, and increased rate of all-cause mortality among older adults.[8,19,34]

In addition to patient-centered outcomes, health literacy is also a significant predictor of emergency department utilization among community-dwelling older adults and is highly correlated with increased health-care expenditures,[34,8] unanticipated post-hospitalization visits, and number of inpatient treatments.[35,8,11] Health literacy also influences engagement in advance care planning (ACP), with studies demonstrating low health literacy being independently associated with poor ACP knowledge.[36] Additionally, health literacy significantly predicts knowledge of hospice care, attitude toward hospice care, and decisions about hospice care.[37]

Furthermore, low literacy affects patient satisfaction with providers, services, and overall care. Specifically, MacLeod et al. explored patient satisfaction with the health-care system and found that sicker patients with inadequate health literacy showed a greater level of dissatisfaction of health care in general, irrespective of physicians, specialists, insurers, or general experiences. Healthier patients with low health literacy also demonstrated dissatisfaction, but primarily with physicians and general health-care experiences.[8]

Assessing Health Literacy and Screening Tools for Research

Currently, there is a lack of data and perceived utility to support the use of health literacy screening tools in medical practice.[38,39] While individual assessment has value in research, more global approaches that prioritize universal health literacy precautions to prevent miscommunication are ideal in common practice.[40] When considering for the purposes of research, it is important to note that different screening tools are specific for different aspects of health literacy such as reading comprehension, numeracy skills, and verbal processing. Also, when utilized for research, a variety of factors should be considered, including, but not limited to, socioeconomic status, cultural background, primary spoken language, visual and hearing limitations, and cognitive dysfunction.[41–45]

Table 59.1 presents an overview of several screening tools primarily used in research.

A comprehensive list of health literacy screening tools can be found on the Health Literacy Tool Shed website, funded by the US National Libraries of Medicine, at http://healthliteracy.bu.edu/about.

While screening for health literacy at the individual level is not recommended, more recent evidence suggests that recognizing communities with low health literacy may yield better health outcomes regionally. For example, one study by Fang et al. (2020) looked at patients over 65 with newly diagnosed diabetes; they found that communities with lower health literacy levels are significantly less likely to initiate antihyperglycemic agents (OHA) and use standard preventive services. Using widely accessible health literacy estimates for communities might allow medical providers and community partners to target public health efforts.[49] These health literacy estimates can be found at http://healthliteracymap.unc.edu.[50] Similarly, Goeman et al. found that codesigning health literacy assessments and interventions across multiple stakeholders in the community led to a more coordinated approach that supported patients in appraising diabetes-related health information.[29]

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Table 59.1 Health literacy screening tools

Tool	Overview	Administration time (mins.)	Languages available	Number of items
Health Literacy Questionnaire (HLQ)	Measures speaking and listening skills, writing and reading skills, and numeracy.	7.5 mins.	English	44 questions
Newest Vital Sign	Patients read a nutrition label and apply the information in 6 questions.	3–5 mins.	English and Spanish	6 questions
Rapid Estimate of Adult Literacy in Medicine (REALM)	Measures word recognition and pronunciation	3 mins.	English	66 medical terms read aloud
Single Item Screening (SIS) Tool	Asks a single question such as, "How confident are you filling out medical forms for yourself?" Measures self-reported confidence in health literacy skills.	1–2 mins.	English or Spanish	1 question
Social Support, Short Test of Functional Health Literacy in Adults (STOFHLA)	Measures both numeracy and reading comprehension.	7 mins.	English and Spanish	36 questions
Test of Functional Health Literacy in Adults (TOFHLA)	Long version: patients participate in 5 scenarios that test comprehension and numeracy skills.	Short version: 7–10 Long version: 18–22	English & Spanish	21 questions

[41–44,46–48]

Communication Strategies to Support Older Adults

Providers often overestimate the health literacy of patients, assuming individuals comprehend more than they actually do.[39] Some studies suggest that less than one half and as little as 20% of what is discussed in a clinical encounter is understood or retained, and many older adults feel uncomfortable asking for clarification.[51,41] As previously discussed, as we age, there can be changes to our senses (particularly vision and hearing), as well as our cognition.[13] This, in addition to advancing comorbidities, can put many older adults in increasingly complex medical and emotional situations where communication is paramount in the physician–patient relationship.[52,53] That being said, there are many strategies that providers can use to help improve verbal communication and rapport with older adults.

Specifically, when providers interact with older adults, it is important to understand how older adults learn and what factors have an effect on their learning. For example, a decrease in vision and hearing, or in short-term memory, along with a tendency to be distracted can all necessitate the need to modify teaching of the older adult.[12]

General Strategies

Before the Visit

- Schedule extra time for patient visits.[13]
- Suggest patients create a list of questions and/or concerns in advance of the appointment.[14]
- Encourage patients to involve caregivers and family in appointments.[14]
- Help patients fill out required forms.[14]
- Escort patients to and from rooms.[14]
- Ascertain the individual's preferred language and use a trained interpreter if unable to speak fluently in that language. Do not rely on family and friends in the room.[41,45]

During the Visit

- To help establish rapport, it is important to use formal language with older adults such as "Mr., Mrs., Ms." Terms of endearment should be avoided (e.g., "hon," "dear," "sweetie") as these can be perceived as patronizing.[14]
- Limit topics/messages discussed to two to three during visits, so patients are not overwhelmed.[52,39]

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- Do not rush patients as this can make them feel unheard and can lead to incomplete information gathering.[14]
- Avoid interrupting as this can cause patients to feel reluctant to share information.[14,53]
- Provide long pauses to allow patients to process and answer questions.[53,52]
- Utilize active listening strategies including frequent, brief responses to show you are engaged throughout the conversation.[14]
- Utilize empathetic phrases to show caring and concern. Examples include, "I am sorry to hear that," "That sounds very frustrating."[14,53]
- Avoid medical jargon. Explaining information in clear, simple language helps empower patients in their own care.[14,39,45] A great resource for translating medical jargon to common language can be found on the Centers for Disease Control and Prevention (CDC) website: "Everyday Words for Public Health Communication": www.cdc.gov/other/pdf/everydaywords-060216-final.pdf.[54]
- Be mindful of language being used that could have alternative meanings for different generations or cultural/ethnic groups.[14]
- Take the time to ask patients/clients about their activities and hobbies so they have a sense that you are interested in them as a person.[53]

At the Conclusion of the Visit

- Write down specific takeaway points or summaries for older adults that they can take with them.[14] Again, limit these to two to three major points.

Communication Strategies for Sensory Deficits

There are many communication strategies that can be utilized to help provide effective communication for patients with both hearing and visual deficits, as illustrated in Tables 59.2 and 59.3.

Specific Communication Tools and Techniques

There are a variety of specific communication tools that are also valuable when communicating with older adults.

- Teach Back:
  - This is sometimes also referred to as the "Show Me Method" or the "Closing the Loop" method. During "Teach Back," the provider asks the patient to explain in their own words the concept or plan. This is not meant to be a test of a patient's knowledge, but rather their understanding. It is important to use open-ended questions to get a better sense of a patient's understanding. Phrases that can be utilized include:
    - "I reviewed several options with you today. Can you tell me in your own words what these options are?"

Table 59.2 Specific tools for communicating with older adults with hearing deficits

• Ensure patient is using hearing devices, if they need them. Alternatively, some offices may utilize "pocket talkers" or other devices to aid in hearing.[14]
• Utilize technology that enhances sound. For example, some mobile devices can be linked directly to hearing aids through Bluetooth.[13]
• Minimize background noises, as they can interfere with hearing. For example, mute televisions and stereos. Temporarily turn off fans.[14]
• Sit face to face with patient, as some patients may read lips to aid with understanding.[13,14] (In an epidemic/pandemic, mask wearing may make this impossible.)
• Speak slowly in short, complete sentences, enunciating words. Offer to repeat information or reword information.[13,14]
• Avoid shouting, as this can make a provider appear as though they are angry. Speak with a lower tone, as high-pitched tones can be hard to hear for patients with sensorineural hearing loss.[14]
• Use hand gestures and nonverbal language to convey ideas and transitions between topics.[14]
• Write information down for patients, and do not solely rely on verbal instructions.[14,13]

Table 59.3 Specific tools for communicating with older adults with visual deficits

• Ensure adequate lighting, including eliminating glare.[14,53]
• If providing written information, make sure writing is clear (typed may be preferred) and large enough for patient to comfortably read (at least 12–14 font).[14]
• Do not solely rely on written instructions.[13]
• If patient has difficulty reading, consider other types of visual aids such as: video animations, health models, large pictures, or diagrams. These can be stored on digital tablets to be utilized in offices and in home-health situations.[14,13]
• Utilize technology that enhances visual aids. For example, some digital tablets and smartphones have features that can enlarge texts, provide extreme contrast, or even read text out loud.[13]

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    - "Can you demonstrate the stretches/exercises that we just went over?"
    - "When will you take this medicine during the day and how will you take it?"[53,41,39,45]
- Ask Me 3:
    - This was developed by the Partnership for Clear Health Communication at the National Patient Safety Foundation. The premise is to encourage all patients to ask and understand three things before leaving an encounter. These three elements are:
        - What is my main problem?
        - What do I need to do?
        - Why is it important for me to do this?[53,41]
- "Brown Bag" Review of Medications:
    - The Brown Bag review of medications is part of the Health Literacy Universal Precautions Toolkit developed by the Agency for Healthcare Research and Quality. The goal of this tool is to have patients bring all their prescribed, over-the-counter medications, and supplements to a visit. During a visit, the provider can ask questions that may help elucidate obstacles a patient may have in taking their medications as prescribed.[55,41,53] Questions helpful to address during a brown bag review of medications include, but are not limited to:
        - What is this medication for?
        - How often are you taking this medication and at what time?
        - Why are you taking this medication differently than the directions on the bottle?
        - How do you organize these medications at home to ensure you are taking them at the correct times?[41,53]

Optimizing Print Resources

Written material can aid in patient comprehension and support verbal communication. In the same way that verbal communication should cater to an individual's current health literacy level, so should written materials.[41] When crafting print materials, keep in mind the communication suggestions described above such as focusing on key information and avoiding medical jargon.

In 2016, Jiggins studied the reading level of printed clinical summaries. Clinical summaries are patient education handouts designed to give patients a comprehensive view of their encounter. Upon review, a sample of 10 clinical summaries from 10 primary care practices were found to require the reading level of a college student.[56] In general, health information should be written at the reading level of your patients.[41,39] The preferred reading level for written texts is at or below the 8th grade level, and ideally at the 5th grade level.[56]

There are a variety of resources online that allow providers to assess the reading level of written material; see Table 59.4.[41] Other strategies that may help in the processing and understanding of print materials include:

- Utilize pictures to illustrate key content.[39]
- Include white space to declutter the reading field.[39]
- Use bullets and headings to clearly delineate information.[57]
- Present information in bulleted lists or question-answer format.[39]
- Write in short paragraphs, avoiding extraneous information.[57]
- Use active voice.[57]
- When using graphs, make sure that they are simple to understand and clearly labeled.[57]
- Use large font (at least 12 font).[57]
- Avoid complex anatomical or physiologic diagrams.[57,39]
- Provide information in the primary language that your patients speak.[55]
- Review written information with patients/clients to assess for understanding and to provide clarification.[41]

Technology and Health Literacy

Health literacy can be supported by repeat exposures to health information through multiple channels. Technological applications to support health literacy include websites, email/text reminders, blogs, online literature searches, online support communities, and patient portal access to one's own electronic medical record. Technology can be used to support motivation and behavior change and build trust between patients and the health-care team. Access to online education can increase self-efficacy and help patients make more informed decisions.[58]

Alternatives to Reading

With the spread of technology, including smart speakers (like Alexa), podcasts, and YouTube, patients can find, sort, and use health information without the need to read.

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Table 59.4 Resources for assessing readability of patient education

AHRQ's Patient Education Materials Assessment Tool (PEMAT) www.ahrq.gov/ncepcr/tools/self-mgmt/pemat.html	Can be used with both print and audiovisual material; allows providers to determine whether patients will be able to "understand and act on information."
The Suitability Assessment of Materials (SAM) www.aspiruslibrary.org/literacy/SAM.pdf	Helps rate materials on aspects that affect readability and comprehension. Takes into account content, literacy demand, graphics, layout and type, learning stimulation and motivation, and cultural appropriateness.
CDC's Clear Communication Index www.cdc.gov/ccindex/index.html?CDC_AA_refVal=https%3A%2F%2Fwww.cdc.gov%2Fhealthcommunication%2Fclearcommunicationindex%2Findex.html	A research-based tool to help providers "develop and assess public communication materials."
Automatic Readability Checker https://readabilityformulas.com/free-readability-formula-tests.php [41,39]	Can quickly ascertain the reading level of text.

Leroy compared health information comprehension via two channels, audio versus text, and found that comprehension was very similar regardless of which mode of presentation was employed. Patients were somewhat more likely to recall information they had read rather than heard, but there was no difference in comprehension. Given these findings, smart speakers may become a useful adjunctive vehicle for health information dissemination.[59]

Barriers to the Use of Technology

Some barriers to technology use exist for older adults who grew up before the information age. Older adults with low health literacy are somewhat less likely to make use of electronic medical resources. This is in part because their motor response time increases, thus increasing the number of errors they make in typing. This can lead to frustration and computer fatigue at a higher level than younger age groups.[58] User interface design with larger buttons and the ability to resize text will help older adults who cannot always manipulate keyboards or see the small screens on smartphones and watches.[58] As with any other new skill, exposure to technology over time builds competence and decreases anxiety.

Educational research in general, and health education research, has shown that older adults can master computer skills. Krajnik provided 8 hours of instruction to 24 rural older adults and documented statistically significant improvements in their ability to locate and critically evaluate health information. Subjects reported markedly improved self-efficacy.[60] In the community, older adults can expand their health literacy skills through one largely untapped resource: public libraries. Librarians and library science students can provide computer and information literacy classes. These classes enable the development of skills in finding and evaluating health information. A growing number of public libraries provide assistance with improving patient–clinician communication and researching health information in order to help patrons be more active participants in their own health care. Healthcare providers can suggest their patients and families seek out these innovative programs and classes provided in their communities. It is also an avenue for free access to the internet for those without computers or the web.[61]

Electronic Medical Record Patient Portals

In addition to using the various technologies described above, many health-care systems also encourage patients to view their electronic medical records through a secure web-based portal. Indeed, this metric is sometimes a quality-of-care indicator in some health systems to measure patient engagement. Price-Haywood et al. found very different concerns among 247 older adult portal users and nonusers. Nonusers failed to perceive the access as valuable and were concerned with issues of privacy and lack of personalization. Portal users saw the value, but they themselves were sometimes dissatisfied by delayed responses to medical questions submitted via the portal. The study concluded that patient training and the ability for caregivers to also access the portal supports patients' use of their personal health information.[62] Patient interest in electronic medical records access is growing. Irizzary et al. conducted a mixed-methods study with 100 community-dwelling older adults; the majority expressed an interest in portal use regardless of their health literacy level.[63]

Conversational Agents/Chatbots

Azevedo et al. also experimented with using conversational agents, also known as chatbots, to overcome the

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limitations of lower health literacy. Chatbots featured human-looking avatar characters who engaged in chat conversations with 360 patients with chronic diseases to improve their understanding of their medications. By translating the prescription instructions into easy-to-understand language and using social cues in the interactions, the chatbots were able to increase comprehension, increase information recall, and elicit positive emotional responses during the interaction. Subjects perceived the avatars as effective teachers and reported that they identified with the avatars.[64]

Co-Management

An underlying theme of many of these studies, and health literacy interventions in general, is that the assistance of another person, either virtually or in person, increases older adults' ability to retain and process health information. Magsamen-Conrad et al. explored online health information-seeking behavior and found the role of a trusted family member to be critical. If family members or other support people can attend appointments, take notes, keep track of questions, and lend technological expertise, but not take over, older adults can more successfully tackle health literacy tasks.[65]

Health Literacy: The Future

Research has suggested that inadequate health literacy predicts health status more strongly than age, income, education, or race,[8] and low health literacy disproportionally affects adults 65 years and older. Fortunately, national organizations such as the DHHS, the Joint Commission on the Accreditation of Healthcare Organizations, and the American Medical Association have identified health literacy as an important patient safety and quality improvement indicator.[66]

Intervention studies thus far have demonstrated variable results to reduce literacy disparities and improve outcomes like health comprehension, disease self-management, compliance with medications, and hospitalizations.[22] An emerging area of research includes community interventions that prioritize the relationship between health literacy, social roles, and social determinants of health.[67] Health-care systems are increasingly recognizing social determinants of health such as access to transportation, safe housing, economic security, and social isolation as significant drivers of health outcomes. Also, specific to older adults, establishing Age-Friendly Health Systems and reimbursement models for preventive care such as the Medicare Annual Wellness Visit has created ways to identify conditions and characteristics impacting health literacy specific to older adults such as sensory and cognitive impairments, as well as medication nonadherence and utilization of preventive health services. In addition to increasing awareness of low literacy and associated outcomes, addressing social determinants of health and health conditions specific to older adults offers promising pathways to optimize health literacy and outcomes among this population.

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Chapter 60

Caregiving

Danielle Snyderman, Judith Heredia, and Susan Parks*

A Caregiver's Story

You have cared for Ms. Jones for over 10 years. She has progressive dementia, likely vascular type. She lived independently for years but her daughter, an only child, began to have concerns about her memory. You did cognitive testing about 5 years ago consistent with mild dementia. Her daughter moved her into a standalone assisted living facility. Unfortunately, finances became an issue about 2 years ago, so the daughter decided to move her out of the assisted living facility and into her own home. Her daughter is currently significantly overwhelmed. Her mother's dementia is worsening. The daughter is also working full-time to make ends meet. She has hired a full-time aide for when she is working but is having several issues with the aide calling out often. The daughter turns to you for help.

Caregiving is defined as "assistance provided to individuals who are in need of support because of a disability, mental illness, chronic condition, terminal illness or who are frail."[1] This can include "attention to any of the needs of the person, including hands on care."[1] This chapter will emphasize informal caregivers, friends and family members, caring for chronically ill older adults. The issue of caregiving has moved into the public spotlight in the past decade. The COVID-19 pandemic has added an additional spotlight on the issue of how we are caring for our older patients. Older patients were disproportionately affected by COVID-19. An overwhelming number of deaths came from the older demographic as well as those residing in nursing home settings. Three months into the pandemic, nursing facilities accounted for 40% of COVID-19-related deaths.[2] This statistic highlights the downstream COVID-19 impact of grief and loss and the emotional toll on families and caregivers. Now, more than ever, our country and society are grappling with how to care for the aging population.

Epidemiology

In 1900 only 4.1% of the population was 65 and older and only 0.2% was 85 and older. By 2050 an estimated 20.7% will be at least 65 years old and 5.0% at least 85.[3] Thus, the population aged 65 or older increased from approximately 3 million in 1900 to nearly 35 million in 2000 and is projected to reach nearly 90 million in the year 2060.[4]

Seventy-three percent of caregivers are spouses or children.[4] The subset of adult daughters makes up 29% of caregivers and wives another 23%.[3] Thus, the overwhelming majority of family caregivers are women. Future cohorts of elderly people may have a different experience, based on trends in the structure of the American family. Household structure has been altered, especially by trends in marriage and fertility. Divorce is a particular concern because of its potential to undermine effective bonds between parents and children, especially fathers and children.[5] Future cohorts of older adults may therefore be less able to rely on spouses and children.[3]

Chronic conditions increase the risk of functional limitations that threaten independence and increase burden on family caregivers. In 2000, more than 100 million Americans had a chronic condition, with that number expected to increase to 158 million by 2040. Chronic conditions are more disabling for the elderly than for younger adults. Nearly 45% of older adults are functionally limited as a result of chronic conditions, the most common being arthritis, hypertension, hearing loss, heart disease, and cataracts.[4]

Trends in disability are encouraging, however. Surveys have consistently documented declines in disability and improvements in instrumental activities of daily living and function in the elderly population.[6] Lower rates of disability portend a better quality of life, greater independence for elders, and lower demands on families and government programs.

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Caregiving and Caregiver Burden

Nevertheless, even in the best-case scenarios of improving health, decreasing disability, and available family caregivers, the projected increase in the older adult population will inevitably place demands on both American families and government programs for the elderly.[4]

The cohort of family caregivers – defined as people who live with and care for a relative with physical or cognitive limitations – in the United States will continue to increase. A wide variety of physical and cognitive disabilities affecting the elderly can require the assistance of a caregiver, including dementias, advanced cancers, and end-stage congestive heart failure.

The term "caregiver burden" was coined in the early 1980s. It has been defined as the "physical, emotional, social, and financial toll of providing care,"[7] or the extent to which caregivers perceive their emotional or physical health, social life, and financial status as suffering as a result of caring for their relative.[8] The term is used in both the medical and lay communities to describe the stress felt by those in the caregiving role.

Despite the abundant literature on caregiver burden, some authors think that the term too broadly tries to encapsulate the stresses associated with this role. In addition, the degree of physical and emotional stress associated with caregiving is very individualized. Thus, we will outline individual components of health and well-being that are affected by caregiving.

The Need for 24-Hour Care

Ms. Jones' daughter calls you during the COVID-19 pandemic. Their aide has called out sick as she is scared to come into their home and provide care during the pandemic. The daughter is required to work from home. The daughter is frantic and feeling overwhelmed. She cannot get her work done at home as she is busy caring for her mother. She is afraid she is going to lose her job. She also thinks her mother has declined significantly during the pandemic, showing weight loss, sadness, and worsened walking ability.

Ultimately, all caregivers, whether family members or friends, will be faced with the decision of how to provide around-the-clock care for the person with dementia. Many caregivers will choose long-term care such as assisted living, nursing homes, and continuing care retirement communities. This decision for long-term care is challenging for many reasons. During COVID-19, the decision for long-term care has been increasingly challenging.

For those in the long-term care setting, the issues have largely centered on safety concerns, high numbers of patients and staff with COVID-19 infections, and family visitation issues. COVID-19 has highlighted the intricacies of the complex caregiving that occurs in long-term care, even during usual times. It is not uncommon that long-term care residents benefit not only from the care of the nursing facility staff but also from family, who should be considered as essential care partners. Though technology has provided a platform to maintain connection to family members, restrictions on communal dining and therapeutic recreation have increased the risk for social isolation and functional decline in long-term care residents, particularly those with severe cognitive impairment. Though the Centers for Disease Control and Prevention (CDC) guidance for visitors limited family members to come only in the setting of end-of-life exceptions, over the course of the pandemic facilities acknowledged and advocated that family members are essential care partners. Many family members provide care that would otherwise prompt a private duty companion such as one-on-one direction and hand-feeding for cognitively impaired residents.[9] Even with the evolving knowledge of the importance of exploring ways to support family visitation, inherent in this is the added logistical challenge of making sure nursing facilities have the appropriate personal protective equipment, not just for the facility staff but for family care partners as well.

In addition to the impact on family visitation and the role of family caregivers in the long-term care setting, COVID-19 has also exposed and amplified a more systemic problem: our inability to invest in and effectively value a long-term care system to meet the needs of aging, frail adults.[10] The financial model is not structured to adequately support long-term dependent care because Medicare only covers a portion of skilled admissions up to 100 days. Additionally, Medicaid finances more than half of long-term care costs for dependent care in activities of daily living but requires beneficiaries to first spend down their assets prior to qualifying. Even prior to the pandemic, the occupancy in nursing homes has declined over the past two decades despite an increasing aging population. Furthermore, as hospitals-at-home increase in popularity, the percentage of skilled nursing facility beds occupied by patients receiving subacute care has also decreased.[11]

The issue of how the pandemic has affected caregiving in the home has been less well publicized or addressed in the medical literature. Caregiving in the home during COVID-19 has particular challenges. One article delineates some of these unique challenges.[12] Use of masks with dementia patients can be particularly vexing. There

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can be a lack of understanding of the situation or why masks are warranted, and forgetfulness can amplify this challenge. Also, increased social isolation can lead to more loneliness as well as challenges getting essential items to home such as food and supplies.

Suggestions to address caregiving issues, including protection from the virus, and mental health and support during the pandemic, have come from multiple sources, including the International Alzheimer's Association. Alzheimer's Disease International (ADI) offers advice and support during COVID-19.[13] In addition, several online resources for caregiver support are available.[14]

Impact of Caregiving

Family caregiving and its effects on health and well-being have been topics of a large body of research since the term was introduced. Some caregivers report high satisfaction with their role. Work by Donelan found that 89% of caregivers feel appreciated by their care recipient, and 71% report that the relationship between them and their care recipient has improved.[15]

Nevertheless, caregiving has well-documented negative effects on mortality, morbidity, and emotional and financial well-being. Higher caregiver burden has been associated with multiple ill effects on the caregiver, the care recipient, and the family as a whole.

Becoming a caregiver can have a significant economic and lifestyle impact.[16] Many caregivers are forced to leave their jobs to provide adequate care for their relative. This can have far-reaching effects on caregivers' financial status and sense of worth. Researchers surveyed 2,000 family caregivers representing different races in July through August 2016 and asked them to keep a diary of their daily expenses. The study found that family caregivers spent an average of $7,000 a year of their own money on the care recipient. These expenses were for medical needs, personal care supplies, transportation fees, and paid help.[17] Likewise, many caregivers feel a sense of isolation, as their new role does not allow them the time to participate in social or self-care activities. Marital and family conflict may also emerge from the stress of providing care.[18] The health impact of caregiving has been well studied and widely reported. A landmark study documented increased mortality, especially in spousal caregivers.[19] In this study, spousal caregivers who experienced burden had a 63% higher mortality rate compared with caregivers who did not experience burden during a 4-year study period.[19] More recent work has determined that mortality rates after the hospitalization of a spouse varied according to the spouse's diagnosis requiring admission.[14] The highest mortality rates among both men and women spousal caregivers occurred when hospitalization was for psychiatric disease or dementia.[20]

Many caregivers experience symptoms of depression and anxiety. Studies have found that the incidence of depression among caregivers ranges from 31% to 46%.[21–23] A large epidemiological study in Ontario, California revealed an increase in any psychiatric diagnosis among caregivers compared with non-caregivers; 20.6% compared with 14.9%. Specifically for anxiety disorders, the increase among caregivers was 17.5% compared with 10.9% for non-caregivers.[24] The only category not higher among caregivers was substance abuse.[24] Two other studies showed equal or less alcohol use among caregivers.[21,25]

Caregiving burden is also associated with increased risk of institutionalization of the care recipient.[26] Institutional placement is more closely associated with family support system collapse rather than with the patient's own health deterioration.[23,27] Also, increased use of formal in-home services is seen in cases of high caregiver burden.[26]

The impact on caregivers has been even more difficult since the COVID-19 pandemic. Some are now juggling working from home while caring for their loved one because they previously enjoyed adult day programs and senior centers, which are no longer open.

Over time, increasing care demands and the costs of long-term care can overwhelm caregivers' lives. Navigating the health-care system and the role as a caregiver is increasingly complex. When the care recipient is seriously ill and discharged home, the caregiver may also be managing equipment, such as feeding tubes, suction machines, and tracheostomy, as well as monitoring the care recipient's condition. Caregivers have to navigate these complex health systems and accept the role of coordinator, sometimes without support. Many caregivers assume this role as full-time employment.

Because caregiving is often a protracted experience and the health conditions that afflict care recipients have changing trajectories, caregiving has been conceptualized as a career.[16] Some researchers have investigated psychological trajectories in the caregiving career, documenting decreasing sense of mastery and competence among many family caregivers.[28]

Dementia Caregiving

Caregiving for dementia patients carries a unique set of stressors. For many dementia caregivers, the strongest

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predictors of burden, depression, and health issues are behavior problems, day/night reversal, wandering, and inappropriate behaviors.[29] Likewise, an important factor is fewer perceived positive or uplifting experiences.[29] Larger social networks can often have a protective effect against the development of burden.[29]

End-of-Life Caregiving

There are distinct issues related to caring for someone at the end of life, which have been explored in the medical literature. The issues of complex bereavement, loss of identifying role, and mental health concerns have been identified. Authors have called for "future study on how and whether providing care for a dying family member is different from providing care for a chronically ill family member."[1]

Identifying Family Caregivers and Assessing Burden

Family caregivers have long been recognized as "hidden patients."[30] Assessment of their physical and mental health status represents an emerging topic in the health-care literature. It has been suggested that primary care physicians are in a unique position to discover patients who may be in the caregiving role by asking about caregiving while obtaining a social history.[31] Caregivers, however, have multiple other entry points into the health-care community, such as when applying for social services. The best time and place for caregiver assessment has been debated; however, it is universally agreed that some form of caregiver assessment is a good practice. An expert panel on caregiving, the 2005 National Consensus Development Conference for Caregiver Assessment: Translating Research into Policy and Procedure, developed a consensus report on caregiver assessment. Their extensive recommendations included identification, assessment of stressors, and current and needed resources.[32] One author described some of the outcomes of caregiver assessment as maintaining caregivers' health and well-being, preventing social isolation of caregivers, and providing appropriate support to caregivers.[33]

Researchers have developed several instruments with which to describe and quantify the degree of burden felt by caregivers. One such instrument, the Zarit Burden Interview (ZBI), has become widely used by researchers studying caregivers.[34] The original form of the ZBI has 22 questions (ZBI-22). Research has studied several shorter versions of the ZBI.[35] A 2018 study found that the three-item version was the most optimal short version, as it provided efficiency for busy clinicians while maintaining similar diagnostic value to the original 22-item version addressing personal strain and role strain[36] (Table 60.1).

Table 60.1 Screening questions (ZBI-S) (adapted from reference 36)

Are you afraid what the future holds for your relative?
Has your health suffered because of your involvement with your relative?
Have you have lost control of your life since your relative's illness?

Table 60.2 Screening questions for health-care providers to assess degree of caregiver burden

Are you taking care of a relative at home?
Do you feel that you are currently under a lot of stress because of your caregiving responsibilities?
Are there family or friends who help you care for your loved one?
Do you have time to take care of yourself on a daily basis?

However, some limitations with cancer caregivers were noted with the ZBI-1. Although this questionnaire was developed for research purposes, primary care providers should screen their patients for the overall degree of burden perceived by their patients who are caring for a relative.

There are additional screening questions that can be used to better clarify the degree of burden perceived by a caregiver (Table 60.2).

Health-care providers should screen patients for depression if they are providing care to a relative. The 15-item Geriatric Depression Scale is a useful clinical tool for elderly caregivers; [37] however, simply asking, "Are you often sad or blue?" is also an effective screening question.

When you talk with Ms. Jones' daughter, you share caregiving tips during the pandemic. However, it becomes clear that the issues are bigger for her on a more personal level. In screening her, you believe that she has symptoms consistent with depression. You explore this with her and provide supportive counseling. You refer her to her own primary care provider. She is grateful for the conversation.

Caregiving Interventions

One of the large caregiving intervention trials in the past decade was the REACH (Resources for Enhancing Alzheimer's Caregiver Health) Study.[38] REACH

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I tested six interventions. Each lasted a few months and there was follow-up at 6, 12, and 18 months. A composite intervention was then developed and tested in REACH II, in five sites. The interventions were largely centered on behavior management and the addition of individualized caregiver stress management. In the original study, both arms resulted in patient behavior improvement, with the stress reduction component helping caregiver burden.[38]

Psychoeducational interventions have been shown to lead to decreased burden and increased overall well-being and satisfaction.[29] Importantly, these effects have only been demonstrated if the intervention includes active participation and skills building for the caregivers.[23] Psychoeducational interventions such as skills training and counseling showed reduced burden, but these effects decreased over time.[39] Support groups had modest effect on decreasing burden.[39] Pharmacologic intervention for the patient did help lessen caregiver burden.[39]

Support for Family Caregivers

A variety of support services are available to assist caregivers, including information and assistance services, technology (including assistive devices and home modifications), education and training, support groups and counseling, respite care, and financial support.

Support groups. There is evidence that support groups can improve the psychological well-being of caregivers.[40] Some evidence suggests that support groups can be most beneficial when they have an educational focus, such as helping with behavior management issues for those caring for a loved one with dementia.[41] Other benefits of support groups include sharing difficult issues, sharing coping strategies, and lessening the feelings of isolation.[42]

Respite services. Respite services provide family caregivers with much-needed rest from their caring responsibilities. Respite can be provided either inside or outside the home. Adult day care programs are considered a form of respite care. Such care has been shown to have a varied effect on caregiver burden[42–45] (Table 60.3).

Financial support. Family caregiving places substantial economic strain on caregivers, especially caregiving of older adults with physical and cognitive impairments. This risk is more evident among low-income caregivers with limited resources and caregivers with limited or no access to paid benefits. Balancing work and caregiving responsibilities can be a difficult task. Many caregivers are forced to reduce work hours and forgo benefits and career opportunities in order to provide care. Area Agency on Aging (AAA) caregiver support programs provide financial assistance, education, and training for caregivers at home.[46] Financial support is provided on a sliding scale based on the income of the household where the care recipient resides. In 2014, President Obama signed a presidential memorandum that allows federal workers to request flexible working arrangements. Policies and practices that support the working caregiver benefit not only the employee but also the employer by reducing absenteeism and improving productivity. The Federal Family and Medical Leave Act (FMLA) enacted in 1993 allows employees to take family leave to care for an ill family member.[47]

Table 60.3 Practical interventions to aid caregivers (adapted from reference 32)

Encourage caregiver as a member of the care team
Proactively explore possible caregiving challenges
Emphasize importance of caregiver's self-care and well-being
Offer resources for education and online support
Utilize other members of care team (interprofessional teams including physical therapist, occupational therapist, nursing, social workers, Area Agency on Aging)
Validate strain and encourage caregivers to access respite care when appropriate

Financial support may have a profound effect on those in the full-time caregiving role. On the federal level, the National Family Caregiver Support Program (NFCSP) exists under the Older Americans Act programs administered by the Administration on Aging and state and local aging agencies. The NFCSP had its first year of funding in 2001. This program is earmarked for caregivers of those 70 years and older to provide information about existing services, assistance to access these services, counseling, support groups, and training around issues of problem-solving, respite care, and other services such as home modification.[42] In 2002, the NFCSP provided information on services to 4 million people, counseling to 182,000 people, and respite services to 76,000 people.[42,48] Table 60.4 details national resources for caregivers.

You have a follow-up conversation with Ms. Jones' daughter. She saw her own primary care physician and is getting medication and counseling for depression. You tell her you are committed to supporting her and you share with her the website for the Family Caregiver Alliance and ask her to schedule a follow-up with you in 1 month.

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Table 60.4 Caregiver resources

Online resources	
AARP	www.aarp.org/families/caregiving
American Society on Aging	http://asaging.org/node/1459
Best Practice Caregiving	https://bpc.caregiver.org/#home
Family Caregiver Alliance	www.caregiver.org
National Alliance for Caregiving	www.caregiving.org
National Family Caregivers Association	www.nfcacares.org
Next Step In Care, United Hospital fund	www.nextstepincare.org
The Global Caregiver	https//theglobalcaregiver.com
National Transitions of Care Coalition	www.ntocc.org/WhoWeServe/Consumers.aspx
Disease-specific caregiving	
Cancer	
American Cancer Society	
www.cancer.org/Treatment/Caregivers	
COVID-19	
National Council on Aging	
www.ncoa.org/ncoa_acf/covid-19-resources-for-older-adults	
National Cancer Institute	
www.cancer.gov/cancertopics/copingfamilyfriends	
Dementia	
Alzheimer's Association	
www.alz.org/living_with_alzheimers_caring_for_alzheimers.asp	
Heart Failure	
American Heart Association	
www.heart.org/HEARTORG/Caregiver/Caregiver_UCM_001103_SubHomePage.jsp	
Stroke	
American Stroke Association	
www.strokeassociation.org/STROKEORG/LifeAfterStroke/ForFamilyCaregivers/CaregivingResources/Caregiver-Resources_UCM_463834_Article.jsp	
Resource locators	
ARCH National Respite Network	www.archrespite.org
Eldercare Locator, Administration on Aging	www.eldercare.gov
Meals on Wheels	www.mowaa.org
Medicare	www.medicare.gov
Paying for Senior Care	www.payingforseniorcare.com
Veteran Directed Care Program	www.acl.gov
Training and education	
AssistGuide Information Services	www.alz.org/care/alzheimers-dementia-care-training-certification.asp
Family Caregiver Alliance	https://caregiver.org/classes-events
Dementia Caregiver Center	www.alz.org/care/alzheimers-dementia-care-training-certification.asp
Video Caregiving	www.videogcaregiving.org
Best Practice Caregiving	https://bpc.caregiver.org/#home

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Cross-Cultural Perspectives

Caregiving experiences vary across different countries and cultures. Recent work by Belle et al. has shown that a multicomponent intervention improves quality of life and depression among caregivers across different ethnic and racial groups within the United States.[49] Outside the United States, some cultures consider family caregiving as a key component of their long-term care. Germany, Australia, and the United Kingdom are examples of countries where caregivers are considered integral to the functioning of their long-term care systems. Legislation has dictated this. In other cultures, however, caregiving is considered the cultural norm. The provision of care by families is expected, not considered the exception. In those societies, nursing homes are few and families provide care to their elder relatives in the home setting. In the United States, though people of color comprise 25% of the workforce, they comprise more than half of the home-care workforce. Because of low wages, these home-care workers earn less than $14,000 yearly.[50]

At a systemic and organizational level, cultural competency training is essential in providing comprehensive patient-centered caregiving. Key components should involve development of policies and training focused on providing cultural competency training to home-care workers and translator services, and ensuring access to printed patient information in the patient's primary spoken language. Nonfamily caregivers should strive to learn and embrace the patient's communication preferences, cultural traditions, and values that may impact how they view wellness and dying.

Conclusion

As our country faces an enormous increase in the number of older Americans, the role of family caregivers will become increasingly important. It is the role of many health-care professionals, especially primary care physicians, to identify people in the caregiving role and to screen for caregiving stress or burden. It is likewise important to screen for health effects including depression, anxiety, and substance abuse. It is also important to link caregivers to local resources. These include the Area Agencies on Aging, the Alzheimer's Association, support groups, individual counseling, respite services, and adult day services. Caregivers should also be assisted in applying for services through available financial resources, especially through the NFCSP or other organizations that exist in most communities. We need to learn from the lessons of the COVID-19 pandemic and work together to prevent future disproportionate impact upon our older patients and their caregivers.

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Chapter 61

Practice Transformation for Better Care of Older Adults

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Overview

Transformation of our health-care systems is required to better meet the complex needs of our aging population as we confront the rise of health-care costs around the world. Older adults with multiple chronic health conditions can receive care that is fragmented, incomplete, inefficient, and ineffective. Care delivery and coordination of the complicated needs of older adults resides primarily in outpatient practice, both subspecialty and primary care. However, the overall coordination is dependent on primary care practices, which through transformation can be designed and equipped to guide comprehensive care for all patients. The term "practice transformation" refers to a process of change in the organization and delivery of care to advance quality improvement and patient-centered care. Practice transformation is a continuous process that involves leadership, goal-setting, workflow changes, quality improvement, and reporting of outcomes. It requires adapting organizational tools and processes to support advances in models of team-based care.

Primary care serves as the backbone of health care. While governmental policies and payment models differ country to country, the principles first espoused in 2007 by the US Institute for Healthcare Improvement as the "Triple Aim"[1] (seeking improved health-care quality and patient experience while reducing cost) have been adopted as a roadmap for most countries seeking more effective health care. Global efforts are underway to review and enhance the effectiveness of primary care. The European Commission published in 2018 an extensive review of the status of primary care in individual European nations, in efforts to advance primary care performance.[2] Internationally, health-care researchers are reporting on recent primary care transformation efforts,[3,4,5] many of them modeled on US efforts that have been underway for decades to transform primary care. We provide an overview of these efforts, highlighting key components for practice transformation and best practice strategies, with special attention to the care of older adults.

Of all the changes envisioned as part of the transformation to improved and more patient-centered primary care, perhaps none is more promising and more challenging than the transition to team-based delivery of care. Well-implemented team-based care has the potential to improve the comprehensiveness, coordination, efficiency, effectiveness, and value of care, as well as the satisfaction of patients and clinicians. To achieve this potential, the transition to team-based primary care requires, for most practices, profound changes in the culture and organization of care, in the nature of interactions among colleagues and with patients, in education and training, and in the ways in which primary care personnel and patients understand their roles and responsibilities.[6]

The typical primary care practice cares for a diverse panel of patients where different strategies are required. The transformed primary care practice can identify needs and goals unique to their elderly patients and build capabilities and processes to optimally address them.[7,8]

Transformed primary care practice incorporates several guiding principles in the care of older adults:

- The highest priority is to maximize and maintain function and independence.
- "Aging in place" is the goal for most. Enabling patients to age in place can be facilitated by a primary care practice with a comprehensive, coordinated approach toward care, including partnering with community resources and using telemonitoring technologies. Effective partnering with family caregivers also promotes this goal.
- Challenging social determinants of health and behavioral health issues have a larger impact on the elderly population. Loneliness, depression, and dementia are more prevalent. This requires enhancing practice capability for addressing patient resource and behavioral health needs.

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We will describe in this chapter how to incorporate these principles into practice by reviewing transformation requirements and best practice strategies.

The Importance of Primary Care Practice Transformation for Better Elder Care

Since the 1980s, variation in health-care practice and outcomes has been analyzed in the USA using Medicare data in the Dartmouth Atlas of Healthcare.[9] Regions with a greater proportion of primary care physicians, and a lower number of specialists and hospitals, tended to have lower costs, higher quality, and lower rates of avoidable hospitalizations.[10] When considering the population over age 65, primary care takes on even greater importance in promoting prevention, chronic disease management, and patient goals for end-of-life care.

The Triple Aim, advanced by the Institute for Healthcare Improvement (IHI),[1] serves as an aspirational guide for this transformation: improve the quality of care, reduce costs, and promote optimal patient experience and engagement. This has been enhanced to include health-care provider experience (the Quadruple Aim) while concerns mount about health-care worker burnout caused by work demands. Recent research provides evidence that team structure and culture, key features of practice transformation, are associated with lower burnout of physicians and support staff in primary care.[11] Practice transformation supports achievement of the Quadruple Aim while successfully meeting the complex needs of elderly patients.

The Evolution of Practice Transformation

The patient-centered medical home (PCMH) is a US model for comprehensive care, featuring a primary care provider who is augmented by a proactive team and information technology. In 2013, report of a survey of five European countries suggested that more elements of the PCMH could improve primary care. "Such a model could prove useful for advanced European systems as they strive to improve primary care, particularly for chronically ill patients. We conclude that despite strong organizational structure, European primary care systems need additional efforts to recognize chronically ill patients as partners in care and can embrace patient-centered medical homes to improve care for European patients."[12]

The concept of a PCMH to coordinate the care of children with chronic conditions was first introduced by the American Academy of Pediatrics (AAP) in 1967. In 2002, the AAP expanded the concept to include the following operational characteristics: accessible, continuous, comprehensive, family-centered, coordinated, compassionate, and culturally effective care. By 2005, the American College of Physicians (ACP) had developed an advanced medical home model, which involved the use of evidence-based medicine, clinical decision-support tools, the Chronic Care Model, medical care plans, "enhanced and convenient" access to care, quantitative indicators of quality, health information technology (HIT), and performance feedback. A pediatric concept no longer, the PCMH now provides care for people of all ages and medical conditions.

There is evidence supporting the effectiveness of this approach. For example, patients with hypertension, hyperlipidemia, diabetes, and coronary atherosclerosis enrolled in a PCMH were shown to experience reductions in emergency department visits. Specifically, patients 65 and older also experienced reductions in pharmacy expenditures, with a parallel reduction in pharmacy standardized cost, indicative of a reduction in the intensity of drug utilization.[13]

In 2008 the National Committee for Quality Assurance (NCQA) released a PCMH Recognition program, the first evaluation program in the USA based on the PCMH model. Because NCQA PCMH Recognition was associated with lower costs and better quality, many insurance payers offered incentives, support, or physician distinction to reward practices that earned and maintained NCQA PCMH Recognition.[14]

Building upon this structure, in 2012 the Centers for Medicare and Medicaid (CMS) launched the Comprehensive Primary Care (CPC) initiative, a 4-year multi-payer initiative designed to strengthen primary care across five national regions. Five key functions were outlined in the CPC:

(1) Risk-stratified care management
(2) Access and continuity
(3) Planned care for chronic conditions and preventive care
(4) Patient and caregiver engagement
(5) Coordination of care across the medical neighborhood.

(The "medical neighborhood" is defined as a clinical–community partnership that includes the medical and social supports necessary to enhance health, with the PCMH serving as the patient's primary "hub" and coordinator of health-care delivery.)

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The program provided a blueprint that drove significant transformation of primary care practices. The program also incorporated financial rewards, tied to meeting budgetary and quality goals.

The Comprehensive Primary Care Plus (CPC+) program, a 5-year advanced primary care medical home model launched in 14 US regions in January 2017, integrated many lessons learned from the CPC, including insights on practice readiness, the progression of care delivery redesign, actionable performance-based incentives, necessary health information technology, and claims data-sharing with practices.

Beginning in 2021, a new program, "Primary Care First," will further the primary care practice as centers of responsibility for providing comprehensive, quality care while accepting increasing responsibility for the total cost of care for its patients. There is one payment model for general primary care populations and a second for Seriously Ill Patients (SIPs). Primary Care First-SIPs focuses on primary care practices that have an increased focus on high-need, high-cost patients.

The Role of Outpatient Subspecialty Care

Subspecialty practice partnership with primary care is critical for comprehensive health-care management. Outpatient specialists are major drivers of health-care spending through the use of procedures and hospitalizations. Using the PCMH model as a guide, the Patient-Centered Specialty Practice (PCSP) program was developed, and similarly designated for recognition by the NCQA, in 2013. It distinguishes subspecialty practices that excel in delivering high-quality, patient-centered care with a focus on proactive coordination and the sharing of information with other providers. Everyone in the practice works as a team to coordinate care with primary care providers, other referring clinicians, community resources, and secondary services.

New Payment Models: Value-Based Care

Value-based care (VBC) is a health-care delivery model in which providers, including hospitals and physicians, are paid based on patient health outcomes, instead of volume of services. While payment structures vary across different economies around the world, the goals of VBC align with the international need to improve quality while reducing costs of health care. The US experience outlined here illustrates how VBC principles translate into practice.

Increasingly, primary care practices in the USA are being required to participate in and lead initiatives in VBC. This is particularly challenging in the care of high-need, high-cost patients, many of whom are elderly. VBC differs from the traditional fee-for-service models where providers are paid based on the number of health-care services they deliver. Rather, the "value" in value-based health care is derived from measuring health outcomes against the cost of delivering the outcomes.[15] Practices that participate in these models aim to avoid wasteful or unnecessary care and preventable hospitalizations, and strive to better coordinate care. By successfully achieving these goals, the assumption is that the cost of care would be reduced.

CMS payment in the US Medicare program is increasingly tied to VBC models, because patients over age 65 (and those younger with disability) are more likely to have higher needs (and higher costs).

New payment mechanisms acknowledge the need for care to be managed in ways outside of the traditional problem-based office visit and to reimburse clinicians for care that more comprehensively meets the needs of older and more complex patients. Examples include additional payment for performing the Medicare annual wellness visit, which is meant to deliver a holistic view of the patient and includes areas not routinely managed by many clinicians. There are now also specific payments to manage patients after a hospitalization, to manage patients with multiple chronic conditions, and to address end-of-life issues (Table 61.1).

Additionally, the Psychiatric Collaborative Care Model and Cognitive Impairment Assessment and Care Planning address mental health needs of the elderly; payment is provided for more comprehensive ongoing services provided by the primary care practice in collaboration with behavioral specialists.

Table 61.1 New payment mechanisms supporting care outside of the traditional office visit

Service	Purpose
Annual wellness visit (AWV)	Annual review of wellness, risks, social determinants of health, and prevention for patients over age 65
Transitional care management (TCM)	Follow-up visit and 30-day management of patients transitioning between venues of care (usually after a hospitalization)
Chronic care management (CCM)	Monthly payment for documented management of a patient with complex needs outside of office visits
Advance care planning (ACP)	Payment for time spent addressing advance directives with patient and family

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In the USA, payers estimate and budget for covered patients' anticipated costs, and predict and allot funds, based upon the patient's complexity. The capture of the complexity of patients determines the practice's budgetary benchmarks, which are used to determine savings and risk. Accurate Hierarchical Conditions Category (HCC) Coding is critical to practice financial stability. HCC relies on ICD-10 coding to assign risk scores to patients, then costs are predicted using algorithms. Elderly patients with multiple chronic conditions would be expected to have higher health-care utilization and costs as long as their complexity is accurately captured through coding.

Practice transformation underlies success in the following categories of VBC programs.

Quality Performance Programs

Measurement of and improvement in quality is expected of health-care providers, as it has been in other industries for decades, and is tied to payment incentives. In 1991, the NCQA began its accreditation program for medical care organizations. Since 1992, the NCQA has collaborated with medical care organizations (MCOs), academic researchers, corporate purchasers, and consumer representatives to create a performance measurement set known as the Health Plan Employer Data and Information Set (HEDIS). Both governmental and private payers in the USA tie performance incentives to quality, based upon HEDIS metrics. All primary care practices are familiar with reporting on quality metrics, such as mammography screening rate and diabetes control (HbA1c). They develop processes to capture, report, and improve on performance.

Specific measures focus on care for the elderly. For example, there are HEDIS measures specific to older patients, built around the Beers Criteria for high-risk medications in the elderly.[16]

Potentially Harmful Drug–Disease Interactions in the Elderly

Assesses adults 65 and older who have a specific disease or condition (chronic renal failure, dementia, history of falls) and were dispensed a prescription for a medication that could exacerbate it.

Use of High-Risk Medications in the Elderly

Assesses adults 65 and older who had at least one dispensing event for a high-risk medication or who had at least two dispensing events for the same high-risk medication.

Alternative Payment Models (APMs)

With legislation through the Affordable Care Act in 2010,[17] there has been a rapid introduction of alternative payment models in the USA, many developed by CMS and designed for Medicare recipients. These all focus on reducing costs through improving preventive, comprehensive, and coordinated care processes, with concomitant reduction of error and waste. Health-care providers (health systems and clinical providers) earn rewards for meeting quality goals, but are increasingly required to be at risk for financial losses. Transformed primary care practices are foundational for meeting the challenge of these APMs.

The first APM, the Medicare Shared Savings Program, established Accountable Care Organizations (ACOs) in 2013. This program fundamentally aligned financial outcomes of organizations and providers with the outcomes of patients.

Increased risk was built into its successor, the "Pathways to Success" ACO Program, in 2019. These increasingly "Advanced" Alternative Payment Models (A-APMs) bear significant financial risk in exchange for greater shares in savings.

Other A-APMs include:

- Bundled Payments for Care Improvement Advanced (BPCI-A)
- Comprehensive End Stage Renal Disease Care (CEC)
- Comprehensive Primary Care Plus (CPC+)
- Risk-bearing MSSP ACO Models: Pathways to Success Track D Basic, and Advanced, and Next Gen ACO Model
- Oncology Care Model (OCM)
- Comprehensive Care for Joint Replacement (CJR)

A comprehensive overview of A-APMs can be found at https://qpp.cms.gov/apms/overview.[18]

MACRA/MIPS

In 2015, the Sustainable Growth Rate (SGR), a US congressional method for determining payment to providers, was replaced by the Medicare Access and CHIP Reauthorization Act (MACRA). For organizations not in a risk-bearing Advanced Alternative Payment Model (A-APM), providers must participate in MACRA's Merit-Based Incentive Payment System (MIPS). MIPS outlines a value-based payment modifier applied to CMS payments that incorporates four areas of performance: quality, efficiency (resource use), clinical process improvement, and meaningful use (MU) of certified

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electronic health record (EHR) technology. MU has been renamed performance interoperability (PI).

In order to succeed in these payment programs, a primary care practice must be able to successfully measure, report, and improve upon quality and efficiency (financial) performance. These capabilities are developed through practice transformation.

More detailed explanations of these programs are available at www.aafp.org/practice-management/payment/medicare-payment/aapms/cms-primary-cares-initiative.html.[19]

Innovative Care Models

The opportunity to reduce total cost of care and profit from shared savings has led to innovative care models that focus resources exclusively on high-need, high-cost patients, many of whom are elderly. This focus allows the practice to concentrate on the sickest population and surround complex patients with a team of providers to maximize care, while avoiding unnecessary hospitalizations and specialty care. The investment results in better-coordinated and less wasteful care. Medicare Advantage programs (who partner with CMS, private insurers, and health-care providers)[20] and private equity firms can realize significant returns on investment in these innovative models.

Key Components for Transformation

Practice transformation is an ongoing and expensive process, and requires substantial resources, new skill sets, new tools, and often new members of the health-care team.[21,22] New payment models have begun to provide resources to support primary care practices to engage in this challenging process.

Technology

Few practices have been able to seamlessly integrate clinical care with the myriad sources of data in a format that would allow for efficient, comprehensive performance assessment and improvement, and reporting.

Agency for Healthcare Research and Quality (AHRQ) discussions with experts and representatives of exemplary primary care organizations suggested that to effectively use health information technology (HIT) for quality improvement (QI), primary care practices require four interconnected factors, none of which is sufficient in isolation:

- a practice culture with a strong commitment to using HIT for QI
- high-functioning HIT tools to enable tracking and extraction of data
- practice clinical team and staff knowledge and skills related to both HIT and QI
- workflows that incorporate effective use of HIT for QI.

Ideally, these factors are supported by financial incentives to offset capital, training, and clinician and staff time related to QI activities and transformation assistance, to build practice skills, processes, and workflows that may include consultation with experts in IT and QI, practice facilitation or coaching, and access to recognized best practices.[23]

Data management tools that integrate information are integral to the success of strategies that lead to better care for complicated older patients. Practices need to assimilate data from multiple sources in order to provide actionable reports. That data includes:

- clinical information captured in EHRs. Clinical information from hospital and specialty care may not reside in the same system as the care recorded in the outpatient primary care setting, complicating information capture
- payer reports, derived from submitted billing claims
- patient-reported experience from surveys, such as Consumer Assessment of Healthcare Providers and Systems (CAHPS), both inpatient (Hospital-CAHPS, or HCAHPS) and outpatient Clinician and Group CAHPS surveys (CG-CAHPS)
- public health data, including measures of social determinants of health, and registries (immunization registries, for example).

Electronic Health Records

The promise of efficiency in the conversion from paper to electronic charts has been tempered by the challenge of incorporating the technology into clinical encounters. Adoption of EHRs is promoted by the Medicare promoting interoperability (PI) program that requires providers and hospitals to utilize standardized 2015 certified electronic health record technology (CEHRT). A scoring methodology requires reporting in four categories:

- electronic prescribing (e-prescribing)
- health information exchange (supporting electronic referral loops by sending and receiving health information with regional information systems)

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- provider to patient exchange (providing patients electronic access to their health information, such as through patient portals)
- public health and clinical data exchange (reporting to two of six possible exchanges, such as immunization registry, or electronic reportable laboratory results to public health systems).

The challenges are significant for primary care practices, and complex interactions between technology and the surrounding work environment can lead to problems. Workflows may be disrupted, providers may find that EHRs adversely impact their productivity, or nursing staff may find their time overly committed to technical requirements. The Federal Government in partnership with AHRQ developed a workbook to address unintended consequences of EHR use.[24] The following list of recommendations for improving EHR safety are designed to prevent adverse unintended consequences that may occur during day-to-day EHR use:

- Actively involve clinicians and staff in the reassessment and ongoing quality improvement of technology solutions.
- Continuously monitor for problems and address any issues as quickly as possible, particularly problems obscured by workarounds or incomplete error reporting.
- Use interdisciplinary brainstorming methods for improving system quality and giving feedback to vendors.
- Carefully review skipped or rejected alerts.
- Require departmental or pharmacy review and sign-off on orders that are created outside the usual parameters.
- Provide an environment that protects staff involved in data entry from undue distractions when using the technology.
- Continually reassess and enhance safety effectiveness and error-detection capability, including the use of error tracking tools and the evaluation of near-miss events.
- Use manual or automated surveillance techniques to continually monitor and report errors and near misses or close calls caused by technology.
- Pursue system errors and multiple causations through root cause analysis (finding the real cause of the problem and dealing with it rather than simply continuing to deal with the symptoms) or other forms of failure-mode analysis.

Telehealth and Telemedicine

Telehealth and telemedicine both refer to the application of technology for health care to remotely monitor patients and provide more convenient "virtual" visits. The COVID-19 pandemic truly accelerated their adoption, underscoring the benefit of the technology for promoting coordinated care. Though not universally available or easily used, telehealth provides improved access to care for many patients. Utilizing these tools in the care of elderly patients poses additional challenges; lack of technology or comfort with that technology and/or cognitive impairment may impede the benefits of adoption. On the other hand, older patients are more likely to accept telehealth technology if it is part of the care ordered by their primary care provider.[25] Homebound patients can be more closely linked with their medical care providers using this technology, resulting in safer and more comprehensive care.

Telemedicine use can be provided either through patient portals (asynchronously) or for appointed visits with their provider (synchronously). These require adequate internet access and, ideally, videoconference capabilities on patient-held devices for visits. When the devices are available, family caregivers can facilitate their use for elderly patients who are uncomfortable with or unable to use them independently.

Sources of Data

The collection and analysis of data are critical to successful performance of the transformed primary care practice. Data capture in usable format, and the ability to analyze and report this data for performance improvement, often represent the biggest challenge to practices. Technical expertise is required. Major sources of data include the following categories.

Electronic Health Records

In the USA, an estimated 1,100 EHR system vendors had products available in 2014. A primary care practice has to choose a system based upon capabilities and affordability. The myriad EHRs are independent and lack the ability to easily share information across systems. Frequently, a practice is faced with converting to a new EHR, requiring laborious transfer of data.

"Front-end pain for back-end gain" reflects the difficulty faced with capturing clinical information at the point of care, usually required of the patient-facing

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clinical team members. EHRs are frequently cited as the top reason for physician burnout.[26]

The wealth of information collected must then be converted to a usable format in order to guide safe and coordinated clinical care. Information capture must be in fields and forms that allow for reconstruction of that data into actionable reports. In addition, the reporting of measures of quality and efficiency is required for value-based payment.

The cost of adopting and enhancing this capability is a rising expense for practices.[27] However, these capabilities are required of the transformed clinical practice.

Payer Reports/Claims Data

Claims submitted for reimbursement to private or governmental insurance companies or other payers provide important cost and clinical data useful for managing a panel of patients. By analyzing claims data, practices can identify clinical opportunities for care improvement. High-cost diagnoses and treatments, patients with excessive utilization patterns, and hospitalizations for ambulatory sensitive conditions (which, when properly managed, don't lead to hospitalization) are studied to develop strategies for intervention. Claims data can also be used to measure continuity of care between providers, and to identify opportunities for improved access to care (through study of emergency department utilization). Patients can be segmented for outreach and care coordination. For example, certain claims are associated with and can identify frailty, a marker of higher risk in elderly patients. Claims data can also support quality performance assessment of preventive and chronic care where payment for tests or procedures such as mammography, colorectal cancer screening, and diabetes testing is found.

Patient-Reported Experience

Patient input is critical to practice success in achieving the Triple Aim. For older patients, feedback from family caregivers is of added importance. Feedback can be collected through numerous strategies such as a local practice-developed survey, an anonymous "patient comments and suggestions" box, and/or patient-family advisory councils (PFACs). A PFAC consists of patients and their caregivers who have received care at a practice. It provides a mechanism to seek and learn from the patient and family perspective and promote a culture of patient- and family-centered care within an organization.

The practice team can recognize all patient comments as potential opportunities for improvement, and systematically report this feedback for review. Surveys required by payers, such as CG-CAHPS, provide scores with benchmarks, which can be used to improve experience of care. Data captured in patient experience surveys is usually separate from other sources of data, such as EHRs or claims, and needs to be integrated.

Public Health Data/Social Determinants of Health

Primary care and public health have also tended to operate without significant collaboration. With the increasing recognition of the impact of Social Determinants of Health (SDOH) on the health and wellness of our populations, the need for partnership is being addressed through educational and legislative efforts to share information. As mentioned earlier, primary care practices are being required through PI to support public health data reporting. Six different opportunities can be reported, although only two are required for PI reporting:

- syndrome surveillance
- immunization registry
- electronic care
- public health registry
- clinical data registry
- electronic reportable laboratory result.

SDOH factors significantly impact the health and experiences of aging, especially the ability to live independently and age in place. Strategies for addressing SDOH will be further explored in Best Practice Strategies below.

Practice Culture

As the population becomes older and ever more complex care is moved to the ambulatory environment, traditional practice culture based upon acute-care visits is being forced to innovate. The idea of a model with a physician leader and subordinated staff members must transform itself to become a collaborative team practice culture. Culture, as defined by John Kotter, is "a group of norms of behavior and the underlying shared values that help keep those norms in place."[28] Care quality (the degree to which patients receive appropriate care based upon evidence and guidelines), patient experience, care safety (the reduction of error), and clinical outcomes are all improved by a culture that promotes effective teamwork.

Primary care practices personify unique and distinct cultures. Whether a solo or multi-physician practice, cultures vary widely, and unique subcultures often

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emerge among larger groups. The transformation of primary care to a common culture will occur if a large enough group of stakeholders decide that old ways are not working, develop a vision for change, act differently, and then enlist others to do the same. Physician leadership is often a key to success.

Teamwork

Team-based care is defined by the US National Academy of Medicine as "the provision of health services to individuals, families, and/or their communities by at least two health providers who work collaboratively with patients and their caregivers – to the extent preferred by each patient – to accomplish shared goals within and across settings to achieve coordinated, high-quality care."[29]

Team-based care offers many potential advantages including:

- expanded access to care (more hours of coverage, shorter wait times)
- more effective and efficient delivery of additional services that are essential to providing high-quality care, especially for elderly and complex patients, such as patient education, behavioral health, self-management support, and care coordination
- increased job satisfaction, and an environment in which all medical and nonmedical professionals are encouraged to perform work that is matched to their training and licensure.[1]

Studies support that physician burnout, increasingly recognized especially amongst primary care providers, is lessened for clinicians and staff who work in effective team-based practices.[26]

Roles and responsibilities and well-defined expectations need to be understood by all members of the team. This structure is foundational in order to develop a sense of team accountability and shared responsibility for patient care. "Practice to the top of your license" is a principle that maximizes the utilization of, and job satisfaction derived by, individual team members. For example, a medical assistant can do more than "room" a patient with vital signs and a chief complaint. They can also address quality gaps and preventive measures, initiate an annual wellness visit, and provide patient education.

The practice team usually includes a group of interprofessional clinical personnel that can include any or all of the following: physicians, nurse practitioners, physician assistants, nurses (for direct clinical care as well as care coordination), nursing/medical assistants, dietitians and diabetes educators, pharmacists, and social workers – as well as nonclinical staff, such as receptionists and peer counselors. Increasingly, behavioral health counselors (psychologists or clinical social workers) are also being integrated into primary care teams. The composition of any practice team is guided by the needs of the practice's patients and the financial resources available to the practice. Patients can be incorporated into the team through PFACs and as central members of their personal care teams.

Engagement with Providers

Physicians and advance practice providers who are not educated with or accustomed to work with teams may resist practice transformation to a team culture. Beyond "buy-in" to changes, providers ideally are "engaged" with the practice. Engagement occurs when the provider is involved in the entire improvement process. Providers must be integrated into the infrastructure discussed above. There is evidence that supports a phased, collaborative process that employs practice talk, a term that describes naturally occurring, collegial conversations among members of clinical teams.[30]

Culture of Safety

A culture of safety, where errors are minimized and each practice member is comfortable speaking up with any concerns, is particularly critical for the care of complex elderly patients. A team-based practice culture can promote safety.

The concept of safety culture originated outside of health care, in studies of high-reliability organizations. These consistently minimize adverse events despite carrying out intrinsically complex and hazardous work, and rely upon:

- acknowledging the high-risk nature of an organization's activities and the desire to achieve consistently safe operations
- a blame-free environment where individuals are able to report errors or near misses without fear of reprimand or punishment
- encouragement of collaboration across ranks and disciplines to seek solutions to patient safety problems
- organizational commitment of resources to address safety concerns.

Embracing Diversity

Embracing increasing societal diversity in transformed primary care practices will promote patient-centered

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care. The older US adult population is becoming more racially and ethnically diverse. In 2015, roughly 8 out of 10 people aged 65+ (78%) in the USA were White non-Hispanic. However, fewer than 6 out of 10 older Americans (57%) in the USA will be White by 2060. From 2015 to 2060, the number of Black older adults in the USA will nearly triple and the number of Hispanic older adults will more than quintuple, while the number of Whites will less than double.[31] These demographic changes have enormous implications for meeting diverse personal and family caregiver preferences, providing services with cultural sensitivity, and training the paid health-care workforce in cultural competence.

Team members will need to provide culturally sensitive care. Language barriers will need to be addressed for older patients and their caregivers through provision of interpreter services. The importance of increasing diversity in the older population has been highlighted by the dramatic impact of the COVID-19 pandemic on older adults of color in the USA compared to White elders.

Low (Health) Literacy

The transformed primary care practice, in order to provide optimal care, must be aware of and develop strategies to address low health literacy. Low health literacy is defined as "the degree to which individuals have the capacity to obtain, process, and understand basic health information and services needed to make appropriate health decisions." Low health literacy levels are predictors of negative health outcomes. Patients with low health literacy use emergency services more frequently, have higher health-care costs, utilize preventive services such as vaccinations and mammograms less frequently, and have higher mortality rates. Socioeconomic status, age, race, cognition, and education level are considered contributing factors of health literacy levels, with increased age as one of the highest correlates of low health literacy.[32]

The primary care practice approach to addressing low health literacy begins with team education about the issue, and increased recognition of patients who may be struggling. Sensitive tools used to verify patient comprehension of information, such as "teach back,"[33] are beneficial in the care of older adults. The advantages of recognizing and addressing low health literacy include improved health-care decisions, communication, adherence to treatment plans, and improved health status.

Best Practice Strategies

Successfully transformed primary care practices focus on multiple initiatives that strategically address key opportunities for improved care and financial performance. The following is a discussion of key strategies, with special attention to the needs of the elderly.

Segment by Need or Risk

A primary care practice panel encompasses segments of patients with different needs, and with different levels of health risk. Recognizing this can help the practice implement different care models to more efficiently meet the diverse needs of patients. Termed risk stratification, this process is often employed by analyzing data for specific cohorts of patients, and ranking them by risk of hospitalization, complication, high cost, and/or death.

High-cost, high-need patients can often be most easily identified through claims/billing data. Many of them may be transiently high cost, because they can be expected to recover (patients with motor vehicle accident recovery or nonterminal cancer treatment, as examples). About 40% of the group, most of whom are older adults, will remain high-cost, high-need over longer periods of time.

While high-cost, high-need patients may benefit from outreach and care coordination, care can also be organized by stratified chronic conditions. For example, needs of patients with congestive heart failure (CHF) vary with their stage of CHF; while Class 1/Stage A CHF may need only regular follow-up, patients with advanced Class 4/Stage D CHF may need home monitoring, virtual visits, medication-assisted treatment (MAT) provided by a pharmacist, and a dedicated nurse care manager.

Categories of care – prevention, acute minor illness, chronic disease management, medication management, and care near the end of life – can guide strategy. These are all important areas to focus upon in care of older adults.

Prevention. An annual wellness visit is an opportunity to comprehensively review and promote prevention measures. Has the practice addressed how and when to offer and schedule preventive visits? Can preventive measures be systematically addressed in all routine visits? Best practice strategy includes the development of work flows, facilitation of the visit with the EHR, and providing the service through the practice team (for example, a nurse or supervised medical assistant can conduct a majority of the visit).

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In patients over 75, the United States Preventive Services Task Force (USPSTF) recommends *against* some preventive activities that are routine for younger patients, such as breast and colon cancer screening.

Acute illness. While in younger patients most acute illness is minor and resolves with time, acute illness in an older patient may need prompt primary care triage. While a younger patient may receive advice over the phone, an older adult with chronic illness may warrant a telemedicine or in-person acute care visit.

Chronic disease management. This becomes more impactful for "rising-risk" patients – those with chronic diseases where attention could prevent or delay the rise in risk from worsening or new conditions as they age. Although many middle-aged patients fall into this category, healthy older patients are at increasing risk for chronic disease development and progression as well.

Frailty is a common clinical syndrome in older adults that carries an increased risk for poor health outcomes including falls, incident disability, hospitalization, and mortality. Clinical frailty scales have been developed to identify this segment of patients for special initiatives.[34] Common frailty markers are impaired mobility, impaired physical activity, and decreased strength. Simple algorithms can be applied to claims data to further identify patients with frailty. Recognition of frailty may help the practice distinguish those patients who need additional medical and social support, support of family caregivers, and advance care planning.

Medication management. Polypharmacy represents significant potential harm for the elderly. "Deprescribing" is a useful strategy to mitigate polypharmacy risk. The Beers Criteria[16] identify high-risk drugs for the elderly, and can be used in conjunction with deprescribing to promote elder patient safety. Dangerous but necessary drugs, such as warfarin, can be more closely monitored. Many of these strategies can be driven by a pharmacist if the practice is lucky to have one on their team.

Strategies addressing medication use can also drive more cost-effective care. Generic drugs comparable to brand-name can be promoted for cost-reduction. Safer prescribing also avoids adverse drug interactions and the hospitalizations and deaths that can result.

Care near the end of life. While advance directives and end-of-life goals of care should be addressed in all elderly patients, palliative and hospice care are critical to helping terminally ill elderly patients achieve their goals of care, and to maximize their quality of life. Care coordination is instrumental.

Prognostication has been deemed a lost art in primary care. In care of chronically ill elderly patients, both patients and providers overestimate the expected outcome and duration of life, even in patients who are terminally ill with end-stage organ failure or cancer. Prognostication tools can trend clinical course and provide a more realistic expectation of quality and length of life for patients, their family, and primary care providers.[35]

Payment is available to practices for advance care planning discussions with patients and their caregivers. Providers need to be educated to provide and document these services for successful reimbursement. Office workflows can be set up to identify the need for and promote attention to advance care planning. Reports on the percentage of elderly patients with advance care plans on record can be run as a quality performance measure.

Social determinants of health (SDOH). For older adults in particular, SDOH significantly impact their health and aging experiences, especially their ability to live independently and age in place. Socioeconomic factors, transportation limitations and food availability, low health literacy levels, loneliness, and social isolation can contribute to greater risk of loss of function and independence, harm, and death. Increasingly, as SDOH are being systematically captured in primary care practices, partnering with community resources becomes a challenging but critical strategy to mitigate the risks. Regional and national registries of community services, such as Aunt Bertha,™ provide a means to access a comprehensive database for patient referral.

Mental health needs. How are mental health needs identified and addressed in primary care? The World Health Organization reports that the most common mental and neurological disorders in the elderly are depression and dementia, which affect approximately 7% and 5% of the world's older population, respectively.[36] Anxiety disorders affect 3.8% of the older population, substance use problems affect almost 1%, and about a quarter of deaths from self-harm are among people aged 60 or above. Substance abuse problems among older people are often overlooked or misdiagnosed. The need for integration of behavioral care into primary care cannot be met by a relatively small behavioral health infrastructure, but transformed primary care practices are increasingly able to meet the need.

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Special payment for coordinated care of mental illness and dementia, as mentioned, is offered by CMS. Provision of this care requires collaboration between the primary care practice and behavioral health providers, and documentation of those efforts in order to submit for reimbursement. Patients and their caregivers must also be incorporated into the care process.

Care Coordination

Care coordination encompasses a variety of roles and procedures foundational to the success of primary care practice. Care coordinators are traditionally nurses with specified roles to promote the safety and continuity of care for patients, especially those with complex medical needs. Nurse care coordinators can function within a practice or remotely. If in a practice, their role needs to be clearly defined to avoid diversion to duties not related to the care coordination. Nurses frequently perform care coordination remotely by phone. Two major tasks assigned to care coordinators are transitions in care, and coordination of care for high-need, high-cost patients.

Care coordinators facilitate transitions of care, especially for recently hospitalized patients. Systems to identify admissions and discharges must be in place to guide outreach. Specific, timely activities must be documented for payment. Inclusion of complex patients' family caregivers, especially in elderly patients, is important to the coordination of care. Documentation systems designed to track and coordinate these activities, either within or alongside the practice EHR, are essential.

As previously discussed, risk stratification, when applied to a practice population, can be used to identify highest-risk patients for care coordination outreach. The care coordinator can then develop a broad perspective on the patient's problems and challenges, and facilitate agile ways to include patient outreach and support, facilitation of communication between treating physicians, medication reconciliation, patient and caregiver education, and arrangement for appropriate health-care and community-based services needed to optimize patient care.

Although nurse care coordinators can be involved in planning care for prevention and chronic conditions and closing quality gaps, these responsibilities can be met with EHR systems that notify patients. Other team members such as medical assistants or pharmacists can perform outreach and facilitate orders for care.

EHR Workflows

We've discussed the key challenges to incorporation of EHR technology into practice; the ability to improve practice processes around EHR use is fundamental to transformation success.

Transformed practices may institute initiatives designed to assist providers with clinical data capture, such as incorporating scribes to enter data real-time into the EHR, or enhancing voice recognition technology to allow capture of clinical narratives. Technical and analytic expertise are employed, whether "in-house" or "outsourced" (purchased through the EHR vendor), to convert collected into reportable data.

Electronic patient portals enable more efficient reporting and communication with patients and are built into most EHRs. Portal use helps to meet performance interoperability requirements. Practices are challenged not only to manage this additional patient service, but also to promote its use. Engaging elderly patients, who may not be as comfortable with portal technology, can involve special communications, programs to promote portal activation, and the enlistment of caregivers or adult children to assist with technologic requirements. Use can enhance access and coordination of care, which is of special benefit for complex patients and their caregivers.

Medical record documentation to accurately reflect patient complexity assists with coordination of care and characterization of a patient panel's complexity. In the USA, ICD-10 diagnosis reporting to capture HCC codes and reflect the complexity of a practice's patients, especially those with complex conditions, is a key component for success in VBC. EHRs have varying capability to prompt providers to enter key HCC codes during patient visits; not all of these codes are directly applicable to the care being delivered at the point of care. Prompts and processes are developed to promote capture. Provider education is required, and feedback on HCC code capture performance can be tied to incentives. Sometimes, outside vendors are employed to use more advanced algorithms to promote HCC capture.

Likewise, EHRs can be optimized to capture the care related to prevention, transitions of care, chronic care management, and advance care planning for older patients.

Measure and Improve Quality of Care

In order to support clinical care and quality improvement work, high-functioning teams develop healthy habits that include:

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- interactions with effective communication
- brief, regular meetings (huddles)
- a collective approach to problem-solving
- agreement on an action plan.

As previously mentioned, primary care practices report on quality through standardized metrics, some of which are specific for elderly patients. Once performance is collected and converted to reportable data, presentation to the practice team is important to promote engagement and improvement. Practice success is ideally reported through a dashboard, the graphic presentation of current practice performance, trends, and progress toward specified goals. Key performance indicators (KPIs) are goals reflecting top practice strategies, and can be identified as targets for incentives.

A standardized process, for example the Plan-Do-Study-Act (PDSA)[37] cycle, promotes transformation success for quality improvement initiatives.

Assure Access and Continuity

A transformed primary care practice will fail if not easily accessible to patients, whether over the phone, portal, or at visits. A frequent challenge is to accommodate acute needs with chronic care and preventive needs in a daily schedule. Usual methods for easier access include assuring an adequate number of same-day appointments, offering appointments during weekends and evenings, reducing no-show visits, "max-packing" office visits to include attention to prevention and quality gaps (with team support) when a patient is in a visit, and considering innovative scheduling processes such as "advanced access" scheduling.[38]

Phones and front office staff can be barriers to practice accessibility. Phone volumes can be studied and mitigated through alternative strategies such as encouraging portal use, automatically refilling chronic medications without a call or visit required when medically appropriate, and making communication with provider or scheduling of a visit simpler.

Continuity is of greater concern for elderly patients. Increasingly, needs of a "regular" office visit can be met through involvement of team members, phone and portal outreach, and coordination of care with family caretakers.

Maintain Financial Sustainability of the Practice

A focus on transformation and team-based care, to maximize efficiency, is critical to financial sustainability of the primary care practice. In the USA, primary care practices are challenged to provide more comprehensive VBC of their patient population within a payment system that is still predominantly office visit- and relative value unit (RVU)-based. Nowhere is this more challenging than in the care of elderly patients. In the USA, straddling a fee-for-service system while building capacity to achieve VBC requires the primary care practice to capture RVUs and revenue while addressing KPIs to maximize quality and shared savings payments. For example, capturing payment for services provided outside of a problem-based office visit, such as for care coordination and annual preventive visits, brings additional revenue while improving quality through better prevention and chronic disease management.

Mitigating losses in risk-based contracts is a growing challenge as US primary care practices are increasingly expected to address financial outcomes. As VBC grows to become the dominant payment model, the transformed primary care practice will be best positioned to provide comprehensive care for the elderly while managing the total cost of care.

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Chapter 62

Health-Care Organization and Financing

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An understanding of the organization of the health-care system enables the clinician to more optimally navigate the system on behalf of the patient and provides important insights that improve the ability of health-care professionals to succeed. These insights also increase the likelihood of financial survival. The US health-care system is frequently described as "broken." To understand how it can be improved requires an appreciation of how it is configured currently. The purpose of this chapter is to provide an overview of fundamental principles of financing health care and insurance. The most relevant programs for the elderly, Medicare and Medicaid, are addressed. There are multiple complex and interacting segments that comprise the health-care delivery and financing system. Where the money comes from, where it goes, and why so much is spent warrant careful consideration. Possible responses to a system under stress that faces the challenges of changing demographics and relentless cost trends are reviewed.

The Health-Care "System"

It is useful to remember that the care of the elderly patient occurs in a complex system that provides and finances care for the entire population including well children, disabled adults, and the uninsured. The many elements of this financing and delivery system are interdependent. Cross-subsidization occurs. This results in all elements being relevant to the elderly patient; however, it is Medicare that has come to symbolize the health-care system for the geriatric patient. Medicare is far more complex than a governmental insurance company paying claims for persons older than 65 years of age. It also pays for care provided to the disabled and those on dialysis; it contracts with private health plans and drug plans to provide required and supplemental benefits to beneficiaries. It has a major role in financing medical education, researching the impact of system change through demonstration projects, and shaping the delivery system. Private insurance often mimics Medicare payment and benefits policies. Medicare is a financing system, not the delivery system. Health care is delivered by providers including doctors and hospitals. There are suppliers including drug and device manufacturers who provide important tools for improving health and preventing disease. They significantly impact the cost and politics of health care. The system also includes private and governmental payers other than Medicare. These payers ostensibly act on behalf of and respond to demands of employers and the voting populace and finance a greater dollar volume of health care than Medicare. Their impact on the system is substantial. Private insurance is important for the elderly, too, as most have some form of coverage beyond traditional Medicare, whether it is employee or retiree coverage, a Medicare Advantage plan, or "gap" coverage. Medicaid is critical for low-income seniors and those in long-term care institutions. The Veterans Administration plays an important role in funding and delivery of care. The most important parts of the health-care system are the patients and their families. The elderly, as a population subset, are individuals of great diversity in employment status and in cultural, behavioral, financial, educational, health, and functional attributes.

There is general popular agreement that our system is seriously flawed.[1] It is often stated that aligning incentives will improve the likelihood that the system will better serve the needs of society. There is agreement on a common goal of achieving a healthy population, but the uniformity of viewpoints stops there. Complex systems also have great inertia, because any change in one part of the system tends to affect another. The ripple from any change may be predictable, based on study of the past, such as service volume increasing in response to price reduction, or for unknown reasons, the use of services by a healthy baby boomer cohort that is accustomed to aggressive consumption of new technologies. The health-care system also exists within a social milieu in which

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health care consumes resources that might otherwise be used for education or housing or income equality, which may have greater effects on health than medical care. Health-care professionals may not believe that the growth rate of the economy is as relevant to their practice of medicine as is the latest treatment innovation for a condition of interest, but it is. Furthermore, the financing and delivery system often seems to be no system at all, with many conflicting interests. Yet clinicians impact the system one patient at a time and have a unique potential to lead system change: a potential that comes with the experience of operating daily at the interfaces of finance, delivery, and caring for patients.

International Perspectives

This chapter focuses on the US system. Much can be learned from other countries. The United States spends more per capita and a greater portion of its GDP than other nations, yet is generally considered to have lower health status than its peer nations.[2] Social factors, universal coverage, the balance of regulation and entrepreneurial liberty, per-capita wealth, age of the population, diversity and homogeneity, national budget policy, delivery system, payment amounts, and benefits/coverage policy are all factors in this equation. For many of these factors there are significant differences between the United States and its fellow developed nations. However, there are also many consistent issues such as human behavior and economic principles and the need for political will to create change.

Insurance Basics

Health insurance serves two fundamental goals: prepay predictable expenses and provide financial protection against the cost of infrequent very costly events. It is well established that the uninsured and underinsured do not receive recommended and necessary services. Traditionally, health insurance allowed the individual to obtain treatment for a major, typically acute illness that would otherwise be unaffordable and forgone. The reason to prepay predictable expenses relates to payment for prevention and to encourage appropriate use of services that will prevent disease, disability, and future medical expenses. Insurance also sets a contracted price compared to charges. It can fund care for persons with essentially no resources, such as those with poverty-level incomes. A growing share of insurance finances go to health care for chronic illness. Insurance also finances, and thus shapes, the delivery system infrastructure.

Insurance is foremost a pooling or spreading of risk across a large population. It works by everyone paying a little into a pool that few will use. Those few who use it will exhaust the pool, less administrative costs and some reserves for unpredicted insurance costs, such as a new drug for hepatitis C. The reserves, besides being a cushion, produce investment income, lowering the amount needed to be collected from each person, known as the premium. In this manner, health insurance is no different than automobile, homeowner, or professional liability insurance.

According to the *CMS Chronic Conditions Chartbook*, in 2017, the 17% of Medicare beneficiaries with six or more chronic conditions accounted for roughly half of the program expenditures.[3] The 32% with 0–1 chronic conditions accounted for only 6% of expenditures. Those with multiple chronic conditions and functional limitations are the costliest. In younger populations where the prevalence of chronic illness is lower, the contrast between the few who spend a great deal and those who use little to no services is starker.[4,5,6] The high-use population is composed of different types of people. Some have one-time high-cost events, such as those who survive major trauma without disability or those who die after an expensive course of care. Others have chronic illness punctuated with rare high-cost events such as coronary artery disease with bypass surgery or chronic illness with recurrent costly events such as congestive heart failure and recurrent inpatient care. But the point is that this variability of high- and low-cost subpopulations allows the application of insurance principles.

The effect of spreading risk works best when it is spread over the largest possible population. Any segmentation creates the potential for the effects of what is known as "adverse selection" or "cherry picking." The United States does not have a system in which the entire population is enrolled in one insurance program. Therefore, adverse selection is very relevant. Consider two health plans: plan A has 5% of its members with a serious chronic illness; plan B has 6% of its members with the same condition. The difference at first appears trivial; however, because the sickest 5% generally account for 50% of all expenses, that means that each 1% change in the sick population will result in a 10% difference in total expenses. If plan B is to make ends meet, it may raise its premiums to cover its greater costs. This will lead people who do not use services and see less value in health insurance to leave to a lower-premium plan, which then only decreases the pool of healthier people over which to spread the risk. Plan B spirals into insolvency. Risk or case

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mix-adjusted premiums might work, but the high-risk people may not be able to afford the higher premium. Adjusting premiums to risk also defeats the principle of pooling risk, unless premiums are paid from a pool, such as premium support programs in a federal health exchange under the Affordable Care Act. Risk adjustors account for a limited amount of the per-person variance, because there remains a significant degree of unpredictability in expenses. Medicare risk adjusts payments to Medicare Advantage plans based on factors such as age, diagnoses, and whether the person is institutionalized. One of the challenges in a voluntary health insurance system is setting premiums for well young adults at a level at which they will purchase coverage. They are indeed low risk and often see little value in insurance that they perceive as inadequately risk adjusted. Thus, incentives or mandates are used to create the largest pool.

"Benefits" are what insurance covers or pays for. Benefit design is a critical element in understanding health-care financing, as it defines what is being paid by insurance. If hospital care is covered by insurance, then the price and volume of hospital services must be calculated and spread over the population paying premiums. If long-term nursing home care is not covered, then its cost and volume is irrelevant. Insurance is a legal contract between the insurer and the insured. Certain services may be essential to the health of the patient, but that fact does not make them covered. Until Medicare created a prescription drug plan, there was no coverage for medications, yet medications were more important than many covered services to the health of most Medicare patients. Medicare does not cover long-term care in a nursing facility, yet it is necessary for many. Benefit design may create or minimize adverse selection risk. Presume our example plan B had a different mix of member complexity from plan A not by random chance but because plan B had slightly different benefits that made it much more attractive to people with chronic illness. By providing better benefits, plan B created its adverse selection. Standard benefits could eliminate this issue, but also reduce choice. Provider network may also create selection risk for a health plan. This same type of issue can affect physicians or physician groups who accept some of the risk by becoming paid by capitation (a per-patient monthly rate). Group A has no geriatricians and has an average complexity patient panel. Group B has geriatricians who attract complex patients. If the two groups are paid the same amount per patient, group B will have to do a far superior management job to make ends meet. There is a great deal of unexplained variation or waste in health-care services utilization, but the disease burden of the patient, not the management skill of the clinicians, will dictate most of the cost of caring for the patient.

Long-term care provides a good example for two other important points: calculating cost offsets or "savings" and behavior change based on who pays. Insurance coverage of a service will make it more accessible. This will expand its use and create a market for providers who will seek to expand it further. If home care is paid fully out of pocket, some will utilize it. If it is insured, the use will grow. Behaviors change with coverage. It is almost certain that some nursing home residents can be cared for more cost-effectively at home with home-care services supporting other caregivers. If $50 per day on custodial home care (activities of daily living support) would prevent $150 per day of nursing home care, it would seem a good buy. It is a gross miscalculation, however, to base savings on the experience expected for one person. Insurance covers populations. In structuring the home-care benefit to cover that nursing home patient who could be cared for at home, it might be that five people at home who need custodial home care, but who are presently paying out of pocket, now become eligible for insurance to pay. The $150 per day "saved" results in $300 per day of home care for six patients, not $50 per day for one. Perhaps five caregivers missed less work, saving their employer costs and boosting productivity and society was better off. The health-care insurance costs were not reduced, however.

This brings us to the point of total costs, insurance costs, cost shifting, and returns on investment. One way to control the insurance cost is to shift medical costs to the patient by limiting the benefit. Medicare Part D drug coverage has beneficiary cost sharing so that it could be affordable to society (at least to the degree that Congress determined). States and the federal government have different insurance programs, but a single patient may be a beneficiary of both. This may create cost shifting and fragmentation. It may create unintended harmful gaps or increase total costs compared to one global coverage. Multiple programs also create multiple administrative processes and added expense. Costs may also be shifted over time periods by making an investment. A diabetes program that delays diabetic morbidity may save an employer-sponsored private insurance company money if an upfront investment in the member's care can be amortized over years of membership premiums and the costliest episodes are prevented or deferred to an age of Medicare eligibility and thus shifted to the Medicare

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program. The insurance company will see a return on the investment, assuming the member remains with them long enough. One of the ironic consequences of Medicare programs helping a person survive another year is that there will be another year of Medicare expense. Medicare is not funded primarily by annual beneficiary premiums; therefore, if Medicare is to have a return on investment, it must reduce lifetime expenditures, which is more difficult. For Medicare, a smaller population is less costly. Private insurance has a per-member premium. Reducing membership reduces income and is not financially desirable.

An insurance company can control costs in limited ways. It can reduce the price of a service or it can reduce the frequency of a service: pay less or buy less. One way to reduce the price is to shift some cost to the member or to not cover the service at all. The provider can be paid less, but payment reductions may stimulate providers to increase volume to make up for lost income. Payment reductions may make a service unavailable because of provider dropout. If the reduction is achieved by shifting the cost to the member, the member may forgo the service. If the service is particularly cost-effective, this would be deleterious. The price change could result in substitution with a costlier service or result in disease progression to costlier stages because of the lack of service. If the service was not cost-effective, the frequency reduction achieved is positive. Attempting to reduce frequency by utilization controls can have the same risk/benefit calculus. Administrative costs will be incurred when implementing programs designed to reduce the volume of services. Seemingly simple actions can have complex unanticipated results.

Financial Principles

In health care it may appear that standard business principles do not apply; however, the delivery system is a business and fundamentals generally apply. The financing system may contribute to confusion over fundamentals. For example, payments may be based on historical charges and not based on current true costs or competitive pricing. That said, every business has income and expenses. It has a product that it provides and makes money per some unit of product. It can have loss leaders or provide charitable services, but it cannot lose money on every service and survive. Inappropriate payment can affect service availability by overstimulating growth of a highly profitable service and causing undervalued services to wither.

A practitioner running his or her office must have a good sense of these realities. There are the rent, the utilities, the employees and their benefits, professional liability insurance, and supplies. Then there is income that is typically fee for service. Each patient is a little different, but overall, there is a predictable gross income per unit of patient service. Income must exceed expenses. There are also variable and fixed costs to consider. One more patient on the schedule will not change the rent or staffing, which are the fixed costs. That patient may use another paper gown as a variable cost, but mostly the one extra patient is pure net income. At some number of additional patients, more staff will need to be added or another doctor asked to join the practice, and there will be expenses associated with a new physician who will not earn their salary on day one.

The same basic principles apply for health-care systems, but there are many confounders. A hospital-employed geriatrician may directly produce income that exceeds their expenses and salary plus benefits and be profitable. The geriatrician might be using only variable costs, if the clinic space and staffing is fixed anyway, but it is likely that fixed costs would be proportionally allocated to each user. The geriatrician may be a feeder of patients to the specialists and hospital. Hospitals have huge fixed costs. A few extra admissions incur few variable costs and are usually highly desirable. If the hospital believes that the geriatrician's patients would not have been at the hospital were it not for their employment, this might be recognized as their contribution to margin. The geriatrician's patients may also give generously to the hospital capital campaigns. A geriatrics service acute and post-acute care unit may reduce hospital length of stay, readmissions, and complications, important in value-based payments. On the other hand, if the hospital concludes that the geriatrician's patients always incur costs of care exceeding reimbursement, the hospital may wish to no longer employ the doctor even if they earn their salary based on the office/clinic income. The bottom line is that regardless of the setting, there is a calculation of productivity or income by some measure and a calculation of costs or resources used. There is either profit and sustainability, or loss and eventual demise. These calculations may be complex and involve assumptions that are controversial or flawed. Ultimately, actual revenue and real expense exist and are likely to be the major consideration.

Other basics warrant mention. The most salient is that in the financing and delivery dyad, costs to one party are income to another. Any cost reductions for payers result in income loss for the providers in the aggregate,

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although not necessarily for an individual provider. The tension is obvious.

Efficient production units lower health-care costs without losing profitability of the unit. They can maintain the same profit margin by reducing expenses even while lowering the price of their services. This is the typical economic paradigm in competitive markets; however, health-care markets are distorted by many factors. A cost-effective clinician is not the same. Cost-effective providers could reduce their income because fewer of their services are used or increase their incomes because they shared in the cost savings, or because the more cost-effective services were the more profitable services for them. Only so much throughput is feasible.

Another important point concerns growth rates and baseline expenditures. In a mortgage, a lower principal will save great sums when interest is applied over time, but the interest rate can be more significant. Baseline expenditures and the growth trend are principal and interest in another setting. An extremely high trend in a service that accounts for a minute portion of total expenditures has an insignificant effect compared with modest growth in services that are responsible for a high proportion of total costs. Sustained high growth rate can, however, make a service that was once an inconsequential expense become very significant. An example of this in health care is the costs of high-cost "specialty" drugs such as the biological agents that once were minor contributors to total costs and, after extreme growth rates, now rival physician expenditures.

Medicare

Medicare was established in July 1966 by Title XVIII of the Social Security Act for people 65 and older regardless of income or health status. It has played a major role in reducing poverty rates among the aged and increasing access to care. It has funded infrastructure development, education, and training, and been a major part of the social transformation of changing health care from a service paid by cash or barter to one paid by insurance. It accounts for one in five dollars spent on health care in America and 15% of the federal budget. It only pays for two thirds of the health-care expenditures of the Medicare beneficiary, with the maximum out-of-pocket limit set at $7,550; average Medicare payments per beneficiary in 2019 were $14,150.[7–9] It has been surpassed by Medicaid as the biggest governmental program by enrollment. Nonetheless, the impact of Medicare on the health-care system and the lives of the elderly would be difficult to overstate. Medicare is an "entitlement" program, contrasted to Medicaid, which is "welfare" or a needs-based program. Medicare benefits and costs were historically the same regardless of beneficiary income or wealth. In 2007, Medicare introduced a variable premium for Part B services based on income. Part D, the pharmacy benefit, which originated in 2006, is supported by income-adjusted beneficiary premiums.

There are four parts to Medicare, and each has different funding and benefits. They are: Part A (hospital insurance); Part B (medical insurance); Part C (Medicare Advantage); and Part D (prescription drug plan). In 1972, Medicare added eligibility for the disabled. People with end-stage renal disease (ESRD) are also eligible. People are entitled to Medicare if they or a spouse are eligible for Social Security, Railroad Retirement, or equivalent federal benefits due to age, or they are eligible due to disability (i.e., they receive Social Security Disability Income [SSDI]), or if they have ESRD. The aged are eligible the first day of the month of their 65th birthday. SSDI recipients are eligible after 2 years of Social Security eligibility. ESRD and people with amyotrophic lateral sclerosis are eligible at the time of initiating dialysis or Social Security payment eligibility, respectively. There are approximately 60 million people covered by Medicare. Of these, 15% are not elderly, but are disabled. Twenty-two percent are also eligible for Medicaid, and half had incomes below $26,200. Twenty-two percent of the beneficiaries have five or more chronic conditions; one third have limitations in activities of daily living; 12% are older than age 85; and 15% are younger than age 65. The population is varied, and as expected, age, gender, and lifelong socioeconomic status are factors.[7]

Medicare has benefits that have deductibles, copayments (set dollar amounts), and coinsurance (percent dollar amounts); that is, the beneficiary pays part of the bill or cost shares for any specific service. Parts B and D of Medicare have premiums; that is, the beneficiary must pay to enroll in that part of Medicare, whereas this is generally not the case for Part A. The "payment gap" created by deductibles, coinsurance, and benefit limits is often covered by "gap" insurance. Many beneficiaries purchase "gap" coverage offered by private insurers. Those eligible for Medicaid have coverage for this liability from Medicaid. Nineteen percent have no gap coverage.[7] The benefits structure for private insurance gap coverage is regulated by Medicare, but there are variations allowed.

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Medicare Part A

Part A is hospital insurance, but it also covers skilled nursing care and hospice. It covers home health care following an inpatient stay. Nearly 40% of Medicare spending is on inpatient services. Part A is mostly funded by a payroll tax of 2.9% of earned income, with additional income from interest on savings in the trust fund and taxes on high-income earners' Social Security checks. The funds are held in the Hospital Insurance Trust Fund and may not be used for other purposes. Expenses are anticipated to outstrip income and savings so that around 2026 the Hospital Insurance Trust will be insolvent (2020 estimate).[8,10] The trust fund has frequent calculations of solvency and has had adjustments made in taxes and other income sources to support it. Like Social Security, it is payroll based and therefore meeting the burden of rising expenditures falls to working people. Part A has benefits and deductibles in a given benefit period. The benefit period is defined by a 60-day continuous break without hospital or skilled nursing care. In 2020, there is a $1,364 deductible per benefit period for a hospital stay. There is a $176 per day co-payment for days 21–100 of a skilled nursing facility (SNF) stay. There is no co-payment for SNF days 1–20, and days over 100 are entirely the responsibility of the beneficiary.[11,12] Hospice covers those with a terminal illness who are expected to live 6 months or less. It provides drugs, medical care, and support for those enrolled in an approved program. It serves over 1.5 million beneficiaries annually and over half of the Medicare decedents.[13] It covers services typically not covered by Medicare such as respite and grief counseling. Hospice care in the home has no beneficiary cost sharing.

Medicare Part B

Part B covers medically necessary services furnished by physicians in settings such as the hospital, office, or ambulatory surgery center. Home health (not following an inpatient stay), ambulance, durable medical equipment and surgical supplies, clinical laboratory and diagnostic services, and services by certain practitioners (e.g., nurse practitioners, clinical psychologists, and physical therapists) are paid by Part B. It covers the facility and professional components of outpatient services as well as drugs and biologicals that are not self-administered or otherwise part of Part D. Physician payments account for 14% of Medicare expenditures, with hospital outpatient departments receiving 10%. Part B is funded mostly by the general treasury, with premiums from beneficiaries accounting for 25% of program support. Premium payments in 2020 were $145 per month for 94% of beneficiaries. Premiums are adjusted annually. Premiums vary for higher-income individuals. (In 2020, monthly premiums were between $145 and $492 per month for the beneficiaries who had the highest income.) Funding for Part B is through the Supplemental Medical Insurance Trust Fund. The Supplemental Medical Insurance Trust has a Part B and a Part D fund within it. Part B services have an annual deductible ($198 in 2020) and a coinsurance of 20% of the allowance. This means the beneficiary is responsible to pay 20% of the allowed charge (Medicare's fee for a provider). For certain preventive services there is no cost sharing. Note that the allowed charge is made up of the amount Medicare pays and the amount the beneficiary (or the gap insurance) pays.

Medicare Part C

Part C is the Medicare Advantage (MA) program. These are private health plans that receive Medicare payments to provide actuarially equivalent benefits. The Part A and Part B benefits are both provided, and the deductible, co-payments, and coinsurance are typically covered by a premium charged by the MA plan. In addition, the beneficiary must continue to pay their Part B premium. Although MA plans cover the Medicare deductibles, co-payments, and coinsurance, they typically have their own forms of cost sharing, such as office visit co-payments, which can be substantial in the aggregate for high utilizers. Most MA plans include Part D. Therefore, these plans are typically A, B, D, and gap coverage all rolled up into one product. Covered benefits must include all services covered by Medicare, but typically additional services such as more extensive preventive care are added. Part C accounted for 30% of Medicare spending in 2017. With the advent of Part D and other factors, plans have grown, and approximately one in three beneficiaries is in an MA plan. For some high-service-use beneficiaries, out-of-pocket costs may be higher than traditional Medicare in these plans. However, for many it is a low-cost alternative.[7,14] Because MA plans represent a subset of the population, adverse selection or the converse "cherry picking" becomes a concern. This is worsened if beneficiaries can move freely back and forth between plans and traditional Medicare. If the MA plan is restrictive but less costly, patients may move into the traditional program when they become ill and the loss of premium savings is outweighed by ease of service use

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found in traditional Medicare.[15] For this reason, the Centers for Medicare and Medicaid Services (CMS) has created lock-in provisions restricting movement.

Medicare Part D

Part D is the prescription drug benefit that 45 million Medicare beneficiaries have. Other Medicare enrollees have coverage through plans such as retiree or employee benefits so that more than 90% of Medicare beneficiaries now have drug coverage. It is funded with premiums (averaging $40/month, varying by plan selected), general revenues, and state revenues that previously were used for Medicaid prescription drug coverage. Part D coverage is provided by private insurers approved by CMS. There is a low-income subsidy for premiums. Part D is not optional for Medicaid enrollees who are also Medicare beneficiaries (the dual-eligibles). There are phases of coverage: the deductible, initial coverage, gap ("donut hole"), and catastrophic coverage. The standard plan for 2020 had a $435 deductible and 25% coinsurance after the deductible. In 2020 the coinsurance remained 25%, eliminating the gap effectively. Beneficiaries may receive help in the out-of-pocket expenses from family or state assistance programs, but not the Part D plan. After $9,719 of total drugs costs ($6,350 in out-of-pocket spending), there is a 5% coinsurance. However, plans do not use this standard design. They have copays rather than coinsurance and may eliminate deductibles. Part D plans have formularies with preferred drugs having different coverage than other drugs or noncoverage for some drugs. CMS requires certain drugs to be on all formularies, and designated classes must have a representative drug. Physician-administered drugs remain on Part B. The Part D benefit is costly, with 13% of Medicare expenditures, very nearly the same as physician payments, going to Part D.[16]

What Medicare Does Not Cover

Medicare does not cover acupuncture, routine vision care and eyeglasses, dental services, hearing examinations and aids, and long-term custodial care. Care outside the United States is generally not covered. It does not cover all preventive care, specifically an annual physical examination for preventive purposes other than one for new enrollees. With the advent of the Affordable Care Act, the recommendations of the US Preventive Services Task Force now affect coverage. Because Medicare covers skilled nursing care and skilled home care, the greatest misconception of the public regarding coverage relates to these services. Many beneficiaries believe long-term nonskilled or "custodial" nursing home care is covered. That is incorrect. Medicare benefits are set by statute. This means Congress defines the coverage, not CMS. CMS may provide greater detail but is not able to add benefits outside an existing category of covered service. Medicare beneficiaries spent an average $5,460 in 2016 for health-care costs because of premiums (42%), cost sharing, and services that are excluded from coverage.[17]

Medicaid

Medicaid was begun in 1965 as a state and federal shared program to help the poor with medical expenses. It is Title XIX of the Social Security Act. The program was established so that it would be funded by state funds with federal matching monies. States could determine eligibility (with some restrictions set by the federal government), select the covered services and payment rates, and administer the program. It is "needs based" with eligibility determined by income and asset limits; that is, it is a welfare program. Over time, the program has changed, and the federal government has placed some further requirements on the states for them to receive matching funds, but fundamentally it remains state specific. Therefore, eligibility and benefits do vary, and a beneficiary who moves from one state to another may be at risk for losing coverage. Medicaid covers poor women and their children, the blind or disabled, and the medically needy, and it funds the Program of All-Inclusive Care for the Elderly (PACE) along with Medicare. The medically needy elder is typically a nursing home resident who was always poor or who has exhausted his or her savings and "spent down" to eligibility. PACE participants receive all their services through the program and require a nursing facility level of care, but are managed in an alternative setting by using flexible benefits not typically provided by Medicare. Seventy-five million Americans have Medicaid. It funds 16% of all personal health-care spending.[18] Twenty percent of all Medicare recipients are "dual-eligibles" who also have Medicaid. Over 60% of all nursing home residents are funded by Medicaid, though Medicaid spends more on community-based long-term care. It provides gap coverage for deductibles, copayments, and coinsurance for Medicare benefits, and prior to 2006, paid for prescription drugs.[19] Because of the high per-person costs for the elderly nursing home

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resident and the disabled, the aged and disabled comprise only 25% of the Medicaid population, yet account for two thirds of Medicaid spending.[20]

Delivery System Financing and Organization

There are multiple components of the delivery system, but we will describe only a few key provider types.

Hospitals

There are approximately 5,200 hospitals in the United States. In the past two decades, the number of inpatient days per 1,000 persons has steadily dropped.[21] Over the same period, the outpatient hospital services growth has been strong. Hospitals have also consolidated, giving them much greater market power in negotiating rates with private payers. This has been useful as they have seen their profit margins from Medicare decline. Since the 1980s, hospital inpatient services have been paid by Medicare with a prospective payment system called Diagnosis-Related Groups (DRG). A hospital receives a single payment for an inpatient admission based on the diagnosis that caused the admission. Procedures and complications affect the DRG selection, and there are special allowances for extreme outlier stays. The goal of such a system is to create incentives for the hospital to improve efficiency. Hospitals also receive payment updates or penalties based on quality performance measures. Private payer inpatient payment methodologies vary by payer and hospital.

Physicians

Physician supply has steadily increased; however, most of the increase has been in non-primary care specialties. The growth in larger group practices that could support infrastructure investments such as electronic health records is significant. The physician workforce is changing; women now outnumber men in medical schools, and more physicians are employees than in the past. Physicians are paid by Medicare on a fee-for-service or "piece-work" basis. In general, the physician payment methodology by CMS has a financial incentive to perform more services. Since 1992, all Medicare fees are determined by a conversion factor and relative value units (RVUs). This is called the resource-based relative value system (RBRVS). Services of all types (procedural and evaluation and management) are ranked in a relative manner based on physician work. Work is determined by time, technical skill and effort, mental effort and judgment required, and stress due to risk to the typical patient. RVUs are assigned using a previously valued service as the reference or comparator. Valuation based on relativity to a reference service is the origin of the term "relative value." Practice expenses are then calculated based on the expenses incurred for clinical staff, supplies, and other costs associated with a specific procedure, and converted to RVUs using a mathematical formula. Finally, professional liability costs related to a specific procedure are estimated and given an RVU amount. The three components of the RVUs are converted to dollars by using a conversion factor (CF). For example, five RVUs with a CF of $30 would result in an allowance of $150. Practice expenses vary by whether a procedure is done in a facility, whereby the facility incurs the expense, or whether the service was non-facility; that is, in the doctor's office.

A committee of physicians representing multiple specialties (including primary care-oriented specialties and geriatrics) is convened by the American Medical Association (AMA) to oversee the valuation process and make recommendations for RVUs to CMS. CMS can accept the recommendations or not, but the acceptance rate is high. The RBRVS methodology replaced a physician charge-based system and was intended to rationalize payment and reduce a disparity favoring procedure-oriented specialties. Congress defined the basics and CMS determines significant details. The ultimate valuations are based on work, practice expense, and liability costs, which is the basis for it being called "resource based." It is noteworthy, and a source of some criticism, that the current methodology does not create payment based on concepts such as cost-effectiveness, quality, value to society, or the need to support a primary care infrastructure. Other payers do not necessarily use the Medicare payment methodology, but the majority use some variant.

The highest volume and highest total cost service in medicine is the office visit. The cost per service is relatively low, but the volume is staggering. RBRVS values office visits and other evaluation and management services based on different levels defined by the medical decision-making or time (effective 2021). Included in the valuation is some amount of work before the face-to-face service, such as reviewing a record sent in advance, and some amount of work after the service, such as dictating a consult note or following up on a laboratory result with the patient. The evidence that the valuations inadequately recognized the work and practice expense of care coordination required for the medically complex,

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chronically ill patient has led to some payment changes such as Medicare paying for chronic care management on a monthly basis starting in 2015. These services have been found to be cost-effective.[22]

The process by which a physician is paid starts with the reporting of that service to a payer using a current procedural terminology (CPT) code. CPT is the nomenclature system maintained by the AMA. Payers (in Medicare, CMS) assign each code a coverage determination (covered, not covered, or included in the payment of another service) and a fee. The claim a physician submits will be processed through an electronic claims system that may have "edits" based on multiple factors such as diagnoses or coverage criteria. The rules for correct procedure code selection, knowing what Medicare covers, and payment processing steps across different payers can be confusing and require education.

CMS assigns an allowance for every procedure. Physicians who participate in Medicare (i.e., agree to the Medicare fee as payment) are paid the allowance, less coinsurance and deductibles, directly by Medicare. Physicians who do not participate in Medicare may bill the patient, but only up to 109.25% of the allowance, and then the physician must collect from the patient who is the recipient of the Medicare payment. Physicians who "opt out" of Medicare privately contract with the patient and are not bound by any allowance, and neither the patient nor the doctor may receive Medicare funds for the doctor's service. The physician who opts out is completely out of Medicare. The patient who sees such a physician may still receive services paid by Medicare from other providers. Medicare physician participation rates and rates of physicians willing to see new Medicare patients remain high and steady despite Medicare physician fee updates not keeping up with practice cost inflation. Physician payment is likely to undergo significant change in the years ahead as Medicare seeks to move away from a fee-for-service method. Quality metrics are increasingly applied. Demonstrations in advance primary care practices (patient-centered medical homes) and accountable care organizations are harbingers of different incentives to organize and transform.

Nursing Facilities

Approximately 4% of all Medicare payments are for skilled nursing facility (SNF) care. SNF reimbursement is based on a variation of the DRG. Facilities receive a global per diem payment that covers all services such as nursing, therapies, room and board, and medications. The payment is based on a Patient-Driven Payment Model related to the type of care the patient is receiving.

Perspectives on Health-Care Costs

Health-care costs have outpaced inflation and the growth of the economy so that an increasing percentage of the GDP is devoted to health care. If the GDP pie grows sufficiently, the non-health-care portion or slice in absolute size may be larger in successive years, even if it is a smaller portion of the pie. Consumption of health care does correlate with wealth in international comparisons and by socioeconomic class in America. All the GDP will be used for something – why not health care? Increased life years, including when adjusted for quality of life (function), have resulted from the effectiveness of the health-care system and almost certainly not just from other effects such as secular dietary changes and no-smoking policies. A part of the reason for increased volume of services is that more meaningful care can be provided for an expanding group of patients. We now do better controlling diabetes and hypertension and detecting breast cancer in early stages. Costly drugs really do help people with otherwise disabling or lethal conditions. The cost of health-care insurance, however, is rising at a rate that is suppressing wages and leading employers to discontinue or reduce coverage. Many elderly people are using a growing portion of their income for health care. The federal budget is strained by cost trends that are independent of demographic change, which itself will further stress the budget. Health is determined by more than health care. Spending on housing, education, and nutrition may be much more effective in promoting the next advance in function or longevity than spending on, for example, a new drug to treat advanced malignancy. Studies on regional variation in spending on health care indicate that higher expenditures are inversely correlated with measurable quality. Concerns about quality, value, and sustainability indicate that the cost trend warrants critical review.[23]

Many have examined costs in America. Although there may not be unanimity of opinion, there are some summary conclusions that can be reasonably made. It is useful to note that there are likely many factors, and an improvement in any one area should not be cast away as irrelevant. No magic bullet is likely. Demographics account for a relatively small aspect of the trend. Care at the end of life does consume a large percentage of total expenditures, but this is true at all ages. The costs are growing at the same rate as other

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services, and the costs for the very old are not due to futile intensive care unit days, but long-term and palliative care. Competition has had mixed effects. When payers with market clout forced hospitals and others to compete for patients by price, there was a period of trend reduction. More typically, competition has resulted in one facility trying to outdo the other in technology arms races that fuel the fire of cost growth. Price is a factor, with drugs and devices costing more than in other international systems that negotiate better with industry. Physicians earn more relative to the rest of the population when compared with western European countries. Administrative costs are significant, typically described as the low single digits for Medicare and 10–15% for commercial insurance.[24,25] This has been fairly stable, is not the bulk of costs, and may include investments in technology and services that will improve efficiency and quality. There is still an opportunity to create savings from better administrative efficiency and reduced marketing costs. Disease frequency, some of which is preventable by lifestyle interventions, may account for significant differences in per-capita spending in international comparisons. Many feel that the biggest factor is the use and rapid diffusion of new technology, typically without a strong evidence base of utility established at the time of diffusion. Cost-effectiveness is rarely assessed.

Attempts to rein in costs have had mixed effects. Fraud, although rare, exists and must be rooted out continuously. Fraud reduction programs generally more than pay for themselves. Payer-created price reductions often resulted in cost shifting to other payers or increased volume of services to maintain gross income. Copayments or deductibles designed to moderate patient demand obviously shift cost to the patient, but the sickest patients account for most of the costs. Their services are typically the least discretionary, so savings from reduced utilization may not result. Removing some cost insulation may lead to consumers being more aware of and engaged in solving a national social problem. A forceful control on technology diffusion does forestall costs for a while but requires a political will that is currently lacking. Systems that operate within a budget such as an integrated delivery system that also provides financing or assumes financial risk do best in cost control. In our pluralistic payment system, budgets are not like those of single-payer systems in other countries and accordingly are harder to enforce. Furthermore, the delivery system is not a system organized with the capacity, authority, and information systems necessary to control expenditures within a national or regional budget. A primary care focus has had success, although restrictions by "gatekeepers" have not.

It is hoped that costs can be controlled by reducing duplication of care, errors, and other waste through use of an electronic health record and other delivery system changes. The same information systems can facilitate more effective use of evidence-based medicine. In an example of misaligned incentives, the cost of the system changes may be borne by a provider, but the benefit may accrue to the financing system. Changing payment from one that favors new procedures and volume of services to one that supports chronic disease care using teams, nonface-to-face interventions, and community resources (Edward Wagner's Chronic Care Model [CCM]) may be effective, if not in saving money, then in improving care.[26,27] Chronic care management and management of transitions has had favorable results.[28] There needs to be a substantial investment into better understanding comparative cost-effectiveness of medical interventions. Only now, after years of resistance, are we developing ways to measure quality and efficiency and make them transparent and actionable for the provider and consumer alike. However, although the notion of "value-based payment" is an admirable concept that is growing in real-dollar relevancy, much remains to be done in proving its validity. Ideally, it will fund transformation and stimulate positive change rather than just be used as a reward for a nice report card. There will be stumbling along the way as this process matures. The financing and payment system must support a transformation in health care, even while there is legitimate concern about the predictability of the result of many interventions. There will be those who are adversely affected by change and will oppose it. Ultimately, all these efforts to improve our system will still require a social calculation of value. The transformational process is underway. All members of our society must make it work for the patient.

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Chapter 63

Ethical Decision-Making

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One might ask, if the ethics of medicine is meant to represent common and immutable truths, why would we need any special attention to ethics in the geriatric patient, above and beyond that which applies to everyone? The fact is that even though ethics may concern itself with universalizable rights and wrongs, its proper application must vary with the clinical situation and therefore with the individual patient. It is not irrelevant that a patient may be aged, but this alone will not justify a universalized approach, because the effects of age may range from nil in the independent and competent elderly patient, to being the most important factors, ethically and medically, in the patient with age-related disability or dementia.

Just as a clinical assessment of an older person looks to functional capacity, mental status evaluation, and the recognition and treatment of reversible illness rather than simply stopping with age and a list of diagnoses, so ethical decision-making in the care of the elderly uses ethical principles in assessing a particular decision but must fully comprehend the actual situation of the older person.

Our recent experience with COVID-19, which has disproportionately affected the elderly as well as causing widespread clusters of deaths in long-term care settings, lends urgency to considering ethics in the way we care for older persons. Some guidelines were published that looked at age alone as one criterion to limit care or the use of scarce resources for older persons ill with the COVID-19 virus. Using age rather than a careful look at the individual person risks unfair allocation decisions and the potential for loss of lives that would otherwise be able to recover.

General Principles

It would be good, therefore, to start with some general principles, and from there consider particular applications. A variety of approaches to ethical analysis exist; one of the most recognized is based on prima facie principles.[1] These include *autonomy*, *beneficence*, *nonmaleficence*, and *justice*. Autonomy (from the Greek for self-rule) requires both liberty and the capacity for intentional action. Nonmaleficence is the avoidance of doing harm, and beneficence is the obligation to do good. Justice in this context is usually concerned with the fair distribution of resources. In applying these principles to ethical analysis, they prove most helpful in characterizing an ethical dilemma as a conflict between two principles, both of which we would wish to honor. Therefore, a wish to honor a patient's autonomy, for instance, could conflict with our wish to provide beneficent treatments or minimize avoidable harms. Because autonomy in particular can weigh heavily in moral dilemmas with elderly patients whose capacity may become impaired, it is useful to remember that autonomy means not only "self-rule" but is also based on "respect for persons." Thus, we may be able to find ways to respect an individual's autonomy even as their capacity for decision-making may diminish.[2]

Although principles are very useful for framing an ethical problem, they may be less effective for determining a proper resolution. Other philosophical systems, such as deontology, consequentialism, or virtue-based ethics, may prove useful here. Deontology proposes to search for the right rules, which, if followed, will lead inevitably to ethical actions. Another approach, consequentialism, will look to a good outcome as the indicator of a morally good action, and is often formulated as doing the greatest good for the greatest number. In recent years, a revitalization of virtue-based ethics places the focus on the character (and virtues) of the one doing the action, as well as the intention underlying that action. This is far from an exhaustive list; other methodologies are often employed, ranging from feminist, care-based, narrative, communitarian, to theological ethics, all having their uses, as well as their strengths and weaknesses. In reality, most individuals other than philosophers, when approaching an ethical dilemma, do not typically employ a single methodology for every case, but may adapt their thinking to the case at hand, often using a somewhat blended approach.

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The Patient as Decision-Maker

When faced with the need to make decisions that are both medically and morally challenging, the ideal situation would involve a patient who is an autonomous adult with full decision-making capacity. In many cases the patient's full decision-making capacity seems self-evident. In doubtful cases a variety of evaluative instruments have been proposed.[3]

It should be remembered that capacity not only varies with age and state of health, but also is specific for certain types of decision-making. Therefore, one might have capacity (a medical judgment) for making treatment decisions for oneself, even after the same patient has been declared incompetent by a court (a legal determination) for handling his or her own financial affairs. Unfortunately, patients' capacity to make their own decisions is often not called into question until and unless they disagree with the recommendations of their physicians. A determination that a patient no longer has decision-making capacity should follow the procedures prescribed by hospital policy or state law. Most frequently the determination may require the concurrence of two physicians, and may even require that one of them be a psychiatrist.

For those patients who are deemed nonautonomous – that is, with diminished or absent capacity – there are still methods to honor their autonomy. Foremost among these is the application of their previously indicated wishes. These may be no more than verbal declarations, but in the best case they would come in the form of a legally binding advance directive. Advance directives are a group of differing documents that attempt to preserve the informed wishes of an individual for health care if that person is no longer able to participate in medical decision-making. This can occur in the course of an acute illness or injury or during a chronic process, such as a dementing illness, that renders the person incapable of decision-making. The point of advance directives, from an ethical perspective, is to honor the autonomy of persons so that they control what happens with their medical care when they cannot speak for themselves. Advance directives allow autonomous desires expressed prior to illness to direct care in these situations.

An advance directive comes into effect when a person is deemed no longer able to make medical decisions. Different jurisdictions have differing laws governing the determination of medical incapability to make decisions; in some areas a single physician can make this determination, in others two are required. Once such a decision is made, the wishes expressed in the advance directive, or the decisions made by a surrogate or proxy, in consultation with the treating physician, are generally to be respected. In the absence of an advance directive, clinicians are forced to look to the next of kin. Again, what constitutes the next of kin depends on different localities. Although normally it would be the spouse, in the absence of a spouse there can be a variety of possibilities about how a sibling, a child, or a person with close personal knowledge of the person can be a valid spokesperson. On occasion, uncertainty and disputes among family members can lead to a court appointing a spokesperson. The creation of advance directives is meant to avoid these conflicts and confusion.

Limitations on respecting advance directives and not following the wishes expressed in the document or conveyed by a surrogate could occur in at least two situations. The first is when there is concern about coercion in the formulation of an advance directive and a reasonable suspicion that there was pressure put on the person to make choices that are inconsistent with their previous wishes. The second is when the advance directive or surrogate directs choices that are ethically impermissible for the clinician to follow. This could include directives to euthanize or assist in the suicide of a person or to withhold all feeding or hydration, including ordinary feeding by mouth. The creation of advance directives that stipulate deliberately life-ending actions at a certain time is also ethically problematic. Directives such as these could be made by a person early in the course of cognitive impairment when their dementia reaches a certain degree. Aside from the problem of physician participation in assisted death, such a document raises the question of whether a person who is now cognitively impaired would at this point consider their life worth living yet not have the opportunity or means to express dissent from the directive made earlier.

Advance directives can take many different forms; it is worthwhile to think of advance directives as a genus with varying types of advance directives as species. One of these species is the fairly common decision of an individual to choose not to be resuscitated, intubated, or hospitalized. These orders, DNR, DNI, and DNH, usually come into play in an acute crisis that makes it obvious the individual is not able to speak up.

Other advance directives provide more fulsome guidance, with different types legally valid in different jurisdictions. One form is a Living Will where a person will express wishes about life-sustaining treatment. Living Wills can be a general statement or a checklist of specific

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medical procedures. These can be problematic if the statements are overly broad or if the checklist of choices leads to confusion in situations that might not have clear application in a specific clinical scenario. An alternative approach is the appointment of a durable power of attorney for health care (or a health-care proxy). In these situations, a surrogate spokesperson is appointed to consult with physicians when the patient is no longer capable. It is important to recognize that the durable power of attorney for health care or the health-care proxy is expected to act in the best interests of the person and not substitute their own preferences. This is even more emphatically true if there is any record of specific preferences indicated by the patient prior to the crisis. It is best that the physician provide a recommendation or a series of reasonable choices and ask the surrogate their best opinion of what the patient would decide if they were able to make a choice. A durable power of attorney for health care must not be confused with the more general durable power of attorney, which allows an individual to make business, legal, and financial decisions for an incompetent patient, but does not empower them to make health-care decisions in any way.

Documents referred to as POLST (physician orders for life-sustaining treatment) and MOLST (medical orders for life-sustaining treatments) orders are relatively new species in the genus of advance directives. Unlike Living Wills or durable power of attorney for health care, these are orders signed by a physician and are meant to provide guidance, particularly to emergency responders, if a patient becomes suddenly and seriously ill and cannot speak for themselves. They are perhaps most useful in the setting of a hospice or terminally ill patient at home who may have a medical emergency but would not want resuscitative efforts initiated. These documents are brightly colored and should be placed in a prominent location so first responders can rapidly review the orders. Some POLST and MOLST forms allow for a variety of orders about antibiotics, hydration, and other interventions. Moving beyond orders about the immediate emergency, however, does raise the risk of an unforeseen clinical situation where the document would limit some treatments, including those that are potentially of a palliative nature, when the clinical circumstances would suggest such a course. Arguably, a POLST or MOLST is best used in the initial crisis, and then a surrogate designated as power of attorney for health care or health-care proxy could best work with the physician and care team to ensure good choices are made in a variety of situations that a MOLST or POLST does not consider.

The process of formulating an advance directive has two parts: a conversation and then documentation. Those physicians who care for older persons should initiate the discussion or assist if the patient brings up the topic of life-sustaining therapy. Although it makes sense for people of all ages to provide guidance about their care in an advance directive, for an older population with the increasing risk of illness and sudden incapacity, it is even more crucial to formulate advance directives. Physicians can have these discussions as part of routine care and assessment as well as when there is evidence of a developing illness. Particularly important are situations when an older person is scheduled for a major surgical procedure with enhanced risk. It is tragic when a prolonged course of aggressive and possibly physiologically futile treatment follows postoperative complications, and there was no attempt to secure an advance directive preoperatively. Likewise, in the setting of an early cognitive disorder, beneficent behavior on the part of the physician would lead the patient to discuss treatment goals and develop an advance directive prior to a loss of decision-making capacity.

The role of the physician, as in any treatment discussion, reflects the need for clinical insight to ascertain the patient's goals and work to provide the appropriate means to meet those goals. Some older people would reject the possibility of mechanical ventilation outright. A physician knowledgeable in the frequency of reversible illness in older persons, especially previously healthy individuals, would inquire about a preference if there was a good chance ventilator support would have a favorable outcome. Advance directives made in lawyer's offices are less likely to consider reversible illness and more likely to view choices for medical interventions as yes/no binary options, as well as to neglect the possibility of time-limited trials. The point is to ascertain the goals of a patient and help in creating an advance directive that has clinical flexibility in meeting those goals, usually with the addition of a surrogate as proxy or durable power of attorney for health care who can assist when outcomes are unclear and decisions need to be made after an immediate crisis. Likewise, involvement of the physician and care team is vital in accessing an advance directive in an emergency. Documents kept in safety deposit boxes or an attorney's office will not be accessible on nights and weekends. Keeping the document in the medical record can assist in rapid recovery, particularly in settings where an electronic medical record is available.

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The documentation process can be accomplished by a physician and patient, although there may be a desire on the part of the patient to have an attorney assist in drawing up the final document and also attending to other advance planning matters such as creating a will and a durable power of attorney for financial concerns. Most jurisdictions do not require an attorney to create a valid advance directive. There are, however, specific regulations about who can be appointed as proxy or durable power of attorney for health care, the need for witnesses, and who are considered appropriate witnesses.

Unfortunately, nothing about a legally mandated hierarchy ensures that a specifically ordered relative would have knowledge of the patient's values and preferences or even be inclined to employ them. In those less common cases where there is no morally or legally valid proxy and no indication of the patient's prior preferences or values, decisions can only be made using a best-interest standard, frequently after the appointment of a guardian by the court. Attempting to apply such a standard can be particularly problematic. Some have suggested an actuarial approach to predict patient preferences;[4] others suggest that the patient's apparent acceptance and comfort with their current status should be considered more determinative than their previously stated wishes, as a truer reflection of respect for persons.

Goals of Care

All medical decision-making should focus on, and be in accord with, goals of care that have been determined by the treatment team, and agreed to by the patient or the patient's surrogate. These discussions should be routinely triggered when discussing prognosis or treatments with low probability of success, or the patient's hopes and fears, or any time the physician can anticipate a remaining lifespan likely limited to 6 to 12 months. A model for this decision-making interaction has been described as "Beneficence in Trust."[5] In this model, the physician and patient work together for an outcome that is mutually beneficial, and is both right (the medically appropriate action) and good (the desirable action, according to the patient's values). The physician's expertise is crucial in determining what is medically right, and therefore making an appropriate recommendation. Yet, we must remember that nothing about a medical education makes the physician's judgment about what constitutes a good outcome for a particular patient take precedence over the patient's own values. Although focus must remain on the patient's good, the locus of the conversation may vary widely. Ideally such conversations would be included in some fashion in every outpatient encounter. In reality, a detailed discussion rarely takes place in this setting, even when the patient has a progressive, potentially terminal disease. It is a difficult conversation at best, made more problematic by the time constraints in an outpatient setting, and unfortunately represents uncompensated time when many physicians are held responsible for all their billable minutes. A less desirable location is in the lawyer's office; although patients should be encouraged to have a last will and testament and any other necessary financial arrangements, this is not the optimal setting for discussions about their end-of-life care. In this setting, conversations that are primarily medical, not legal, may result in an advance care document that is more legally than medically appropriate. When a patient is admitted to the hospital or a nursing home, especially when they have recently been hospitalized for a severe progressive illness, a discussion of end-of-life care becomes more urgent. If the patient is facing a poor prognosis with the prospect of severe suffering or imminent death, or themselves have brought up hospice, palliative care, or a desire to die, such a conversation is crucial. Inexplicably, even these conditions do not always trigger timely discussions.

Actions and Intentions

A discussion of goals may lack clarity when the motives or intentions behind certain actions are not made explicit, either by the physician or the patient. It is true that not every bad outcome is associated with bad intentions; in fact, this is not usually the case. Good actions can also lead to a bad outcome that may be foreseeable but unintended, and can be justified by applying the Principle of Double Effect. According to this, a morally good or neutral act may be justifiable even when a bad outcome can be foreseen, if certain conditions are met. Only the good outcome must be intended; the bad outcome, even if foreseeable but unintended, and the bad act cannot be the means to the good outcome. This must take place in a situation where the risks are proportional to the good that can be achieved (see Box 63.1). The classic example is the use of morphine in sufficiently high doses for pain relief in a terminal patient, even when there is a foreseeable risk of an earlier death to the patient due to respiratory suppression. The intention here is pain relief for the patient, but not pain relief by means of terminating the patient's life. The true intention is made clear when escalation of the morphine dose is stopped at

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Box 63.1 Principle of double effect

I. **Act** – Morally good or neutral
II. **Intention** – Only the good effect desired
III. **Means** – The bad effect is not the means to the good effect
IV. **Proportionality** – Good effect outweighs the bad effect

the point of pain relief, but at a point that does not necessarily result in an accelerated death to the sufferer.

Other axioms can also prove useful in seeking appropriate goals, such as those regarding quality of life, and withholding and withdrawing treatments. The first guideline is that quality-of-life judgments should be made according to the individual's own value of their life and is not to be a judgment made by a third party. This will have a great bearing and even change the nature of discussions about the benefits and burdens of a particular treatment. Although it is not uncommon to hear someone say, "I wouldn't want to live like that," the important point is not whether you would want to live like that, but whether the patient is willing to do so. We are notoriously bad at predicting the tolerability of conditions experienced by others; only they can ultimately make a proper judgment about whether their life is worth living and medical interventions are worth enduring. Also, it is routinely accepted that both the withholding and withdrawing of life-sustaining treatments are morally equivalent. Although they may at times feel different to the involved parties, the comparability of such actions in a dying patient is found not only in their similar outcomes, but also in their equivalent intentions. Both are aimed at allowing the patient to die without an unwanted prolongation of their dying by burdensome but ineffective life-sustaining interventions.

Actions That Result in Death

Finally, the question must be asked: Are there goals or intentions that should be considered morally impermissible? Most commonly, these would arise in a discussion of killing versus allowing to die. Most recently, the discussion has extended to the morality of euthanasia and physician-assisted suicide (PAS). Let us consider each of these in turn. The distinction between killing and allowing to die has enjoyed a long tradition in medical morality. The direct and intentional killing of one's patient has been discouraged or forbidden in the Hippocratic and Judeo-Christian traditions throughout the centuries. Some confusion or even denial regarding the difference has played a significant role in the discussions of legalization of euthanasia and PAS. It is important, therefore, that if the distinction is real, it should be understood clearly for what it is and what it is not.

Some would argue that the difference is based on active versus passive "killing." This will not take us far, because the act of turning off the respirator in a terminally ill patient, a classic instance of allowing to die, is clearly active and not passive. Causation and intentionality have also been considered important, where the death is expected to be the result of an underlying illness. In order to clarify the differences, these definitions have been offered: "Killing is an act in which an agent creates a new lethal pathophysiological state with the specific intention of causing a person's death" and "Allowing to die is an act in which an agent removes an intervention, which is opposing a pre-existing fatal condition, or does not begin that intervention."[6] The second definition was phrased in that way to acknowledge that allowing to die could occur with the intention of causing the death (passively) or without directly intending the person's death. Under this concept, all killing would be wrong, but also some allowing to die could be wrong, when it is done with the intention of making the individual die. For those who do not intend the death of the patient in a withdrawal of support (which allows them to die), there may be no discernible difference in the action itself. If I unplug the ventilator, how does anyone know what my intentions were? I might be intending a patient's death or I might not. It is in this ambiguity between concrete actions and discernible or opaque intentions that the confusion about the real difference is found.

It might even be difficult for the physician performing an "unplugging" to be certain of his or her own motives. Nevertheless, there are some indicators that may clarify the different intentions. For instance, if the patient is taken off the ventilator but then begins to spontaneously breathe, does this make the physician think it was a failure? If the life-sustaining treatment is withdrawn without the subsequent death of the patient, is the next question asked, "Well, now what can we do?" Wouldn't these reactions be indicators of an intention not to simply remove an unwanted burdensome or ineffective intervention? Doesn't it begin to look like the burden to be removed is the burden of the patient or the patient's life?

In traditional medical ethics, intentionally killing our patients is considered morally wrong, and therefore the

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distinction between killing and allowing to die has been important. In the present era, the challenge lies not only in maintaining the distinction, but also in countering a proposed refutation of the immorality of killing itself. Although the traditional proscriptions against directly killing patients (euthanasia) or supplying them the means to kill themselves (PAS) are still maintained in most locales and by most physician groups,[7] this is no longer universally true. Following practices first started in the Netherlands and Belgium, at least three states in the USA have legalized PAS through a popular referendum or legislative activity, and others through judicial opinions. The stated justification is usually one of compassion – that is, relieving a patient of the burden of their suffering – which then extends to relieving them of the burden of their life. In the United States, only PAS has been practiced legally, as is the case in Switzerland. In Belgium and the Netherlands both PAS and euthanasia are now options. At the present time, these options are limited to actions by physicians. Although this may be seen as an attempt to maintain societal control, no serious discussions have taken place as to why these acts should be restricted to physicians. In fact, in venues where these actions are not legalized, they still have been done by nonprofessionals, sometimes with the guidance of publications from the Hemlock Society or Compassion and Choices. These groups may provide detailed instructions about medications to use, doses to ingest, and even supplementary actions to ensure completion of the desired effect; for example, tying a plastic bag around one's head.[8] Some physicians who are willing to participate in PAS are less willing to take an active role in directly euthanizing a patient, with objections that are not restricted to its illegality in the United States. However, with growing acceptance of PAS, there may be growing demands for direct euthanasia by physicians. This is almost inevitable, not only because of some reports of messy attempts by nonphysicians, but also because not everyone can maintain the capability to participate in PAS as a patient. In fact, if members of the public and the profession come to see this as a benefit, as a good thing, to be offered by the profession, then its extension from PAS for the terminally ill, to euthanasia or PAS for a variety of nonlethal conditions in other patients is logically inevitable. In fact, this is no longer conjecture; there are multiple examples already of this sort of "mission creep" – the Swiss organization Dignitas has assisted in suicides already for patients whose problems ranged from uncomplicated old age to loss of good looks. In the Lowlands, deaths have been arranged for depression, hearing loss, and anticipated bad prognoses for infants and children.[9]

This has inevitably resulted in reaction from those members of the profession who do not see this as a good thing.[10] Those arguing against it point out that euthanasia/PAS are not normally required for beneficent care. Pain relief is almost always possible, and suffering that may be psychological, emotional, or existential is not best treated by causing the patient's death. Moreover, what appears to be an autonomous action on the patient's part could easily devolve into a sense of obligation or expectation, if one's continued life is sensed to be burdensome to others, either financially or emotionally. Such a shift in traditional medical practice diminishes the value of palliative care, as it diminishes the self-worth of the patient as well, at a time when their declining capabilities may make them question their continued purpose in life.[11] Moreover, assuming such a role may do irreparable harm to the profession itself. Patients may justly be concerned if the physician pledged to safeguard their life and health is the same one offering to usher them out of it. If support for PAS and euthanasia grows, and with it the expectation that this will become the responsibility of the medical profession, it may lead to one of the most crucial ethical and moral dilemmas facing the profession in the near future.

Case 1: Disclosing a Diagnosis of Dementia

An 82-year-old woman is brought into your office by her daughter. The daughter tells you about her mother's gradually worsening memory loss with increasing forgetfulness over the past few years. In addition, the patient has expressed paranoid thoughts that her daughter wants to put her in a nursing home and that people are stealing her possessions. The patient lives alone, but near to her daughter. The patient is adamant about continuing to live on her own and has expressed that living in a nursing home is a fate worse than death. She currently dresses, feeds, and bathes herself. The daughter conducts most of her mother's finances, arranges and administers her medications, and also does her shopping. There have been no significant accidents or incidents in which the mother is wandering or becomes lost. After a thorough evaluation, you feel this patient most likely has dementia caused by Alzheimer's disease. When you express this to the patient's daughter, she requests that you not tell her mother, as it would only anger, depress, or worry her. The daughter would like you to start a medication for

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dementia, but asks you not to tell the patient the real purpose of the medication.

Discussion

The primary professional obligation of physicians is to the patient's good. How is this good to be determined and carried out? The doctor must offer beneficent treatment and care options, avoid harm, and respect the autonomy of the patient. These principles cannot be adequately followed without employing certain specific virtues. Along with virtues such as competence, fidelity, and compassion, this situation calls for honesty. Unless patients are treated with the respect due them, they can't be expected to respond appropriately and be capable of autonomous decision-making. Therefore, patients have a right to be told the truth about their condition. This right cannot be abrogated by third parties, even loving family members, whose own anxiety may cause them to make an error in judgment about the patient's capabilities.

For the same reason, a beneficent physician is not allowed to make a paternalistic and unilateral decision to withhold potentially troubling information. Both ethics and the law support this approach; however, it is not without exceptions. A patient may decide to waive their right to information. This may be prompted by cultural, psychological, or emotional aspects of an individual patient. Such a waiver, however, must be explicit, and can be determined in advance. When a physician suspects that a patient may prefer not to hear unpleasant or troubling medical facts, the patient should be asked, "How much do you want to know?" And then, "Whom should I tell, and what should they be told?" Such an explicit waiver should then clearly be documented in the medical record.

An additional and more common exception occurs when a patient has such diminished capacity that they can't fully comprehend, or deal with, the information, or act on it in a fully autonomous way. When this status is suspected, the evaluation of capacity should be formal, and typically doctors are required to determine that an advance directive (if available) should now be activated. Unfortunately, even after a predictable decline, too many patients have failed to indicate what their preferences might be or who a suitable proxy decision-maker would be. One of our obligations as physicians should be to ensure these discussions take place when the patient is still capable of indicating their preferences. This should then obviate the need to seek legal guardians' help.

What is not sufficient is the assertion by a family member, no matter how insistent, that a patient should not be told, and cannot deal with information. Nor should the doctor and loved ones go around or talk over the head of the patient. Age and state of health don't automatically impair the geriatric patient's autonomy. In fact, it may be a quarrelsome disposition of the patient that triggers such caregiver behaviors, ones that will likely be worsened if the patient feels that she or he is no longer treated with respect. The children do not automatically assume the parent's role, nor do they automatically have rights to information and decision-making. It is imperative that these issues be fully discussed early in the course of diagnosis and treatment.

There may be good reasons for a patient to know their diagnosis of early dementia. These include the opportunity for further planning for legal, financial, and long-term care issues. Moreover, patients may then become free to decide how to spend their time with people and things that are most important to them and to reconcile themselves with others when necessary. Additionally, some patients may seek the opportunity to participate in treatment trials for their condition, but only if that condition has been made known to them. Reasons to avoid disclosure, in addition to those already discussed above, often focus on concerns about depression and the fear of suicide. The best preventive for this may be the provision of emotional and spiritual support in addition to adequate health care and support for the tasks of daily life. It is crucially important that patients not be made to feel that they are a burden either to the system or to their families. Rather, it should be possible to focus on the positives that do exist, even as the patient's condition devolves from one stage to another.

Case 2: Substituted Judgment and Medical Futility

An 89-year-old woman has a history of diabetes, hypertension, congestive heart failure, chronic obstructive pulmonary disease, and advanced dementia. She has been a resident in a nursing home for the last 5 years. You meet this patient and her family for the first time when she is hospitalized for a hip fracture. Soon after her hip repair, the patient develops pneumonia. She requires oxygen therapy to maintain appropriate oxygen saturation; however, the patient has become delirious and frequently takes off her oxygen mask. The house staff order restraints to keep the oxygen therapy in place. Furthermore, the patient's intake of fluids and nutrition

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has substantially decreased during this illness. She develops acute renal failure associated with dehydration. An intravenous catheter is placed to administer fluids that, despite her restraints, is quickly torn out by the patient. Further attempts to place an intravenous catheter are unsuccessful.

You meet with the family to discuss this patient's complicated hospital course, her prognosis, and the goals of care for the patient. You ask the family about the patient's wishes regarding resuscitation and other life-sustaining medical therapies. The patient has not executed an advance directive. One daughter says that the patient did not previously express treatment wishes, but she believes that her mother would want everything done to keep her alive and would not want a DNR order. She requests the patient be transferred to the intensive care unit for progressive management.

Discussion

This clinical scenario is unfortunately all too common. In this case, the physician has no clear indication of the patient's wishes, no clear surrogate decision-maker, and only an indirect estimation of the patient's probable values that might impact subsequent care. The estimations provided by family members regarding a patient's probable preferences have not been shown to be highly reliable,[12] and only the treating physician's estimations were shown to be less reliable. The first task will be to decide on the morally appropriate and legally valid surrogate decision-maker. As we have seen, this might be designated by local law; however, even such laws do not typically help choose between proxies at the same level of hierarchy, such as children or siblings. The physician, sometimes with the help of an ethics consultant, can seek to determine who knows the patient best, and would have his or her best interests at heart. With a large number of family members who may be clamoring for information or a vote in the decision-making, it is frequently helpful to ask them to designate a spokesperson. This family member can then be the conduit for delivering information to the rest of the family and giving feedback on decision-making. Proxies or surrogate decision-makers must be made to understand that decisions should not be made according to their preferences, but on the preferences of the patient only. This substituted judgment is often the most difficult aspect of surrogate decision-making.

With the patient who has such multiple and chronic problems, and a predictably poor prognosis, physicians may be tempted to discourage certain treatment options on the basis of medical futility. Families, such as in this case, may be resistant to couching the problem in such terms. There is no common definition of futility that is likely to be universally accepted, other than that of strict physiological futility. If an intervention is completely incapable of achieving a desired goal, everyone can agree that it would be futile. However, this is not the case with most of those interventions that we think would be of dubious value and inadvisable. In fact, families often perceive that what we might describe as futility is in fact a disguised value judgment. It is not so much that an intervention would have no effect, but rather that we think its continued effect would not be worthwhile. It is these judgments that typically shape our recommendations to forgo ventilator support, dialysis, and other life-sustaining interventions near the end of life.

The problem at this juncture usually resides in differing concepts of futility.[13] If strict medical futility occurs when (a) there is a goal, (b) an intervention is aimed at achieving this goal, and (c) there is virtual certainty that the intervention will fail in achieving this goal, then the failure of agreement usually revolves around the acceptable goal. Hippocrates stated, "The physician must not treat the patient who is overmastered by his disease, realizing in such cases medicine is powerless."[14] Physicians often see futility in situations other than those that have no possible physiological benefit. Other goals of aggressive treatment, such as to prevent bodily death, may be thought to serve no useful purpose, when their idea of benefit is more oriented toward pain reduction or a peaceful death. If goals of care have not been explicitly agreed upon, conflict may well follow.

It is better to prevent than try to remedy conflicts with the proxy decision-makers. Thorough and explicit discussions may help to redirect requests that "Everything be done." The treating physician should reassure the family that she or he wants everything done that can provide real benefit under the circumstances. Reasons a possible intervention is not thought beneficial should be explained and alternatives detailed. Engaging in such a dialogue rather than giving an immediate refusal is more likely to be successful. Physicians must also remember that nonmaleficence is an important principle; physicians are not obligated to provide treatments they believe are ineffective or harmful. A patient or surrogate's right to participate in the decision-making process is a negative right; that is, they can accept or reject proposed treatments. It does not constitute a positive right to demand treatments

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that are not medically indicated. The effort to avoid doing harm, while respecting the patient's values, requires patience, humility, and good communication skills. It may be rewarded by avoiding unnecessary intensive care, yet providing those interventions that may still prove beneficial, sometimes as a time-limited trial.

Case 3: Nutrition and Hydration

A 77-year-old man with end-stage Alzheimer's and multi-infarct dementia is admitted to a geriatric psychiatric ward for worsening aggressive behavior, after physically attacking his wife. He lives at home with his wife, who is assisted by live-in nursing help. The patient's oral intake has substantially decreased, and he has lost approximately 15 pounds in the past 3 months. On admission, the patient's laboratory values indicate likely dehydration and malnutrition. You place a line intravenously to rehydrate him and the nutrition consultant asserts that the patient requires a feeding tube to address malnutrition. On meeting with the patient's wife, she asks you not to give her husband fluids or a feeding tube. "What good would it do in the end? Can we just let him go?" she asks you.

Discussion

In the recent past, perhaps nothing has generated more controversy, headlines, or policy statements than issues regarding the provision of nutrition and hydration. When considering a decision to withhold (or withdraw) nutrition and hydration, candidate patients generally fall into three categories: the unfeedable (those with a short gut or bowel obstruction), the terminal, and the stable, but neurologically devastated. Controversy does not usually attach to the first two categories. There is no moral obligation to do the impossible, and there is little evidence that extraordinary measures to provide feedings to the terminally ill patient are actually beneficial. In fact, the evidence would point the other way. The third category has triggered the most discussion and controversy, and we will consider how it applies to patients with dementia.

The question becomes: Should patients with Alzheimer's and advanced dementia fit into the second or third category? It is anticipated that all patients who live long enough with Alzheimer's will experience difficulty with eating. There is a loss of appetite, as well as motor function, which then interferes with intake, chewing, and swallowing. This can be considered a normal part of the disease process as it is nearing its end. Although a feeding gastrostomy can be placed, the available evidence indicates that it would not serve the purpose of improving nutrition or prolonging life under the circumstances. Careful explanation of these facts to the patient and family long before the apparent need would arise can obviate difficult discussions and emotional angst when the time comes. Most families can accept that this is a normal and irreversible event at the end of a dementia patient's life. Moreover, when patients and physicians understand the limitations of tube feedings in this situation, enthusiasm fades. Although the most common diagnostic category for patients receiving tube feedings is dementia, tube feedings have not been shown to reduce the risk of aspiration, to maintain weight or nutritional status, to prevent pressure sores, or to delay mortality in such patients.[15]

The initial justifications given for withholding or withdrawing nutrition and hydration were that they were artificially supplied. Because this required a medical procedure, it was argued that it could be refused or discontinued as burdensome, like any other medical intervention. The fact of the matter is that the primary burden of the feeding tube, and its most important risks, occurs in its placement, not in its maintenance. Moreover, further discussions about supplying nutrition and hydration have not focused on the presence of a feeding tube at all, but rather the provision of these by any route, including oral. It is certainly preferable to offer hand feedings orally to a dementia patient near the end of life rather than put in a feeding tube. Currently, arguments are being made that neither approach is necessary; that no feeding at all needs to be done. There are serious discussions about VSED, the voluntary stopping of eating and drinking. This is being proposed for those patients who still have the capacity to choose it, and also to be done to those who have lost capacity to accept that choice.[18]

It should be noted that the examples given above are less controversial than the withholding or withdrawing of nutrition/hydration from neurologically devastated but stable patients. The concern revolves around the intention of such cessation. These patients are not dying, but it is the feedings that are keeping them alive. A decision to withdraw nutrition and hydration usually follows a decision that their lives are no longer worth sustaining. Therefore, such a withdrawal is rightly seen as intended to end their lives, rather than relieve them of any medical burdens involved in the feeding.[19] Because of this, individuals and institutions who see this withdrawal as an unacceptable act of euthanasia have chosen not to participate. Consequently, withdrawal of nutrition and hydration from medically stable patients will not be done in Catholic hospitals and nursing homes.[20] This

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restriction would not apply to the unfeedable patients, or those who are imminently dying anyway, such as patients with advanced dementia.

Conclusion

Every interaction with a patient has an ethical component. In many cases, it is implicit and uncontroversial. Sometimes, it is explicit and becomes the central focus of activity. Resolution of ethical dilemmas can be aided by a systematic approach guided by principles and axioms. In all cases, the good of the patient is a foremost consideration, balanced by the realization that the physician is also a moral agent. This means that physicians are not mere servants to autonomy as they seek to balance the myriad factors involved, but always must seek that which truly benefits the patient.

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Chapter 64

Epidemic and Pandemic Impacts on the Elderly Population

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Introduction

Pandemics wreak global devastation and historically have been more damaging than any other type of natural disaster. The pandemic caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has had marked morbidity and mortality and significantly impaired the functioning of society and the world economy. The coronavirus disease 2019 (COVID-19) pandemic, however, was not the first infectious threat, nor will it be the last. COVID-19 impacted the world at an unprecedented scale, and lessons from prior infectious disease outbreaks have guided our ability to address this formidable event.

In the past several decades, outbreaks of severe acute respiratory syndrome (SARS), H1N1 "swine" influenza, and Ebola resulted in significant concern throughout the international community. The human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS) epidemic that shook our country in the 1980s/1990s continues to have a significant footprint within the USA and worldwide. The threat of novel viral pathogens is persistent, and lessons learned from these prior events as well as from COVID-19 will arm us for the future.

The impact of pandemics on economies and healthcare systems around the world is enormous. The individual impact of pandemics, however, is different for specific populations. The negative effects are felt most harshly in marginalized populations,[1] including older adults, refugees, and people living in heavily populated areas with substandard housing such as shanty towns. There are multiple reasons older adults are vulnerable to the impacts of pandemics. Increasing age leads to increasing comorbidities and polypharmacy. Additionally, older adult populations face challenges from decreased social support and availability of high-quality nutrition. Communal living conditions also can greatly increase their vulnerability to infection.[2] Policy decisions and actions to curb pandemics may aggravate these conditions, resulting in poor access to health care, drug shortages, limited food supplies, and movement restrictions.

Defining Pandemics

The amount of disease that is typically present in a community is referred to as the *endemic* level of disease. This baseline level is not necessarily the desired level of disease, which may be zero, but instead is the observed level. Occasionally, the amount of disease in a community rises above the expected, endemic, level. *Epidemic* refers to an increase, often sudden, in the number of cases of a disease above what is normally expected in that population. *Outbreak* has the same definition as epidemic, but often describes a more limited geographic area. *Pandemic* refers to an epidemic that has spread over several countries or continents, usually affecting a large number of people.[3]

The common conditions for the occurrence of an outbreak or epidemic are listed below. A pathogen is defined as an infectious or noninfectious substance that is capable of producing a pathologic process that can lead to disease.

- The introduction of a new pathogen, or an increased amount of virulence of a known pathogen, from an infected human, animal, birth, or arthropod vector, or from the environment.
- An adequate number of exposed and susceptible persons.
- An effective means of transmission between the source of the pathogen and the susceptible persons.[4]

Older Adults during Pandemics, Epidemics, and Outbreaks in History

COVID-19

The first case of coronavirus disease 2019 (COVID-19) was reported in Wuhan in the Hubei province of China on December 12, 2019. As of June 8, 2021 there were over

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170 million confirmed cases of SARS-CoV-2 infection causing COVID-19, with up to 3.7 million deaths worldwide.[5] The risk of hospitalization and death increases with age.[6,7] There is an increased case fatality rate in patients with advanced age (2.3% in the general population compared to 14.8% in patients over 80 years old[8]), with the highest susceptibility to infection in patients 65 years old and older.[9] There are several potential mechanisms that may be increasing the severity of this infection in older patients, in particular viral transmission, and physical reserve and immune system changes with age.[10]

One proposed mechanism to explain the high incidence in older COVID-19 patients may be increased viral shedding. A small study of 26 cases from Hong Kong detected higher peak viral load in respiratory specimens of older COVID-19 patients.[11] More severe COVID-19 cases may have a higher viral load, and patients with more severe COVID-19 are older than patients with milder infections (mean age 56 compared to 44 years old).[12] Since older patients have higher peak viral loads, they are likely to shed more virus.

Other considerations that may have led to COVID-19 having a significant impact on older populations include atypical presentations of illness, difficulty ensuring quarantine, and increased comorbidities. Examination of older patient presentations at the emergency department found atypical presentation of illness that mimicked other common geriatric conditions such as falls, and many patients lacked any specific symptoms of underlying infection. For example, older persons may not develop a fever and may only show cognitive or functional decline.[13,14]

Preventing the spread of the infection by adequate hygiene measures and quarantine may be difficult in elderly patients when there are cognitive dysfunctions. These challenges are amplified in communal living situations such as in nursing homes and can be further exacerbated when there is a shortage in testing and personal protective materials, as was witnessed at the beginning of the COVID-19 pandemic.

The impact of COVID-19 on older patients may also play into the case fatality rates of certain geographic regions, as associations have been found between increased population median age within a region and COVID-19 mortality.[15]

HIV-1/AIDS

The HIV-1 pandemic was first identified in the United States in 1981 when two groups of gay men were found to have rare diseases. One group in Los Angeles was found to have pneumocystis pneumonia, while one group in New York was found to have a rare and unusually aggressive cancer – Kaposi's sarcoma. These conditions are both associated with people who have weakened immune systems, and all the cases within these clusters were deadly.[16] By late 1981, cases were being seen in heterosexuals who used injection drugs, and health experts were concerned about a blood-borne pathogen as the cause. These suspicions were confirmed as experts identified cases that had likely transmission through heterosexual contact or through the blood supply. This led to the creation of "universal precautions," including wearing gloves when exposed to blood and other bodily fluids as well as establishment of safe needle disposal equipment.[17] In 1983, a new human retrovirus was isolated from patients with AIDS. A serological test was developed to allow for large sero-epidemiological studies, which confirmed that HIV causes AIDS. This work laid the foundation for the first diagnostic test for HIV infection.[18]

The HIV virus infects white blood cells in the body's immune system. In particular, it targets the T-helper cells (also called CD4 cells). Initially, there is a flulike syndrome, followed by a period where symptoms subside. Over time, however, immunity decreases until the final stages of the infection, which often involve malignant tumors and terminal infections.[19] It was determined that initial infections of HIV occurred 10 to 15 years before end-stage illness developed. This period of symptomatic quiescence meant that, by the time the initial AIDS cases were recognized, the pandemic had been going on for more than a decade.

The HIV pandemic continues to this day, and at the last HIV Surveillance Report in 2018, over half (51%) of people in the United States and dependent areas with diagnosed HIV were 50 years of age or older. (See Chapter 25.) New HIV diagnoses are overall declining in this group; however, around one in six HIV diagnoses in 2018 were in people aged 50 and older. Older people in the USA have a greater likelihood of having an AIDS-defining diagnosis at, or within 3 months prior to, their first presentation for HIV care. This, along with findings of lower CD4 counts on initial HIV diagnosis compared to those younger than 50 years of age, demonstrates that older people in the USA are more likely than younger people to have late-stage HIV infection at the time of diagnosis.[20]

There are several challenges unique to older Americans in regard to HIV prevention. The population over 50 has been found to visit their doctors more

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frequently; however, their providers are less likely to discuss sexual or drug use behaviors. Additionally, older patients may not consider themselves to be at risk for HIV, or may mistake HIV symptoms for those associated with normal aging.[21]

Older adults are additionally at risk for falling victim to the stigma surrounding HIV. People aged 50 and older may be hesitant to seek care to disclose their HIV status as they may already face isolation due to illness or loss of family or friends. Finally, aging with HIV has challenges in relation to other diseases that are often comorbidities of both HIV and advancing age. HIV and increasing age both increase patients' risk for cardiovascular disease, bone loss, and certain cancers. People aged 50 and older face challenges with potential polypharmacy, including possible interactions between HIV treatments and medications for other common comorbidities such as hypertension, diabetes, and elevated cholesterol.

Severe Acute Respiratory Syndrome (SARS)

The SARS epidemic of 2002 was the first new global epidemic of the twenty-first century. The first cases occurred in Guangdong Province, China and spread throughout Asia and beyond between 2002 and 2004. There were a total of 8,422 cases and 916 fatalities around the world. SARS is caused by a coronavirus spread through airborne transmission. SARS impacted older adults and those with weakened immune systems at a disproportionate rate. These patient populations faced much higher fatality rates than seen from other infectious agents that cause a similar constellation of symptoms.[22,23]

The case–fatality ratio for SARS was found to be 15% in Hong Kong. The case–fatality ratio, however, was markedly different for young versus older adults. Patients younger than 60 in Hong Kong had a case–fatality rate of 6.8%; for patients over 60 it was 55%.[24] This high case–fatality rate among older adults of course not only posed a serious threat to their physical health but also had a significant impact on their mental well-being.[25] SARS negatively impacted the emotional wellness of the geriatric population, to the point of there being a significant increase in suicide deaths among those aged 65 and over in Hong Kong. There was a 31.7% increase in the suicide rate among persons aged 65 and over in Hong Kong in 2003 compared to 2002.[26] Studies evaluated choice and personal motivation of the individuals who committed suicide and uncovered two phenomena.

1. Some older adults at risk did not have the necessary care and support from families or friends. These older adults were isolated because of quarantine requirements in order to minimize exposure risks.
2. Vulnerable elderly persons were more pessimistic, paying less attention to positive information about SARS. They were overwhelmed by negative news and developed fears about the epidemic, which induced mental anxieties. The feelings of isolation, helplessness, and reduced social integration all contributed to an increased number of suicidal acts.[27,28]

There have additionally been found to be long-lasting community impacts of an infectious disease outbreak, even among those who did not develop the disease. After the resolution of the SARS pandemic, 9.2% of residents in more highly impacted communities reported feeling more pessimistic overall about life, with older adults, high school seniors, and those worried about recurrence being most affected.[29]

H1N1 Influenza Virus

The H1N1 influenza pandemic of 1918 to 1919 infected 500 million people around the globe and killed between 50 million and 100 million, ultimately becoming one of the most deadly events in recorded human history.[30] Since that time, the genome of the 1918 influenza virus has been sequenced, but many questions about the pandemic it caused remain unanswered. In 2009, a pandemic of swine-origin H1N1 influenza began in Veracruz, Mexico. Within a few months the virus had spread around the globe. In late April 2009, the World Health Organization (WHO) declared its first ever "public health emergency of international concern" and in June 2009, the WHO and the US Centers for Disease Control and Prevention (CDC) declared the outbreak a pandemic.[31] Similar to the pandemic of 1918, the resurgence of H1N1 differed from many other infectious outbreaks, as it commonly infected previously healthy individuals. Symptoms typically lasted a little less than a week, although a small group of patients developed sudden worsening of fever and shortness of breath, leading to acute respiratory distress syndrome (ARDS).

Unlike most strains of influenza, H1N1 did not show a predilection for the elderly.[32] Before the pandemic was declared over in late 2010, the number of fatalities attributed to H1N1 infection was approximately 200,000, with 87% of deaths occurring in those under 65 years of age.[33] This impacted vaccine distribution of the limited

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2009 H1N1 vaccine. The CDC did not include people aged 65 and older in the groups recommended to get the initial doses of the 2009 H1N1 vaccine, citing that rates among younger persons were 15 to 20 times higher.[34] In the uninfected population, 33% of those >60 years old had preexisting neutralizing antibodies against the 2009 H1N1. This suggested that influenza strains that circulated 50 to 60 years prior may have provided cross-protection against the swine-origin 2009 H1N1 influenza virus.[35] Interestingly, in the 1918 H1N1 influenza pandemic, the mortality in older persons was less pronounced than that in other age groups, implying partial protection from disease, possibly due to previous exposure to related influenza viruses.[36]

Ebola

The Ebola outbreak that began in early 2014 was the largest and deadliest in recorded history. With more than 28,000 cases and 1,000 deaths, Ebola shook West Africa before being brought under control in 2015.[37] Ebola virus disease (EVD) is an acute, serious illness first discovered in two simultaneous outbreaks in South Sudan and the Democratic Republic of the Congo (DRC) in Central Africa in 1976. The first symptom is usually fever. Symptoms often then progress to include fatigue, myalgias, severe headache, red eyes, sore throat, vomiting, and diarrhea. There can additionally be impaired renal and liver function. At times both internal and external bleeding can occur (such as oozing from the gums or bloody diarrhea). A person infected with Ebola cannot spread the disease until symptoms develop.[38] Ebola's highly infectious and virulent nature, rapid progression, and high fatality rate created significant fear in the international community, leading to the United Nations Security Council declaring the situation a global threat to international security.[39]

The primary focus during the Ebola outbreak was on adults and children. Consequently, the casualty data for older adults is limited. Study findings, however, have shown that people over the age of 45 years have had more than double the risk of death.[40] The higher risk of mortality in older patients is likely multifactorial. Age-related decline of the immune system, causing a high viral load and contributing to multiorgan failure, is likely a significant contributor. The roles of older adults in West African society likely also were a factor. It is common for children in this region to be raised and cared for by extended family members, in particular stepmothers and grandmothers. This practice has been increasing, in part because of civil war, but also because of increasing opportunity and need for birth mothers to earn an income around urban centers. There are many documented cases where local outbreaks can be traced to children visiting their birth mothers in an urban center then returning back to their more rural villages, bringing the virus with them. Additionally, older people in rural areas may follow different health-seeking practices than younger urban counterparts because of the distance to health facilities, a greater level of poverty, and greater confidence in non-biomedical remedies.

Risk: General Risks to Elderly Populations during Pandemics

Patients with advanced age are commonly affected disproportionately by pandemics. There are multiple factors that increase the risks older populations face during infectious outbreaks, epidemics, and pandemics. Advancing age is associated with physiologic changes that put patients more at risk for disease and for increased severity of disease. These changes include increasing comorbidities as well as decreased ability to mount the needed immune response to overcome an infectious threat. Additionally, there are socioeconomic and social determinants of health that impact elderly patients, who are often more isolated and may be living on a fixed income. Finally, the burden of isolation and fear can have significant deleterious effects on older adults' mental health.

Immunosenescence

Many infectious diseases in older adults are more frequent and severe than in the younger population. There are likely several components to why these patients are more prone to infection. One, termed "immunosenescence," refers to the notion that there is an age-related dysfunction of the immune system. Research is ongoing in this area, as studies often have contradictory results, but one consistency is that decreased immune function in older adults does exist. Two examples that highlight these changes are reactivation of tuberculosis in older adults[41] and a decreased effectiveness of the influenza vaccine.[42]

Comorbidities

The world's population is aging, and an important part of this demographic shift is the increasing development of chronic illness. Certain chronic diseases tend to cluster

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with others, either because of underlying risk factors, complications of the primary disease, or factors such as an increased inflammatory state.[43] The increased incidence of infection and mortality for many infectious diseases is likely in part related to the increased number of comorbid conditions that accompany advanced age. Comorbidity results in reduced innate immunity, the nonspecific defense mechanisms of the body. These include nonspecific barriers such as skin integrity, cough, and mucociliary clearance. For example, in chronic obstructive pulmonary disease (COPD) the impaired mucociliary clearance, alveolar macrophage dysfunction, and suppressed cough mechanism that accompany COPD substantially increase the risk for lower respiratory tract infection.

Social Determinants of Health

The living environment of older individuals can also influence their exposure to infections, with long-term care facilities and hospitals bringing greater risk than living at home.[44,45] Nursing home residents share dining, recreation, and therapeutic facilities. They can also be highly dependent upon health-care workers for assistance with activities of daily living. This chronic contact with health-care workers can increase risk of exposure to infectious organisms during an outbreak.

Public health emergencies such as global pandemics can also impact older adults through disruption in their ability to obtain needed household food and supplies. Changes in the types of foods eaten due to changes in food availability during shelter-in-place orders, for example, may precipitate the exacerbation of heart failure. Lack of exercise due to isolating at home may lead to deconditioning with subsequent weakness and falls. A decrease in cognitive stimulation that occurs with socializing and engaging with the wider world may worsen cognitive and behavioral symptoms of dementia.[46]

Mental Health

Particular attention should be given to the psychological aspects of isolation and infection control, especially in the geriatric population. Individuals respond differently to stressful situations such as an infectious disease outbreak that requires social distancing, quarantine, or isolation. However, common responses may include anxiety, worry, or fear. This fear can be related to their own health status, or day-to-day challenges related to securing needed items, such as groceries and personal care items. Additionally, there can be uncertainty or frustration about how long the situation will last. Finally, there can be loneliness associated with isolation, as well as boredom, frustration, and depression.[47]

Guarding against isolation through continued contact with family, friends, and support networks should be encouraged through phone, email, and virtual meetups to assist with these mental health aspects. Older patients face significant consequences from depression and loneliness, which have been found to be related risk factors for worsening cognition.[46] Practical suggestions for the psychological and mental health of elderly individuals living in a pandemic such as COVID-19 are outlined in Table 64.1.

Response

As the course of a public health emergency unfolds, geriatricians and other primary care providers may be called upon to adapt roles beyond bedside care. This may include creating algorithms for testing strategies and indications, supporting optimal infection prevention and control measures in the community and in nursing homes, and adapting frameworks for vaccine distribution.

Providers caring for the geriatric population may be needed to participate in data collection efforts to track and control the event as well as understand the natural history of a disease. Finally, the geriatric care provider is often a trusted source of information for patients, the community, and the media. There is therefore a need to be prepared to adapt to the varying roles that may be needed of the geriatric clinician.

Infection Prevention and Control

Infection prevention must be a priority in any setting where health care is delivered. This includes primary care and geriatric offices, hospitals, nursing facilities, and long-term care facilities. When a suspected public health emergency is identified, immediate implementation of infection and control procedures as well as informing local health authorities is imperative. Clinicians must work with administration and infection control/public health officials to ensure rapid activation of policies and implementation of procedures and needed equipment such as personal protective equipment (PPE).

For pathogens with preventive vaccines available, it is important to include immunizations in the response plans. First responders and health-care workers are priority groups in the event of a pandemic, both because it

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Table 64.1 Practical suggestions for the psychological and mental health of the elderly during COVID-19

Considerations	Practical suggestions
(Psychological health) Stress caused by COVID-19	(1) Stop reading, watching, and listening to news, including social media, about the pandemic, because repeated exposure can cause stress and anxiety. (2) Refrain from spreading unofficial information. (3) Understand that it is normal to feel stress and fear in unpredictable situations. (4) Take deep breaths, stretch the body, and perform yoga or meditation. (5) Give attention to one's needs, emotions, and thoughts. (6) Determine actions after considering collective and social influences. (7) Refrain from discriminating or blaming specific individuals or groups for the infection. (8) Take care of and encourage oneself. (9) Those with disabilities, such as mental illnesses or drug abuse, may be particularly vulnerable in an emergency, and thus must continue with treatment. Notice new symptoms or the worsening of existing symptoms, in which case, ask for medical help.
(Social health) Social support	(1) Maintain contact with family and friends. (2) Maintain regular religious activities and contact with the local community. (3) Be informed, in advance, of where and how to receive counseling and other supporting services. (4) Notify close family and friends when symptoms of sadness, depression, and anxiety occur.
(Physical health) Physical activity	(1) Maintain a daily schedule and exercise pattern. (2) Have regular habits to maintain good health. (3) Make time for leisure activities and find enjoyable activities. (4) Maintain a healthy and balanced diet. (5) Obtain enough sleep. (6) Avoid excessive drinking and drug use. (7) Take prescription medicine as usual.

Source: Consideration of the psychological and mental health of the elderly during COVID-19: A theoretical review.[48]

reduces the likelihood they will fall ill themselves and it decreases the likelihood of transmission to patients and other health-care staff. It will be vital to work with health authorities in the event of an emerging public health crisis to determine other priority groups based on who is most at risk or vulnerable to the pathogen and develop strategies for timely disbursement to these groups. Older adults are often among the most vulnerable to emerging pathogens, and ensuring access to vaccines as well as encouraging elderly patients to obtain the vaccines when available are important roles for health-care providers.

Reporting

Each state has different requirements regarding reporting of communicable diseases to allow for epidemiological surveillance. State health departments as well as the US CDC are available daily, every day of the year, to allow for timely reporting and response. The outpatient primary care or geriatric clinician may be among the first to notice unusual signs and symptoms, or atypical manifestations of a disease process that signal suspicion of an outbreak. If you suspect an outbreak, it is imperative to contact your local health department for assistance with further investigation and control.

Diagnostics

Diagnostic testing should be directed by presenting symptoms and signs, with early diagnostic sampling and empiric treatment while awaiting results if symptoms are severe, or watchful waiting in clinical scenarios that are not progressing as rapidly. In scenarios where diagnostic testing is limited, working with public health authorities to develop and implement algorithms for appropriate testing scenarios will be important until greater numbers of tests can be developed.

Therapeutics

Therapeutic remedies may not always be available at disease onset, as pathogens causing epidemic- or pandemic-level emergencies are often novel and without effective treatment. If treatment exists, timely distribution to infected patients is paramount. In public health emergencies without a known treatment, supportive care and ensuring patients have a caregiver during their illness

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(in particular vulnerable elderly patients, especially those living alone) are key to success in the outpatient setting. Timely admission to the hospital is needed when the level of disease is too severe or patients cannot care for themselves and are without a caregiver. Health-care providers must remain attuned to breaking health news and stay informed with local authorities to ensure rapid acquisition and distribution of therapeutics that are developed during outbreaks, epidemics, and pandemics.

Communication

Communication and dissemination of timely and accurate information are important initial responses to pandemic emergencies. Fear commonly accompanies outbreaks of infectious diseases, and communication of what is known as well as what is not known allows for effective establishment of trust while avoiding false reassurances. Communication between health-care providers and patients as well as patients' family members and the surrounding communities allows for the disbursement of scientific, fact-based advice and helps quell rumors that might interfere with actions taken by or involving suspected or confirmed patients. Cultivating messages that are complete, clear, and succinct, as well as providing specific information regarding what to do and which health behaviors to avoid, are the keys to effective communication in a large-scale public health emergency.

Later Pandemic Response and Recovery

During the later stages of pandemics that persist, or as time passes and the pandemic begins to subside, the response and recovery includes sustaining and rebuilding community function. In-person social supports may be impacted during prolonged pandemic periods while movement or contact is hampered because of concern for infection spread. Virtual contact can be immensely helpful through phone, video, and web applications. Continued participation in functions that patients hold in importance and are part of their regular lives in normal circumstances, such as religious activities and social or support groups, should be attempted as able, such as through virtual means. Instilling a sense of normalcy and helping reestablish routines can be effective for fostering resiliency. Health-care providers should assess for mental health impacts of a prolonged pandemic, as depression and anxiety can be heightened in this scenario. This will allow for identification of patients who would benefit from provision of mental health resources.

Preparedness

The best response to a public health event is built on the strong structure of systems built prior to an outbreak. Despite outbreaks, epidemics, and pandemics often arising with little to no warning, there are steps that can be taken to prepare for emergencies prior to occurrence. These include developing procedures and policies in advance, and conducting training and drills as well as ensuring a well-stocked supply of equipment, medications, and clinical supplies.

Physicians working with nursing facilities and other long-term care facilities can take steps to assess and improve the preparedness of responding to a public health emergency. Checklists can be made and or adapted from available resources (CDC COVID-19 preparedness checklist for nursing homes:[49] www.cdc.gov/coronavirus/2019-ncov/downloads/novel-coronavirus-2019-Nursing-Homes-Preparedness-Checklist_3_13.pdf).

In the setting of an outbreak or public health crisis, nursing facilities and primary care clinics must be prepared for an increase in ill patients. Distinguishing individuals responsible for monitoring public health advisories allows for quick response to emerging situations. Infection control policies need to be in place to prevent transfer of infection while caring for sick individuals. Timely communication through phone trees or other methods of communication can be used to inform staff of urgent changes to policy or needs of the facility. Continued education and training of personnel allows all members of the institution to be up to date on infection control and management. Educational topics may need to include the signs and symptoms of illness, proper hand hygiene, and selection and use of PPE, as well as reinforcing the importance of staying home when ill. There may additionally need to be a plan for surge capacity, should members of the care team fall ill. Ensuring staffing to provide other services, including environmental services and security, is also essential. Being involved in establishing preparedness plans prior to a public health emergency is preferable to developing them during a crisis. Geriatrics-trained health-care professionals can and should provide leadership for these events.

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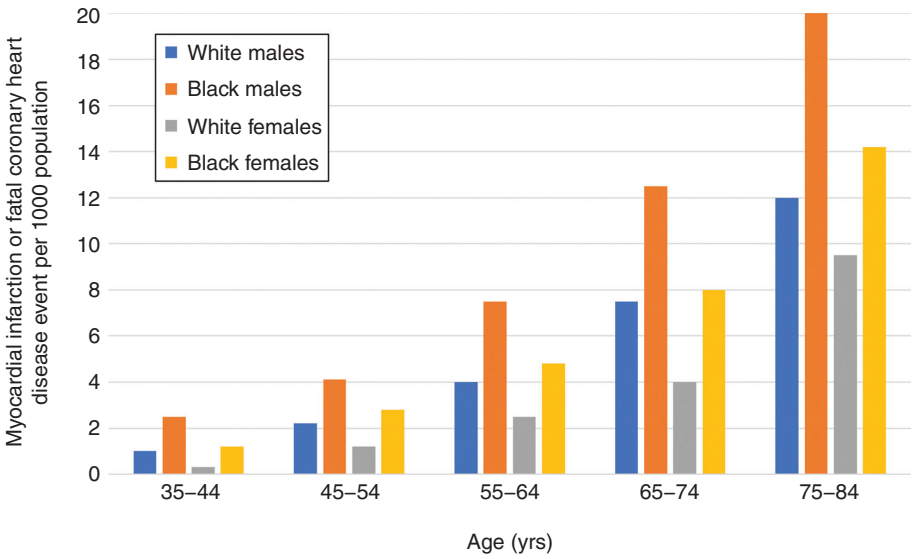

Figure 13.1 Incidence of myocardial infarction or fatal coronary heart disease in the Atherosclerosis Risk in Communities study, 2005 to 2014. (A black-and-white version of this figure will appear in some formats.)

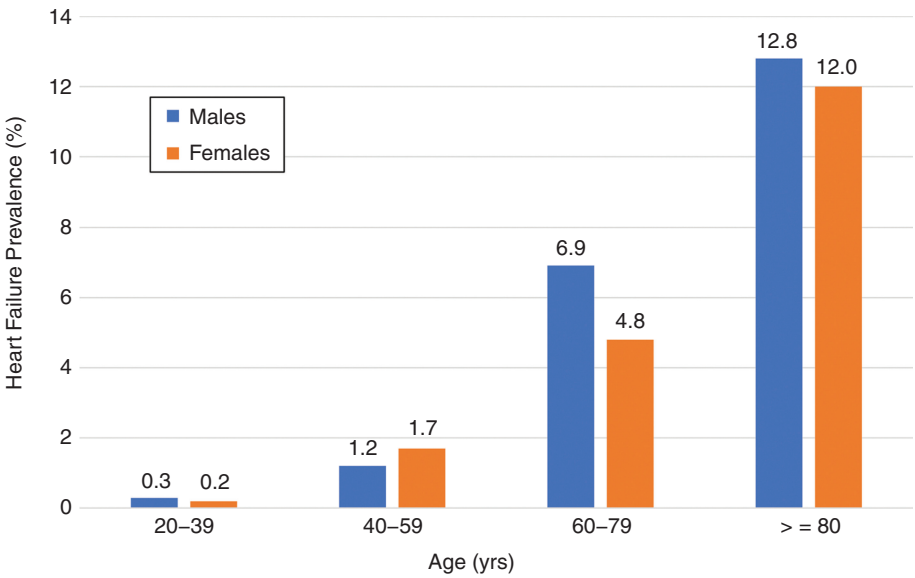

Figure 13.2 Prevalence of heart failure among adults in the National Health and Nutrition Examination Survey (NHANES) between 2013 and 2016. (A black-and-white version of this figure will appear in some formats.)

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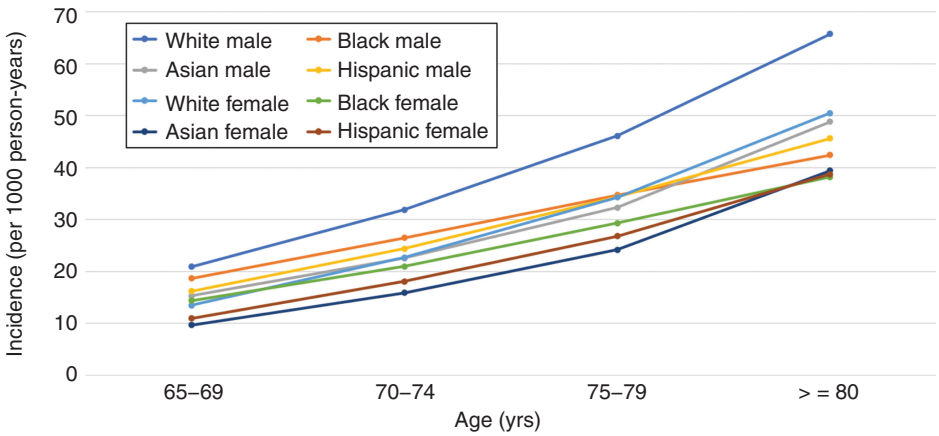

Figure 13.3 Incidence of atrial fibrillation by age, sex, and race in California between 2005 and 2009. (A black-and-white version of this figure will appear in some formats.)

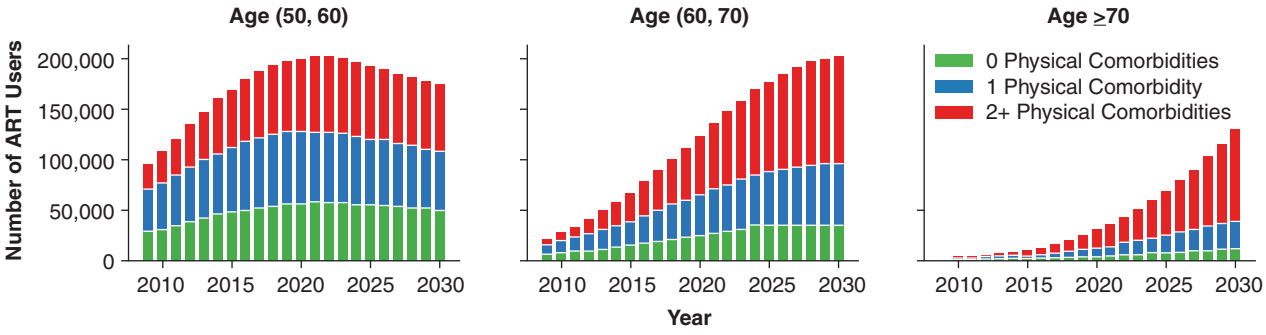

Figure 25.3 Projected burden of multimorbidity by age.[41] (A black-and-white version of this figure will appear in some formats.)

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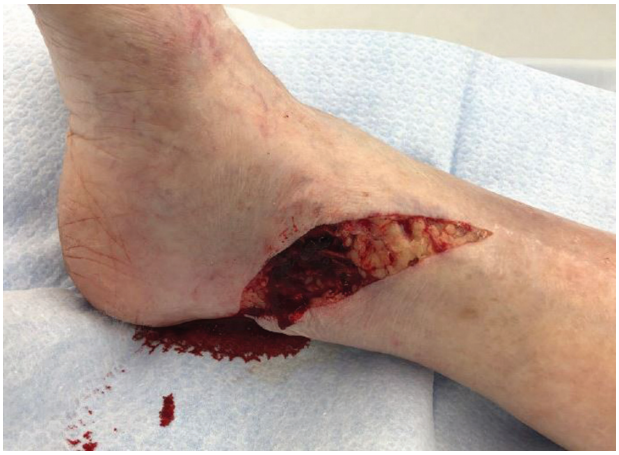

Figure 37.1 Clinical image of a 95-year-old female who sustained an open ankle fracture. The medial, oblique wound is a result of tensile failure of the skin as the displaced distal tibia tears through it. (A black-and-white version of this figure will appear in some formats.)

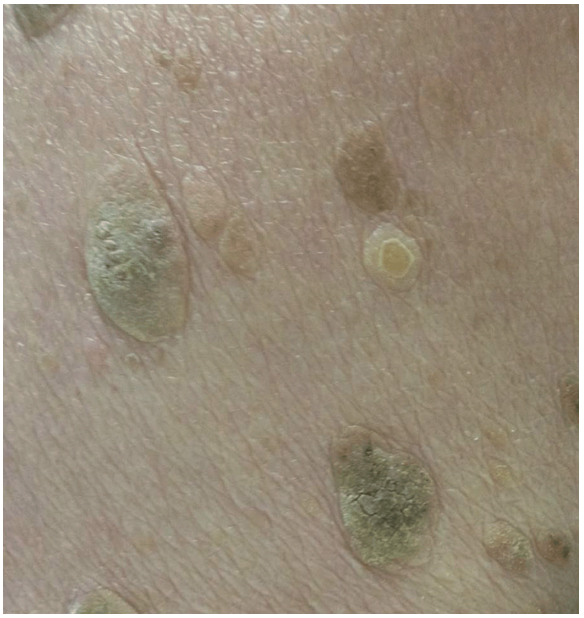

Figure 38.1 Seborrheic keratoses. Stuck-on, waxy, crumbling, hyperpigmented papules and plaques. (A black-and-white version of this figure will appear in some formats.)

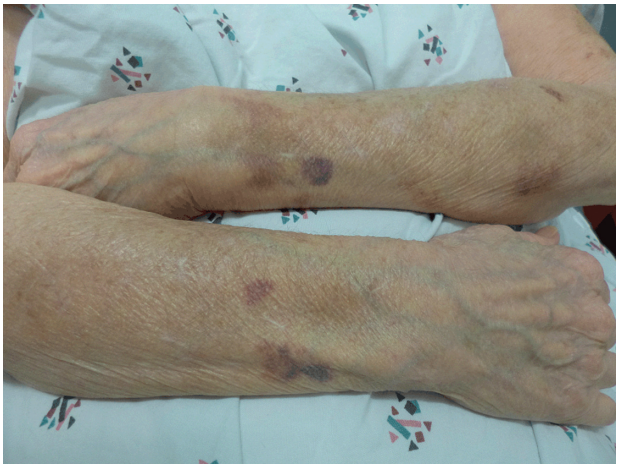

Figure 38.2 Actinic purpura. Purpura classically located on the forearm with absence of yellow-green hues. (A black-and-white version of this figure will appear in some formats.)

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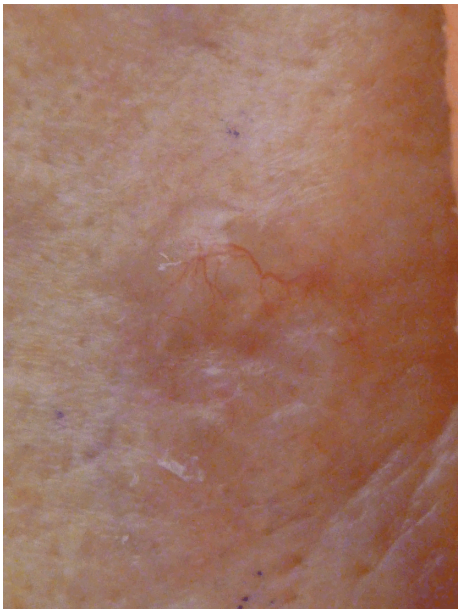

Figure 38.3 Basal cell carcinoma. Pearly papule in the retroauricular area with arborizing telangiectasias. (A black-and-white version of this figure will appear in some formats.)

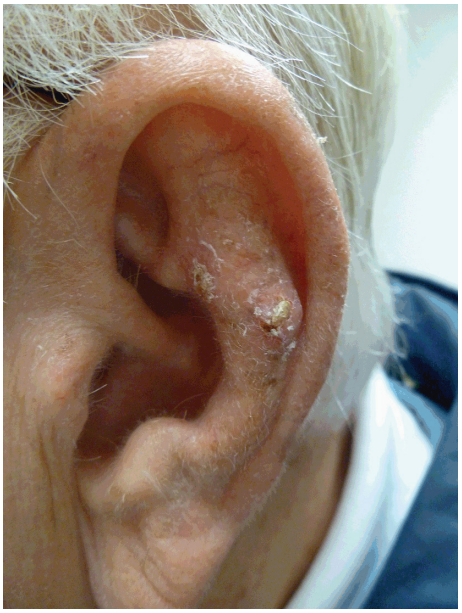

Figure 38.4 Squamous cell carcinoma. Crusted nodule on the antihelix, a high-risk location. (A black-and-white version of this figure will appear in some formats.)

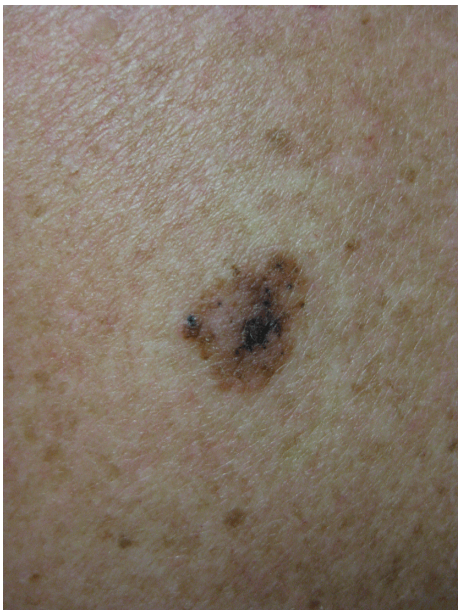

Figure 38.5 Superficial spreading melanoma. Irregularly shaped, pigmented plaque with variegated pigment. (A black-and-white version of this figure will appear in some formats.)

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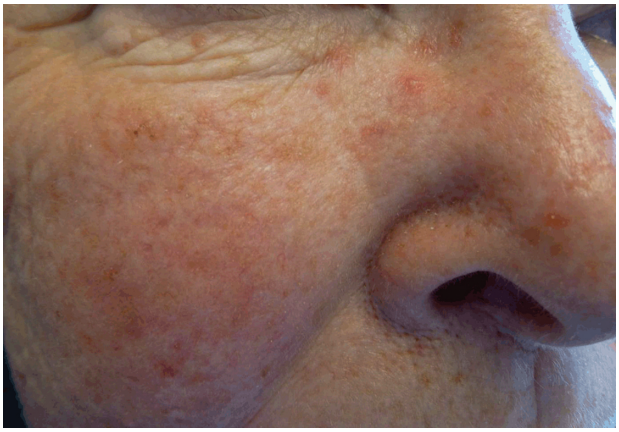

Figure 38.6 Rosacea. Erythema, telangiectasias, and scattered papules and pustules. (A black-and-white version of this figure will appear in some formats.)

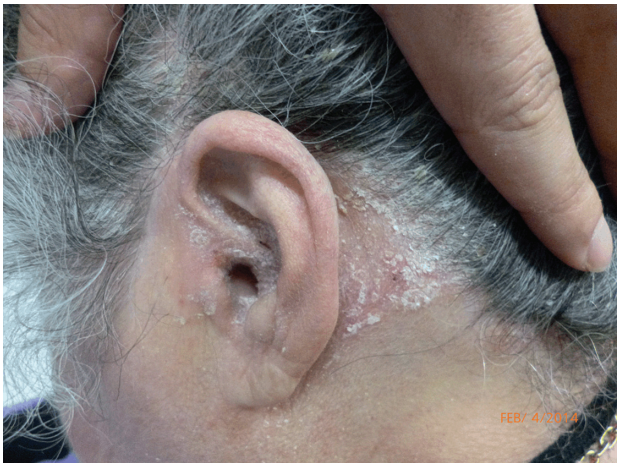

Figure 38.7 Seborrheic dermatitis. Erythematous plaques that are flaky, adherent, and slightly greasy affecting the scalp, ear, and retroauricular sulcus. (A black-and-white version of this figure will appear in some formats.)

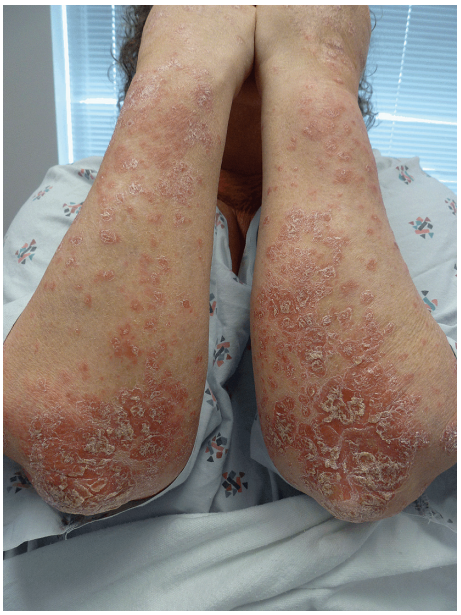

Figure 38.8 Psoriasis. Scaly, erythematous plaques with a predilection for extensor surfaces. (A black-and-white version of this figure will appear in some formats.)

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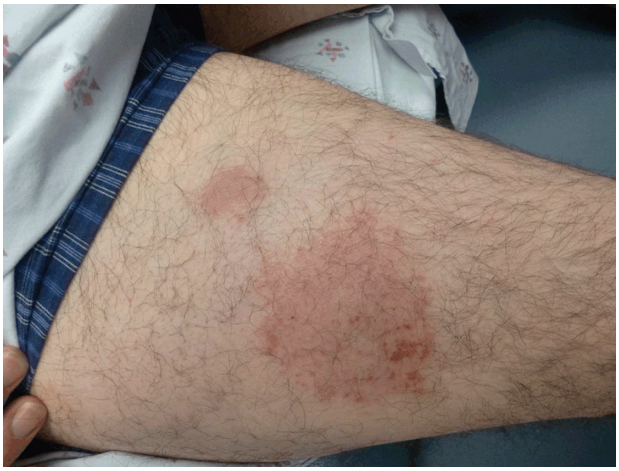

Figure 38.9 Nummular dermatitis. Annular, slightly scaly, pruritic plaques on the extremities. (A black-and-white version of this figure will appear in some formats.)

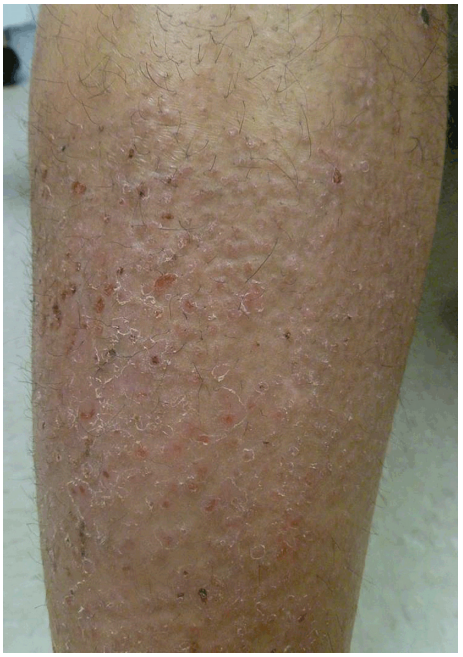

Figure 38.10 Lichen simplex chronicus. Thick, excoriated, lichenified plaque on the lower leg at a site of frequent scratching due to contact dermatitis. (A black-and-white version of this figure will appear in some formats.)

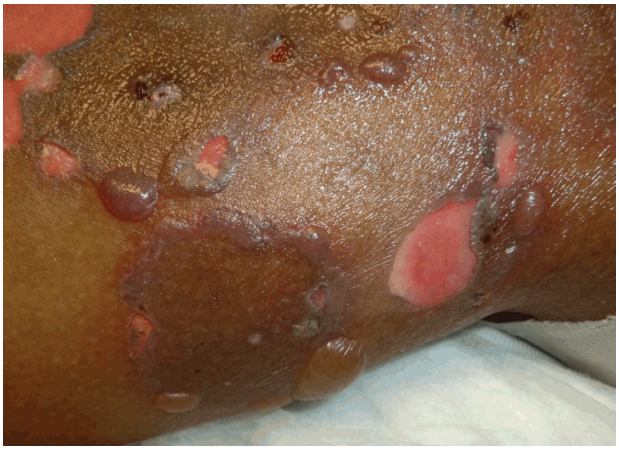

Figure 38.11 Bullous pemphigoid. Widespread, tense bullae erupting diffusely on the trunk and extremities. Shallow ulcers are present where bullae have ruptured. (A black-and-white version of this figure will appear in some formats.)

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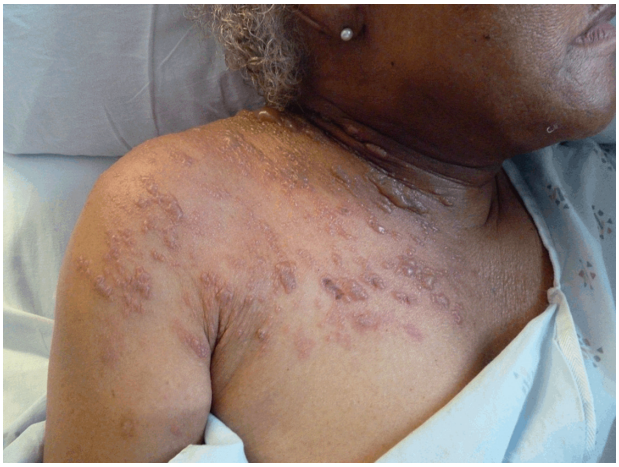

Figure 38.12 Herpes zoster. Clustered vesicles on an erythematous base in a dermatomal distribution. Note the sharp demarcation at midline. (A black-and-white version of this figure will appear in some formats.)

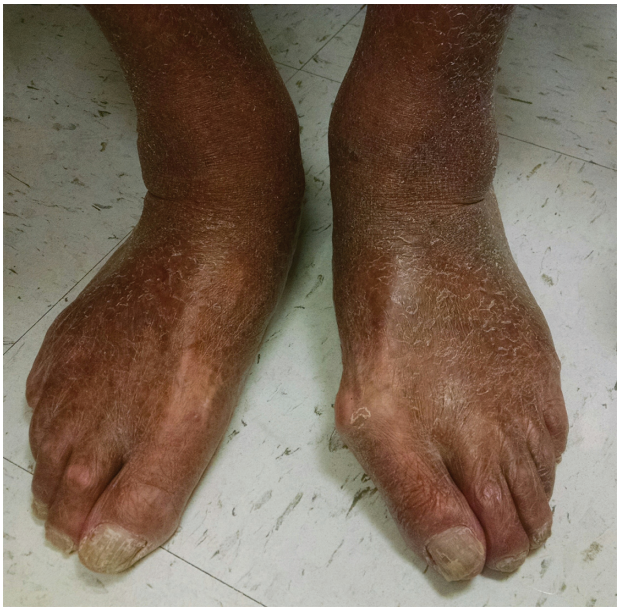

Figure 45.1 Feet of an older adult showing many of the conditions that can happen with aging. Xerosis is present on both legs and feet. Onychomycosis is present bilaterally. Hallux valgus (bunion) is noted on the left foot. Digiti flexus (hammertoe) is present in the right second toe. (A black-and-white version of this figure will appear in some formats.)

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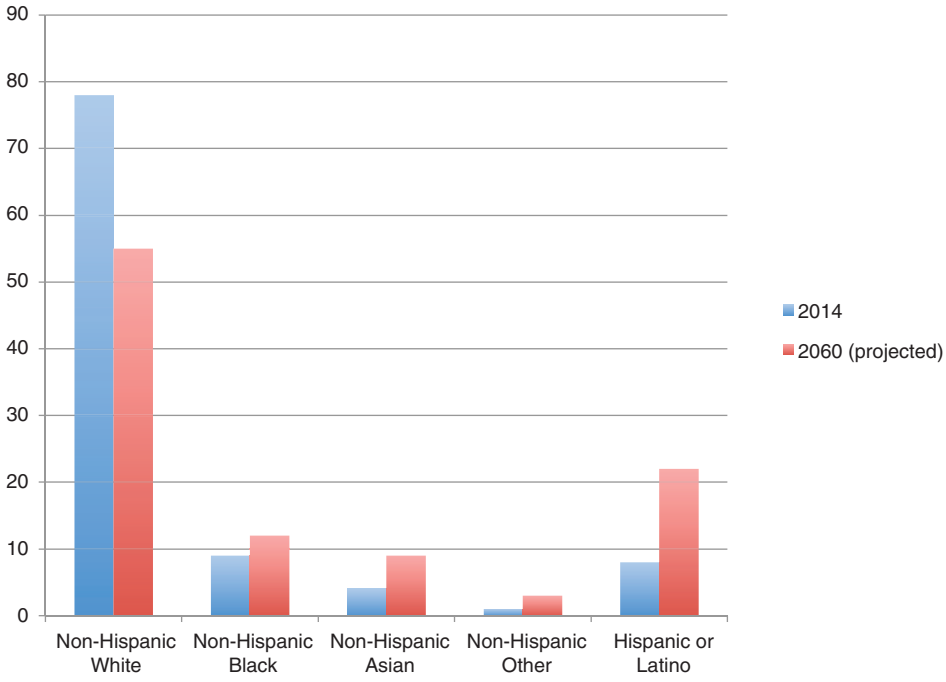

Figure 58.1 a. Percent of US population aged 65 and over by race and Hispanic origin 2014 and projected 2060. (A black-and-white version of this figure will appear in some formats.) b. Projected percent of ethnic-minority elders.

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