

Trauma in the Elderly

Considerations for Anesthetic Management

Shawn E. Banks, MD^{a,*}, Michael C. Lewis, MD^b

KEYWORDS

- Geriatric trauma • Physiology of aging • Geriatric anesthesia • Hip fracture
- Pulse pressure variation • Systolic pressure variation • Regional anesthesia

KEY POINTS

- The volume of geriatric trauma patients is expected to increase significantly in coming years.
- Recognition of severe injuries may be delayed because they are less likely to mount classic symptoms of hemodynamic instability.
- Anesthetic medications and opiates will require reductions in doses for elderly patients.
- Monitoring pulse pressure and systolic pressure variations can provide an accurate assessment of volume status in mechanically ventilated patients.
- Regional anesthesia for hip fractures may offer better patient outcomes than general anesthesia.

INTRODUCTION

Geriatric patients comprise a rapidly expanding age segment of the population of developed countries. It is the fastest growing population segment in the United States. Individuals older than 65 years of age currently represent 13% of the US population, but the US Census Bureau projects that by 2050, they will represent more than 21%.¹ Accidental injury was the fifth most frequent cause of mortality among the elderly in 2009 and 2010,² and utilization of trauma care services is expected to increase significantly among older age groups. It is projected that geriatric patients will account for almost 39% of trauma admissions by 2050.³ Our understanding of best practices in geriatric trauma and anesthesia care continues to expand, as it does in all other areas of medicine.

DEFINITIONS OF AGING

Aging is a progressive process depicted as maintenance of life with a diminishing capability for adjustment.⁴ Senescence results in a progressive decline in cellular

^a Ryder Trauma Center, University of Miami Miller School of Medicine, Miami, FL, USA;

^b University of Miami Miller School of Medicine, Miami, FL, USA

* Corresponding author. 1611 Northwest 12th Avenue Suite SW303, Miami, FL 33136.

E-mail address: sbanks2@med.miami.edu

function, resulting in a loss of organ performance. Cells lose their capacity to respond to injury and eventually die. Senescence is associated with impaired adaptive and homeostatic mechanisms, resulting in an increased susceptibility to the effects of stress. Function may seem to be unchanged, yet physiologic reserve diminishes. Any disruption of homeostasis that is well tolerated by younger adults might precipitate functional decline in the elderly population. The situation is further compounded by variable response to medications and comorbidity.

A standard age-based definition for the term “geriatric” does not exist. Most researchers have used the breakpoint of 65 years of age and more, perhaps somewhat arbitrarily. Geriatric trauma has historical roots in the establishment of the social security system in the United States. Using a strict age-based criterion is also difficult because it is generally believed that aging adults will be entering their “geriatric” years in better health than they did in preceding generations and that preinjury function may significantly influence recovery from injury.

PHYSIOLOGIC CHANGES IN THE ELDERLY POPULATION

As a result of the aging process, there is a wide set of physiologic changes each of which result in a decreased ability to maintain homeostasis during stress.

Between the ages of 20 and 80 years there is a progressive decline in cardiac function, estimated to be as great as 50%. The progressive stiffening of the arterial tree and resultant adaptations of the aging myocardium can result in ventricular hypertrophy as shown in [Fig. 1](#). This impaired cardiac function, paired with decreased sensitivity to catecholamines, complicates the management of the hemodynamically compromised older patient.

Above the age of 50 years, renal mass is lost, with a corresponding fall in glomerular filtration rate.^{5,6} Renal tubular function is also compromised. These changes decrease the ability of the elderly to cope with large volume resuscitation.

Respiratory function is also compromised in the elderly population. There is an observed loss of lung elastic recoil and significant reduction of the vital capacity. Moreover, there is impaired mucociliary clearance of bacteria, dependence on diaphragmatic breathing, and reduced ability to cough.⁷ There is a disruption of the normal matching of ventilation and perfusion. Forced expiratory volume in 1 second, forced vital capacity, and peak expiratory flow rate are decreased.⁸ Such changes in diffusion capacity, ventilation-perfusion mismatch,⁹ and closing volumes mean that there is a decrease in baseline arterial oxygen tension with age.¹⁰ Alterations in compliance result in an increased work of breathing.¹¹ The combination of these factors means that there is an increased risk of respiratory failure in the elderly patient, resulting in a higher incidence of mechanical ventilation,¹² acute lung injury, and ventilation-associated pneumonia as a consequence of longer intensive care unit stay and high morbidity.

There are age-related changes of endocrine function. The tissue responsiveness to thyroxine and its production is reduced with aging.¹³ Secretion of cortisol does not seem to change with aging.¹⁴

FUNCTIONAL RESERVE

When an organism maintains a steady state in the face of increased physiologic demand, it is said to demonstrate a good functional reserve. [Fig. 2](#) illustrates this divergence between “baseline” and “stress” situations. Imbalance within the system therefore results in a breakdown of homeostatic compensation. A decline in functional

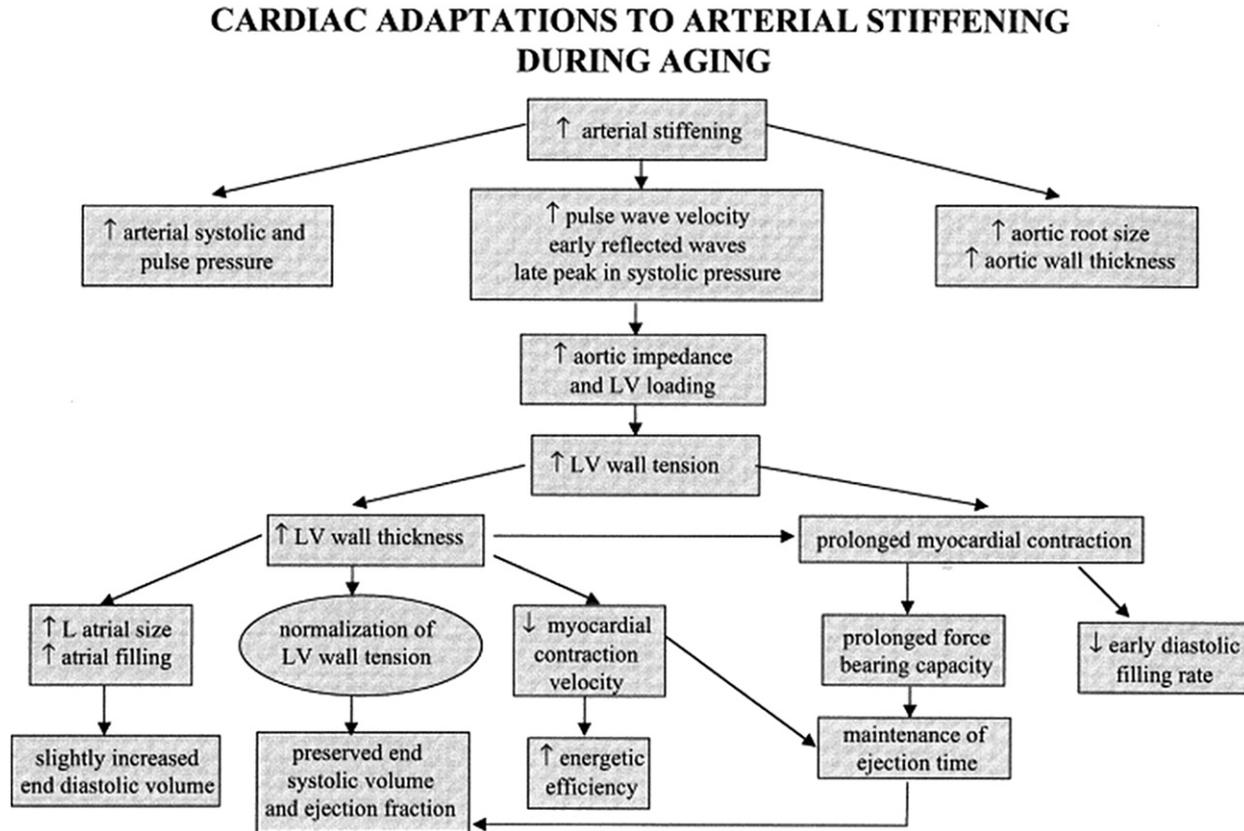


Fig. 1. Loss of elasticity in the arterial tree is thought to be responsible for increasing left ventricular (LV) afterload. The resulting ventricular hypertrophy may lead to diastolic dysfunction. (Reprinted from Lakatta EG, Sollott SJ. Perspectives on mammalian cardiovascular aging: humans to molecules. *Comp Biochem Physiol A Mol Integr Physiol* 2002;132(4):699–721. Elsevier, with permission.)

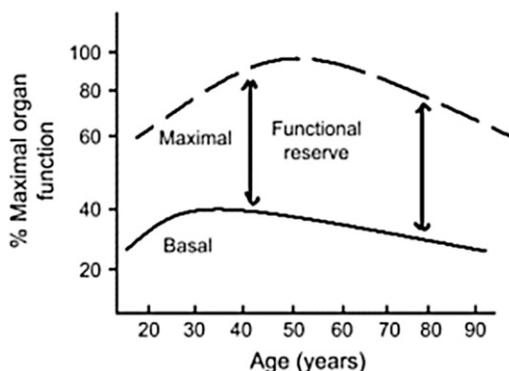


Fig. 2. The functional reserve is the difference between basal function (solid line) and maximal function (broken line). Even in healthy individuals, this functional reserve is reduced. (From Muravchick S. *Gerianesthesia: principles for management of the elderly patient*. St Louis (MO): Mosby 1997; with permission.)

reserve may in the elderly patient precipitate a serious decline in performance when the elderly patient is exposed to stress and increases the risk of age-related disease.

Because of decreasing functional reserve, the older patient is less able to preserve homeostasis in face of such a physical insult.¹⁵ Fortunately, this functional decline does not occur in all individuals at the same pace. A significant amount of variability occurs. This unevenness is rooted within lifestyle choices, environmental factors, genetics, and the presence of age-related disease. Older trauma victims do not cope as well as younger adults.^{16,17} After injury, elderly patients are more likely to arrive in the emergency department in hypotensive shock and to be hypothermic, 2 factors that portend worse outcomes at any age. Decreased functional reserve contributes to the higher percentage of the geriatric trauma victims that appear in the early trauma mortality statistics. It affects infection-related deaths and multiple organ dysfunction syndromes. Diminished reserve, manifested as comorbid disease states, seems to have a negative predictive value for outcome.¹⁸ However, when aggressive treatment is initiated, the outcome difference between younger and older adult decreases.¹⁹

PREINJURY MEDICAL CONDITIONS AND THERAPIES

As the risk of cardiovascular disease increases with age, so does the prevalence of β -blockade and oral anticoagulation therapies. These therapies may have direct impact on evaluation and early management of traumatically injured patients. A retrospective study across ten years at one Level I trauma center demonstrated that preinjury β -blocker therapy was associated with an odds ratio for mortality of 2.1 when head injuries were excluded. β -Blocker therapy was also positively associated with the incidence of other coexisting diseases like diabetes, hypertension, renal failure, and dyslipidemia.²⁰ Recognition of injury severity and appropriate resuscitation may be delayed if the classic symptom of tachycardia is blunted.

Head-injured patients on preinjury warfarin therapy have demonstrated significantly worse outcomes in multiple studies; the risk of mortality is more than doubled.^{21,22}

MECHANISMS OF INJURY

Elderly patients are victim to many of the same injury mechanisms as their younger counterparts (**Table 1**). Low-energy falls remain the most common mechanism for

Table 1
Leading causes of injuries for persons older than 65 years of age in the United States in 2009

Rank	Nonfatal Injuries	Fatal Injuries
1	Fall	Fall
2	Struck by/against object	Motor vehicle traffic
3	Overexertion	Suicide firearm
4	Motor vehicle occupant	Unintentional unspecified
5	Cut/penetrating injury	Unintentional suffocation

Data from Centers for Disease Control and Prevention National Center for Injury Prevention and Control. Available at: <http://www.cdc.gov/injury/wisqars/LeadingCauses.html>. Accessed August 29, 2012.

traumatic injury in the elderly.^{23,24} Various factors predispose elderly persons to falls, such as unsteady gait, orthostatic hypotension, and slow reaction time.²⁵ Falls can lead to significant injuries,²⁶ even if from standing height. It has been estimated that falls can account for more than 50% of eventual trauma-related deaths.²⁷ Minor mechanisms of injury may lead to severe injury with much greater frequency as age increases from 55 years,²⁸ and the death rate is especially high in octogenarians.²⁹

Traffic accidents involving drivers or pedestrians are also leading causes of injuries in the elderly population.²³ Underlying diseases, decreased hearing or vision, muscle weakness, and reduced reaction times³⁰ are contributing factors.³¹

Thermal injuries occur more frequently in the elderly population. This increased risk could be attributable to a reduced sense of smell, impaired hearing or vision, or reduced mobility and reaction time. These injuries are inclined to be more serious in terms of surface area and depth.³² The propensity to more severe thermal injury may be attributable to age-related alterations in skin morphology and diminished visual, olfactory, and auditory senses.

Elder mistreatment should be considered when evaluating the injured older patient. Investigations suggest that excess of 2% of elders are abused or neglected.^{33,34}

INITIAL PREHOSPITAL EVALUATION: TRIAGE AND TREATMENT

Limited physiologic reserve means that the prognosis of the elderly injured patient is much better when the patient is rapidly transported to a trauma center.^{35,36} Patient outcomes may be improved when care is provided by higher-volume trauma centers with broader experience in care of the elderly.³ One Level II trauma center demonstrated improved patient outcomes with regard to infections, respiratory failure, and overall survival by creating a designated trauma care team for the elderly.³⁷ Despite this observation, there is a continuing phenomenon of under-triaging the elderly to trauma centers. One review of a statewide trauma network showed that the elderly were significantly less likely to receive trauma team activation when compared to younger patients with similar injuries. The under-triaged elderly patients were found to have 4 times the risk of mortality and disability compared to their younger counterparts.³⁸

INJURY ASSESSMENT AND RESUSCITATION

Airway and Breathing

In conscious patients, initial airway assessment can begin by asking questions as simple as "What is your name?" A clear, appropriate response indicates that the

airway is patent and that the brain is adequately perfused. Unconscious or obtunded patients require further attention.

Laryngeal reflexes may be diminished or absent. Because of a widespread loss of all muscular and neural elements, laryngeal structures undergo a gradual deterioration in function. Older patients may also exhibit a decrease in protective airway reflexes.³⁹ Edentulous patients may suffer from oropharyngeal obstruction when maintained in a mandatory supine position. In addition, the airway can be physically obstructed as a result of direct injury, edema, or foreign bodies. There is increased concern for cervical spine injuries in elderly patients suffering from blunt mechanism of injury. Fractures of the first and second cervical vertebrae are more common in geriatric patients.⁴⁰

Pulmonary contusion is one of the most common blunt thoracic injuries. Rib fractures are expected to occur with greater frequency than in younger patients. Noninvasive ventilation via a continuous positive airway pressure mask has been described in the literature for the management of hypoxemia caused by lung contusion and for other medical conditions.²⁶ This effect may serve as a pathophysiology-directed therapy for hypoxemic patients who have blunt chest injury in whom endotracheal intubation is not required. The use of thoracic epidural analgesia has not been consistently shown to influence the rates of pulmonary complications but provides analgesia that is superior to other modes. It should be considered early in the pain management plan.

Orotracheal intubation under anesthesia with planned neuromuscular blockade and manual in-line stabilization remains the safest and most effective method for airway control in the severely injured patient.⁴¹ Confirmation of intubation should be established with capnography or capnometry, and continuous pulse oximetry should be used. Recommendations concerning the doses of medications used to facilitate intubation are shown in **Table 2**. In the older adult, the doses of many of the sedative agents used to facilitate intubation may have to be further altered. Their pharmacokinetics could be altered due to the trauma⁴² or to physiologic changes associated with aging.^{10,43,44}

To avoid hypotension in the elderly trauma patient, the doses of the etomidate,⁴⁵ barbiturates,⁴⁶ and benzodiazepines^{47,48} should be reduced. For example, an 80-year-old trauma patient needs less than half the amount of etomidate to reach the same electroencephalographic endpoint as a 22-year-old patient.⁴⁵ This reduction of blood pressure in the elderly patient is especially marked when the patient is hypovolemic. Ketamine is commonly used in the trauma scenario. In the geriatric patient, this drug has a reduced clearance and is expected to have a longer duration of action.

The opioids have an increased activity or alterations in pharmacokinetics in the elderly patient. A reduction in the dosage of morphine,⁴⁹ alfentanil, fentanyl,⁵⁰ and remifentanil⁵¹ is recommended. The only exception to this rule is meperidine, for which no changes in clearance rate or terminal elimination half-time value have been

Table 2

Dosage alteration for the anesthetic drugs used to facilitate intubation in the elderly patient

Class of Medication	Change of Dose in the Elderly Patient
Sedatives	Reduction of 50% of bolus dose
Depolarizing neuromuscular blocking agent (succinylcholine)	No reduction in bolus dose
Nondepolarizing neuromuscular blocking agents	No reduction in bolus dose
Opioids	Reduction of 50% of bolus dose

shown.⁵² However, because of its central nervous system–active toxic metabolite, normeperidine, its use is not advocated in elderly patients.

In the geriatric population, a reduction in physical activity theoretically should result in a reduction in sensitivity because of up-regulation to neuromuscular blockers. In contrast, augmented exercise increases sensitivity to neuromuscular blocking drugs receptors.⁵³ Clinical doses of neuromuscular blockers are usually unchanged.⁵⁴

Circulation

Significant reductions in coronary blood flow can occur in the absence of known coronary artery disease.⁵⁵ The aging myocardium is also less able to respond to circulating catecholamines.⁴¹ Therefore, the geriatric patient may not develop tachycardia in the presence of hypovolemia. Elderly patients may also be taking medications such as β -blockers that alter their heart rate response. Often, geriatric patients have hypertension; therefore, a normal or borderline blood pressure should be treated with a degree of suspicion.

In recent years, it has been suggested that the minimum acceptable systolic blood pressure (SBP) during initial evaluation of geriatric patients should be significantly higher than the acceptable limits for younger adults. Similarly, a lower threshold for the definition for tachycardia may be warranted. Advanced Trauma Life Support guidelines from the American College of Surgeons classify severe hemorrhage (Class 3 or 4) when the SBP is less than 90 mm Hg and heart rate is greater than 120 bpm. It has been suggested that these thresholds may not reliably indicate severe hemorrhage in the elderly. Investigators have suggested hypotension be defined as SBP less than 100 to 110 mm Hg and tachycardia greater than 90 bpm.^{56,57} Along those lines, it has also been proposed that the definition of hypotension continues to change with age, with 120 mm Hg for ages 50 to 69 years and 140 mm Hg for ages 70 years and greater as cutoff points for minimum acceptable SBPs.⁵⁸ Severely elevated SBP in younger patients without significant head injuries has been associated with worse outcomes, but this trend does not seem to hold in the geriatric population. Elevations in SBP have actually been associated with improved outcomes in the elderly.⁵⁹ Despite these findings, there is still an insufficient amount of data to warrant changes to existing evaluation criteria from specialty organizations.

Given the challenges of interpreting vital signs, it is important to measure other markers of perfusion as early as possible. Lactic acid levels and base deficit calculations are particularly useful. One center reported that 42% of its elderly trauma admissions were found to have significant alteration of lactate levels or base deficits, despite “normal” vital signs.⁵⁷ In patients who are “normotensive” by standard definitions, elevated serum lactate levels are associated with increased mortality. One investigator showed that lactate levels in excess of 4 mmol/L resulted in a mortality rate near 40% for patients older than 65 years of age. The mortality rate for younger patients with similar lactate levels was 12%. Base deficits greater than 6 were associated with similar outcomes.⁶⁰

The elderly trauma victim as compared with their younger counterpart may be less able to compensate for changing oxygen demands by increasing cardiac output. It has been proposed that oxygen carrying capacity of blood should be optimized by maintaining adequate hemoglobin levels at all times.¹⁶

Monitoring

Early invasive monitoring with hemodynamic optimization has been associated with improved survival in the geriatric trauma patient.⁵⁵ Primarily based on these findings, the Eastern Association for the Surgery of Trauma (EAST) 2001 guidelines for

resuscitation state that any severely injured geriatric patient should undergo invasive monitoring.⁶¹ This study has not been duplicated and the topic has not been reviewed in more recent guideline updates from EAST, and the extent to which routine invasive monitoring is applied in geriatric trauma patients is not known.

In recent years, there has been a renewed focus on measurement of dynamic parameters of hemodynamics including pulse pressure variation (PPV), systolic pressure variation (SPV), and stroke volume variation (SVV) as indicators of fluid responsiveness in mechanically ventilated patients. Although SVV will require additional equipment that may not be available in many hospitals, SPV and PPV can be followed via arterial blood pressure catheters alone. There are no prospective trials focused on the geriatric population in trauma. However, meta-analysis of available literature from 1998 to 2008 demonstrated that the accuracy of these variables is significantly better at determining volume responsiveness in the critically ill than central venous pressure or indices of the left ventricular end-diastolic area and global end-diastolic volume. Among the 3, PPV was shown to have the greatest accuracy.⁶²

COMMON SPECIFIC INJURIES OF THE ELDERLY

Hip Fractures

Osteoporosis and tendency to fall increase the incidence of hip fractures, which is the most common cause of traumatic injury in geriatric patients, mainly in women. Hip fracture can occur as part of a multitrauma or as an isolated injury. Hip fracture in multitrauma is associated with other bone and soft-tissue injuries, intra-abdominal and intrapelvic injuries, major blood loss, head and neck injuries, and other extremity injuries. Overall, an inability to return to a preinjury level of mobility results in precipitous functional decline, a loss of independence, quality-of-life reduction, and depression in older persons. There are data to suggest that outcome in these patients is superior when they are managed by a specialized multidisciplinary team.⁶³

The timing of hip surgery in the elderly may play a significant role in morbidity and mortality. Numerous small studies have produced mixed findings, but larger analyses have provided some clarification. This finding was demonstrated in a 2008 meta-analysis that reported both an increased 30-day and 1-year mortality (odds ratio 1.41 and 1.32, respectively) for delays greater than 48 hours.⁶⁴ Looking more closely, the investigators commented that early surgery was probably of greatest benefit to younger patients and those with a low baseline risk of mortality in one year. Patients with higher baseline risk did not necessarily receive benefit from earlier surgery. A more recent prospective cohort study of patients found that mortality was not increased by delays up to 120 hours, when adjusted for severity of comorbid disease.⁶⁵ Decisions about timing should therefore be made with regard to a patient's baseline risk and comorbidities. In patients without significant comorbid conditions, there is a definite benefit to early operation within the first 24 to 48 hours after injury. Although there is evidence that patients with uncontrolled comorbid conditions may not fare worse after up to 5 days of delay, efforts should be directed at timely optimization to allow the earliest possible surgical correction of the hip fracture.

Early ambulation and daily physical therapy after hip fracture surgery should be encouraged. Delayed ambulation after hip fracture surgery is related to the development of new-onset delirium, postoperative pneumonia, and increased length of hospital stay.⁶⁶

The best choice of anesthetic technique for hip fracture surgery has been debated for quite some time, with small studies resulting in mixed findings. The most recent, and possibly best, study was a large retrospective analysis of hip fracture patients

from 126 New York hospitals. This finding indicated that regional anesthesia was associated with a 29% reduction in mortality and 25% reduction in pulmonary complications while in hospital.⁶⁷ These data are similar to data that were published earlier in the veteran population.⁶⁸ Based on these findings, it is most likely appropriate to choose regional techniques for hip fracture surgeries when feasible. There appears to be no significant difference in postoperative cognitive functioning between the 2 techniques.⁶⁹

Head Injuries

Head injuries of any severity have a worse prognosis in the geriatric population. The risks of mortality and poor functional outcomes increase progressively with age. Researchers have also consistently found that higher severity injuries, lower Glasgow Coma Scale (GCS) scores on admission, preinjury anticoagulant therapies are all associated with poor neurologic outcomes in this population. Increased vascular vulnerability is characteristic of the aging brain. Subdural hematoma can result in changes in mental status, headache, disturbances in ambulation, or nonfocal neurologic findings.

One meta-analysis of 5600 severe head-injured patients treated in the late 1980s to 1990s demonstrated a risk of poor outcome that increased 40% to 50% per decade of life. At that time, the 6-month mortality for those aged more than 65 years was around 72%.⁷⁰ Outcomes have not necessarily improved over the years. In a more recent report from one state trauma network, geriatric patients with moderate to severe brain injury had an overall in-hospital mortality rate approaching 30%, and no patient with an admission GCS score less than 9 had good outcomes; the mortality for that subgroup was 80%.⁷¹

Even those patients with mild head injury are expected to fare worse than their younger counterparts, as mortality rates are elevated⁷² and postdischarge function is reduced. Although better outcomes were associated with increased use of specialty consults and multidisciplinary care, it has also been reported that older patients tend to receive lower-intensity care for their injuries.⁷³

Anesthetic management for the head-injured older patient follows the same general principles applied to younger patients. Etomidate hydrochloride and propofol are used to induce anesthesia before intubation; no single agent has been shown to be superior. Each decreases the systemic response to intubation, blunts intracranial pressure changes, and decreases the cerebral metabolic rate for oxygen.⁷⁴

The ability of the aging brain to autoregulate blood flow may be similar to that of younger patients under normal conditions.⁷⁵ Autoregulation is often severely impaired after head injury in any patient, but this impairment may be exaggerated in the old. It is unclear if this is related purely to age or the presence of comorbid conditions.⁷⁶ Maintenance of adequate cerebral perfusion pressure (CPP) should be a priority during anesthetic care, but there is currently no recommendation to adjust CPP based on age. Current recommendations indicate that CPP should be maintained at least 60 mm Hg. There is no evidence that artificially elevating it greater than 70 mm Hg is beneficial.⁷⁷

SUMMARY

Elderly patients represent the most rapidly expanding segment of our population, and a significant portion will rely on trauma care services during their lives. The progressive functional decline that occurs with normal aging, especially when chronic disease states are superimposed, makes these patients more physiologically fragile. They

are less capable of sustaining the stresses of traumatic injury and their injuries are often much worse than those sustained by younger patients via similar mechanisms. Geriatric patients have been shown to benefit from focused, intensive care and from multidisciplinary teams with geriatric experience. Despite these facts, the geriatric population in developed countries may be the least likely to be triaged into trauma care systems that can best deal with their injuries.

Routinely administered anesthetic medications should be adjusted appropriately for age, often being reduced by as much as half. Volume resuscitative therapies should be directed to maintain cardiac output and oxygen carrying capacity, and a lower threshold for conducting invasive cardiac monitoring is recommended. Normal-appearing vital signs may not indicate occult hypoperfusion.

Hip fractures are among the most common injuries sustained by elderly patients. There is reasonable evidence that the surgical repair of such fractures should be achieved as soon as possible to minimize risks for mortality. The use of regional anesthesia for such cases may significantly reduce morbidity and mortality. Head injuries of any severity may place geriatric patients at increased risk of mortality, but there are currently no geriatric-specific treatment recommendations that differ from usual adult guidelines.

REFERENCES

1. United States Census Bureau. Projected population of the United States, by age and sex: 2000 to 2050. Available at: <http://www.census.gov/population/www/projections/usinterimproj/natprojtab02a.pdf>. Accessed August 29, 2012.
2. Murphy SL, Xu J, Kochanek KD. Deaths: preliminary data for 2010. *Natl Vital Stat Rep* 2012;60:1–52.
3. Pandya SR, Yelon JA, Sullivan BS, et al. Geriatric motor vehicle collision survival: the role of institutional trauma volume. *J Trauma* 2011;70:1326–30.
4. Travis KW, Mihevc NT, Orkin FK, et al. Age and anesthetic practice: a regional perspective. *J Clin Anesth* 1999;11:175–86.
5. Muhlberg W, Platt D. Age-dependent changes of the kidneys: pharmacological implications. *Gerontology* 1999;45:243–53.
6. Buemi M, Nostro L, Aloisi C, et al. Kidney aging: from phenotype to genetics. *Rejuvenation Res* 2005;8:101–9.
7. Janssens JP. Aging of the respiratory system: impact on pulmonary function tests and adaptation to exertion. *Clin Chest Med* 2005;26:469–84.
8. Williams JM, Evans TC. Acute pulmonary disease in the aged. *Clin Geriatr Med* 1993;9:527–45.
9. Cardus J, Burgos F, Diaz O, et al. Increase in pulmonary ventilation-perfusion inequality with age in healthy individuals. *Am J Respir Crit Care Med* 1997;156:648–53.
10. Nickalls RW, Mapleson WW. Age-related iso-MAC charts for isoflurane, sevoflurane and desflurane in man. *Br J Anaesth* 2003;91:170–4.
11. Thompson LF. Failure to wean: exploring the influence of age-related pulmonary changes. *Crit Care Nurs Clin North Am* 1996;8:7–16.
12. Chalfin DB. Outcome assessment in elderly patients with critical illness and respiratory failure. *Clin Chest Med* 1993;14:583–9.
13. Mooradian AD. Normal age-related changes in thyroid hormone economy. *Clin Geriatr Med* 1995;11:159–69.
14. Barton RN, Horan MA, Clague JE, et al. The effect of aging on the metabolic clearance rate and distribution of cortisol in man. *Arch Gerontol Geriatr* 1999;29:95–105.

15. Frankenfield D, Cooney RN, Smith JS, et al. Age-related differences in the metabolic response to injury. *J Trauma* 2000;48:49–56.
16. Demarest GB, Osler TM, Clevenger FW. Injuries in the elderly: evaluation and initial response. *Geriatrics* 1990;45:36–8.
17. Swab C, Shapiro M, Kauder D. Geriatric trauma: patterns, care and outcomes. In: Mattox K, Feliciano D, Moore E, editors. *Trauma*. New York: McGraw-Hill; 2000. p. 1099–113.
18. Gubler KD, Davis R, Koepsell T, et al. Long-term survival of elderly trauma patients. *Arch Surg* 1997;132:1010–4.
19. DeMaria EJ, Kenney PR, Merriam MA, et al. Aggressive trauma care benefits the elderly. *J Trauma* 1987;27:1200–6.
20. Neideen T, Lam M, Brasel KJ. Preinjury beta blockers are associated with increased mortality in geriatric trauma patients. *J Trauma* 2008;65:1016–20.
21. Franko J, Kish KJ, O'Connell BG, et al. Advanced age and preinjury warfarin anticoagulation increase the risk of mortality after head trauma. *J Trauma* 2006;61:107–10.
22. Lavoie A, Ratte S, Clas D, et al. Preinjury warfarin use among elderly patients with closed head injuries in a trauma center. *J Trauma* 2004;56:802–7.
23. Pudelek B. Geriatric trauma: special needs for a special population. *AACN Clin Issues* 2002;13:61–72.
24. Wofford JL, Moran WP, Heuser MD, et al. Emergency medical transport of the elderly: a population-based study. *Am J Emerg Med* 1995;13:297–300.
25. McMahon DJ, Shapiro MB, Kauder DR. The injured elderly in the trauma intensive care unit. *Surg Clin North Am* 2000;80:1005–19.
26. Hurst JM, DeHaven CB, Branson RD. Use of CPAP mask as the sole mode of ventilatory support in trauma patients with mild to moderate respiratory insufficiency. *J Trauma* 1985;25:1065–8.
27. Mosenthal AC, Livingston DH, Elcavage J, et al. Epidemiology and strategies for prevention. *J Trauma* 1995;38:753–6.
28. Velmahos GC, Jindal A, Chan LS. “Insignificant” mechanism of injury: not to be taken lightly. *J Am Coll Surg* 2001;192:147–52.
29. Lambert DA, Sattin RW. Death from falls, 1978–1984. *MMWR CDC Surveill Summ* 1998;37:S21–6.
30. Ruhle R, Wolff H. Psychological aspects of traffic fitness of aging car drivers. *Z Gesamte Hyg* 1990;3:346–50 [in German].
31. Sjogren H, Eriksson A, Ostrom M. Role of disease in initiating the crashes of fatally injured drivers. *Accid Anal Prev* 1996;28:307–14.
32. Linn BS. Age differences in the severity and outcome of burns. *J Am Geriatr Soc* 1980;28:118–23.
33. Kennedy RD. Elder abuse and neglect: the experience, knowledge, and attitudes of primary care physicians. *Fam Med* 2005;37:481–5.
34. Elder abuse and neglect: council on scientific affairs. *JAMA* 1987;257:966–71.
35. Finelli FC, Jonsson J, Champion HR, et al. A case control study for major trauma in geriatric patients. *J Trauma* 1989;29:541–8.
36. Phillips S, Rond PC, Kelly SM, et al. The failure of triage criteria to identify geriatric patients with trauma: results from the Florida Trauma Triage Study. *J Trauma* 1996;40:278–83.
37. Mangram AJ, Mitchell CD, Shifflette MD, et al. Geriatric trauma service: a one-year experience. *J Trauma* 2011;72:119–22.
38. Lehmann R, Beekley A, Casey L, et al. The impact of advanced age on trauma triage decisions and outcomes: a statewide analysis. *Am J Surg* 2009;197:571–4.

39. Pontoppidan H, Beecher HK. Progressive loss of protective reflexes in the airway with the advance of age. *JAMA* 1960;174:2209–13.
40. Lomoschitz FM, Blackmore CC, Mirza SK, et al. Cervical spine injuries in patients 65 years old and older: epidemiologic analysis regarding the effects of age and injury mechanism on distribution, type, and stability of injuries. *Am J Roentgenol* 2002;178:573–7.
41. Adnet F, Lapostolle F, Ricard-Hibon A, et al. Intubating trauma patients before reaching hospital—revisited. *Crit Care* 2001;5:290–1.
42. Berkenstadt H, Mayan H, Segal E, et al. The pharmacokinetics of morphine and lidocaine in nine severe trauma patients. *J Clin Anesth* 1999;11:630–4.
43. Eilers H, Niemann C. Clinically important drug interactions with intravenous anaesthetics in older patients. *Drugs Aging* 2003;20:969–80.
44. Vuyk J. Pharmacodynamics in the elderly. *Best Pract Res Clin Anaesthesiol* 2003;17:207–18.
45. Arden JR, Holley FO, Stanski DR. Increased sensitivity to etomidate in the elderly: initial distribution versus altered brain response. *Anesthesiology* 1986;65:9–27.
46. Homer TD, Stanski DR. The effect of increasing age on thiopental disposition and anesthetic requirement. *Anesthesiology* 1985;62:714–24.
47. Reves JG, Fragen RJ, Vinik HR, et al. Midazolam: pharmacology and uses. *Anesthesiology* 1985;62:310–24.
48. Smith MR, Bell GD, Quine MA, et al. Small bolus injections of intravenous midazolam for upper gastrointestinal endoscopy: a study of 788 consecutive cases. *Br J Clin Pharmacol* 1993;36:573–8.
49. Kaiko RF, Wallenstein SL, Rogers AG, et al. Narcotics in the elderly. *Med Clin North Am* 1982;66:1079–89.
50. Shafer SL. The pharmacology of anesthetic drugs in the elderly patients. *Anesthesiol Clin North America* 2000;18:1–29.
51. Minto CF, Schnider TW, Egan TD, et al. Influence of age and gender on the pharmacokinetics and pharmacodynamics of remifentanyl: I. Model development. *Anesthesiology* 1997;86:10–23.
52. Herman RJ, McAllister CB, Branch RA, et al. Effects of age on meperidine disposition. *Clin Pharmacol Ther* 1985;37:19–24.
53. Martyn JA, White DA, Gronert GA, et al. Up and down regulation of skeletal muscle acetylcholine receptors. *Anesthesiology* 1992;76:822–43.
54. Rupp SM, Castagnoli KP, Fisher DM, et al. Pancuronium and vecuronium pharmacokinetics and pharmacodynamics in younger and elderly adults. *Anesthesiology* 1987;67:45–9.
55. Scalea TM, Simon HM, Duncan AO, et al. Geriatric blunt multiple trauma: improved survival with early invasive monitoring. *J Trauma* 1990;30:129–34.
56. Heffernan DS, Thakkar RK, Monaghan SF, et al. Normal presenting vital signs are unreliable in geriatric blunt trauma victims. *J Trauma* 2010;69:813–20.
57. Martin J, Alkhoury F, O'Connor J, et al. 'Normal' vital signs belie occult hypoperfusion in geriatric trauma patients. *Am Surg* 2010;76:65–9.
58. Edwards M, Ley E, Mirocha J, et al. Defining hypotension in moderate to severely injured trauma patients: raising the bar for the elderly. *Am Surg* 2010;76:1035–8.
59. Ley EJ, Singer MB, Gangi A, et al. Elevated systolic blood pressure after trauma: tolerated in the elderly. *J Surg Res* 2012;177(2):326–9.
60. Callaway DW, Shapiro NI, Donnino MW, et al. Serum lactate and base deficit as predictors of mortality in normotensive elderly blunt trauma patients. *J Trauma* 2009;66:1040–4.

61. Jacobs DG, Plaisier BR, Barie PS, et al. Practice management guidelines for geriatric trauma: the EAST practice management guidelines work group. *J Trauma* 2003;54:391–416.
62. Marik PE, Cavallazzi R, Vasu T, et al. Dynamic changes in arterial waveform derived variables and fluid responsiveness in mechanically ventilated patients: a systematic review of the literature. *Crit Care Med* 2009;37:2642–7.
63. Khasraghi FA, Christmas C, Lee EJ, et al. Effectiveness of a multidisciplinary team approach to hip fracture management. *J Surg Orthop Adv* 2005;14:27–31.
64. Shiga T, Wajima Z, Ohe Y. Is operative delay associated with increased mortality of hip fracture patients? Systematic review, meta-analysis, and meta-regression. *Can J Anaesth* 2008;55:146–54.
65. Vidan MT, Sanchez E, Gracia Y, et al. Causes and effects of surgical delay in patients with hip fracture: a cohort study. *Ann Intern Med* 2011;155:226–33.
66. Kamel HK, Iqbal MA, Mogallapu R, et al. Time to ambulation after hip fracture surgery: relation to hospitalization outcomes. *J Gerontol A Biol Sci Med Sci* 2003;58:1042–5.
67. Neuman MD, Silber JH, Elkassabany NM, et al. Comparative effectiveness of regional versus general anesthesia for hip fracture surgery in adults. *Anesthesiology* 2012;117:72–92.
68. Radcliff TA, Henderson WG, Stoner TJ, et al. Patient risk factors, operative care, and outcomes among older community-dwelling male veterans with hip fracture. *J Bone Joint Surg Am* 2008;90:34–42.
69. Berggren D, Gustafson Y, Eriksson B, et al. Postoperative confusion after anesthesia in elderly patients with femoral neck fractures. *Anesth Analg* 1987;66:497–504.
70. Hukkelhoven CW, Steyerberg EW, Rampen AJ, et al. Patient age and outcome following severe traumatic brain injury: an analysis of 5600 patients. *J Neurosurg* 2003;99:666–73.
71. Utomo WK, Gabbe BJ, Simpson PM, et al. Predictors of in-hospital mortality and 6-month functional outcomes in older adults after moderate to severe traumatic brain injury. *Injury* 2011;40:973–7.
72. Susman M, DiRusso SM, Sullivan T, et al. Traumatic brain injury in the elderly: increased mortality and worse functional outcome at discharge despite lower injury severity. *J Trauma* 2002;53:219–23.
73. Thompson HJ, Rivara FP, Jurkovich GJ, et al. Evaluation of the effect of intensity of care on mortality after traumatic brain injury. *Crit Care Med* 2008;36:282–90.
74. Unni VK, Johnston RA, Young HS, et al. Prevention of intracranial hypertension during laryngoscopy and endotracheal intubation. *Br J Anaesth* 1984;56:1219–23.
75. Yam AT, Lang EW, Lagopoulos J, et al. Cerebral autoregulation and ageing. *J Clin Neurosci* 2005;12:643–6.
76. Thompson HJ, McCormick WC, Kagan SH. Traumatic brain injury in older adults: epidemiology, outcomes, and future implications. *J Am Geriatr Soc* 2006;54:1590–5.
77. Bratton SL, Chestnut RM, Ghajar J, et al. Guidelines for the management of severe traumatic brain injury. IX. Cerebral perfusion thresholds. *J Neurotrauma* 2007;24:S59–64.