# **Trauma in Elderly**



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## 13.1 Introduction

The average life length has increased considerably throughout the world; over the next 20 years, the population over 65 years of age is projected to double insides, reaching 82.3 million by 2020 [1]. This implies an increase in healthcare costs for the treatment of this kind of patient. In fact, this age group represents 40% of hospitalized adults (in the US population) [2].

In western countries, old people keep driving cars as long as possible. As a consequence, 17% of all traffic fatalities involved older people, who are the 9% of all people injured in traffic crashes during the years [3]. However, physiological changes and decline make the body's compensatory mechanisms less effective following a trauma. Still, the higher comorbidity rates (66%) make complications more frequent during hospitalization. Blunt trauma is the principal kind of trauma (80%) in elderly patients (EPs). Falls are 70% of blunt trauma, caused by physiological change with age and comorbidities affecting the muscle-skeletal system and the brain causing tremor, rigidity, and dementia. The rest of the trauma causes are penetrating trauma and suicide attempts. Moreover, domestic abuses and assaults represent an underrated cause of trauma with an estimate 25.886 elderly people treated in emergency departments yearly in the US for lacerations, contusions, or fractures. By now there are no specific guidelines addressing the management and treatment of the trauma in geriatric patients. EPs even if seriously injured are underestimated in 25% of cases. This bias results in significant delay in treatment. In 43% of the EPs at the time of access to the emergency department had experienced a cardiogenic shock not associated with alteration of the hemodynamic state at the time of admission; consequently, they are erroneously considered hemodynamically stable.

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Fifty-four percent of these cases result in death [4]. Mortality among those patients may be substantially reduced by employing "age over 70", as an activation code for the trauma team [5]. There is a strong relationship between age, Injury Severity Score (ISS), and mortality, so a clear trauma classification is essential for the management and treatment of the EPs at presentation. The invasive procedures are widely accepted in ISS > 18, whereas in ISS < 18 the indication needs to be continuously evaluated according to risks and benefits balance [6]. A recent study showed that the integration of new standard operative procedures (such as early whole-body CT scan, damage control surgery, and the use of goal-directed coagulation management) was associated with a lower mortality rate in severely injured geriatric trauma patients [7]. This same benefit was not demonstrated for a moderately injured patient (ISS 9–15). It has been evaluated that a poor pre-injury functional status is a strong predictor of undesirable outcome than the admitting diagnosis [8]. Respiratory complications are most frequent among all and increase the risk of in-hospital mortality [9]. The most important predictor of long-term mortality is age. Quickly accessible factors independently associated with mortality, besides age, are coagulopathy, acidosis, and a lower level of consciousness. These parameters can be defined as follows [10]:

- Coagulopathy: International Normalized Ratio—INR  $\geq$  1.4;
- Acidosis: arterial base excess—ABE  $\leq -6$ ;
- Level of consciousness: Glasgow Coma Scale—GCS score  $\leq 8$ .

## 13.2 The Physiology of Aging

The body response to trauma may be related to the physiological change occurring with age (Table 13.1). In cases of trauma with bleeding, the first body reaction is the release of catecholamine hormones to raise blood pressure and provide more blood to muscle and brain. In EPs, the cardiovascular system responds to this stimulation with a consistent delay [11]. Furthermore, the heart becomes stiffer as fat and fibrous tissue replace cardiac muscle cells. This leads to a decreased responsiveness to endogenous stimulation and a limited capability to increase cardiac output. Also, medications such as beta-blockers or diuretic medications may mitigate the hormones' effect and exacerbate a forced status of hypotension and hypoperfusion. EPs try to compensate by increasing systemic vascular resistance which results in an unreliable normal pressure. It will be necessarily a continuous parameters revaluation in EPs during the very first admission period; for example, Heffernan et al. found that mortality increased in EPs with a systolic blood pressure <110 mmHg and >130 beats/min, respectively whereas the same results were obtained when the blood pressure was <95 mmHg and >90 beats/min, respectively in youngers [12].

There are substantial differences between the physiology of adult patients' population and EPs. Blood vessels may be extremely narrow and stiff; the cardiac reserve

Organ system	Changes	Considerations and management
Head and Brain	<ul> <li>Cortical atrophy</li> <li>Cerebrovascular vessel plaque</li> <li>Cognitive decline</li> <li>Cerebellar functions decreased → worse balance</li> </ul>	<ul> <li>SDH are four times more common than in younger patients and may manifest later</li> <li>EDH are rare</li> <li>Combination of polypharmacy and injury may lead to delirium</li> <li>High incidence of ICH in anticoagulated patients (even for minor head trauma)</li> </ul>
Cardiovascular	<ul> <li>Loss of connective tissue elasticity         → stiff veins and arteries</li> <li>Death of cardiac muscle cell and         replacement by fibrous tissue →         stiff heart</li> <li>Decreased responsiveness to         stimulation (catecholamines)</li> </ul>	<ul> <li>Medications such as beta-blockers or diuretics → forced status of hypotension and hypoperfusion</li> <li>Unreliable vital signs → SBP &lt; 100 mmHg; HR &gt; 130 b/min</li> <li>Limited physiologic reserves and rapid deterioration</li> <li>Blood loss is not well tolerated, transfuse early</li> </ul>
Pulmonary	<ul> <li>Increased chest wall stiffness, decreased pulmonary compliance</li> <li>Reduced respiratory rate, impaired gas exchange (reduced PO<sub>2</sub>)</li> <li>Increased residual volume (air trapping)</li> </ul>	<ul> <li>Higher risk of aspiration, atelectasis, pneumonia, and pulmonary embolism</li> <li>Early intubation recommended</li> <li>Do not underestimate severe trauma in EPs with "normal" respiratory function → frequent rapid decompensation</li> </ul>
Renal	<ul> <li>Less effective filtration system</li> <li>Loss of renal mass (glomerular tissue replaced by fibrous tissue)</li> </ul>	<ul> <li>Decreased tolerance to hypotension and nephrotoxic drugs</li> <li>Higher risk of acute kidney injury</li> </ul>
Bones	<ul> <li>Osteoporosis and arthritis → decreased bone density</li> </ul>	• Higher risk of more severe and multiple fractures
General	<ul> <li>Decrease immune system functions</li> <li>Malnutrition</li> <li>Loss of adequate thermoregulation</li> </ul>	<ul><li>Decreased wound healing</li><li>Higher risk of hypothermia</li></ul>

Table 13.1 Physiological aging and management strategies

SDH subdural hematoma, EDH epidural hematoma, ICH intracranial hemorrhage, SBP systolic blood pressure, HR heart rate, EPs elderly patients

is limited; the respiratory rate and pulmonary compliance is decreased. Furthermore, lung tissue becomes fibrotic and this worsens pulmonary compliance. Hence, early intubation is recommended in patients with borderline respiratory function [13].

The kidneys filtration system is less functional, for those reasons, even temporary hypotension and hypoperfusion or nephrotoxic drugs represent a trigger for an acute kidney injury [14].

Osteoporosis and arthritis decrease bone density and make EPs susceptible to fracture, even in low-energy trauma [15]. Lastly, the loss of adequate thermoregulation may drive to hypothermia after a mild or moderate trauma, resulting in a coagulopathy worsening, and consequent raising of mortality risk [16].

#### 13.3 Neurotrauma

The most common mechanism for traumatic brain injury (TBI) is the fall. To prevent such injuries several studies listed a number of strategies to decrease the probability of these traumatic events happening, like the careful choice of the more appropriate antihypertensive (thiazide-type diuretic therapy reduces hip and pelvic fracture risk compared with other antihypertensive medications), and the promotion of physical exercise to strengthen their musculoskeletal systems [17, 18]. In the general population TBI physiology is divided into two phases: the first phase consists of cellular death that leads to a neurological impairment and the second phase results in microenvironmental changes associated with inflammation and the consequent edema. In EPs, physiological age-related changes exacerbate the phases of TBI. Additionally, TBI conduces to further cellular loss and may boost the progression of preexisting diseases [19]. In Eps, the maintaining of an appropriate blood pressure and, consequently, a correct cerebral oxygenation is crucial, given the high rate of EPs with hypertension [20]. In EPs, the heart ejection should be precisely calculated to determine the fluid management. Currently, the mainstays in TBI treatment are as follows: (1) balance hypoxia and hypotension; (2) carefully avoid hypoperfusion from hyperventilation; (3) administer anticonvulsants over the first 7 days after the trauma; and (4) do not administer steroids [21]. For the typical physiological change in EPs (reduced brain volume; enlarged dead space between the brain and the head bone), a brain swelling with a consequent increased intracranial pressure needing surgical intervention is rare. Despite mannitol may worse cerebral edema, it remains the most efficient drug in the control of increased intracranial pressure in these patients. More recently, several studies have shown that statins reduce the risk of both in-hospital mortality and 12-month adverse outcome [22, 23]. From the surgical perspective, guidelines currently recommend evacuating an acute subdural hematoma when it is >10 mm and/or midline shift is >5 mm on CT scan, regardless of the patient's Glasgow Coma Scale (GCS) [21]. While this recommendation is well established for young patients, it is vague and controversial regarding EPs. Though, EPs who underwent craniotomy for hematoma evacuation showed acceptable outcomes (in-hospital mortality 5-16%) and a similar ability to return to the baseline when compared to younger counterparts. The difference is that EPs may result in a longer hospitalization and rehabilitation. Historically, most subdural hematoma cases were treated conservatively and resulted in a chronic event. Patients have occasionally reported headache and/or minor mental changes; less than 10% have reported substantial neurological symptoms [20]. As shown in some recent studies chronic subdural hematoma (CSDH) may lead to complications in the long-term. In the intermediate long-term CSDH in EPs may lead to a mortality rate of 26.3% and 32%, respectively at 6 and 12 months [24]; hip fractures have similar intermediate long-term results [25]. It might be postulated that CSDH may exacerbate preexisting comorbidities and affecting the brain functions may result in a mortality rate increase [25]. Further studies looking at specific TBI cohorts of EPs indicated "male gender" as a predictive factor for a worse outcome. In women better outcomes are likely due to estrogen and progesterone release, which act in the reparation process [26]. Another parameter that deeply affects the TBI outcome is the anticoagulation therapy commonly used in EPS. Even a ground-level fall in anticoagulated geriatric patients is a mechanism of injuries significantly associated with intracranial hemorrhages (ICH) [27]. Anticoagulants also make treatment much more demanding and challenging [28]. Howard et al. found a relationship between an increased risk of mortality in EPs and those on warfarin who fall [29]. However, not all the author agrees with this assertion. Indeed, over the last decade, new oral anticoagulants (NOACs) are available worldwide. NOACs are as efficient as warfarin but relatively safer, even though there is a significant risk of delayed ICH for elderly patients on NOACs, so repeated evaluation is recommended [27]. The action mechanism consists in the direct inhibition of the coagulation cascade. NOACs have a shorter half-life compared to warfarin (8–16 h vs. 1 week, respectively) and are, thus, easier to manage. The major limitation of NOACs is the lack of a specific antidote [30]. The RE-LY trial documented that low doses of NOACs decrease the risk of intracranial hemorrhage as compared with warfarin; conversely, high doses result in a similar risk [31].

#### 13.4 Rib Fracture

Rib fractures after chest wall trauma are a common injury, especially in EPs. The age-related bone loss and osteoporosis make the bone easily fractured even with minor trauma. Furthermore, aging processes such as impaired mucociliary clearance of bacteria, reduced cough effectiveness, and reduction in lung and chest wall compliance make the respiratory performance globally reduced [32]. As a consequence, relatively simple rib fractures can lead to pneumonia and other pulmonary complications as respiratory failure. Rib fractures in patients older than 65 years are associated with an average mortality risk of 20% and a pneumonia acquisition risk of 19% [33]. They also carry a 10% increase in mortality risk for each additional rib [33]. Treatment options consist of oxygen support, pulmonary toilette, and analgesia. Pain control is critical in multiple rib fractures. In EPs is suggested to avoid polypharmacy or narcotic administration, epidural anesthesia and early intubation are strongly recommended. A decreased risk of delirium using regional analgesia in EPs with multiple rib fractures has been demonstrated [34]. Some authors have also suggested to consider rib plating as a valid treatment option, even in EPs, due to an improvement in outcome measures such as survival and quality of recovery [32, 35].

### 13.5 Pelvic Fracture

The pelvis is the strongest bone unit in the body and its fracture is secondary to high-energy injury (13–18%). However, comorbidities (osteoporosis, arthritis, and osteopenia), preexisting conditions (previous surgery, metastases), and age may weaken the bone pattern and make it prone to fracture even in cases of low-energy trauma [36]. In EPs, the most common cause of pelvic fracture is low-energy trauma (usually falls from standing) [37]. Therefore, prevention of falls is fundamental to decrease the risk factors. Pelvic fractures are more prevalent among males in

younger patients and among females in EPs. The difference in prevalence between sexes is presumably attributable to the hormonal changes in females (estrogen and progesterone decline) which unleash subsequent osteoporosis [38]. In 64% of pelvis fractures, preexisting osteoporosis is present and this rises to 94% in <60-vear-old patients [39]. Mortality in EPs (7.6%) is mostly related to hemorrhage; this is four times higher than in younger patients, where morbidity is due to incomplete healing and/or nerve damage [40]. Pelvic fracture management is standardized. According to ATLS, the first assessment is based on clinical findings and pain sites. Physical examination is essential to determine the anatomical site entailed in the trauma, which requires further investigations. Bimanual compression of the iliac wings can rule out either vertical or rotational instability [41]. Pelvic X-ray represents the first radiological investigation. Inlet, outlet, or judet views may offer additional data. CT scan may be useful in suspected active bleeding in order to define the site of a subsequent angiographic embolization if necessary [42]. When underlying osteoporosis is suspected, an extensive investigation, including levels of calcium, thyroid function, and sexual hormones in the serum, should be carried out [43]. As previously mentioned, management is primarily based on clinical presentation. In EPs comorbidities should be considered, especially for potential medication interactions. It is likely that home therapy might interact with drugs administered over the course of hospitalization, causing confusion and obnubilation. Is necessaries correct those therapies in order to avoid any aggravation of the EPs. A prompt correction of coagulopathy in patients on anticoagulants is essential, while desmopressin may help to treat patients with chronic kidney disease [44]. The clinical examination and the radiological investigations looking at potential bleeding define the diagnostic phase. Even if the patient is hemodynamically stable, there could be an identified bleeding, so a conservative treatment (bed rest, minimally invasive interventions, and pain control) is preferred. If there is a bleeding, can happen that the sites of hemorrhage are multiple. Besides, in one-third of pelvic fractures, sources of bleeding are outside of the pelvic borders [45] and that makes their identification more difficult. If the patient is hemodynamically stable and there is a small amount of bleeding, it can be controlled by putting in place a pelvic binder which stabilizes the fracture. Although arterial bleeding is unusual, its presence should be investigated with radiological investigations when suspected. Such bleeding is unlikely to be controlled with immobilization, and more invasive treatment (i.e., angioembolization and/or surgery) is required [46]. To prevent bone resorption and to decrease the risk of pulmonary infection and vascular or gastrointestinal complications a mobilization as soon as possible is recommended. The average hospital stays for EPs who sustain a pelvic fracture is around 21 days. A long rehabilitation is then necessary, but rarely there is a complete reestablishing for EPs [47].

#### 13.6 Penetrating Trauma

While blunt trauma discussed so far (falls, motor vehicle injuries, or pedestrian collision) is the most common and with outcomes favorable [48], the same cannot be said for penetrating trauma, which is relatively rare but with an extremely poor prognosis. This poor prognosis is due to the scarce physiological reserve in association with several comorbidities, as such preexisting cardiovascular disease, which requires the administration of anticoagulants. Penetrating trauma is most commonly due to a suicide attempt. Social context (urban setting) and comorbidities (depression and chronic pain) may deeply affect EP quality of life. Self-inflicted injury rates increase with aging (46.2% between 65 and 74 years of age and 51.5% over 75 years of age). This type of injury represents a clear public issue [49]. In 80% of suicide attempts, firearms are predominantly employed, and the head is the site most commonly involved (54.2%), with an extremely high mortality (over 90%). Further anatomical sites of penetrating trauma are the thorax (13.5%) and abdomen (8.2%). In assaulted patients, the thorax and abdomen are the most commonly involved area (43%), followed by the extremities (16.9%). Higher mortality is recorded in suicide attempts (60%) as compared to assault-related patients (25%) or unintentional penetrating injuries (9%) [49]. Given the complex history and home therapy often present in EPs, these patients should be closely assessed and monitored. As mentioned above, it has been demonstrated that the classic hemodynamic criteria (systolic BP <90 mmHg or heart rate >120/min), which are typically applied in trauma team activation, are inadequate in EPs [12]. As soon as the patient is admitted, it is crucial to gather the detailed clinical history and the home therapy must be scrupulously evaluated. The anticoagulant therapy is frequent in EPs, so the anticoagulants need to be promptly interrupted and the coagulopathy corrected if the ISS is high [50]. Multiple blood tests may help in assessing EPs whose vital signs are modified by anticoagulants and beta-blockers. In these patients, vital signs "wrongly" considered stable may conceal potentially life-threatening bleeding. ATLS guidelines should be adopted. Radiological investigations need to be completed while monitoring vital signs continuously, and angioembolization or aggressive interventions should be considered in the first phase of the evaluation [51]. Fluid administration needs to be managed carefully because the physiological response between hypovolemia and edema in EPs is extremely thin. However, a regulated hydration and bicarbonate administration reduce nephropathy. In trauma patients, the thermoregulatory response may be dysregulated causing hypothermia, acidosis, and coagulopathy. Therefore, hypothermia prevention through the administration of warm fluids represents a mainstay in the management of trauma [52]. Surgical management is related to the injured area and whether vital signs are stable. If vital signs are stable, further investigations (such as CT scan, endoscopy, and bronchoscopy) should be considered and a conservative treatment evaluated. Patients with penetrating trauma to the neck need an emergency operation if the vascular or aerodigestive system is involved and vital signs are unstable. Historically, less than 15-20% of penetrating neck injuries need surgery [53, 54]. In cases of chest trauma with hemodynamic instability, an emergency thoracotomy or median sternotomy, depending on the site involved, is required. Most cases of penetrating trauma to the chest (nearly 80%) may be treated with a chest drain [55]. Penetrating trauma to the abdomen which results in peritonitis or in hemodynamic instability requires an emergency laparotomy [56]. An emergency laparoscopy should be taken into consideration in left thoracoabdominal trauma and penetrating trauma to the lower part of the left thorax, in order to rule out lacerations to the diaphragm [51] (Fig. 13.1).

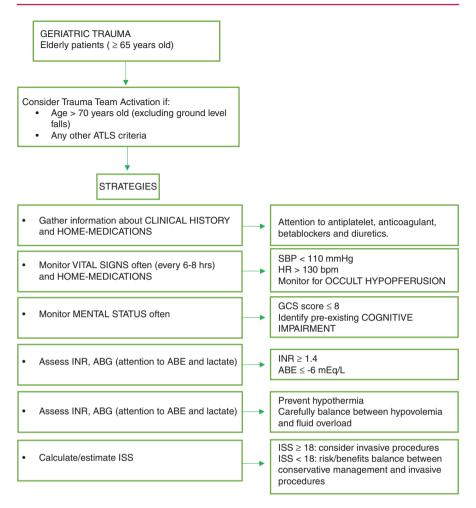


Fig. 13.1 Management strategies in elderly trauma patients

# References

- Projections of Future Growth of the Older Population. 2016. http://www.aoa.gov/aoaroot/ aging\_statistics/future\_growth/future\_growth.aspx#age. Accessed 24 Feb 2017.
- Wier LM, Levit K, Stranges E, et al. HCUP facts and figures: statistics on hospital-based care in the United States, 2012. Rockville: Agency for Healthcare Research and Quality; 2014. http://www.hcup-us.ahrq.gov/reports.jsp.
- 3. NHTSA Traffic Safety Facts. Older population. 2014
- 4. 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.
- Demetriades D, Sava J, Alo K, Newton E, Velmahos GC, Murray JA, et al. Old age as a criterion for trauma team activation. J Trauma. 2001;51(4):754–6; discussion 6–7.
- 6. Tornetta P, Mostafavi H, Riina J, Turen C, Reimer B, Levine R, et al. Morbidity and mortality in elderly trauma patients. J Trauma. 1999;46(4):702–6.

- Peterer L, Ossendorf C, Jensen KO, Osterhoff G, Mica L, Seifert B, et al. Implementation of new standard operating procedures for geriatric trauma patients with multiple injuries: a single level I trauma centre study. BMC Geriatr. 2019;19(1):359.
- Maxwell CA, Mion LC, Mukherjee K, Dietrich MS, Minnick A, May A, et al. Preinjury physical frailty and cognitive impairment among geriatric trauma patients determine postinjury functional recovery and survival. J Trauma Acute Care Surg. 2016;80(2):195–203.
- 9. Labib N, Nouh T, Winocour S, Deckelbaum D, Banici L, Fata P, et al. Severely injured geriatric population: morbidity, mortality, and risk factors. J Trauma. 2011;71(6):1908–14.
- Pape HC, Lefering R, Butcher N, Peitzman A, Leenen L, Marzi I, et al. The definition of polytrauma revisited: an international consensus process and proposal of the new 'Berlin definition'. J Trauma Acute Care Surg. 2014;77(5):780–6.
- Haddad F, Hunt SA, Rosenthal DN, Murphy DJ. Right ventricular function in cardiovascular disease, part I: anatomy, physiology, aging, and functional assessment of the right ventricle. Circulation. 2008;117(11):1436–48.
- Heffernan DS, Thakkar RK, Monaghan SF, Ravindran R, Adams CA Jr, Kozloff MS, et al. Normal presenting vital signs are unreliable in geriatric blunt trauma victims. J Trauma. 2010;69(4):813–20.
- Bonomo L, Larici AR, Maggi F, Schiavon F, Berletti R. Aging and the respiratory system. Radiol Clin N Am. 2008;46(4):685–702, v–vi. E.
- Candela-Toha AM, Recio-Vazquez M, Delgado-Montero A, del Rey JM, Muriel A, Liano F, et al. The calculation of baseline serum creatinine overestimates the diagnosis of acute kidney injury in patients undergoing cardiac surgery. Nefrologia. 2012;32(1):53–8.
- Lach HW, Lorenz RA, L'Ecuyer KM. Aging muscles and joints: mobilization. Crit Care Nurs Clin North Am. 2014;26(1):105–13.
- Vaughan MS, Vaughan RW, Cork RC. Postoperative hypothermia in adults: relationship of age, anesthesia, and shivering to rewarming. Anesth Analg. 1981;60(10):746–51.
- Kendrick D, Kumar A, Carpenter H, Zijlstra GA, Skelton DA, Cook JR, et al. Exercise for reducing fear of falling in older people living in the community. Cochrane Database Syst Rev. 2014;28(11):Cd009848.
- Puttnam R, Davis BR, Pressel SL, Whelton PK, Cushman WC, Louis GT, et al. Association of 3 different antihypertensive medications with hip and pelvic fracture risk in older adults: secondary analysis of a randomized clinical trial. JAMA Intern Med. 2017;177(1):67–76.
- 19. Powers R. Neurobiology of aging. In: Cummings J, Coffey CE, editors. Textbook of geriatric neuropsychiatry. Washington: American Psychiatric Press; 2000. p. 33–79.
- 20. McIntyre A, Mehta S, Aubut J, Dijkers M, Teasell RW. Mortality among older adults after a traumatic brain injury: a meta-analysis. Brain Inj. 2013;27(1):31–40.
- Bratton SL, Chestnut RM, Ghajar J, McConnell Hammond FF, Harris OA, Hartl R, et al. Guidelines for the management of severe traumatic brain injury. I. Blood pressure and oxygenation. J Neurotrauma. 2007;24(Suppl 1):S7–13.
- Schneider EB, Efron DT, MacKenzie EJ, Rivara FP, Nathens AB, Jurkovich GJ. Premorbid statin use is associated with improved survival and functional outcomes in older head-injured individuals. J Trauma. 2011;71(4):815–9.
- 23. Wible EF, Laskowitz DT. Statins in traumatic brain injury. Neurotherapeutics. 2010;7(1):62–73.
- 24. Miranda LB, Braxton E, Hobbs J, Quigley MR. Chronic subdural hematoma in the elderly: not a benign disease. J Neurosurg. 2011;114(1):72–6.
- 25. Shoda N, Yasunaga H, Horiguchi H, Matsuda S, Ohe K, Kadono Y, et al. Risk factors affecting inhospital mortality after hip fracture: retrospective analysis using the Japanese diagnostic procedure combination database. BMJ Open. 2012;2(3):e000416.
- Gibson CL, Gray LJ, Bath PM, Murphy SP. Progesterone for the treatment of experimental brain injury; a systematic review. Brain. 2008;131(Pt 2):318–28.
- Cocca AT, Privette A, Leon SM, Crookes BA, Hall G, Lena J, Eriksson EA. Delayed intracranial hemorrhage in anticoagulated geriatric patients after ground level falls. J Emerg Med. 2019;57(6):812–6.

- De Bonis P, Trevisi G, de Waure C, Sferrazza A, Volpe M, Pompucci A, et al. Antiplatelet/anticoagulant agents and chronic subdural hematoma in the elderly. PLoS One. 2013;8(7):e68732.
- Howard JL, Cipolle MD, Horvat SA, Sabella VM, Reed JF, Fulda G, et al. Preinjury warfarin worsens outcome in elderly patients who fall from standing. J Trauma. 2009;66(6):1518–22; discussion 23–4.
- Kundu A, Sardar P, Chatterjee S, Aronow WS, Owan T, Ryan JJ. Minimizing the risk of bleeding with NOACs in the elderly. Drugs Aging. 2016;33(7):491–500.
- 31. Eikelboom JW, Wallentin L, Connolly SJ, Ezekowitz M, Healey JS, Oldgren J, et al. Risk of bleeding with 2 doses of dabigatran compared with warfarin in older and younger patients with atrial fibrillation: an analysis of the randomized evaluation of long-term anticoagulant therapy (RE-LY) trial. Circulation. 2011;123(21):2363–72.
- 32. Christie DB 3rd, Nowack T, Drahos A, Ashley DW, et al. Geriatric chest wall injury: is it time for a new sense of urgency? J Thorac Dis. 2019;11(Suppl 8):S1029–33.
- 33. Bulger EM, Arneson MA, Mock CN, et al. Rib fractures in the elderly. J Trauma. 2000;48:1040-6.
- 34. O'Connell KM, Quistberg DA, Tessler R, Robinson BRH, Cuschieri J, Maier RV, et al. Decreased risk of delirium with use of regional analgesia in geriatric trauma patients with multiple rib fractures. Ann Surg. 2018;268(3):534–40.
- 35. Fitzgerald MT, Ashley DW, Abukhdeir H, Christie DB 3rd. Rib fracture fixation in the 65 years and older population: A paradigm shift in management strategy at a Level I trauma center. J Trauma Acute Care Surg. 2017;82(3):524–7.
- 36. Lorich D, Gardener M, Helfet D. Trauma to the pelvis and extremities. In: Norton J, Barie P, Bollinger R, Chang A, Lowry S, Mulvihill S, et al., editors. Surgery basic science and clinical evidence. New York: Springer; 2008. p. 505–20.
- Stahel PF, Hammerberg EM. History of pelvic fracture management: a review. World J Emerg Surg. 2016;11:18.
- Morris RO, Sonibare A, Green DJ, Masud T. Closed pelvic fractures: characteristics and outcomes in older patients admitted to medical and geriatric wards. Postgrad Med J. 2000;76(900):646–50.
- Krappinger D, Kammerlander C, Hak DJ, Blauth M. Low-energy osteoporotic pelvic fractures. Arch Orthop Trauma Surg. 2010;130(9):1167–75.
- Balogh Z, King KL, Mackay P, McDougall D, Mackenzie S, Evans JA, et al. The epidemiology of pelvic ring fractures: a population-based study. J Trauma. 2007;63(5):1066–73; discussion 72–3.
- 41. Coccolini F, Stahel PF, Montori G, Biffl W, Horer TM, Catena F, et al. Pelvic trauma: WSES classification and guidelines. World J Emerg Surg. 2017;12:5.
- Schicho A, Schmidt SA, Seeber K, Olivier A, Richter PH, Gebhard F. Pelvic X-ray misses out on detecting sacral fractures in the elderly—Importance of CT imaging in blunt pelvic trauma. Injury. 2016;47(3):707–10.
- Bazylewicz D, Konda S. A Review of the definitive treatment of pelvic fractures. Bull Hosp Jt Dis. 2016;74(1):6–11.
- 44. Kaufmann JE, Vischer UM. Cellular mechanisms of the hemostatic effects of desmopressin (DDAVP). J Thromb Haemost. 2003;1(4):682–9.
- 45. White CE, Hsu JR, Holcomb JB. Haemodynamically unstable pelvic fractures. Injury. 2009;40(10):1023–30.
- 46. Wagner D, Ossendorf C, Gruszka D, Hofmann A, Rommens PM. Fragility fractures of the sacrum: how to identify and when to treat surgically? Eur J Trauma Emerg Surg. 2015;41(4):349–62.
- 47. Ojodu I, Pohlemann T, Hopp S, Rollmann MF, Holstein JH, Herath SC. Predictors of mortality for complex fractures of the pelvic ring in the elderly: a twelve-year review from a German level I trauma center. Injury. 2015;46(10):1996–8.
- Nagy KK, Smith RF, Roberts RR, Joseph KT, An GC, Bokhari F, et al. Prognosis of penetrating trauma in elderly patients: a comparison with younger patients. J Trauma. 2000;49(2):190; discussion 3–4.

- Lustenberger T, Inaba K, Schnuriger B, Barmparas G, Eberle BM, Lam L, et al. Gunshot injuries in the elderly: patterns and outcomes. A national trauma databank analysis. World J Surg. 2011;35(3):528–34.
- Talving P, Benfield R, Hadjizacharia P, Inaba K, Chan LS, Demetriades D. Coagulopathy in severe traumatic brain injury: a prospective study. J Trauma. 2009;66(1):55–61; discussion -2.
- Biffl WL, Leppaniemi A. Management guidelines for penetrating abdominal trauma. World J Surg. 2015;39(6):1373–80.
- Brown CV, Shoemaker WC, Wo CC, Chan L, Demetriades D. Is noninvasive hemodynamic monitoring appropriate for the elderly critically injured patient? J Trauma. 2005;58(1):102–7.
- Montorfano MA, Pla F, Vera L, Cardillo O, Nigra SG, Montorfano LM. Point-of-care ultrasound and Doppler ultrasound evaluation of vascular injuries in penetrating and blunt trauma. Crit Ultrasound J. 2017;9(1):5.
- 54. Colip CG, Gorantla V, LeBedis CA, Soto JA, Anderson SW. Extremity CTA for penetrating trauma: 10-year experience using a 64-detector row CT scanner. Emerg Radiol. 2016;24(3):223–32.
- 55. Kuhajda I, Zarogoulidis K, Kougioumtzi I, Huang H, Li Q, Dryllis G, et al. Penetrating trauma. J Thorac Dis. 2014;6(Suppl 4):S461–5.
- Hajibandeh S, Gumber AO, Wong CS. Laparoscopy versus laparotomy for the management of penetrating abdominal trauma: a systematic review and meta-analysis. Int J Surg. 2016;34:127–36.