Fred A. Luchette
Jay A. Yelon
Editors

Geriatric Trauma and Critical Care

Second Edition

Springer

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Changing Demographics of the American Population

1

Stephanie Gordy

Summary

- The geriatric population comprises about 15 % of the American population.
- The elderly population is getting increasingly older even within itself as the >85 population is expanding.
- The aging population will increase the cost and complexity of medical care.
- Trauma is the fifth leading cause of mortality in the elderly.
- Comorbidities, concomitant medications, and unique physiology add complexity to the care of the geriatric trauma patient.
- Conversations regarding advance directives and end-of-life care are of paramount importance.

Introduction

Geriatric citizens in the United States are the most rapidly growing segment due to the aging baby boomer generation. This generation will live longer than the preceding and will have access to improved healthcare. Because these physically active elderly will remain living independently and longer, traumatic injuries can be expected to increase. In addition, there are numerous physiologic alterations that occur with aging, and special consideration should be given to the elderly patient from a medical and surgical standpoint. The use of multiple medications and presence of multiple comorbidities may also be present in this population leading to higher complications, longer hospital stays, and a higher case fatality rate. Moreover, disposition barriers often exist and include the need for short- and long-term rehabilitation. Finally, traumatic injuries have the ability to change the patient's independent living status and increase the need for admission to skilled nursing facilities. Complex end-of-life decisions and discussions are often also required in this population. Trauma and acute care surgeons should be knowledgeable about the specific needs of the geriatric critically ill patient.

The Aging Population

The definition of elderly has not been definitively established in the trauma literature, but the consensus is that it lies somewhere between the ages of 45 and 75 years [1]. In 2013, the population over 65 years of age was 44.7 million. This represented 14.1 % of the American population at that time. As the "baby boomer" generation reaches the golden years, this demographic is expected to show continued growth. The 15.3 % increase from 2000 to 2010 in the over 65 portion of the population was nearly double the increase (8.7 %) for all ages younger than 65. It is projected that by 2020, this number will increase by 36 % to 55 million. In 2010, the 65–74 age category was ten times larger than in 1900 at 20.8 million. In contrast, the 75–84-year-old demographic was 17 times larger at 13.1 million. Moreover, the >85 group was 45 times larger at 5.5 million. This reveals that the elderly population itself is getting increasingly older even within itself. By 2020, the population over the age of 85 years is projected to increase from 5.5 million in 2010 to 6.6 million. Furthermore, the centenarian (greater than 100) population is steadily increasing. In 2010, 53,364 persons were 100 years old or greater which is greater than a 50 % increase from the 1990 values [2].

Since 1900, improvements and accessibility to healthcare services in addition to improved life expectancy have allowed the number of individuals over 65 years to more than triple. This is not only due to the post-World War II baby boom but to an increased life expectancy as well. A child born in 2009

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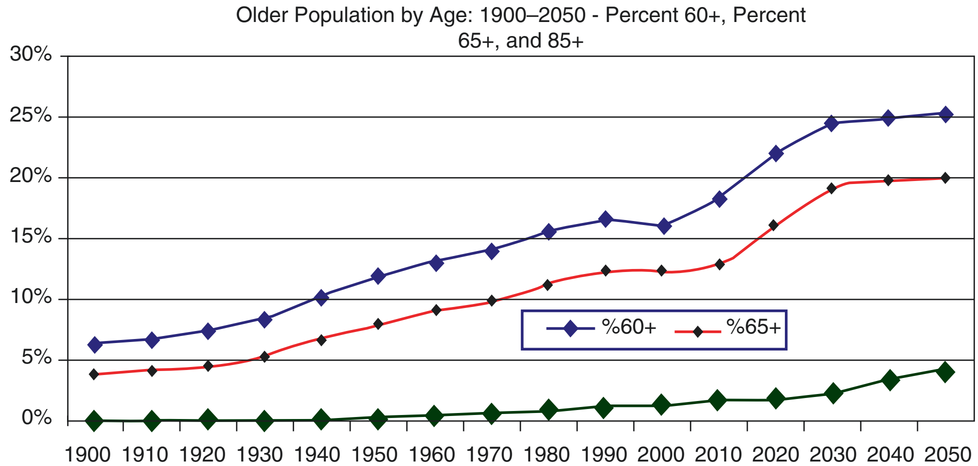
Fig. 1.1 The administration on aging



can expect to live 78.2 years which is 30 years longer than the life expectancy of a child born in 1900. The older population will continue to increase due to maturation of the baby boomer generation (Fig. 1.1). While the population growth slowed in the Great Depression era, it will continue on the upswing as those born between 1946 and 1964 get older. The elderly population will reach its peak by the year 2040 as the greater than 65-year-old population is expected to be 21.7 % of the entire populace [3]. As this segment of the population increases, the number of injured elderly will also grow [1].

The Cost of Caring for the Elderly

As the elderly population increases, the need for healthcare services and the cost of healthcare are expected to rise. The elderly represented 40 % of all hospitalized adults in 2008 [4]. Similarly, even though this population comprises only 13 % of the citizens in the United States, nearly half of all healthcare dollars spent are on the elderly. Additionally, the population over 85 years of age represents only 1.8 % of the total population but accounts for 8 % of all hospital discharges. Hospitalizations and healthcare spending for older adults are expected to rise as the number of elderly increases [5].

More healthcare resources will be necessary to care for the aging population which will pose an additional burden on an already strained federal budget. This cost will not only be reflected in dollars but in resource utilization including acute and long-term care. In 2002, the elderly made up 13 % of the US population, but they consumed 36 % of the total US personal healthcare expenses. The average healthcare expense in 2002 was $11,089/year for elderly people but only $3352/year for those younger than 65 years [6]. Furthermore, older Americans spend 13.2 % of their total expenditures on health, more than twice the proportion spent by the younger citizens (6.6 %) [2]. The five most costly illnesses include heart disease, cancer, trauma, mental disorders, and pulmonary conditions. Heart disease and trauma ranked first and second as the two costliest diseases in terms of total healthcare spending [7].

Thirty percent of total Medicare payments each year are for 6 % of the beneficiaries who died that year. Payments for the last 60 days of life constitute 52 % of the total dollars spent annually by Medicare. Inpatient services consume 70.3 % of the Medicare budget of which the majority of the funds are spent on critical care [8]. In summary, the sickest, eldest patients with a high incidence of morbidity and mortality consume the majority of the Medicare budget [9].

Effects of Aging on Organ Function

Understanding the medical physiology pertinent to this population is particularly important, because it affects the physiologic reserve and compensatory mechanisms required to respond to a traumatic injury, an acute illness, and major operations. The elderly population has a high incidence of comorbidities which can confound the physicians' ability to assess for injury. In injured geriatric patients, the incidence of preexisting medical conditions is 66 %. Moreover, 81 % of nonagenarians have medical comorbidities [10]. Nearly every organ system is affected by changes due to aging. A detailed discussion of this is beyond the scope of this chapter but a brief synopsis follows.

Traumatic brain injury (TBI) has a bimodal age distribution, with the first peak at 15–19 years and the second appearing in those over 65 years of ages. The most common cause of TBI in older patients is falling from standing. Despite this low-energy mechanism, the brain is more susceptible to injury due to the progressive volume loss and atrophy that results in increased space for shear injury [11]. Elderly patients with traumatic brain injuries have worse outcomes when compared to similar injuries in the young [12].

Cardiovascular changes that occur in this population include arterial atherosclerosis which can lead to an elevation in baseline systemic vascular resistance (SVR). Disruption in coronary autoregulation from scarring can result in ischemia. The typical tachycardia in response to hypovolemia may also be blunted in these patients due to medications. The increase in SVR may produce a falsely elevated blood pressure. If these patients are chronically hypertensive, a normal systolic blood pressure (SBP) may be relatively hypotensive for an individual patient and may result in end-organ ischemia [13]. The effect of age on the pulmonary system is impaired gas exchange due to a reduced alveolar surface area [14]. Chest wall compliance is decreased and may result in a blunted cough reflex leading to increased risk for aspiration [15]. There is also a risk for renal failure following trauma. The renal tubular function declines with increasing age as indicated by a decrease in the glomerular filtration rate (GFR). Chronic diuretic use may predispose to electrolyte abnormalities and a contracted plasma volume. The collecting tubules may not concentrate or retain appropriate electrolytes and are at risk for acute kidney injury/failure due to medications and/or ischemia [16]. Changes in the gastrointestinal system result in increased reflux disease and dysphagia resulting in a higher risk for aspiration in the elderly. Aging causes a slower transit time and colonic disturbances ranging from constipation to diarrhea. The musculoskeletal system is also affected. Lean body mass decreases at a rate of 10 % per decade after the age of 50. The reduction in the number and size of myocytes results in progressive weakness with increasing age. This loss of muscle mass combined with osteoporosis leads to an increased risk of fall-related fractures. Hip fractures are a common injury in the elderly and result in an eightfold increase in all-cause mortality within 3 months after the fall [17]. The endocrine and immune systems are also affected by aging. Extensive hormonal changes occur and thermoregulation may be impaired. Elderly patients are also more susceptible to infections and concomitantly are less able to mount a normal immune response. Moreover, malnutrition is common in the elderly requiring nutritional supplementation to prevent profound catabolism [18]. In summary, every organ system is affected by the aging and predisposes to injury, infection, and disability. Medications for preexisting illnesses may also complicate the physiologic response to injury and resuscitation. It is paramount to take these changes into consideration when caring for a geriatric trauma patient.

The functional decline that occurs with aging can lead to an increase in traumatic injuries due to changes in the ability to do activities of daily living (ADLs). ADLs include bathing, dressing, eating, and mobilization. They are important in assessing an individual's ability to function independently. In noninstitutionalized Medicare recipients, 27 % had difficulty in performing one or more ADLs. The ability to conduct ADLs is worse for institutionalized recipients, and 95 % reported difficulties with one or more activity. Additionally, 74 % of those surveyed had difficulty with three or more activities. Limitations in ADLs related to chronic conditions that increase with age and can predispose to traumatic injuries [2]. An increase in the frequency of ground-level falls in this group can reflect a decline in the ability to perform daily activities. This decline in their ability to perform ADLs suggests that the elderly may become more prone to injuries with advancing age.

Trauma in the Elderly

Trauma is the fifth most common cause of death in the elderly. The mechanism of injury in this demographic is primarily blunt forces, and falls are the most common mechanism of injury in this group (Figs. 1.2, 1.3, and 1.4) [1]. The increased population of older adults with active lifestyles has led to a dramatic increase in geriatric traumas. In 2008, adults in the United States aged 65 years and older accounted for more than 5.8 million emergency department visits for injuries, contributing to 30 % of all visits by older adults and almost 14 % of all injury-related emergency department visits [19]. The increase in life expectancy and independent living will lead to an increase in elderly drivers. It is estimated that between 20 and 30 million licensed drivers are currently older than 65 [20]. This number is projected to increase to 50 million by 2030. This explosion in geriatric drivers will be associated with an increase in motor vehicle collisions and/or pedestrians struck and result in an increase in the mortality rate.

The geriatric trauma population poses a special challenge to the trauma team. The mechanism of injury is different than those seen in younger patients. Injuries sustained are more severe in older versus younger adults, and with the increased presence of comorbid disease and independent effects of age, this leads to increased morbidity and mortality in older patients [21]. Several studies have reported an age-related increase in mortality rates, for all injury mechanisms and ISS [22–24].

Multiple mechanisms that result in trauma exist in the elderly population. Of those patients that fall, it is usually a repeated occurrence, and 71 % of falls result in an injury requiring medical care [25]. Additional mechanisms of blunt trauma include motor vehicle collisions, pedestrians struck, and burn injuries. According to the NTDB, less than 5 % of deaths are due to penetrating injuries in this age group [26]. Elderly patients who sustain blunt chest trauma with rib fractures have a morbidity and mortality rate twice that compared to those younger than 65. For each additional rib fracture in the elderly, mortality increases by 19 % and the risk of pneumonia by 27 % [27]. Moreover, when considering

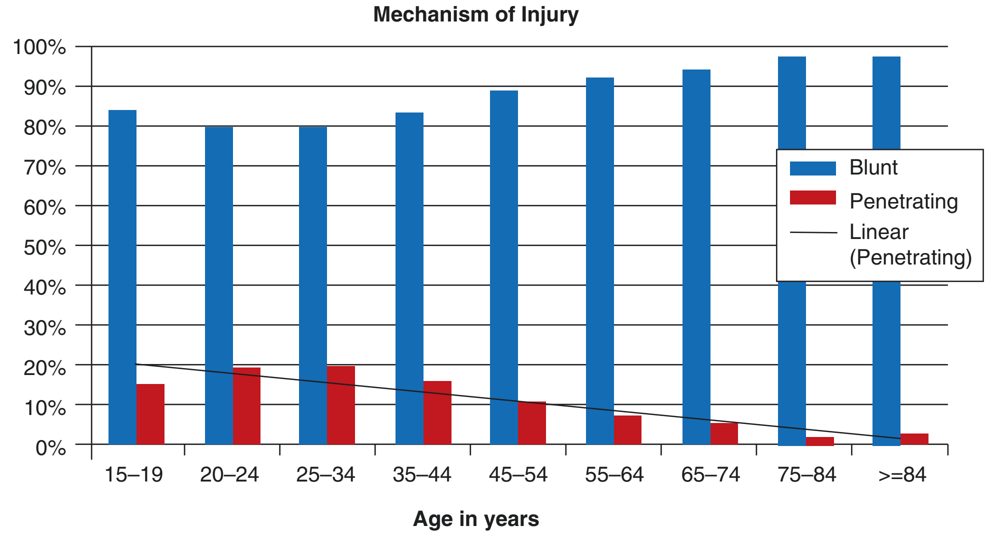


Fig. 1.2 Mechanism of injury in the elderly [1]

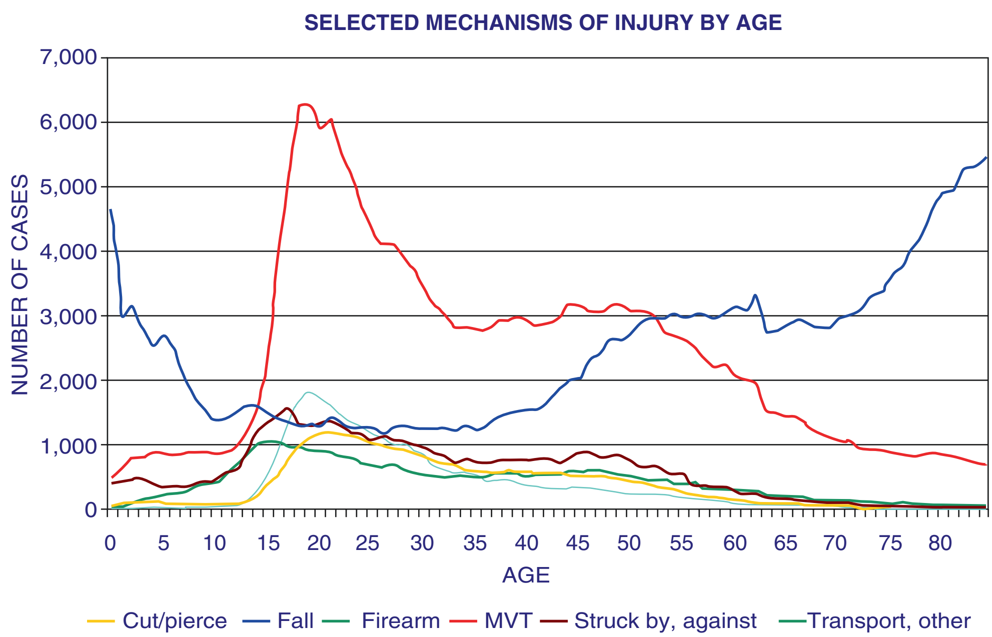
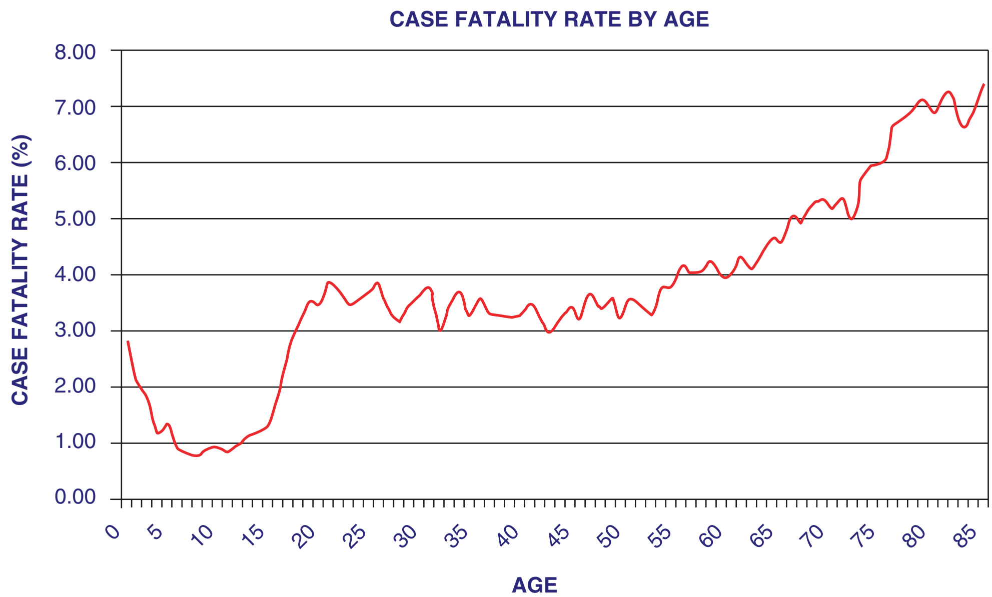


Fig. 1.3 Mechanism of injury by age

rib fracture injuries, "elderly" has been shown to be as young as 45 and older [28]. Clinical pathways that aggressively treat the pain and attempt to prevent the associated respiratory complications have shown to be successful in reducing morbidity and mortality [24].

Once an elderly person is injured, the prehospital system is not reliable in identifying those that are severely injured. This results in a significant undertriaging of these patients. Demetriades et al. found that 63 % of elderly patients that were severely injured (ISS >15) and 25 % of those critically injured did not meet the trauma center's standard trauma activation criteria. They concluded that patients older than 70 years should be considered for trauma team activation based on age alone [29, 30]. The EAST guidelines recommend that geriatric patients should be triaged to a trauma center, but do not use age as an impetus to activate the trauma team [31]. The state of Ohio has implemented a specific geriatric triage based on age [32]. Furthermore, once the elderly are in the emergency department, they may not be easily identified as in shock. Physiologic changes that occur in the elderly may alter the typical physiologic signs and manifestations of shock. Scalea et al. studied patients older than 65 involved in motor vehicle collisions and found their physiology allowed them to present with a higher than expected systolic blood pressure (SBP) due to an elevated systemic vascular resistance (SVR). Of those initially deemed hemodynamically stable with a normal SBP at presentation, 43 % were found to actually be in cardiogenic shock, and 54 % of these patients died [33].

Fig. 1.4 Case fatality by age



Accordingly, base deficit may correlate better with mortality in the elderly trauma population. In those older than 55 years, a base deficit greater than 10 was associated with an 80 % mortality rate. In contrast, a base deficit between 3 and 5 equated to a mortality rate of 23 % [34]. Geriatric blunt trauma patients warrant increased vigilance despite normal vital signs on presentation. It has been suggested that criteria for the elderly include a heart rate greater than 90 or a systolic blood pressure less than 110 mm [35]. The National Trauma Triage Protocol (NTTP) has additionally recognized that SBP less than 110 mmHg may represent shock in those older than 65 years, and practitioners should be vigilant when such a patient presents [36].

Additionally, medications taken prior to an accident can confound the diagnosis of significant injury as well as the need for resuscitation of these patients. Over 80 % of patients that fall are treated with a drug that could have contributed to the fall including antidepressants, antihypertensives, and sedatives [37]. Beta-blockers prescribed for hypertension can blunt the normal tachycardiac response to hemorrhage resulting in a false sense of security that the patient is stable. Anticoagulants, including warfarin, Plavix, aspirin, and new novel anticoagulants, can result in increased bleeding. This can be especially detrimental when traumatic brain injuries are present and expeditious reversal should occur. Patients that do present while on an anticoagulation medication have a higher risk of death [38]. Moreover, increasing numbers of elderly Americans take novel anticoagulants such as direct factor Xa inhibitors ("xabans," rivaroxaban) or direct thrombin inhibitors (dabigatran) for a variety of indications. Although these therapeutic agents benefit patients at risk for thrombotic or embolic events, they increase the risk for post-injury hemorrhage and alterations in the post-discharge destination [39]. Additionally, patients taking these medications have a higher risk of death if they present with a head injury and should speak with their prescribing physicians regarding the risk/benefit of taking these oral anticoagulants, especially if they are prone to falls [40].

Furthermore, delirium, which is common in the elderly, may add to the difficulty in assessing the injured patient in the emergency department. Delirium affects up to 10 % of elderly patients presenting to the emergency department and can confound assessment of these patients. Delirium is often the first presentation of sepsis in the elderly and is unrecognized which may lead to an increase in mortality [15]. As early sepsis can result in falls and therefore traumatic injuries, sepsis screening in the emergency department should be implemented early in these patients [41].

Once admitted to the hospital, geriatric patients pose a unique challenge to the trauma service due to their abnormal response to shock and injury. Bradburn et al. established a geriatric protocol that significantly reduced mortality in their patient population. The protocol included a geriatric consultation, a lactate level, arterial blood gas, and echocardiogram [42]. An additional study by Lenartowicz et al. showed that a proactive geriatric consultation resulted in decreased delirium and short time for discharge to long-term care facilities [43].

In a large series of elderly patients, mortality was demonstrated to correlate closely with ISS. It was also influenced by blood and fluid requirements as well as the GCS score. Regression analysis revealed that ISS predicted adult respiratory distress syndrome, pneumonia, sepsis, and gastrointestinal complications; fluid transfusion predicted myocardial infarction; and need for surgery and transfusion requirements predicted sepsis. These complications, in turn, were significant risk factors for mortality [23]. Additionally, geriatric patients requiring intubation and blood transfusion or suffering from head, C-spine, or chest trauma have an increased likelihood of death. Inhospital respiratory, gastrointestinal, or infectious complications also predict a higher mortality [44]. A prognostic tool for geriatric mortality after injury called the "Geriatric Trauma Outcome Score" (GTOS), where GTOS = [age] + [ISS × 2.5] + [22 if transfused any PRBCs by 24 hours after admission], has been developed, and the data available at 24 hours post-injury accurately predicts inhospital mortality for injured elderly patients [45].

Early Inpatient Rehabilitation

Weakness associated with impaired function is commonplace in the injured elderly patient. Admission to the intensive care unit (ICU) often results in increased muscle weakness, and the need for short- and long-term rehabilitation is frequent. Implementing early physical therapy in the ICU can result in increased strength as well as decreased length of ICU and hospital stay. More importantly, preventing core muscle wasting and preserving strength can reduce mortality [46].

Multiple hospitalizations increase in the last few months of life, as does the use of intensive care services, suggesting an increase in intensity of care. Other studies have also found an increase in the aggressiveness of care at the end of life. On the other hand, the sustained growth in hospice payments indicates that palliative and supportive care services are becoming utilized more as well. Some patients receive both types of care, undergoing aggressive treatment for some time and then entering a hospice program a short time before death. The relationship between hospice utilization and other services is unclear. Whereas hospice may substitute for more aggressive care in some cases, it may be used in addition to conventional care services in others [47].

Geriatric trauma patients have an overall higher mortality rate for equivalent injuries when compared to younger patients. Additionally, their likelihood of dying within 5 years after an injury increases significantly with time. Despite this elevated mortality rate, a portion are able to go home and resume a good quality of life. Of those that are discharged from the hospital, 52 % are to home, 25 % are to skilled nursing facilities, and 20 % are to rehabilitation facilities. The discharge process can be complex from a financial and emotional standpoint as a traumatic event often results in the need for additional care and loss of independence.

End-of-Life Issues

Advanced care directives and honoring a patient's end-of-life wishes are salient in the geriatric population. Laws regarding these medical decisions arose to preserve a patient's autonomy in a critical care scenario. Patients often fear prolonged suffering, emotional and financial burdens on their families, and concerns about lack of control of their end-of-life care [48]. Of 3746 older adults (>60 years old), 42.5 % required end-of-life decision-making. Of these patients, 70.3 % lacked capacity. Patients with an advance directive were significantly more likely to want limited or comfort care and have their wishes honored [49]. Additionally, the elderly are more likely to have a do-not-resuscitate (DNR) status at the time of death. The increased rate of DNR could be either a reflection of increased injury or poor physiology and inability to tolerate resuscitation [31].

As the elderly portion of the population has increased, the prehospital presence of advanced directive decisions has also increased. In 1994, the SUPPORT study showed that only 21 % of seriously ill patients had an advanced directive, while a 2010 study revealed that 67 % had an advanced directive [50]. A retrospective study by Trunkey et al. evaluated the decision-making process for those geriatric patients that were at risk for death. This study revealed that the elderly frequently have more concerns about long-term disability rather than death. Notably, the families were initially reluctant to discuss the topic of end-of-life care, but ultimately the majority of end-of-life discussions centered on withdrawal of therapies and establishing comfort care measures. Moreover, surgeon input regarding the projected quality and quantity of life was also instrumental when family members were establishing goals of care [51].

The legal aspects of end-of-life care vary from state to state. The POLST form was implemented in Oregon in 1991. This advanced directive addresses four treatment options: code status, transportation wishes, desire for antibiotic administration, and tube feeding. This form is an easily identifiable bright pink form, and the data collected is entered into a central database that can be easily accessed by EMTs and emergency physicians in case of injury. Multiple studies have evaluated this program's effectiveness in preventing unwanted treatments, hospitalization, and resuscitations. In a review of nursing home patients, 91 % had DNR orders, which were ultimately honored. Additionally in a survey of EMTs, 93 % of them regarded the POLST form favorably, and greater than half reported using the POLST to change a patient's treatment plan [52]. While this plan is more applicable to those in nursing home facilities, it can aid in decision-making when the elderly are injured. Advanced care planning should be addressed upon admission as soon as the patient or their surrogate is present. It belies the trauma team to be proactive in addressing these issues early so the patient's autonomy is protected and specific interventions are not performed.

As the post-World War II generation continues to age and the geriatric population expands, our medical system must likewise mature to provide optimal care for them. The cost of healthcare will continue to increase and will place new strains on an already stressed system. As trauma is a major cause of morbidity and mortality in the elderly, efforts to improve all aspects of acute care should increase to match the growing demands. A high index of suspicion is needed when caring for these patients as they may not follow the standard physiologic response when injured. Additionally an increasing number of elderly patients are prescribed novel anticoagulants, adding complexity to their care. Furthermore, end-of-life discussions are paramount when caring for the elderly. Goals of care should be established early with the patient or their representative in order to maintain the patient's autonomy and to honor their wishes. Expansion of geriatric-centered strategies to improve trauma prevention, triage, resuscitation, critical care, and rehabilitation in the elderly is necessary to meet the needs of this rapidly expanding, complex population.

References

1. Adams SD, Cotton BA, McGuire MF, et al. Unique pattern of complications in elderly trauma patients at a level I trauma center. J Trauma Acute Care Surg. 2012;72(1):112–8.
2. Profile of Older Americans. Available at: http://www.aoa.gov/ aoaroot/aging_statistics /Profile/index.aspx. Accessed 17 Nov 2012.
3. Administration on Aging (AoA). Aging statistics. Available at: http://www.aoa.acl.gov/Aging_Statistics/index.aspx. Accessed 3 Dec 2015.
4. HCUP Facts and Figures 2008: Statistics on Hospital-Based Care in the United States. Available at: http://www.hcup-us.ahrq.gov/reports/factsandfigures/2008/intro.jsp. Accessed 17 Nov 2012.
5. Wier L, Pfuntner A, Steiner C. Healthcare Cost and Utilization Project (HCUP) Statistical Briefs. Rockville (MD): Agency for Health Care Policy and Research (US); 2006.
6. Projections of Future Growth of the Older Population. Available at: http://www.aoa.gov/aoaroot/aging_statistics/future_growth/future_growth.aspx#age. Accessed 17 Nov 2012.
7. The High Concentration of U.S. Health Care Expenditures. Available at: http://www.ahrq.gov/research/ria19/expriach4.htm. Accessed 17 Nov 2012.
8. Kane RL, Kane RL. Essentials of clinical geriatrics. New York: McGraw-Hill Medical; 2009.
9. Lubitz JD, Riley GF. Trends in medicare payments in the last year of life. N Engl J Med. 1993;328(15):1092–6.
10. Bergeron E, Lavoie A, Clas D, et al. Elderly trauma patients with rib fractures are at greater risk of death and pneumonia. J Trauma. 2003;54(3):478–85.
11. Thompson HJ, McCormick WC, Kagan SH. Traumatic brain injury in older adults: epidemiology, outcomes, and future implications. J Am Geriatr Soc. 2006;54(10):1590–5.
12. Mosenthal AC, Livingston DH, Lavery RF, et al. The effect of age on functional outcome in mild traumatic brain injury: 6-month report of a prospective multicenter trial. J Trauma. 2004;56(5):1042–8.
13. Menon V, Greene T, Wang X, et al. C-reactive protein and albumin as predictors of all-cause and cardiovascular mortality in chronic kidney disease. Kidney Int. 2005;68(2):766–72.
14. Kulik AM, Kondrat'eva LN. Combined effects of hypoxia and hypercapnia on the functional state of the respiratory center. Biull Eksp Biol Med. 1975;79(4):39–43.
15. Han JH, Shintani A, Eden S, et al. Delirium in the emergency department: an independent predictor of death within 6 months. Ann Emerg Med. 2010;56(3):244–52. e1
16. Marik PE. Management of the critically ill geriatric patient. Crit Care Med. 2006;34(9 Suppl):S176–82.
17. Haentjens P, Magaziner J, Colón-Emeric CS, et al. Meta-analysis: excess mortality after hip fracture among older women and men. Ann Intern Med. 2010;152(6):380–90.
18. Cahill NE, Dhaliwal R, Day AG, Jiang X, Heyland DK. Nutrition therapy in the critical care setting: what is "best achievable" practice? An international multicenter observational study. Crit Care Med. 2010;38(2):395–401.
19. National Center for Health Statistics. National Hospital Ambulatory Medical CareSurvey: 2008 Emergency Department Summary Tables. Available at: http://www.cdc.gov/nchs/data/ahcd/nhamcs_emergency/2008_ed_web_tables.pdf2008. Accessed 4 Dec 2015.
20. Hakamies-Blomqvist L, Wiklund M, Henriksson P. Predicting older drivers' accident involvement--Smeed's law revisited. Accid Anal Prev. 2005;37(4):675–80.
21. Hollis S, Lecky F, Yates DW, et al. The effect of pre-existing medical conditions and age on mortality after injury. J Trauma. 2006;61(5):1255–126.
22. Richmond TS, Kauder D, Strumpf N, Meredith T. Characteristics and outcomes of serious traumatic injury in older adults. J Am Geriatr Soc. 2002;50(2):215–22.
23. Tornetta 3rd P, Mostafavi H, Riina J, et al. Morbidity and mortality in elderly trauma patients. J Trauma. 1999;46(4):702–6.
24. Holcomb JB, McMullin NR, Kozar RA, Lygas MH, Moore FA. Morbidity from rib fractures increases after age 45. J Am Coll Surg. 2003;196(4):549–55.
25. Aschkenasy MT, Rothenhaus TC. Trauma and falls in the elderly. Emerg Med Clin North Am. 2006;24(2):413–32. vii
26. American College of Surgeons: Trauma Programs: NTDB: NTDB® Data Points. Available at: http://www.facs.org/trauma/ntdb/datapoints.html. Accessed 17 Nov 2012.
27. Bulger EM, Arneson MA, Mock CN, Jurkovich GJ. Rib fractures in the elderly. J Trauma. 2000;48(6):1040–6. ; discussion 1046–1047
28. Testerman GM. Adverse outcomes in younger rib fracture patients. South Med J. 2006;99(4):335–9.
29. Demetriades D, Sava J, Alo K, et al. Old age as a criterion for trauma team activation. J Trauma. 2001;51(4):754–6. ; discussion 756–757
30. Goodmanson NW, Rosengart MR, Barnato AE, Sperry JL, Peitzman AB, Marshall GT. Defining geriatric trauma: when does age make a difference? Surgery. 2012;152(4):668–75.
31. Jacobs DG. Special considerations in geriatric injury. Curr Opin Crit Care. 2003;9(6):535–9.
32. Werman HA, Erskine T, Caterino J, Riebe JF, Valasek T. Development of statewide geriatric patients trauma triage criteria. Prehosp Disaster Med. 2011;26(3):170–9.
33. Scalea TM, Simon HM, Duncan AO, et al. Geriatric blunt multiple trauma: improved survival with early invasive monitoring. J Trauma. 1990;30(2):129–34. ; discussion 134–136
34. Martin JT, Alkhoury F, O'Connor JA, Kyriakides TC, Bonadies JA. "Normal" vital signs belie occult hypoperfusion in geriatric trauma patients. Am Surg. 2010;76(1):65–9.
35. Heffernan DS, Thakkar RK, Monaghan SF, et al. Normal presenting vital signs are unreliable in geriatric blunt trauma victims. J Trauma. 2010;69(4):813–20.
36. Brown JB, Gestring ML, Forsythe RM. Systolic blood pressure criteria in the National Trauma Triage Protocol for geriatric trauma: 110 is the new 90. J Trauma. 2015;78(2):352–9.
37. Nordell E, Jarnlo GB, Jetsén C, Nordström L, Thorngren KG. Accidental falls and related fractures in 65-74 year olds: a retrospective study of 332 patients. Acta Orthop Scand. 2000;71(2):175–9.

38. Ohm C, Mina A, Howells G, Bair H, Bendick P. Effects of antiplatelet agents on outcomes for elderly patients with traumatic intracranial hemorrhage. J Trauma. 2005;58(3):518–22.
39. DiBartolomeo S, Marino M, Valent F, et al. Effects of anticoagulant and antiplatelet drugs on the risk for hospital admission for traumatic injuries: a case-control and population-based study. J Trauma. 2014;76(2):437–42.
40. Inui TS, Parina R, Chang D, et al. Mortality after ground-level fall in the elderly patient taking oral anticoagulation for atrial fibrillation/flutter: A long-term analysis of risk versus benefit. J Trauma. 2014;76(3):642–50.
41. Moore LJ, Turner KL, Todd SR, McKinley B, Moore FA. Computerized clinical decision support improves mortality in intra abdominal surgical sepsis. Am J Surg. 2010;200(6):839–43. ; discussion 843–844
42. Bradburn E, Rogers FB, Krasne M, et al. High-risk geriatric protocol: improving mortality in the elderly. J Trauma Acute Care Surg. 2012;73(2):435–40.
43. Lenartowicz M, Parkovnick M, McFarlan A, et al. An evaluation of a proactive geriatric trauma consultation service. Ann Surg. 2012;256(6):1098–101.
44. Labib N, Nouh T, Winocour S, et al. Severely injured geriatric population: morbidity, mortality, and risk factors. J Trauma. 2001;71(6):1908–14.
45. Cook AC, Joseph B, Inaba K, et al. Multicenter external validation of the geriatric trauma outcome score: a study by the prognostic assessment of life and limitations after trauma in the elderly[PALLIATE] consortium. J Trauma. Available at: http://journals.lww.com/jtrauma/Abstract/publishahead/Multicenter_External_Validation_of_the_Geriatric.99700.aspx. Accessed 4 Dec 2015.
46. Morris PE, Goad A, Thompson C, et al. Early intensive care unit mobility therapy in the treatment of acute respiratory failure. Crit Care Med. 2008;36(8):2238–43.
47. Riley GF, Lubitz JD. Long-term trends in medicare payments in the last year of life. Health Serv Res. 2010;45(2):565–76.
48. Bok S. Personal directions for care at the end of life. N Engl J Med. 1976;295(7):367–9.
49. Silveira MJ, Kim SYH, Langa KM. Advance directives and outcomes of surrogate decision making before death. N Engl J Med. 2010;362(13):1211–8.
50. Gordy S, Klein E. Advance directives in the trauma intensive care unit: Do they really matter? Int J Crit Illn Inj Sci. 2011;1(2):132–7.
51. Trunkey DD, Cahn RM, Lenfesty B, Mullins R. Management of the geriatric trauma patient at risk of death: therapy withdrawal decision making. Arch Surg. 2000;135(1):34–8.
52. Schmidt TA, Hickman SE, Tolle SW, Brooks HS. The physician orders for life-sustaining treatment program: oregon emergency medical technicians' practical experiences and attitudes. J Am Geriatr Soc. 2004;52(9):1430–4.

Effect of Aging on Cardiac Function Plus Monitoring and Support

2

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Key Points

- Elderly surgical patients are common and often require evaluation and support of the cardiovascular system.
- Aging significantly impacts ventricular and vascular anatomy resulting in altered cardiac functionality.
- Physiological changes of aging include a blunted baroreflex and altered beta-adrenergic responsiveness resulting in a decreased dependence on chronotropy and an increased reliance on stroke volume in response to stress.
- Monitoring of the elderly cardiovascular system is valuable and can be achieved in a number of noninvasive and invasive methods.
- Management of shock in the elderly benefits from an understanding of the needs of the specific patient and a recognition of the risks/benefits of each cardiovascular medication.

Introduction

As the world's population continues to grow, advanced age has become an increasingly important risk factor influencing morbidity and mortality. It is estimated that in the United States alone, approximately 20 % of the population will be over the age of 65 by the year 2030 [1–3]. As the fastest-growing population, specialized attention to the physiology of these aging patients is paramount to the successful treatment of the elderly. Specifically, cardiovascular disease remains the most prevalent and influential comorbidity affecting outcomes in the elderly surgical patient. Half of all heart failure cases in the United States are older than 75, and 90 % of heart failure deaths occur in adults older than 65 [4]. Heart failure is also the leading cause for hospitalization in Medicare beneficiaries. With advances in the care of chronic diseases and longer life expectancy, familiarity with the effects of aging and how to treat elderly patients in all fields of medicine is required to successfully treat this population. The unique physiology of the aging cardiovascular system as well as the impact of these changes during the stress of surgery is outlined in Table 2.1. Understanding these changes and their implications to the treatment of the elderly patient will improve care and outcomes in this population.

Effect of Aging on the Right Ventricle

The right ventricle is connected in series to the left ventricle and is therefore obligated to pump the same stroke volume. As the cardiovascular system ages, this relationship is not always maintained, and right heart flow may not always equal left heart flow. Radio-nucleotide studies and echocardiography have demonstrated impairment in both systolic and diastolic right ventricular function. The mechanism for this reduction is believed to be secondary to a gradual age-related increase in pulmonary arterial vascular resistance, clinically evident by increased pulmonary artery systolic pressures [3]. Using M-mode echocardiography in combination with Doppler technology, right ventricular impairment is demonstrated by observing a reduction of tricuspid annular plane systolic excursion. The tricuspid annular plane systolic excursion (TAPSE) estimates the longitudinal contractile properties of the right ventricle. These modalities demonstrate a significant reduction in TAPSE in otherwise healthy subjects as they age. Pulsed tissue-derived measurements of right ventricular systolic function have confirmed these findings agreeing with findings of older studies demonstrating reduced systolic function on echocardiography.

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Table 2.1 Summary of the effect of aging on the cardiovascular system

Cardiovascular element	Alteration in the elderly
Right ventricle	Reduced systolic function Reduced diastolic function
Left ventricle	Left ventricular hypertrophy Dependence on atrial contribution Age-related impaired contractility and relaxation
Vascular structures	Increased arterial stiffness Systolic hypertension
Cardiac output	Preserved resting cardiac output Preserved ejection fraction
Changes in physiology	Blunted baroreceptor reflex Decreased adrenergic responsiveness
Response to stress	Decreased reliance on heart rate Increased cardiac output due to increased stroke volume

Inefficient rotational motions and non-longitudinal muscular movement contribute to the age-related decrease in right heart systolic function.

The aging process also affects right heart diastolic function. Diastolic functional properties can be characterized by determining right atrial pressure (RAP), tricuspid inflow velocity (E), myocardial early diastolic velocity (Ea), and atrial peak velocity (Aa) [5, 6]. Age is significantly correlated with progressive increases in Aa and decreases in Ea. Additionally, there is a negative relationship between the Ea/Aa ratio and increasing age, indicating less filling velocities in the ventricle despite higher atrial velocities [5, 6]. In the same way that systolic functional decline is attributed to increasing stiffness of the pulmonary vasculature, diastolic functional changes are attributed to increased right heart afterload [5].

Effect of Aging on the Left Ventricle

Years of ongoing stress on the heart result in changes in cardiac function related to increased workload. As aging blood vessels stiffen leading to elevated systolic blood pressure, the left ventricle (LV) changes in response. The heart is required to perform greater amounts of stroke work (stroke volume times blood pressure) in the presence of sustained elevations in systolic pressure resulting in LV wall thickening in elderly patients [7]. Structural changes observed on cardiac MRI demonstrate a significant increase in myocardial thickness as a result of increased cardiomyocyte size. The overall shape of the heart also changes from an elliptical to spheroid shape with asymmetric increase in the intraventricular septum as opposed to free wall hypertrophy [8, 9].

Resting diastolic filling rates decline with age as evidenced by studies utilizing M-mode echocardiography and gated blood pool scans. Diastolic filling of the ventricles occurs in passive and active phases. As individuals age, the heart fills more slowly, and the bulk of ventricular filling shifts to late diastole, with less passive filling. As a result, ventricular filling during diastole becomes more dependent on the active phase. Atrial enlargement is observed as atrial contraction contributes more and more to ventricular filling [8].

Studies using cardiac MRI have investigated LV structure and function. These studies have demonstrated the development of LV hypertrophy and fibrosis leading to diastolic dysfunction and heart failure with preserved systolic function. Indices of cardiac function (such as ejection fraction and ejection velocity) were preserved despite reductions in both LVEDV and LVESV [10]. It appears that modest hypertrophic changes in the left ventricular wall are adaptive to preserve cardiac function at rest. However, exercise capacity is reduced. Fibrotic cardiac remodeling plays an important role in the development of diastolic heart failure with age, and adaptive changes to maintain cardiac output play a pivotal role in senescent cardiac function [9, 11]. These adaptations of the aging LV to maintain cardiac output include prolonged contraction, atrial enlargement, and increased contribution to LV filling [12].

Effect of Aging on Vascular Structures

Increasing arterial stiffness is the predominant change that occurs within the cardiovascular system in the setting of advanced age. The degree of arterial stiffness is proportionally greater in the diseased cardiovascular system. Potential energy released during the cardiac cycle stretches elastin fibers in the arteries and subsequently transmits this energy smoothly downstream to the muscular arterioles and capillary beds [13]. The aging process causes elastin to become depleted and replaced with increased amounts of non-distensible collagen and calcium [14].

The depletion of elastin and replacement with calcium and collagen results in systolic hypertension syndrome that is characterized by an increase in systolic pressures with a lowering or maintenance of the diastolic pressure level resulting in a widened pulse pressure [15]. These changes in the walls of vascular structures predispose to non-laminar and turbulent blood flow, which increases tensile and shear forces on the vessel wall resulting in progressive injury. To compensate for arterial stiffening, cardiac changes result in increased blood velocity to overcome the increased afterload of the stiffened central arterial tree [16].

Vascular changes that occur due to aging result in compromised diastolic filling and subsequently the ability of elderly patients to tolerate the stress of injury or surgery. Central arterial elasticity decreases with age and is paralleled by increased pulse wave velocity occurring in the forward and backward (reflected) direction. Based on the intrinsic compliance of their vessels, young patients have pulse wave reflections occurring in diastole that augment coronary perfusion and ameliorate tensile shear forces of pulsatile blood flow [16]. Blood flow in less compliant vessels has enhanced shear due to turbulent flow and does not augment diastolic filling of coronary vessels that are already at risk due to atherosclerosis. A widened pulse pressure is the manifestation of stiffened central arteries due to a cardiac impulse transmitted downstream with greater force, causing reflected waves to return at end or peak systole [16].

Effect of Aging on Cardiac Output

With healthy aging the overall resting systolic function does not change. Cardiac imaging utilizing both echocardiography and radio-nucleotide studies has confirmed the preservation of systolic function [11]. The maintenance of myocardial performance was felt to be due to increases in left ventricular thickness, a prolongation of contraction times, an enlargement of the atria, and an increase in the contribution of the atrium to left ventricular filling [17]. With the development of cardiac MRI has come an advanced understanding of the performance of the heart in the elderly. It is now recognized that although older myocytes do increase in size, there is an overall myocyte depletion that is associated with increased collagen deposition and nonenzymatic cross-linking [18]. While the older ventricle increases in overall mass, it does not increase in functional mass, as evidenced by increasing left ventricular mass to volume ratios and associated declines in LVEDV in relation to left ventricular mass. Although the resting EF is preserved, absolute stroke volume does not remain comparable [19]. While both LVEDV and LVESV decrease with age, the decrease in LVEDV is proportionally greater than the decrease in LVESV, which leads to an overall age-related decline in resting stroke volume [19]. It was previously felt that the preservation of EF meant that elderly patient could respond to stress similar to their younger counterparts. Though the preservation of the net systolic function remains unaltered, with exercise the effects of aging are more evident. The reduction of cardiac reserve is a result of multiple factors including increased vascular afterload, arterial-ventricular mismatch, reduced contractility, impaired autonomic regulation, and physical conditioning.

Effect of Aging on the Beta-Adrenergic Response

The response of the cardiovascular system to surgical stress relies greatly on adrenergic stimulation. Exercise and stressors stimulate sympathetic output to increase heart rate, augment contractility and relaxation, and decrease afterload. Unfortunately, one of the consequences of the normal aging process is a decreased responsiveness to beta-adrenergic stimulation. Maximal heart rate (HRmax) decreases in the setting of aging and is responsible for decreases in aerobic work capacity. Decreases in HRmax are independent of gender, regular exercise, and other factors [6, 20]. This attenuation of heart rate responsiveness contributes significantly to an age-related reduction in maximal cardiac output and therefore determines aerobic exercise capacity [20].

The decrease in chronotropic responsiveness (HRmax) to exercise seen throughout the normal aging process remains poorly understood [20]. Proposed mechanisms for the decreased cardiovascular response to adrenergic stimulation are alterations to the conduction pathways as well as decreased receptor expression. With generalized increase in collagenous tissue and fibrosis of the cardiac myocytes, changes in the cardiac conduction system develop. Variable degrees of fibrosis and calcification of the cardiac skeleton can impact AV nodal conduction as well as the development of atrioventricular conduction block. Fat accumulation around the SA node is also observed with aging. This may cause partial or total separation of the SA node from the surrounding atrial tissue and may lead to decreased intrinsic heart rate. However, SA node dysfunction is not always identified in the setting of myocardial remodeling and instead may indicate a molecular change in the pacemaker cells [21]. The number of pacemaker cells also significantly declines with advanced age further decreasing the cardiovascular response to adrenergic stimulation [8]. Other observations have shown reductions in calcium channel proteins, which may lead to decreased sinus node depolarization reserve and thus suppression of action potential formation and propagation [22].

Another mechanism proposed for the decreased adrenergic responsiveness is a decrease in cardiac adrenergic receptor density. Elevated adrenergic neurotransmitter levels have been observed in the elderly and appear to be a compensatory response to decreased receptor expression and deficient NE uptake at nerve endings [8]. With prolonged adrenergic expression and deficient uptake, neurotransmitter depletion can contribute further to the blunted cardiac response and LV systolic performance seen with exercise and stress in the elderly.

Effect of Aging on the Baroreflex Response

In the normally functioning cardiovascular system, the baroreflex serves as an efficient component of a complex feedback loop that maintains adequate cardiovascular function. The effect of aging on the baroreflex has been studied by relating pulse interval to changes in systolic blood pressure after phenylephrine injection. This work revealed a linear relationship between pulse interval and change in systolic blood pressure as well as a distinct decrease in the baroreceptor reflex sensitivity in the elderly [23, 24]. Others have found that an age-related decline in baroreflex sensitivity is independent of systolic blood pressure and systemic adrenergic levels [25]. Decreased baroreceptor reflex sensitivity was also demonstrated in a study of healthy volunteers examining cardiac response to angiotensin II (ANG II) infusions. The elderly, unlike younger patients, do not exhibit decreases in heart rate when blood pressure is increased via ANG II infusion [24, 26].

Effect of Surgery on the Geriatric Cardiovascular System

Much of what we know about the response of the aging cardiovascular system to surgery has been elucidated from a body of work evaluating the impact of exercise. Surgery results in a substantial amount of physical and metabolic stress on the body due to blood loss, the inflammatory response, and the effects of anesthesia. The effects of stress vary greatly depending on the age of the patient and the presence of associated comorbidities. Exercise provides a controlled stress state that allows some understanding of the effects of surgery on the elderly.

The normal response to exercise and presumably to surgical stress consists of an increase in cardiac output to meet the elevated metabolic needs of the body. Initially, it was believed that the elderly demonstrated a depressed cardiac output. Subsequent studies that excluded patients with coronary artery and myocardial disease showed a more appropriate increase in cardiac output although the mechanism appears to be different than in the young [27, 28]. Older patients cannot increase cardiac output with the typical increases in heart rate secondary to decreases in HRint and B-adrenergic responsiveness. The elderly optimize the Frank-Starling mechanism by increasing their end-diastolic volume and stroke volume during exercise, thereby increasing cardiac output without substantially increasing heart rate. While the elderly are able to augment stroke volume during exercise, the increase in ejection fraction is less than that observed in younger counterparts secondary to a decreased ability to reduce end-systolic volume. This physiologic response is similar to that which is seen in young patients administered with exogenous beta-blockade and then stressed with increasing exercise loads.

Surgery and injury are frequently associated with hypovolemia secondary to blood loss and capillary leak commonly leading to cardiovascular compromise. Free water loss, chronic poor oral intake, pharmacologic vasodilation (home medications), and decreased plasma oncotic pressure (poor nutrition) also commonly lead to further intravascular volume depletion. Given the dependence on the Frank-Starling modulation of cardiac output rather than chronotropy, the elderly patient is particularly sensitive to preload reductions. While Shannon and colleagues showed that elderly patients mount a blood pressure increase and slight HR increase similar to younger patients during tilt tests, this response is negatively affected by hypovolemia [29]. When the same test is performed after preload reduction with diuretics, elderly patients sustain a symptomatic fall in blood pressure due to an inability to mount a tachycardic response in contrast to younger patients who exhibit an appropriate increase in both heart rate and blood pressure [29].

The body of literature evaluating the effects of exercise on the aging cardiovascular system has demonstrated the ability to maintain cardiac output in response to the stress of surgery [27, 28]. The mechanism appears to depend upon stroke volume by increasing end-diastolic volumes and contractility rather than through the augmentation of heart rate [31]. Newer technology including cardiac MRI and pulsed tissue Doppler echocardiography in 2D and 3D has shown that while elderly patients can mount a cardiac output response to stress, this is of lesser magnitude than in their younger counterparts due to decreased cardiac reserve [12, 30, 31]. It is clear that elderly patients generate increased cardiac performance in the face of stress; however, the magnitude of this response is attenuated and less robust than that of younger counterparts.

Effect of Comorbidities on Cardiovascular Function: Atrial Fibrillation

Elderly patients rely heavily on prolonged contraction times and increased atrial contribution for adequate left ventricular filling. Atrial fibrillation (AF) is particularly problematic because atrial arrhythmias result in inconsistent and often inadequate ventricular filling due to limited contraction and decreased filling time. Age-related increases in left (and right) atrial size in older patients are a risk factor for the development of AF [32]. Additional age-related risk factors include inflammatory cytokines, local and systemic stress, altered calcium handling, and electrical remodeling on a chronic basis [33]. In the acute setting, pulsatile mechanical atrial stretch and inflammatory cytokines (from surgery, injury, or sepsis) contribute to arrhythmogenesis [33]. Numerous cytokines may contribute to the development of AF including interleukin-6, interleukin-8, and hsCRP [33]. These same cytokines are present in high levels in the serum of injured patients and can be used to predict progression to multiple organ failure in the injured patient [34]. Surgical patients are exposed to other risk factors including large-volume resuscitation causing atrial stretch, increased endogenous catecholamine release, rapid fluid and electrolyte shifts, hypoxia, and hypercarbia [35, 36]. Another common risk factor is withdrawal from chronic beta-blockade in the elderly following surgical procedures.

Atrial fibrillation significantly impacts elderly surgical patients and frequently complicates the postoperative course. Chronic AF should be managed with the main goal being control of heart rate as this results in more optimal long-term outcomes [37]. Maintenance management of AF usually consists of beta-blocker, calcium channel blocker, or antiarrhythmia medications such as amiodarone. These should be continued through the perioperative period as much as possible although this can be challenging in the setting of hemodynamic compromise and limited gastrointestinal function. In the setting of significant surgery or severe injury, acute AF is common and results in prolonged hospital length of stay. There is no superior treatment regimen, and therapy is usually tailored to meet the unique patient care needs present at the time of diagnosis. Trauma patients have been found to benefit from beta-blockade due to the commonly high levels of catecholamines present at the time of injury [38]. For postoperative patients, beta-blockade and calcium channel blockade are the most common and efficacious approaches in the presence of adequate perfusion. Patients with hemodynamic compromise at the time of AF onset may require synchronized cardioversion or the initiation of antiarrhythmics such as amiodarone. Often, therapy for acute AF is only needed during the perioperative period of time and can be discontinued as the body heals and the cytokine environment returns to normal. Nevertheless, AF should be diagnosed and managed expeditiously in the elderly due to greater expected reductions in cardiac output secondary to loss of atrial kick and need for longer diastolic filling times.

Effect of Comorbidities on Cardiovascular Function: Ischemic Heart Disease

Surgical patients are at significant risk for acute myocardial ischemia given the associated endogenous catecholamine release, systemic inflammation, and increased myocardial oxygen demand. Additionally, hyperdynamic blood flow during resuscitation and its associated turbulent and non-laminar blood flow increase vessel wall shear forces. This increased shear may cause the rupture of coronary atherosclerotic plaques and predispose to myocardial infarction (MI) [39]. The risk of MI is compounded in the elderly in whom arterial pulse wave indices do not support diastolic filling of coronary vessels and arterial stiffening only exacerbates conditions of turbulent arterial blood flow. Elderly patients are also at greater risk due to preexisting coronary artery and intrinsic cardiac disease. Perioperative MI represents an important disease entity to address as it is associated with worse outcomes especially in the aged [40].

The elderly are the most at risk to experience an MI after surgery, are the most likely to suffer poor MI-related outcomes, and subsequently are the most likely to benefit from intervention. Due to atypical symptomatology and presentation, MI is difficult to diagnose in the critically ill elderly patients. A high index of suspicion and liberal use of diagnostic modalities such as ECG and serial troponin measurements are required to identify acute myocardial ischemia. Myocardial ischemia should be considered in the setting of unexplained vital sign decompensation after hemorrhage and hypovolemia are ruled out. Echocardiogram may be valuable to identify wall motion abnormalities in the face of non-diagnostic troponin elevation [41]. Cardiology consultation should be obtained liberally in the setting of acute coronary syndrome as the patient may be a candidate for reperfusion with coronary intervention.

Effect of Comorbidities on Cardiovascular Function: Heart Failure with Preserved Ejection Fraction

Heart failure (HF) with preserved EF is defined as heart failure with an ejection fraction equal to or greater than 50 % and represents up to 40 % of patients with heart failure [42]. This clinical condition is important because patients will appear normal when at rest and this resting EF is often erroneously used in these patients as a surrogate for achievable cardiac performance under stress. Several exercise studies of patients with HF with preserved EF demonstrated an inability to adequately increase LV systolic elastance. Further, these patients demonstrate lower peripheral resistance, increase heart rate, and reductions in ventricular-arterial coupling that result in an intolerance of submaximal and maximal exercise workloads [42]. Patients with HF express maladaptive inotropic, lusitropic, chronotropic, and vasodilatory responses to the physical stress of exercise and are believed to have similar inadequate responses to the physical stress of surgery.

Monitoring the Aging Cardiovascular System

Due to the significant anatomic and physiologic limitations described above, the elderly cardiovascular system often requires multiple monitoring techniques to provide the necessary support during the perioperative period of time. The elderly do not have the same reserve as the young surgical patient and therefore require more exact maintenance of preload, contractility, and afterload to ensure adequate cardiac performance. The initial question that must be answered for any surgical patient should always be, “Is the patient in shock and underperfused?” The answer to this question is provided