Postoperative Complications

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Cardiac Complications

Cardiopulmonary complications are the most frequent type of postoperative complications, followed by thromboembolic, infectious, and hematologic complications [1]. Hypertension, heart failure, and arrhythmias are common cardiovascular complications in older persons after surgery. Evaluation of postoperative hypertension should include non-cardiovascular etiologies such as assessment of pain, urinary retention, anxiety, delirium, electrolyte imbalance, hypercarbia, and hypoxia as well as a review of medications, assessment of volume status, and evaluation of fluid administration. Antihypertensive drugs can be continued throughout the perioperative period. In particular, beta-blocker therapy can decrease perioperative myocardial infarction(MI); thus beta-blocker therapy should be continued or started at least 1 day prior to the surgery in patients with coronary artery disease or risk factors for coronary artery disease unless there are contraindications such as significant hypotension or bradycardia [2, 3]. However, it is not recommended to newly start a beta-blocker in the patient who does not have these clear indications (e.g., hypertension, atrial fibrillation, angina, heart failure, or prior MI) since it increases all-cause mortality and stroke in noncardiac surgery [3].

When non-cardiovascular causes are ruled out for the etiology of postoperative hypertension, alpha- or beta-adrenergic receptor blockers, calcium channel blockers, or angiotensin-converting enzyme inhibitors can be administered orally or intravenously [3]. The potential adverse effects of certain antihypertensive agents should be considered and monitored carefully. For example, hydralazine, a potent vasodilator, should be avoided in patients with hypertrophic cardiomyopathy because rapid vasodilation and reflex tachycardia induced by it can compromise diastolic filling. In addition, the negative chronotropic effect of beta-blockers can mask the physiologic compensatory response of tachycardia due to intravascular volume depletion.

Older patients with congestive heart failure (CHF), coronary artery disease, or long-standing hypertension are also predisposed to postoperative volume overload, as their cardiac reserve is narrow and can be easily compromised. Studies have shown that CHF is associated with increased morbidity and mortality after noncardiac surgery in patients aged 65 years and older [4, 5]. CHF can develop as a result of any conditions that give rise to excessive intravascular volume overload. Acute cardiac ischemia,

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tachy- or brady-arrhythmia, and iatrogenic volume overload from intravenous fluid therapy are common causes of postoperative CHF. When patients present with acute symptoms or signs of CHF such as shortness of breath, edema, weight gain, tachypnea, abnormal respiratory sounds, hypoxia, new S3 gallop, or elevated JVP, meticulous evaluation and treatment should be conducted immediately.

The approach to CHF treatment is reducing vascular volume and cardiac afterload. Diuretics and vasodilators are commonly used for systolic heart failure. Although it is often difficult to accurately evaluate volume status in older patients based on physical examination, laboratory, and imaging studies, more invasive monitoring by Swan–Ganz catheter has no proven mortality benefit in high-risk patients [6–8].

In older adults, the mortality of perioperative MI has been reported to be as high as 40%, and the incidence of postoperative MI is associated with a high prevalence of underlying coronary artery disease [9]. Thus, early recognition and proper treatment are critical in the treatment of perioperative MI. The most common cause of postoperative MI is thought to be thrombosis due to coagulation cascade activation by surgery [10]. Older patients do not always present with "typical" features of myocardial ischemia such as chest pain. It can manifest with subtle mental status changes, gastrointestinal symptoms, and shortness of breath or even without any symptoms. Especially in cognitively impaired patients who are unable to communicate effectively, electrocardiograms should be included in the assessment of postoperative complications.

Cardiac arrhythmias are correlated with MI and heart failure. Postoperative arrhythmias can be an indicator of new cardiac ischemia or heart failure, and arrhythmia itself can lead to these complications [11, 12]. Therefore, when new arrhythmia develops postoperatively, the diagnostic work-up is directed toward investigation of possible underlying causes. Often, noncardiac problems such as electrolyte imbalance (hypokalemia, hypomagnesemia), infections, or anemia can give rise to arrhythmia. Common arrhythmias in the postoperative settings are supraventricular tachycardia, atrial fibrillation, and ventricular tachyarrhythmia [1, 12, 13]. Polanczyk et al. [13] identified risk factors for perioperative supraventricular tachycardia to be valvular disease, age greater than 70, prior history of supraventricular tachycardia, asthma, heart failure, and premature atrial complexes on preoperative electrocardiogram. Adenosine, beta-blockers, and calcium channel blockers can be used to control ventricular rate as well as promote conversion to sinus rhythm. When atrial fibrillation is diagnosed, anticoagulation should be considered to reduce the risk of stroke, especially if it persists more than 24 hours. Electrical or chemical cardioversion and rate control with beta-blockers or calcium channel blockers are the mainstays of treatment for atrial fibrillation, with each method having different indications, advantages, and disadvantages [14, 15].

Anemia, Bleeding, and Hematoma

Anemia is very common in the elderly. About 9.5% of people aged 65 and older were reported to have anemia, with the incidence and prevalence increasing with age, according to the National Health and Nutrition Examination Survey (NHANES) III study [16]. More specifically, estimates of the prevalence in geriatric patients undergoing orthopedic surgery range from 5% to 45% depending on the definitions of anemia each study applied [17, 18]. Currently the World Health Organization criteria, defining normal hemoglobin values as ≥12 g/dl for women and ≥13.0 g/dl for men, are widely accepted and adopted in most studies.

As most elderly patients have multiple contributing etiologies for anemia, it is difficult to identify the most likely etiology, especially if it is present in a preoperative evaluation. In the NHANES III study, causes of anemia were deficiencies of iron, folate, and vitamin B12, renal insufficiency, anemia of chronic inflammation, and unexplained anemia. However, the most common causes of newly developed anemia following surgery are acute or chronic blood loss, followed by nutritional deficiency and anemia of chronic disease [19]. Regardless of the underlying reasons, a limited physiologic reserve and diminished response to blood loss render the elderly population vulnerable to postoperative hematologic complications including anemia. Wu et al. [20] found that preoperative anemia is associated with a higher 30-day mortality rate.

Blood transfusion is commonly used to rapidly restore hemoglobin levels when patients present with symptoms of anemia or are hemodynamically compromised. Carson et al. [21] showed that a liberal transfusion (threshold <10 g/dl) did not reduce the mortality or inability to walk at 60 days in elderly patients compared to a restrictive transfusion (threshold <8 g/dl or symptomatic anemia). Recently, a Cochrane review on red blood cell transfusion for people undergoing hip fracture surgery concluded that there is no significant difference in postoperative mortality, functional recovery, or postoperative morbidity between the liberal and restrictive transfusion groups [22]. On the other hand, Gregersen et al. [23, 24] found that a liberal transfusion (threshold <11.3 g/dl) may improve survival and recovery of activity of daily living in elderly patients from nursing homes compared to a restrictive transfusion (threshold <9.7 g/dl). Further study is needed to clarify the transfusion threshold in elderly patients.

Although deficiency of iron, folate, or vitamin B12 can be easily corrected through oral or parenteral therapy, some geriatric patients do not tolerate oral iron well due to gastrointestinal symptoms, requiring intravenous iron. Several small studies on the roles of perioperative intravenous iron and/or erythropoietin therapy showed safety and moderate effectiveness in reduction of allogenic transfusion, but large randomized studies are still required [25–27]. In addition, intraoperative blood conservation techniques using cell recovery devices and acute normovolemic hemodilution procedures are other options to utilize the patient's own blood and reduce the need of allogenic blood transfusion [28–31].

Several case reports have described bleeding from surgical wounds and neuropathy induced by hematomas leading to significant postoperative morbidity [32–34]. Therefore, it is important to detect early signs and symptoms of postoperative bleeding and hematoma (e.g., unusual pain, ecchymosis, marked swelling, decreasing hemoglobin, any neural deficit). Surgical decompression or conservative management, as well as restoration of appropriate hemoglobin levels, should be instituted when appropriate for the treatment of postoperative bleeding and hematoma.

It is not always simple to manage anticoagulation therapy during the perioperative period. In addition, preventing deep vein thrombosis with prophylactic anticoagulation may not be safe in certain situations. Perioperative management of anticoagulation for deep vein thrombosis prophylaxis is discussed in detail in Chap. 5.

Falls

About 30% of persons 65 years and older experience at least one fall every year, 1.8 million elderly people visit emergency departments for treatment, and more than 13,000 people die as a result of falls [35]. Injuries from falls can vary from bruises and minor contusions to severe soft tissue wounds and fractures [36]. Hip fractures are almost always associated with falls either in the community or inpatient setting [37, 38]. People admitted to hospitals or residing in nursing homes have three times higher incidence of falls than community dwellers. The rate of falls in elderly inpatients ranges from 2.2% to 27%, and the incidence of falls peaks at the second postoperative week when patients begin to resume ambulatory function [39].

Many studies have identified the risk factors for falls. These can be categorized into intrinsic and extrinsic factors. Intrinsic risk factors supported by many studies are age greater than 75, a history of falls, cognitive impairment, functional disability, gait and balance disorders, comorbidities, and visual impairment [40]. Extrinsic factors include environmental factors and polypharmacy or high-risk medications. Stevens et al. identified several environmental hazards (uneven surfaces, cluttered environment, rugs, poor illumination, use of restraints, Foley's catheter placement, IV poles) associated with falls [41, 42]. Of note, use of bedside rails does not decrease or prevent falls and fall-related injuries [43]. Also polypharmacy or "high-risk" medications such as opioids, antihistamines, selective serotonin reuptake inhibitors, tricyclic antidepressants, sedatives, and anticoagulants are considered a strong predictor of falling [44]. The risk of falling increases when multiple risk factors are combined. Tinetti et al. [45] reported that the percentage of falling in persons with four or more risk factors increased from 27 to 78% compared with those with none or just one risk factor.

When a patient sustains a fall, a comprehensive physical examination and a review of predisposing risk factors including medication review should be performed. Vital signs including orthostatic blood pressure should be measured since syncopal episodes resulting from comorbidities or deconditioning from operations are closely related to falls. Special attention should be taken in neurologic evaluation, of which evaluation of gait and balance is an essential part. Mental and psychological status exams should not be excluded since delirium plays a significant role in the risk of falling in acute settings.

At least 24–48 hours of close monitoring is warranted to detect delayed complications of falls, especially in the hospital setting. Along with treatment of injuries, strategies for the prevention of future falls should be sought after falls. Several randomized controlled trials [46–49] have demonstrated that multidisciplinary and multifactorial programs show a modest reduction of the incidence of falls. These multifaceted approaches contain general and individualized safety precautions. Avoidance of physical restraints and unnecessary IV or Foley's catheter placement, encouragement of early ambulation, availability of a call light, the use of low-rise beds and floor mats, proper illumination, and scheduled toileting are examples of general safety measurements [50, 51].

A review of medications to reduce those that pose a high risk and encouragement of physical activities to enhance balance and gait are also crucial components of individualized fall prevention strategies. The individualized assessment and development of fall prevention strategies are important and beneficial, especially for patients with cognitive impairment, osteoporosis, brain injury, or other predisposing diseases that make them vulnerable to falls and fractures [52]. Education for patients and caregivers and discharge instructions for fall prevention should also be ensured in the transition from hospital to primary care. Recently, the American Geriatrics Society published a consensus statement on vitamin D supplementation to prevent falls and concluded that vitamin D supplementation be recommended to maintain a serum 25-hydroxyvitamin D concentration of 30 ng/mL or above [53]. A more detailed description of the approach to falls can be found in Chap. 3 dedicated to falls.

Electrolyte Imbalances, Fluctuating Volume Status, and Nutritional Considerations

The aging kidneys contribute to decreased glomerular filtration rate, reduced urine concentration ability, and an impaired thirst mechanism. Normal physiologic changes of the kidneys with aging, in addition to acute, "non-physiologic stress" from surgery, predispose older patients to develop fluid and electrolyte imbalances. In this section, several common electrolyte imbalances and the importance of postoperative nutrition in older patients will be discussed.

Hyponatremia

As older people have limited cardiopulmonary reserve, they are vulnerable to subtle volume changes postoperatively. A common electrolyte imbalance in patients is hyponatremia [54]. Association between hyponatremia and fracture in the elderly has been suggested [55, 56], and Leung et al. [57] showed increased perioperative mortality and morbidity in patients with preoperative hyponatremia. Postoperatively, prevalence of hyponatremia ranges from 2.8% to 36% depending on observation period, and patients with hyponatremia had longer hospital stays and increased risk of death than patients without hyponatremia [58–61]. Risk factors for postoperative hyponatremia include use of diuretics, proton pump inhibitors, and selective serotonin reuptake inhibitors, as well as polypharmacy, older age, and lower body weight. Other etiologies are dehydration, syndrome of inappropriate antidiuretic hormone, and underlying disease (CHF, salt-wasting nephropathy, adrenal insufficiency, hypothyroidism) [58, 60, 62]. Clinically, hyponatremia is associated with three different states of extracellular fluid (ECF) volume: decreased ECF volume (e.g., gastrointestinal losses, adrenal and renal salt-wasting conditions), increased ECF volume (e.g., heart failure), or normal ECF volume (e.g., syndrome of inappropriate antidiuretic hormone or SIADH) [63]. The most common cause of hyponatremia is SIADH, but regardless of the underlying cause, it is related to an excessive release of antidiuretic hormone, which can be stimulated in the postoperative period by pain, narcotics, nausea, and anesthesia. This hormone mediates retention of free water by increasing reabsorption of water in the kidneys and reducing the concentration of sodium in the body. Moreover, a diminished ability to eliminate excess fluid in older persons exacerbates water retention. The most common manifestation of hyponatremia is alteration in neurologic function, such as mild cognitive impairment, seizure, and coma due to brain edema. Improvement of neurologic symptoms usually follows days after hyponatremia is corrected.

SIADH can be caused by numerous conditions, such as infection, pulmonary and central nervous system disorders, and medications [64]. Well-recognized medications widely taken by the elderly that may cause SIADH include diuretics (thiazide, furosemide), antidepressants (SSRIs, trazodone, venlafaxine, duloxetine), antipsychotics (haloperidol, quetiapine), antihypertensives (ACE-I, ARB, carvedilol), drugs for urinary incontinence (tolterodine, oxybutynin), proton pump inhibitors, and donepezil. Physicians should be aware of these potential offending drugs and administer them cautiously during the immediate postoperative period. The diagnosis of SIADH is made after excluding other hyponatremic conditions; low plasma osmolality (<275 mosmol/kg) and inappropriate urine concentration (urine osmolality >100 mosmol/kg) are of pathognomic value [65].

Although treatment for older patients is not different from younger ones, slow correction and frequent monitoring are key to avoid iatrogenic cardiovascular and/or neurologic complications.

Hypernatremia

Hypernatremia can occur as a result of loss of free water or gain of sodium. Older adults have a decreased ability to concentrate urine and an impaired thirst mechanism, which predisposes to the development of hypernatremia especially in acute settings. Those patients with cognitive impairment or who are not ambulatory are at the highest risk for hypernatremia as well as those with infections, fever, and neurologic disorders. In a retrospective analysis of 15,187 geriatric inpatients, Snyder et al. described the incidence of hypernatremia to be 1%; the most common cause was surgery (21%), followed by febrile illness (20%) [66]. Common medications associated with the development of hypernatremia are diuretics, lithium, demeclocycline, phenytoin, ofloxacin, and amphotericin B. These drugs should be used cautiously in older patients. Recently, increased perioperative 30-day morbidity and mortality was observed in the patient with perioperative hypernatremia [67, 68].

A study of the symptoms of hypernatremia in 150 geriatric patients found that tachycardia, low blood pressure secondary to volume depletion, and cognitive dysfunction due to neuronal cell shrinkage were the most common presenting signs [69]. The declining cognitive function associated with hypernatremia may be difficult to notice in elderly patients who have a preexisting cognitive impairment. Therefore, frequent monitoring of electrolytes and close observation of mental status are important to prevent these events and initiate early treatment. The free water deficit should be replaced slowly (sodium concentration change no more than 10 mEq/L per day), usually over 72 hours by intravenous fluid or oral fluid intake.

Potassium Imbalance

Many elderly patients have a high risk of potassium imbalance, as nephrotoxic insults in the settings of chronic kidney disease are common in the postoperative period. Often drugs such as NSAIDs, angiotensin-converting enzyme inhibitors, potassium supplements, and beta-blockers can lead to hyperkalemia [63]. Many older patients on diuretics take potassium supplementation. Thus, close monitoring of potassium is important especially when those patients are not receiving diuretics post-operatively to manage intravascular volume.

Since hypokalemia affects the cardiovascular, neurologic, muscular, and renal systems, early identification and treatment are key to minimize morbidity and mortality. There are many potential etiologies of hypokalemia in elderly patients during the postoperative period (such as vomiting, diarrhea, and nasogastric suction), all of which can induce metabolic alkalosis, shifting potassium into cells. Renal loss from the use of diuretics, underlying kidney disease, and endocrine problems are also common causes. Correction of underlying condition(s) and appropriate potassium repletion are the major approaches in the treatment of hypokalemia.

Imbalances in Calcium and Other Electrolytes

There are multiple causes for hypercalcemia and hypocalcemia in the elderly, and these are not commonly associated with postoperative complications. However, since many older patients who have sustained fractures are likely to have underlying osteoporosis, physicians should ensure that patients take adequate amounts of calcium and vitamin D to reduce the risk of future fractures. Currently, the National Osteoporosis Foundation recommends a total of 1200 mg of daily calcium from dietary and supplemental sources for the elderly. Even though there is a significant benefit of adequate calcium intake in reducing fracture risk, increased risk of nephrolithiasis and gastrointestinal side effects were observed [70]. Also, a potential causal relationship between calcium supplementation and coronary and cerebrovascular events was suggested and needs further investigation [71, 72].

Renal insufficiency, poor or excessive dietary intake, and certain medications can contribute to alterations in other electrolytes. For example, a high-calcium diet may inhibit zinc absorption, which is an essential dietary element for proper wound healing [63, 73], and hypocalcemia cannot be corrected without correction of hypomagnesemia [63]. Hence, monitoring of calcium and magnesium is imperative in ill geriatric patients.

Nutrition

Older patients are predisposed to under- or malnourishment in the postoperative period. Up to 20% of patients with hip fractures [74, 75] were reported to have malnutrition. In particular, protein depletion adversely affects the outcome of surgery and is associated with poor wound healing and decreased 1-year survival [76, 77]. Patients with low serum albumin (<3.5 g/dl) and low total lymphocyte count (<1500 cells/μl) have longer hospital stays (2.9 times), have higher 1-year mortality (3.9 times), and are less likely to return to their pre-fracture level of function (4.6 times) [78]. By contrast, patients with hip fractures who had received aggressive postoperative nutritional protein supplementation had shorter hospitalizations, appropriate weight gains, and declines in postoperative complications [79–82].

Most patients should be advised to resume oral intake as early as within 12 hours postoperatively. Intake of supplementary formula was found to have a beneficial effect on surgical outcome [83]. If adequate oral intake is not achieved, temporary nasogastric tube feeding should be considered as an alternative or supplementary method [79, 82], although tolerability may be low. It may be prudent to consult with a dietician to meet patient-specific nutritional requirements.

Infections: Surgical Site Infection, Urinary Tract Infection, and Pneumonia

Elderly patients may have decreased immune function resulting from both age-related declines in immunity and comorbidities. Therefore, typical signs and symptoms of infection like fever may be absent in frail older adults with serious infections, and it is not uncommon that they present with confusion, falls, or nonspecific decline in functional status. If these occur in demented patients, it becomes very difficult for physicians to differentiate infection from underlying cognitive impairment; thus, physical and psychological changes in the elderly should be carefully monitored postoperatively.

Surgical Site Infection

Surgical site infection is a serious postoperative complication. A study by Kaye et al. [84] demonstrated that elderly patients with postoperative surgical site infection had three times higher mortality, and their hospital stays were 2 weeks longer on average. *Staphylococcus aureus* was the most common pathogen in surgical site infection, followed by coagulase-negative *Staphylococcus*, *Enterococcus* spp., *Streptococcus*, *E. coli*, and *Pseudomonas*. Of note, 58.2% of the staphylococci were methicillin resistant.

Currently, prophylactic antibiotics are recommended and widely used based on studies which showed a decreased incidence of surgical wound infection in patients who received antibiotics prior to surgery [85, 86]. Clinical guidelines on prophylactic antibiotics were published in 2013 with updated recommendations regarding antibiotic selection, timing and dose of antibiotics, as well as duration of prophylaxis. The optimal timing for initiation of antibiotics is within 1 hour prior to surgical incision and 2 hours prior to surgery in the case of vancomycin or fluoroquinolone prophylaxis. Use of antibiotics less than 24 hours after the surgery is recommended [87].

Pulmonary Complications: Pneumonia

Diminished ability to protect the airway from aspiration and decreased functional capacity make the older surgical patient prone to developing postoperative pulmonary infections. Atelectasis is a common predisposing factor to the development of pneumonia. Arozullah et al. proposed a clinical prediction model for postoperative pneumonia in noncardiac surgical patients based on a database study of 160,000 veterans [88]. Patients were divided into five risk classes based on risk index scores generated from predisposing factors such as type of operation, age, functional status, weight loss, chronic obstructive pulmonary disease, general anesthesia, impaired sensorium, cerebral vascular accident, blood urea nitrogen level, recent transfusion, emergency surgery, long-term steroid use, smoking, and alcohol use. Pneumonia rates were 0.2% among those with 0–15 risk points, 1.2% for those with 16–25 risk points, 4.0% for those with 26–40 risk points, 9.4% for those with 41–55 risk points, and 15.3% for those with more than 55 risk points. This index may be useful in identification of high-risk patients and in providing early preventative measures, including percussion, voluntary cough, incentive spirometry, and encouragement of early ambulation [88]. Once postoperative pneumonia is diagnosed, management should include collection of a respiratory specimen for culture and early initiation of broad-spectrum antibiotics until culture and sensitivity results are available. The most common organisms found in postoperative pneumonia are gram-negative bacteria and *Staphylococcus aureus* [89]. Selection of antibiotics should be tailored based on hospital antibiotic resistance profiles or culture results if available.

Urinary Tract Infection

Postoperative urinary tract infection (UTI) is a common problem in the elderly [90]. The incidence of urinary tract infection after hip fracture surgery was reported to be between 23% and 25% [85, 91, 92]. After the second day of catheterization, the risk of UTI increases by 5–10% [93, 94].

The most common organisms associated with short-term catheter use during an operative procedure are usually from skin colonization and include *Escherichia coli*, enterococci, *Pseudomonas*, *Klebsiella*, *Enterobacter*, *Staphylococcus epidermidis*, *Staphylococcus aureus*, and *Serratia*. The gold standard diagnostic test in patients with an indwelling catheter is urine culture after documentation of significant pyuria by urinalysis (usually >50 white blood cells/high power field). The growth of 10^5 or more colony-forming units of bacteria per milliliter of urine can diagnose UTI with 90% specificity and 37% sensitivity [95]. Treatment of UTI is straightforward, but separate consideration should be given to asymptomatic bacteriuria, which does not need to be treated except in patients who are undergoing implantation of a prosthesis, are immunocompromised, or harbor strains that have a high incidence of bacteremia (e.g., *S. marcescens*) [96]. Treatment with appropriate antibiotics for 10–14 days, depending upon the severity of symptoms and rapidity of response, is generally recommended for UTIs associated with indwelling catheters [97]. Choice of antibiotics should be based upon the urine culture results. If treatment is needed before culture results are available, the urine gram stain, previous culture results, or hospital-specific antimicrobial sensitivity patterns should be considered when initiating empiric antibiotics.

Removing indwelling urinary catheters within 24 hours of surgery has been shown to reduce the incidence of urinary retention [90]. One randomized controlled trial also demonstrated that intermittent urinary catheterization rather than indwelling catheterization accelerated recovery of the normal voiding pattern by 4 days [98].

Physicians also need to be aware of common medications that can contribute to urinary retention such as analgesics, sedatives, and anticholinergics.

Pressure Ulcers

A pressure ulcer is defined by the National Pressure Ulcer Advisory Panel (NPUAP) as localized injury to the skin and/or underlying tissue, usually over a bony prominence, as a result of pressure or pressure in combination with shear and/or friction [99]. The incidence of pressure ulcers in patients with hip fractures is reported to range from 10% to 40% [100, 101]. Common sites of occurrence are the ischium (28%), sacrum (17–27%), trochanter (12–19%), and heel (9–18%) [99, 102–105]. In geriatric patients, the occiput and the thoracic kyphotic areas can also be involved, as those hospitalized are often likely to lie in a supine position for a long period of time. Pressure ulcers are detected by their appearance and location overlying a bony prominence. Pressure ulcers should be distinguished from ulcers associated with diabetic neuropathy, vascular insufficiency, and cellulitis.

The NPUAP published an update on pressure ulcer staging in 2016. These are:

Stage 1. Intact skin with non-blanchable erythema of a localized area usually over a bony prominence.

Stage 2. Partial thickness loss of skin with exposed dermis presenting as a shallow open ulcer with a red or pink wound bed, without slough and an intact or ruptured serum filled blister.

Stage 3. Full thickness skin loss. Subcutaneous fat and granulation tissue may be visible, but the bone, tendon, or muscle is not exposed. Undermining and tunneling may also be seen.

Stage 4. Full thickness skin and tissue loss with exposed bone, tendon, or muscle. These ulcers often include undermining and tunneling. Unstageable: Full thickness skin and tissue loss in which the base of the ulcer is covered by slough (yellow, tan, gray, green, or brown) and/or eschar (tan, brown, or black) in the wound bed and extent of tissue damage can not be determined. Deep tissue pressure injury: Non-blanchable deep red, purple or maroon discoloration of localized area of discolored intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear.

Pathogenesis of pressure ulcers is multifactorial, usually as a result of a combination of external factors (pressure, shearing forces, friction, moisture) and host factors [106]. The host factors include immobility, incontinence, nutritional compromise, poor skin perfusion, and neurologic diseases [107–111]. Haleem et al. [112] identified risk factors for pressure ulcers in patients with hip fractures as increased age, diabetes mellitus, lower mental status test score, lower mobility score, lower admission hemoglobin, and an intraoperative drop in blood pressure.

An increased risk of nosocomial infections and prolonged hospitalization is associated with development of pressure ulcers during inpatient stays [100, 113]. Infection is the most common major complication of pressure ulcers. It can present as superficial infection or more seriously as cellulitis, osteomyelitis, and sepsis. Commonly isolated organisms are *Proteus mirabilis*, group D streptococci, *Escherichia coli*, *Staphylococcus*, *Pseudomonas*, and *Corynebacterium* [114]. Although infection of pressure ulcers is usually indicated by local signs such as warmth, erythema, tenderness, discharge, and the presence of a foul odor [115], delayed wound healing can be the only sign of infection. In the elderly, systemic signs (leukocytosis, fever) are not always present [116].

Distinguishing between bacterial colonization and invasion is challenging as almost all pressure ulcers have bacterial colonization which precedes the development of infection. Wound swab culture is a simple procedure that is often used to guide antibiotic selection for patients who are not candidates for invasive diagnostic procedures such as bone or tissue biopsy. However, guidelines on the treatment of pressure ulcers by the Agency for Healthcare Research and Quality recommend against using swab cultures to define the microbiology of a pressure ulcer as it merely reflects bacterial colonization [117]. The gold standard method of wound culture is still culturing deep tissue specimens sampled from a surgically cleaned and debrided ulcer, along with blood cultures. The general concepts of wound care are keeping the wound moist and intact skin dry, as well as removing dead tissue. Currently, hydrocolloid dressings have been supported by many experts because of their excellent effect on rapid wound healing, fewer dressing requirements, and cost-effectiveness [118]. Among antiseptics, NPUAP specifically discourages the use of hydrogen peroxidase as a preferred topical antiseptic secondary to its high toxicity to tissue. Also, cautious use of iodine products and sodium hypochlorite was recommended [99].

Necrotic tissue prevents wound healing and provides an environment for bacteria to grow and therefore should be debrided. There are several debridement methods including enzymatic, autolytic, mechanical, surgical, and biosurgical (e.g., using sterile larvae from "medical-grade" maggots of the green bottle fly, *Lucilia sericata*) [119]. The choice of method should be based on a consideration of the patient's ability to tolerate the procedure, as some of these (e.g., mechanical, surgical) may cause pain [118, 120].

The strategy of pressure ulcer prevention consists of skin care measures, relief of skin pressure using pressure-relieving devices or frequent position changes, and good nutrition (increased protein intake to 1.50 g/kg) [118]. Placing pillows under the legs to elevate the feet, applying transparent film dressings on the heels, and changing the patient's position regularly and frequently are good examples of pressure ulcer prevention for the heels [118, 120, 121]. Active utilization of pressure-relieving devices such as foam, gel, low air loss, or air-fluidized mattresses also helps to prevent ulcers and accelerate wound healing in high-risk patients. If the nutritional intake is low or there is nutritional deficiency, it is reasonable to optimize the nutritional intake and correct the deficiency [122]. Education of patients, caregivers, and care-providing medical staff is a critical part of pressure ulcer prevention.

Pain Management

Insufficient postoperative pain management can lead to delayed ambulation and a prolonged hospital stay. Conversely, adequate pain control can have a positive impact on the rapid resumption of functional status [10]. Choosing the right analgesics is sometimes challenging to physicians because older patients are vulnerable to side effects of analgesics and drug interactions, and in cognitively impaired patients, it is very difficult to assess the response to medications. Therapy with “round-the-clock” nonnarcotics such as acetaminophen along with short-acting opioids for breakthrough pain has been shown to be effective for pain control in geriatric hip fracture patients [123]. Prevention of analgesic-induced side effects is as important as good pain control. When opioids are used, good bowel regimens should be included to avoid constipation. See Chap. 7 for a comprehensive overview of perioperative pain management.

Deep Vein Thrombosis, Pulmonary Embolism, and Fat Embolism

Deep Vein Thrombosis

Deep vein thrombosis (DVT) is common in patients with fractures. The incidence rate in patients after hip fracture surgery was reported to be 27% [124, 125], and the rate of fatal pulmonary embolism was 1.4–2.5% for the first 3 months after hip fracture surgery [124].

The ninth edition of the “Antithrombotic Therapy and Prevention of Thrombosis” guidelines from the American College of Chest Physicians recommends the use of low-molecular-weight heparin (LMWH), fondaparinux, apixaban, rivaroxaban, low-dose unfractionated heparin, adjusted-dose vitamin K antagonists, aspirin, or an intermittent pneumatic compression device [126]. The guidelines suggest LMWH in preference to the other agents in patients who are undergoing major orthopedic surgery including total hip arthroplasty, total knee arthroplasty, and hip fracture surgery. Based on patient bleeding risk and renal function, the most appropriate agent should be chosen. Platelet count should be regularly monitored to detect heparin-induced thrombocytopenia in patients on heparin products (unfractionated or LMWH). See Chap. 5 for a detailed review of DVT prophylaxis.

Pulmonary Embolism

One of the most serious complications following orthopedic surgery is pulmonary embolism (PE). Immobilization from orthopedic procedures; underlying diseases such as heart failure, malignancy, and myeloproliferative disease; and age itself pose a high risk for developing thrombosis and subsequent PE in the elderly. PE develops most commonly as a result of migration of thrombi from the deep venous system of the lower extremity [127]. The incidence of PE increases with age with an annual incidence of 1.8 per 1000 among persons of age 65–69 as compared with 3.1 in those age 85–89 [128]. Especially after orthopedic surgery, the incidence of PE is reported to be as high as 30% for total hip replacement surgery, 7% for total knee replacement surgery, and 24% for hip fracture surgery [129].

Elderly patients with PE can present with a wide variety of symptoms and signs from mild shortness of breath to sudden cardiopulmonary arrest. Since the symptoms and signs of PE are nonspecific and difficult to differentiate from patients’ underlying cardiopulmonary diseases, it is often mis- or underdiagnosed. Morpurgo et al. reported in 200 autopsy cases that 78% of massive or submassive PE had not been detected by physicians [130]. The most common symptoms and signs include dyspnea at rest or on exertion, cough, pleuritic pain, orthopnea, calf or thigh swelling or pain, tachypnea, tachycardia, and abnormal lung sounds such as crackles and decreased breath sounds [131]. Therefore, when assessing a patient presenting with unexplained tachypnea, tachycardia, or change in mental status in postoperative periods, PE should be included in the differential diagnosis.

The diagnosis of PE can be challenging due to the lack of specificity and sensitivity of these presentations. Therefore, a scoring system that provides a pretest probability has been developed to guide clinicians to consider further diagnostic tests to establish the diagnosis of PE [132]. However, since a clinician's subjective judgment strongly influences the scores, the objectivity and validity of this scoring system are still being debated [133]. Therefore, diagnostic tests should be performed when clinical suspicion is present. Currently, imaging studies such as a ventilation–perfusion lung scan (V/Q scan) and single- or multi-detector computed tomographic angiography (CT angiography) play a more important role than laboratory testing in the diagnosis of PE.

The V/Q scan uses a pattern of a mismatch of perfusion with ventilation, and the results are displayed as normal, high, intermediate, and low probabilities. The Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) study demonstrated that the predictive value of the V/Q scan varies depending on clinical suspicion. For example, when a high-probability V/Q scan is combined with a high clinical pretest probability, the likelihood of PE was 96%, but only 56–88% had confirmed PE when a high-probability V/Q scan is associated with a low clinical pretest probability. In one study, there were up to 70% of patients who remained undiagnosed even after the V/Q scan was performed [134]. Therefore, additional tests are required in many situations, and the study itself is not often feasible in certain patients who are not able to follow the instructions of scan protocols. These limitations make CT angiography a more preferable choice for a diagnosis of PE. In one study, single-detector CT angiography had a sensitivity of 70% and a specificity of 88%, with 84% negative predictive value and 76% positive predictive value [135]. Although it has limited sensitivity in detecting subsegmental emboli, CT angiography has the additional advantage of detecting other pulmonary diseases. Also, negative CT angiography is acceptable in decision-making for the safe withdrawal of anticoagulation therapy [136].

Pulmonary angiography is a definitive diagnostic test for PE, but because it has several disadvantages, including invasiveness and high intravenous contrast exposure, it should be reserved for situations where other tests are nondiagnostic and the angiography results will change treatment plans. Ultrasonography and color-flow Doppler may be helpful in patients with nondiagnostic V/Q scans and CT angiography. Patients with positive test results on ultrasonography may reasonably receive anticoagulation regardless of V/Q scanning or CT angiography results.

The most common EKG findings in PE are sinus tachycardia and a right-sided strain pattern, but classic patterns like S1Q3T3 (S wave in lead I, Q wave in lead III, T wave inversion in lead III) are seen in less than 20% of cases [137]. The chest X-ray can be normal, but the classic "Hampton hump" sign caused by pleural infarction can also be seen. The D-dimer test has been intensively investigated as a noninvasive, radiation-free test for DVT and PE. However, the specificity of this test has been questioned and has been shown to be dependent upon the type of assay [138, 139]. Furthermore, the specificity was found to be lower in geriatric patients. Therefore, this test should be used in conjunction with other imaging studies [140–142]. Arterial blood gas studies are also nonspecific. Arterial hypoxemia and an increased alveolar–arterial (A–a) gradient are common findings, but these cannot be used in the diagnosis of PE since an elevated A–a gradient is not uncommon in the geriatric population [142].

Treatment for PE consists of supportive care, anticoagulation, and close monitoring of bleeding complications from anticoagulation therapy. Adequate intravascular volume (which can be compromised due to a decreased cardiac output) and supplementary oxygen should be ensured. In addition, anticoagulation therapy with either unfractionated heparin or LMWH should not be delayed until the completion of the diagnostic work-up, especially when there is moderate-to-high clinical suspicion of PE, because the mortality rate is highest within the first few hours of clot development.

Oral anticoagulation with warfarin should be started with heparin therapy, and heparin can be discontinued when warfarin reaches therapeutic range, usually with an international normalized ratio

(INR) of 2–3. The novel oral anticoagulants, including factor Xa inhibitors (rivaroxaban, apixaban, edoxaban), can also be used as long-term anticoagulation [143–145]. However, direct thrombin inhibition (e.g., dabigatran) is not recommended in those above age 80 [146]. Duration of anticoagulation therapy depends on possible underlying etiologies. If PE occurs with reversible risk factors such as surgery and immobilization, warfarin therapy should be maintained for 3 months. However, in patients with recurrent thromboembolism or irreversible risk factors (such as factor V Leiden mutation, protein C and S deficiency, or antiphospholipid antibodies), lifelong anticoagulation should be considered. Patients with a first episode of unprovoked thromboembolism should be treated with warfarin for at least 3 months and then reassessed [147].

Thrombolytic therapy should be considered for hemodynamically unstable patients. Although there is no proven mortality benefit from thrombolytic therapy, many studies have demonstrated hemodynamic and radiographic improvement in critically ill patients.

When a patient with PE is not a candidate for anticoagulation therapy due to absolute contraindications such as recent surgery, active bleeding, or hemorrhagic stroke, or when there is a foreseeable fatal clot burden from recurrent thromboembolism, an inferior vena cava filter (IVC filter or Greenfield filter) insertion should be considered [147].

Fat Embolism

Fat embolism (FE) is a potentially fatal condition causing high mortality and morbidity in trauma patients. It can develop as a result of entrance of fat droplets into the circulatory system. The majority of cases are associated with major trauma such as long bone and pelvic fractures. The incidence of fat embolism has been reported from 0.9 to 23% in patients with fractures [148, 149]. A higher incidence was associated with multiple fractures and closed fractures [150, 151].

Clinical manifestations of FE consist of a classic triad: respiratory abnormalities, skin rash, and neurologic changes [151, 152]. These symptoms and signs usually present 24–72 hours after the trauma. Frequently, respiratory abnormalities such as tachypnea, hypoxia, and dyspnea precede other skin and neurologic problems. FE often leads to respiratory failure requiring mechanical ventilation. When these fat emboli go to the central nervous system and the skin after escaping the pulmonary circulation, neurologic changes, including confusion, disorientation, personality change, vision change, paresis, and seizures, can develop, followed by the occurrence of skin rash such as characteristic petechial rash in the conjunctiva, skin folds, and oral mucous membranes [153].

Diagnosis is usually made based on typical clinical features. However, this is often challenging because there is no definitive test to rely on. Thus, several diagnostic criteria (e.g., Lindeque's criteria, Gurd's criteria, and a fat embolism index) have been proposed, but the sensitivity and specificity of these criteria still remain unclear [136, 154].

Laboratory tests may be helpful. An unexplained drop of hematocrit, thrombocytopenia due to intravascular coagulopathy, and hypocalcemia due to calcium binding to fatty acids can be present.

There are several radiographic changes suggestive of FE but not very specific to this condition. The chest X-ray findings vary from normal to diffuse or patchy air space consolidation. The signs of alveolar edema, hemorrhage, and inflammatory changes can be seen on chest CT scan [152, 155]. It may also be useful to confirm the presence of fat globules in urine or sputum in diagnosis of FE [154].

No specific treatment has been developed for FE, which conversely emphasizes the importance of prevention. Early immobilization and operative fixation of fractures and application of surgical techniques that minimize an increase of intraosseous pressure are reported to reduce the incidence of FE [156]. Prophylaxis with corticosteroids decreased the incidence and severity of FE in several studies [136, 157], but the routine use of prophylactic corticosteroid is still controversial. Once the diagnosis of FE is highly suspected, supportive care is a mainstay of treatment plans. Adequate oxygenation and hydration, and proper prophylaxis for DVT and GI bleeding, should be ensured [158]. Overall mortality is reported to be approximately 7% [159].

Postoperative Cognitive Dysfunction: Delirium

Delirium may be the most common postoperative complication in geriatric patients. The incidence was reported to be as high as 62% among hip fracture patients [160]. It is often underdiagnosed and undertreated [98]. There is growing evidence that delirium is associated with increasing mortality, prolonged length of hospital stay, high likelihood of post-discharge nursing home placement, poor functional recovery, and even increased risk of death 2 years after hospital discharge [161–163]. The most common risk factors are advanced age, dementia, sensory impairment, a history of alcohol abuse, electrolyte imbalance, use of psychotropic medications, and changes in sleep–wake cycle [123, 160]. Clinicians can diagnose delirium by utilizing the Confusion Assessment Method (CAM) which incorporates the four key features of delirium. These components are (1) acute change in mental status and fluctuating course, (2) inattention, (3) disorganized thinking, and (4) altered level of consciousness. The diagnosis of delirium requires the presence of components 1 and 2 and either 3 or 4 [164, 165]. Once delirium is suspected, a comprehensive history and thorough physical examination with a focused neuropsychiatric exam, review of medications, assessment of pain, and targeted diagnostic laboratory tests are essential.

The major principles in the prevention and treatment of delirium consist of identification and correction of reversible risk factors. Removing physical restraints as well as unnecessary Foley catheters and intravenous lines, resuming ambulation, minimizing environmental changes, and verbal and visual reorientation are important. When pharmacologic treatment is considered, high potency antipsychotics are preferred due to their favorable side effect profiles [123, 160, 166]. However, prolonged use of antipsychotics should be avoided given increased mortality in dementia patients. Chapter 6 provides a detailed overview of postoperative delirium.

References

1. McLaughlin M, et al. Preoperative status and risk of complications in patients with hip fractures. J Gen Intern Med. 2006;21:219–25.
2. Siddiqui AK, Ahmed S, Delbeau H, Conner D, Maffana J. Lack of physician concordance with guidelines on the perioperative use of beta-blockers. Arch Intern Med. 2004;164(6):664–7.
3. Wijeysundera DN, Duncan D, Nkonde-Price C, Virani SS, Washam JB, Fleischmann KE, Fleisher LA. Perioperative beta blockade in noncardiac surgery: a systematic review for the 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: a report of the American College of Cardiology/American Heart Association task force on practice guidelines. J Am Coll Cardiol. 2014;64(22):2406–25.
4. Hernandez AF, Whellan DJ, Stroud S, Sun JL, O'Connor CM, Jollis JG. Outcomes in heart failure patients after major noncardiac surgery. J Am Coll Cardiol. 2004;44(7):1446–53.
5. Dunlop WE, Rosenblood L, Lawrason L, Birdsall L, Rusnak CH. Effects of age and severity of illness on outcome and length of stay in geriatric surgical patients. Am J Surg. 1993;165(5):577–80.
6. Shah MR, Hasselblad V, et al. Impact of the pulmonary artery catheter in critically ill patients: meta-analysis of randomized clinical trials. JAMA. 2005;294:1664–70.
7. Binanay C, Califf RM, Hasselblad V, et al. ESCAPE investigators and ESCAPE study coordinators. Evaluation study of congestive heart failure and pulmonary artery catheterization effectiveness: the ESCAPE trial. JAMA. 2005;294:1625–33.
8. Shah MR, O'Connor CM, Sopko G, et al. Evaluation study of congestive heart failure and pulmonary artery catheterization effectiveness (ESCAPE): design and rationale. Am Heart J. 2001;141:528–35.
9. Mangano DT. Perioperative cardiac morbidity. Anesthesiology. 1990;72:153–84.

10. Souders J, Rooke A. Perioperative care for geriatric patients. Ann Longterm Care. 2005;13:17–29.
11. Goldman L. Cardiac risks and complications of noncardiac surgery. Ann Intern Med. 1983;98:513–4.
12. Pedersen T, Eliasen K, Henriksen EA. Prospective study of risk factors and cardiopulmonary complications associated with anaesthesia and surgery: risk indicators of cardiopulmonary morbidity. Acta Anaesthesiol Scand. 1990;34:144–55.
13. Polanczyk CA, Goldman L, Marcantonio ER, Orav EJ, Lee TH. Supraventricular arrhythmia in patients having noncardiac surgery: clinical correlates and effect on length of stay. Ann Intern Med. 1998;129:279–85.
14. Khoo CW, Lip GY. Acute management of atrial fibrillation. Chest. 2009;135(3):849–59.
15. Naccarelli GV, Wolbrette DL, Khan M, Bhatta L, Hynes J, Samii S, Luck J. Old and new antiarrhythmic drugs for converting and maintaining sinus rhythm in atrial fibrillation: comparative efficacy and results of trials. Am J Cardiol. 2003;91(6A):15D–26D.
16. Guralnik JM, Eisenstaedt RS, Ferrucci L, Klein HG, Woodman RC. Prevalence of anemia in persons 65 years and older in the United States: evidence for a high rate of unexplained anemia. Blood. 2004;104(8):2263–8.
17. Haljamae H, Stefansson T, Wickstrom I. Preanesthetic evaluation of the female geriatric patient with hip fracture. Acta Anaesthesiol Scand. 1982;26:393–402.
18. Gruson KI, Aharonoff GB, Egol KA, et al. The relationship between admission hemoglobin level and outcome after hip fracture. J Orthop Trauma. 2002;16:39–44.
19. Kulier A, Gombotz H. Perioperative anemia. Anaesthesist. 2001;50(2):73–86.
20. WC W, et al. Preoperative hematocrit levels and postoperative outcomes in older patients undergoing noncardiac surgery. JAMA. 2007;297(22):2481–8.
21. Carson JL, et al. Liberal or restrictive transfusion in high-risk patients after hip surgery. N Engl J Med. 2011;365(26):2453–62.
22. Brunskill SJ, Millette SL, Shokoohi A, Pulford EC, Doree C, Murphy MF, Stanworth S. Red blood cell transfusion for people undergoing hip fracture surgery. Cochrane Database Syst Rev. 2015;4:CD009699.
23. Gregersen M, Borris LC, Damsgaard EM. Postoperative blood transfusion strategy in frail, anemic elderly patients with hip fracture: the TRIFE randomized controlled trial. Acta Orthop. 2015;86(3):363–72.
24. Gregersen M, Borris LC, Damsgaard EM. Blood transfusion and overall quality of Life after hip fracture in frail elderly patients-the transfusion requirements in frail elderly randomized controlled trial. J Am Med Dir Assoc. 2015;16(9):762–6.
25. Munoz M, et al. Role of parenteral iron in transfusion requirements after total hip replace ment. A pilot study. Transfus Med. 2006;16(2):137–42.
26. Garcia-Erce JA, et al. Perioperative stimulation of erythropoiesis with intravenous iron and erythropoietin reduces transfusion requirements in patients with hip fracture. A prospective observational study. Vox Sang. 2005;88(4):235–43.
27. Cuenca J, et al. Perioperative intravenous iron, with or without erythropoietin, plus restrictive transfusion protocol reduce the need for allogeneic blood after knee replacement surgery. Transfusion. 2006;46(7):1112–9.
28. Goodnough LT, Monk TG, Sicard G, et al. Intraoperative salvage in patients undergoing elective abdominal aortic aneurysm repair: an analysis of cost and benefit. J Vasc Surg. 1996;24(2):213–8.
29. Monk TG, Goodnough LT, Brecher ME, et al. A prospective randomized comparison of three blood conservation strategies for radical prostatectomy. Anesthesiology. 1999;91(1):24–33.
30. Bryson GL, Laupacis A, Wells GA. Does acute normovolemic hemodilution reduce perioperative allogeneic transfusion? A meta-analysis. The international study of perioperative transfusion. Anesth Analg. 1998;86(1):9–15.
31. Monk TG, Goodnough LT. Acute normovolemic hemodilution. Clin Orthop Relat Res. 1998;357:74–81.
32. Oldenberg M, Muller RT. The frequency, prognosis and significance of nerve injuries in total hip arthroplasty. Int Orthop. 1997;21(1):1–3.
33. Farrell CM, Springer BD, et al. Motor nerve palsy following primary total hip arthroplasty. J Bone Joint Surg Am. 2005;87(12):2619–25.
34. Fleming RE, Michelsen CB, Stinchfield FE. Sciatic paralysis. A complication of bleeding following hip surgery. J Bone Joint Surg Am. 1979;61:37–9.
35. Centers for Disease Control and Prevention. Fatalities and injuries from fall among older adults – United States, 1993–2003 and 2001–2005. MMWR Morb Mortal Wkly Rep. 2006;55:1221–4.
36. Kannus P, Sievanen H, Palvanen M, et al. Prevention of falls and consequent injuries in elderly people. Lancet. 2005;366:1885–93.
37. Nyberg L, Gustafson Y, Berggren D, Brannstrom B, Bucht G. Falls leading to femoral neck fractures in lucid older people. Incidence of falls in three different types of geriatric care. A Swedish prospective study. J Am Geriatr Soc. 1996;44:156–60.
38. Nyberg L, Gustafson Y, Janson A, Sandman PO, Eriksson S. Incidence of falls in three different types. Scand J Soc Med. 1997;25(1):8–13.
39. Pils K, Neumann F, Meisner W, Schano W, Vavrovsky G, Van der Cammen TJ. Predictors of falls in elderly people during rehabilitation after hip fracture – who is at risk of a second one? Z Gerontol Geriatr. 2003;36(1):16–22.
40. Tinetti ME. Preventing falls in elderly persons. N Engl J Med. 2003;348:42–9.

41. Grey-Micelli D, Capezuti E, Zwicker D, Mezey M, Fulmer T, editors. Preventing falls in acute care: evidence-based geriatric nursing protocols for best practice. 3rd ed. New York: Springer Publishing Company; 2008. p. 161–98.
42. Stevens M, Holman CD, Bennett N. Preventing falls in older people: impact of an intervention to reduce environmental hazards in home. J Am Geriatr Soc. 2001;49:1442–8.
43. Capezuti E, Maislin G, Strumpf N, Evans LK. Side rail use and bed-related fall outcome among nursing home residents. J Am Geriatr Soc. 2002;50:90–6.
44. Leigzig RM, Cumming RG, Tinetti ME. Drugs and falls in older people: a systematic review and meta-analysis: I. Psychotropic drugs. J Am Geriatr Soc. 1999;47:30–5.
45. Tinetti ME, Speechley M, Ginter SF. Risk factors for falls among elderly persons living in the community. N Engl J Med. 1988;319:1701–7.
46. Haines TP, Bennell KL, Osborne RH, Hill KD. Effectiveness of targeted falls prevention programmes in a subacute setting. A randomised controlled trial. BMJ. 2004;328:676–9.
47. Healey F, Monro A, Cockram A, et al. Using targeted risk factor reduction to prevent falls in older hospital inpatients. A randomised controlled trial. Age Ageing. 2004;33:390–5.
48. Reuben DB, Borok GM, Wolde-Tsadik G, et al. A randomized trial of comprehensive geriatric assessment in the care of hospitalized patients. NEJM. 1995;332(20):1345–50.
49. Berggren M, Stenvall M, Olofsson B, Gustafson Y. Evaluation of a fall-prevention program in older people after femoral neck fracture: a one-year follow-up. Osteoporos Int. 2008;19(6):801–9.
50. Tinetti ME. Multifactorial fall-prevention strategies: time to retreat or advance. J Am Geriatr Soc. 2008;56:1563–70.
51. Chang JT, Ganz DA. Quality indicators for fall and mobility problems in vulnerable elders. J Am Geriatr Soc. 2007;55(Suppl 2):S327–34.
52. Naqvi F, Lee S, Fields S. An evidence-based review of the NICHE guideline for preventing falls in older adults in an acute setting. Geriatrics. 2009;64:10–26.
53. American Geriatrics Society Workgroup on Vitamin D Supplementation for Older Adults. Recommendations abstracted from the American Geriatrics Society consensus statement on vitamin D for prevention of falls and their consequences. J Am Geriatr Soc. 2014;62(1):147–52.
54. Gordon M. Restoring functional independence in the older hip fracture patient. Geriatrics. 1989;44(12):48–53. see also pp 56, 59.
55. Tolouian R, Alhamad T, Farazmand M, Mulla ZD. The correlation of hip fracture and hyponatremia in the elderly. J Nephrol. 2012;25(5):789–93.
56. Gankam Kengne F, Andres C, Sattar L, Melot C, Decaux G. Mild hyponatremia and risk of fracture in the ambulatory elderly. QJM. 2008;101(7):583–8.
57. Leung AA, McAlister FA, Rogers SO Jr, Pazo V, Wright A, Bates DW. Preoperative hyponatremia and perioperative complications. Arch Intern Med. 2012;172(19):1474–81.
58. Cumming K, Hoyle GE, Hutchison JD, Soiza RL. Prevalence, incidence and etiology of hyponatremia in elderly patients with fragility fractures. PLoS One. 2014;9(2):e88272.
59. Tinning CG, Cochrane LA, Singer BR. Analysis of hyponatraemia associated post-operative mortality in 3897 hip fracture patients. Injury. 2015;46(7):1328–32.
60. Rudge JE, Kim D. New-onset hyponatraemia after surgery for traumatic hip fracture. Age Ageing. 2014;43(6):821–6.
61. Tambe AA, Hill R, Livesley PJ. Post-operative hyponatraemia in orthopaedic injury. Injury. 2003;34(4):253–5.
62. Sah AP. Hyponatremia after primary hip and knee arthroplasty: incidence and associated risk factors. Am J Orthop (Belle Mead NJ). 2014;43(4):E69–73.
63. Luckey AE, Parsa CH. Fluid and electrolytes in the elderly. Arch Surg. 2003;138(10):1055–60.
64. Hirshberg B, Ben-Yehuda A. The syndrome of inappropriate antidiuretic hormone secretion in the elderly. Am J Med. 1997;103:270–3.
65. Laczi F. Etiology, diagnostics and therapy of hyponatremias. Orv Hetil. 2008;149(29):1347–54.
66. Snyder NA, Feigal DW, Arieff AI. Hypernatremia in elderly patients: a heterogeneous, morbid, and iatrogenic entity. Ann Intern Med. 1987;107:309–19.
67. Leung AA, McAlister FA, Finlayson SR, Bates DW. Preoperative hypernatremia predicts increased perioperative morbidity and mortality. Am J Med. 2013;126(10):877–86.
68. Mc Causland FR, Wright J, Waikar SS. Association of serum sodium with morbidity and mortality in hospitalized patients undergoing major orthopedic surgery. J Hosp Med. 2014;9(5):297–302.
69. Chassagne P, Druesne L, Capet C, et al. Clinical presentation of hypernatremia in elderly patients: a case control study. J Am Geriatr Soc. 2006;54(8):1225–30.
70. Wallace RB, Wactawski-Wende J, O'Sullivan MJ, et al. Urinary tract stone occurrence in the Women's Health Initiative (WHI) randomized clinical trial of calcium and vitamin D supplements. Am J Clin Nutr. 2011;94(1):270–7.
71. Bolland MJ, Avenell A, Baron JA, et al. Effect of calcium supplements on risk of myocardial infarction, cardiovascular events: meta-analysis. BMJ. 2010;341:c3691.
72. Bolland MJ, Grey A, Avenell A, Gamble GD, Reid IR. Calcium supplements with or without vitamin D and risk of cardiovascular events: reanalysis of the Women's Health Initiative limited access dataset and meta-analysis. BMJ. 2011;342:d2040.

73. Portale AA, Lonergan ET, Tanney DM, Halloran BP. Aging alters calcium regulation of serum concentration of parathyroid hormone in healthy men. Am J Phys. 1997;272:E139–46.
74. Garcia Lazaro M, Montero Perez-Barquero M, Carpintero BP. The role of malnutri-tion and other medical factors in the evolution of patients with hip fracture. An Med Interna. 2004;21(11):557–63.
75. Lichtblau S. Treatment of hip fractures in the elderly – the decision process. Mt Sinai J Med. 2002;69(4):250–60.
76. Huddleston JM, Whitford KJ. Medical care of elderly patients with hip fractures. Mayo Clin Proc. 2001;76(3):295–8.
77. Lavernia CJ, Sierra RJ, Baerga L. Nutritional parameters and short term outcome in arthro-plasty. J Am Coll Nutr. 1999;18(3):274–8.
78. Koval KJ, Maurer SG, ET S, et al. The effects of nutritional status on outcome after hip fracture. J Orthop Trauma. 1999;13(3):164–9.
79. Bastow MD, Rawlings J, Allison SP. Benefits of supplementary tube feeding after fractured neck of femur: a randomised controlled trial. BMJ. 1983;287:1589–92.
80. Hartgrink HH, Wille J, Konig P, Hermans J, Breslau PJ. Pressure sores and tube feeding in patients with a fracture of the hip: a randomized clinical trial. Clin Nutr. 1998;17:287–92.
81. Sullivan DH, Nelson CL, Bopp MM, Puskarich-May CL, Walls RC. Nightly enteral nutrition support of elderly hip fracture patients: a phase I trial. J Am Coll Nutr. 1998;17:155–61.
82. Tidermark J, Ponzer S, Carlsson P, et al. Effects of protein-rich supplementation and nandrolone in lean elderly women with femoral neck fractures. Clin Nutr. 2004;23(4):587–96.
83. Avenell A, Handoll HH. Nutritional supplementation for hip fracture aftercare in older people. Cochrane Database Syst Rev. 2006;4:CD001880.
84. Kaye KS, Anderson DJ, Sloane R, Chen LF, Choi Y, Link K, Sexton DJ, et al. The effect of surgical site infection on older operative patients. J Am Geriatr Soc. 2009;57:46–54.
85. Southwell-Keely JP, Russo RR, March L, Cumming R, Cameron I, Brnabic AJ. Antibiotic prophylaxis in hip fracture surgery: a metaanalysis. Clin Orthop. 2004;419:179–84.
86. Gillespie WJ, Walenkamp G. Antibiotic prophylaxis for surgery for proximal femoral and other closed long bone fractures. Cochrane Database Syst Rev. 2001;1:CD000244.
87. Bratzler DW, et al. Clinical practice guidelines for antimicrobial prophylaxis in surgery. Am J Health Syst Pharm. 2013;70(3):195–283.
88. Arozullah AM, Khuri SF, Henderson WG, et al. Development and validation of a multifactorial risk index for predicting postoperative pneumonia after major noncardiac surgery. Ann Intern Med. 2001;135(10):847–57.
89. Montravers P, Veber B, Auboyer C, Dupont H, Gauzit R, Korinek AM, Malledant Y, Martin C, Moine P, Pourriat JL. Diagnostic and therapeutic management of nosocomial pneumonia in surgical patients: results of the Eole study. Crit Care Med. 2002;30(2):368.
90. Skelly JM, Guyatt GH, Kalbfleisch R, Singer J, Winter L. Management of urinary retention after surgical repair of hip fracture. Can Med Assoc J. 1992;146:1185–9.
91. Sigel A, Schrott KM. Disturbances of micturition after general surgical operations. Langenbecks Arch Chir. 1977;345:563–4.
92. Johansson I, Athlin E, Frykholm L, Bolinder H, Larsson G. Intermittent versus indwelling catheters for older patients with hip fractures. J Clin Nurs. 2002;11:651–6.
93. Smith NK, Albazzaz MKA. Prospective study of urinary retention and risk of death after proximal femoral fracture. Age Ageing. 1996;25:150–4.
94. Schaeffer AJ. Catheter-associated bacteriuria. Urol Clin North Am. 1986;13(4):735–47.
95. Sedor J, Mulholland SG. Hospital-acquired urinary tract infections associated with the indwelling catheter. Urol Clin North Am. 1999;26(4):821–8.
96. Nicolle LE. Asymptomatic bacteriuria: when to screen and when to treat. Infect Dis Clin N Am. 2003;17:367–94.
97. Stamm WE. Guidelines for prevention of catheter-associated urinary tract infections. Ann Intern Med. 1975;82(3):386–90.
98. Morrison RS, Chassin MR, Siu AL. The medical consultant's role in caring for patients with hip fracture. Ann Intern Med. 1998;128:1010–20.
99. NEW. Prevention and treatment of pressure ulcers: clinical practice guideline. 2014. http://www.npuap.org/resources/educational-and-clinical-resources/prevention-and-treatment-of-pressure-ulcers-clinical-practice-guideline. Last accessed on Sept 10 2015.
100. Allman RM, Goode PS, Burst N, Bartolucci AA, Thomas DR. Pressure ulcers, hospital complications, and disease severity: impact on hospital costs and length of stay. Adv Wound Care. 1999;12:22–30.
101. Baumgarten M, Margolis D, Berlin JA, et al. Risk factors for pressure ulcers among elderly hip fracture patients. Wound Repair Regen. 2003;11:96–103.
102. Abrussezze RS. Early assessment and prevention of pressure ulcers. In: Lee BY, editor. Chronic ulcers of the skin. New York: McGraw-Hill; 1985. p. 1–9.
103. Allman RM. Pressure ulcers among the elderly. N Engl J Med. 1989;320(13):850–3.
104. Fuhrer MJ, Garber SL, Rintala DH, et al. Pressure ulcers in community-resident persons with spinal cord injury: prevalence and risk factors. Arch Phys Med Rehabil. 1993;74(11):1172–7.

105. Garber SL, Campion LJ, Krouskop TA. Trochanteric pressure in spinal cord injury. Arch Phys Med Rehabil. 1982;63(11):549–52.
106. Reuler JB, Cooney TG. The pressure sore: pathophysiology and principles of management. Ann Intern Med. 1981;94(5):661–6.
107. Barbenel JC, Ferguson-Pell MW, Kennedy R. Mobility of elderly patients in bed. Measurement and association with patient condition. J Am Geriatr Soc. 1986;34(9):633–6.
108. Lowthian PT. Underpadsin the prevention of decubiti. In: Kenedi RM, Cowden JM, Scales JT, editors. Bedsore biomechanics. Baltimore: University Park Press; 1976. p. 141.
109. Berlowitz DR, Wilking SV. Risk factors for pressure sores. A comparison of cross-sectional and cohort-derived data. J Am Geriatr Soc. 1989;37(11):1043–50.
110. Bliss M, Simini B. When are the seeds of postoperative pressure sores sown? Often during surgery. BMJ. 1999;319:863.
111. Inouye SK, Studenski S, Tinetti ME, Kuchel GA. Geriatric syndromes: clinical, research, and policy implications of a core geriatric concept. J Am Geriatr Soc. 2007;55(5):780–91.
112. Haleem S, Heinert G, Paker MJ. Pressure sores and hip fractures. Injury. 2008;39(2):219–23.
113. Nuffield Institute of Health, University of Leeds, NHS Centre for Review and Dissemination. How effective are pressure relieving interventions for the prevention and treatment of pressure sores? Effective Health Care Bull. 1995;2:1–15.
114. Peromet M, Labbe M, Yourassowsky E, et al. Anaerobic bacteria isolated from decubitus ulcers. Infection. 1973;1(4):205–7.
115. Livesley NJ, Chow AW. Infected pressure ulcers in elderly individuals. Clin Infect Dis. 2002;35(11):1390–6.
116. Brown NK, Thompson DJ. Nontreatment of fever in extended-care facilities. N Engl J Med. 1979;300(22):1246–50.
117. Agency for Health Care Policy and Research (AHCPR). Treatment of pressure ulcers. Rockville: US Department of Health and Human Services, Public Health Service, Clinical Practice Guideline Number 15. AHCPR Publication No. 95–0652; 1994.
118. Pompei P, Murphy JB, editors. Geriatric review syllabus: a core curriculum in geriatric medicine. 6th ed. New York: American Geriatric Society; 2006.
119. Mumcuoglu KY. Clinical applications for maggots in wound care. Am J Clin Dermatol. 2001;4:219–27.
120. Bergstrom N, Bennett MA, Carlson CE, et al. Treatment of pressure ulcers. Clinical practice guideline no. 15. Rockville: US Department of Health and Human Services, Public Health Service, Agency for Health Care Policy and Research; 1994. p. 47–9.
121. Lyder CH, Preston J, Grady JN, et al. Quality of care for hospitalized Medicare patients at risk for pressure ulcers. Arch Intern Med. 2001;161(12):1549–54.
122. Bergstrom N, Horn SD, Smout RJ, Bender SA, Ferguson ML, Taler G, Sauer AC, Sharkey SS, Voss AC. The national pressure ulcer long-term care study: outcomes of pressure ulcer treatments in long-term care. J Am Geriatr Soc. 2005;53(10):1721.
123. Marcantonio ER, Flacker JM, Wright RJ, et al. Reducing delirium after hip fracture: a randomized trial. J Am Geriatr Soc. 2001;49(5):516–22.
124. Todd CJ, Freeman CJ, Camilleri-Ferrante C, et al. Differences in mortality after fracture of hip: the east Anglian audit. BMJ. 1995;310:904–8.
125. Anderson FA Jr, Wheeler HB, Goldberg RJ, et al. A population-based perspective of the hospital incidence and case-fatality rates of deep vein thrombosis and pulmonary embolism. The Worcester DVT study. Arch Intern Med. 1991;151:933–8.
126. Guyatt GH, Akl EA, Crowther M, Gutterman DD, Schuünemann HJ. Executive summary: antithrombotic therapy and prevention of thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. Chest. 2012;141(2_suppl):7S–47S.
127. Moser K. Pulmonary thromboembolism. In: Wilson J, Harrison TR, et al., editors. Harrison's principles of internal medicine, vol. 1. 12th ed. New York: McGraw-Hill; 1991. p. 1090.
128. Kniffin WD, Baron J, Barrett J, et al. The epidemiology of diagnosed pulmonary embolism and deep venous thrombosis in the elderly. Arch Intern Med. 1994;154:861–6.
129. Geerts WH, Heit JA, Clagett GP, et al. Prevention of venous thromboembolism. Chest. 2001;119:132S–1325S.
130. Morpurgo M, Schmid C, Mandelli V. Factors influencing the clinical diagnosis of pulmonary embolism: analysis of 229 postmortem cases. Int J Cardiol. 1998;65(Suppl I):S79–82.
131. Stein PD, Beemath A, Matta F, et al. Clinical characteristics of patients with acute pulmonary embolism: data from PIOPED II. Am J Med. 2007;120:871–9.
132. Wells PS, Ginsberg JS, Anderson DR, et al. Use of a clinical model for safe management of patients with suspected pulmonary embolism. Ann Intern Med. 1998;129:997–1005.
133. Schouten HJ, Geersing GJ, Oudega R, van Delden JJ, Moons KG, Koek HL. Accuracy of the wells clinical prediction rule for pulmonary embolism in older ambulatory adults. J Am Geriatr Soc. 2014;62(11):2136–41.
134. The PIOPED Investigators. Value of the ventilation/perfusion scan in acute pulmonary embolism: results of the prospective investigation of pulmonary embolism diagnosis (PIOPED). JAMA. 1990;263(20):2753–9.

135. Stein PD, Hull RD, Pineo GF. The role of newer diagnostic techniques in the diagnosis of pulmonary embolism. Curr Opin Pulm Med. 1999;5:212–5.
136. Lindeque B, Schoeman H, Dommisse G, Boeyens MC, Vlok AL. Fat embolism and the fat embolism syndrome. J Bone Joint Surg. 1987;69B:128–31.
137. Rodger M, Markropoulos D, et al. Diagnostic value of the electrocardiogram in suspected pulmonary embolism. Am J Cardiol. 2000;86(7):807–9.
138. Moores LK, Jackson WL, Shorr AF, Jackson JL. Meta-analysis: outcomes in patients with suspected pulmonary embolism managed with computed tomographic pulmonary angiography. Ann Intern Med. 2007;141:866–74.
139. Egermayer P, Town GI, Turner JG, et al. Usefulness of D-dimer, blood gas, and respiratory rate measurements for excluding pulmonary embolism. Thorax. 1998;53:830–4.
140. Tapson VF, Carroll BA, Davidson BL, et al. American Thoracic Society. Clinical practice guideline: the diagnostic approach to acute venous thromboembolism. Am J Respir Crit Care Med. 1999;160:1043–66.
141. The Task Force on Pulmonary Embolism, European Society of Cardiology. Guidelines on the diagnosis and management of acute pulmonary embolism. Eur Heart J. 2000;21:1301–36.
142. Ginsberg JS, Wells PS, Kearon C, et al. Sensitivity and specificity of a rapid whole-blood assay for D-dimer in the diagnosis of pulmonary embolism. Ann Intern Med. 1998;129(12):1006–11.
143. EINSTEIN–PE Investigators, et al. Oral rivaroxaban for the treatment of symptomatic pulmonary embolism. N Engl J Med. 2012;366(14):1287–97.
144. Agnelli G, Buller HR, Cohen A, et al. Oral apixaban for the treatment of acute venous thromboembolism. N Engl J Med. 2013;369(9):799–808.
145. Hokusai-VTE Investigators, Büller HR, Décousus H, et al. Edoxaban versus warfarin for the treatment of symptomatic venous thromboembolism. N Engl J Med. 2013;369(15):1406–15.
146. American Geriatrics Society 2012 Beers Criteria Update Expert Panel. American Geriatrics Society updated beers criteria for potentially inappropriate medication use in older adults. J Am Geriatr Soc. 2012;60(4):616–31.
147. Kearon C, et al. Antithrombotic therapy for VTE disease: antithrombotic therapy and prevention of thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. Chest. 2012;141(2 Suppl):e419S.
148. Mageral F, Tscherne H. Zur Diagnose, Therapie und proplylaxe der fettembolie. Langenbecks Arch Klin Chir. 1966;314:29.
149. Ganong RB. Fat emboli syndrome in isolated fractures of the tibia and femur. Clin Orthop. 1993;291:208–14.
150. Collins JA, Hudson TL, Hamacher WR, et al. Systemic fat embolism in four combat causalities. Ann Surg. 1968;167:493–9.
151. Johnson MJ, Lucas GL. Fat embolism syndrome. Orthopedics. 1996;19:41.
152. Fulde GW, Harrison P. Fat embolism – a review. Arch Emerg Med. 1991;8:233–9.
153. Kaplan RP, Grant JN, Kaufman AJ. Dermatologic features of the fat embolism syndrome. Cutis. 1986;38:52–5.
154. Gurd AR. Fat embolism: an aid to diagnosis. J Bone Joint Surg Br. 1970;52b:732–7.
155. Van den Brande FGJ, Hellemans S, De Schepper A, et al. Post-traumatic severe fat embolism syndrome with uncommon CT findings. Anaesth Intensive Care. 2006;34:102–6.
156. Riska EB, Van Bonsdorrf H, Hakkien S, Japonia O, Kilivoyo O, Papillainen T. Prevention of fat embolism by early fixation of fractures in patients with multiple injuries. Injury. 1976;8:110–6.
157. Schonfeld SA, Ploysongsang Y, DiLisio R, et al. Fat embolism prophylaxis with corticosteroids. Ann Intern Med. 1983;99:438–43.
158. Mellor A, Soni N. Fat embolism. Anaesthesia. 2001;56:145–54.
159. Bulger EM, Smith DG, Maier RV, Jurkovich GJ. Fat embolism syndrome. A 10-year review. Arch Surg. 1997;132(4):435–9.
160. Bitsch M, Foss N, Kristensen B, et al. Pathogenesis of and management strategies for post-operative delirium after hip fracture: a review. Acta Orthop Scand. 2004;75(4):378–89.
161. Bellelli G, Frisoni GB, Pagani M, et al. Does cognitive performance affect physical therapy regimen after hip fracture surgery? Aging Clin Exp Res. 2007;19(2):119–24.
162. Feil D, Marmon T, Unutzer J. Cognitive impairment, chronic medical illness, and risk of mortality in an elderly cohort. Am J Geriatr Psychiatry. 2003;11(5):551–60.
163. Kiely DK, Bergmann MA, Murphy KM, et al. Delirium among newly admitted post-acute facility patients: prevalence, symptoms, and severity. J Gerontol A Biol Sci Med Sci. 2003;58(5):M441–5.
164. Ely EW, Inouye SK, Bernard GR, et al. Delirium in mechanically ventilated patients: validity and reliability of the confusion assessment method for the intensive care unit. JAMA. 2001;286(21):2703–10.
165. Inouye SK, Van Dyck CH, Alessi CA, et al. Clarifying confusion: the confusion assessment method: a new method for detection of delirium. Ann Intern Med. 1990;113(12):941–8.
166. Trzepacz P, Breitbart W, Franklin J, et al. Practice guideline for the treatment of patients with delirium. American Psychiatric Association. Available at: http://www.psychiatryonline.com/pracGuide/pracGuideTopic_2.aspx