

Skin and Soft Tissue Infections in Older Adults

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Skin and soft tissue infections (SSTIs) are common in the general population and in older persons. Because of changes in skin consistency, immunosenescence, and the presence of underlying skin conditions and comorbid conditions, elderly persons are at high risk for SSTIs. This article discusses SSTIs acquired frequently in the community, such as cellulitis, necrotizing fasciitis, and carbunculosis, and infections that frequently are health care associated, such as pressure ulcers and surgical site infections (SSIs).

Infections in the community

Cellulitis and erysipelas

Overview, pathophysiology, and microbiology

Cellulitis is a common bacterial infectious disease of the skin and is managed by health care providers in various diverse fields. Cellulitis occurs frequently in long-term care facilities, affecting 1% to 9% of residents [1]. Cellulitis is caused by bacteria that breach the skin and sometimes involve the subcutaneous tissue. Thus, peripheral lymphedema, venous stasis, and the presence of tinea pedis are important in the pathogenesis of some cases.

Without the presence of underlying abscess or deeper infectious foci, the bacterial burden in the skin is low. Bacterial exotoxins play a prominent role in the inflammation and infectious symptoms associated with cellulites [2].

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The majority of cellulitis cases are caused by gram-positive organisms, most commonly *Streptococci* (group A most commonly and also groups B, C, and G). *Staphylococcus aureus* is another notable cause of cellulitis. Other pathogens do not cause cellulitis routinely but can in specific circumstances: *Pseudomonas aeruginosa* after puncture wounds through a sneaker, *Haemophilus influenzae* type b in children, *Pasteurella* spp after a cat bite or scratch, *Capnocytophaga canimorsus* after dog bite, *Eikenella corrodens* after a human bite, and *Aeromonas hydrophila* and *Vibrio* spp after trauma and water exposure.

Erysipelas is an infection involving the upper dermis that often is characterized by a raised rash with clearly demarcated borders. Erysipelas is caused most commonly by *Streptococcus pyogenes* (group A streptococcus) and *S aureus*. The presentation, diagnosis, and management for erysipelas are similar to those for other types of cellulitis.

Risk factors, presentation, and diagnosis

Knowledge of the clinical epidemiology of cellulitis almost exclusively comes from studies in the general population; the elderly have not been studied extensively as a group. Risk factors for cellulitis include conditions that compromise the integrity of the skin and associated host defenses, such as obesity, venous or lymph stasis, tinea pedis, recent trauma, and underlying skin conditions, such as eczema. Elderly patients have a high frequency of conditions that are associated with skin fragility, such as edema and trauma, that predispose them to cellulitis. For example, by age 70, approximately 70% of persons have at least one underlying skin problem [3]. Patients often present with rapidly spreading erythema, warmth, and edema. Occasionally, tenderness, lymphangitic streaking, and regional lymphadenopathy are present. Systemic symptoms occur less frequently and often are mild, including fever, tachycardia, hypotension, leukocytosis, and change in mental status. Cellulitis in the elderly, however, may present with atypical symptoms; fever often is low grade or absent and patients might present solely with changes in mental status or declining functional status [3].

The diagnosis of cellulitis usually is a clinical one. Blood cultures are positive in less than 5% of cases and cultures of soft tissue aspirate and punch biopsies generally are negative. Serologic tests sometimes are useful in culture-negative cases. Generally, cultures and serologies are not necessary to confirm a diagnosis of cellulitis, but these modalities might be considered if patients do not respond to standard antimicrobial therapy. The differential diagnosis for cellulitis includes deep venous thrombosis, herpes zoster, gout, and acute venous stasis dermatitis.

Treatment and outcomes

If patients are toxic, are septic, or have severe or rapidly spreading cellulitis, then initial therapy should be intravenous (IV) and patients should be hospitalized. In mild cases, antimicrobials can be delivered orally in an

outpatient setting. Antimicrobial therapy should include agents that are effective against streptococci and *S aureus*. Typical agents include penicillinase-resistant penicillins, first-generation cephalosporins, vancomycin, and clindamycin (the latter two agents typically are used for patients who have life-threatening penicillin allergies). If patients have a history of recent methicillin-resistant *S aureus* (MRSA) infection, ongoing IV drug abuse, or recent health care exposures, then initial therapy should include vancomycin, linezolid, daptomycin, or other agents that demonstrate in vitro activity against MRSA. If patients have purulent cellulitis, then community-acquired MRSA (CA-MRSA) should be considered a pathogen and should be treated with trimethoprim-sulfamethoxazole, vancomycin, clindamycin (if D-test negative), linezolid, or daptomycin. Typical courses of antimicrobial therapy range from 5 to 14 days, depending on severity of infection and clinical response [2]. If therapy is initiated IV, transition to oral therapy can be considered when patients' local and systemic symptoms improve clinically. Elevation of the affected area (eg, a lower extremity) helps to decrease edema by promoting lymph and venous drainage and can accelerate the time from treatment initiation to cure [2].

Prevention

Limiting the severity of edema through medications (such as diuretics) and medical stockings and by elevating affected extremities can prevent cellulitis. Treating macerated feet with topical antifungals also can prevent recurrent cellulitis. For patients who have multiple recurrent episodes of cellulitis, prophylactic antibiotics can be considered. Antibiotics typically used for prophylaxis include penicillin and erythromycin [2].

Necrotizing fasciitis

Overview, pathophysiology, and microbiology

Necrotizing fasciitis is a rare but severe infection of the subcutaneous tissue that tracks along the fascial layers, destroying the fascia (usually the superficial fascia). Necrotizing fasciitis usually develops after a superficial injury. The initial injury might be mild (such as insect bite, abrasion, or cut) and, in approximately 20% of cases, no primary lesion is identified [2].

There are two types of necrotizing fasciitis. Type 1 is a polymicrobial infection and usually follows a surgical procedure. Infection occurs most often in the lower extremities, abdominal wall, groin, or perianal region. Subsets of type 1 necrotizing fasciitis include Fournier's gangrene and cervical necrotizing fasciitis. Pathogens typically arise from the bowel and include a mix of aerobic and anaerobic gram-positive and gram-negative bacteria. Organisms commonly described as pathogens in type 1 infection, including those occurring in the elderly, are coliform bacteria, such as *E coli*, *Klebsiella pneumoniae*, and *P aeruginosa*, and anaerobes [4]. Type 2 infection is a monomicrobial infection. Most commonly, type 2 infection is caused by *S pyogenes*

(group A streptococcus) and represents a variant of toxic shock syndrome. Other pathogens that can cause type 2 necrotizing fasciitis include *S agalactiae* (group B streptococcus; particularly in those who have diabetes mellitus), *S aureus*, *V vulnificans*, *A hydrophila*, and anaerobic streptococci. Toxin production plays an important role in the pathophysiology of disease.

Risk factors, presentation, and diagnosis

Predisposing factors for type 1 necrotizing fasciitis include surgical procedures (typically involving the bowel or bladder), decubiti or pressure ulcers, perianal abscess, IV drug abuse, and presence of a Bartholin abscess or other vulvovaginal infection [2]. Frequently, patients have a history of diabetes mellitus. Type 2 necrotizing fasciitis usually occurs in the lower extremities. Predisposing factors for type 2 infection include diabetes mellitus, peripheral vascular disease (PVD), zoster, blunt trauma, IV drug abuse, exposure to another case of type 2 necrotizing fasciitis, childbirth, and surgery.

It may be difficult to diagnose necrotizing fasciitis when patients first present for medical evaluation. Local symptoms might be mild and unimpressive. Frequently, the affected area is extremely painful and the pain, toxicity, and discomfort are out of proportion to physical examination findings. An overlying cellulitis, typified by erythema, edema, and warmth, often can be identified. After the first few days of illness, the skin changes evolve to dark, reddish-purple lesions, then to blisters and bullae. Systemic toxicity is inevitable and usually prominent with fever, tachycardia, and sometimes hypotension present [2].

Clinical suspicion is necessary to diagnosis necrotizing fasciitis. If patients seem toxic or septic, if patients are not responding to antibiotics, or if patients are deteriorating despite appropriate antimicrobial therapy, necrotizing fasciitis must be considered. CT and MRI of affected areas often provide nonspecific results that might not be diagnostic. Leukocytosis often is present and blood cultures usually are positive. Direct observation of the fascial planes and subcutaneous tissue is critical for diagnosis and usually reveals swelling, dullness, and gray discoloration of the fascia. Often, a brownish exudate is present. Gram's stain and culture of affected tissue often demonstrates the pathogens (often multiple in type 1 and a single pathogen in type 2). Aspiration of fluid at the leading edge of the lesion can also provide good material for Gram's stain analysis and culture.

Treatment and outcomes

The hallmark of treatment is surgical intervention. Surgical drainage and exploration is warranted if local wounds demonstrate necrosis or easy dissection along fascial planes or if a soft tissue infection is accompanied by the presence of gas. Typically, patients have to return to an operating room frequently for additional débridement. Antimicrobial therapy is a useful adjuvant to surgical débridement and should be directed against suspected pathogens. For the mixed, type 1 infection, recommended

therapies include (1) a β -lactam/ β -lactamase inhibitor agent (eg, piperacillin-tazobactam) plus clindamycin plus ciprofloxacin; (2) carbapenem monotherapy; (3) cefotaxime plus metronidazole or clindamycin; or (4) clindamycin or metronidazole plus aminoglycoside or fluoroquinolone. For type 2 streptococcal infection, an important component of initial therapy is clindamycin, which helps to decrease toxin production and modulate cytokine production. Initial therapeutic regimens include (1) penicillin plus clindamycin; (2) vancomycin; (3) linezolid; (4) quinupristin/dalfopristin; or (5) daptomycin. For type 2 *S aureus* infection, therapeutic options include nafcillin, cefazolin, vancomycin, and clindamycin. For clostridial infection, clindamycin and penicillin are therapeutic options [2].

Furuncles, carbuncles, and boils

Overview, pathophysiology, and microbiology

A furuncle, or boil, is an infection of the hair follicle. Typically, a furuncle involves an inflammatory nodule overlying a pustule. When infection involves several adjacent follicles, a coalescent purulent mass or carbuncle forms. In adults, the most common pathogen causing furuncles and carbuncles is *S aureus*. Classically, among patients who do not have health care contact or other risk factors for MRSA, methicillin-susceptible *S aureus* has been the most common type of *S aureus* to cause infection. Recently, however, CA-MRSA has become a common cause of carbunculosis in many parts of the United States. In addition to causing endemic carbunculosis, CA-MRSA also is associated with outbreaks in diverse, previously healthy populations, including children, families, athletes, military recruits, and prisoners.

Risk factors, presentation, and diagnosis

Risk factors for infection include inadequate personal hygiene, exposure to other cases of carbunculosis, and skin injury. Fomites can harbor pathogens causing carbunculosis and can facilitate spread in families and in other populations. For example, among athletes, body shaving, sharing of razors and other personal equipment, and sports equipment itself (eg, wrestling mats) are important risk factors for infection. One might speculate that institutions with common equipment, such as rehabilitation facilities, might place older adults at similar risk, but no data have been generated specifically to answer this question. CA-MRSA seems to have a predilection for infecting younger individuals and occurs less frequently in persons 65 or older, but the reasons for this are unclear [5,6].

Treatment and outcomes

Drainage of purulent lesions is critical to cure carbunculosis. For small lesions, application of moist heat to promote drainage might be all that is required. For larger lesions, however, incision and drainage often are

necessary. If carbuncles are small (eg, <5 cm) and not associated with cellulitis or systemic infection, then systemic antibiotics might not be needed [7]. If lesions are large or there is associated cellulitis, however, then antibiotics should be prescribed in addition to incision and drainage.

Prevention

If exposed to individuals who have carbuncles, practicing good hygiene, bathing with antibacterial soaps, and not sharing personal items all can help prevent the spread of boils, in particular those resulting from CA-MRSA. If individuals have repeated attacks of carbunculosis, abnormal host immune response should be ruled out as a predisposing cause. Attempts can be made to decolonize individuals. One approach involves applying mupirocin to the anterior nares twice daily for 5 days. This approach is reported to reduce recurrences by 50% [8]. This regimen often is conducted in conjunction with daily chlorhexidine gluconate showers for 5 days. This regimen should be used judiciously, however, as investigators report the rapid emergence of resistance of MRSA to mupirocin [9]. For recurrent attacks despite these approaches, some investigators report limited success with long courses of low-dose antibiotics, including rifampin, trimethoprim-sulfamethoxazole, or clindamycin [2]. If providers want to decolonize patients using long courses of systemic antibiotics, consultation with an infectious diseases expert should be considered.

Health care–associated skin and soft tissue infections

Decubiti or pressure ulcers

Overview, pathophysiology, and microbiology

Pressure ulcers are common yet often preventable and often occur in high-risk populations, such as patients who have physical impairments or older persons. In fact, more than two thirds of decubiti occur in persons older than 70 years of age. The incidence rate for pressure ulcers ranges from 2% to 24% in long-term care settings and 0.4% to 38% in acute care settings [10]. The majority of pressure ulcers occurring in hospitals develop during the initial 5 days of hospitalization. An estimated 2.5 million pressure ulcers are treated annually in the United States. In addition to having an adverse impact on patients clinically, pressure ulcers prolong duration of hospitalization and lead to excess health care costs.

Pressure ulcers occur when prolonged pressure and tissue compression cause local ischemia and the accumulation of toxic metabolites and cell death, eventually leading to ulceration and necrosis [11]. For example, excessive pressure on the heels of patients on an operating room table can lead to necrosis if the duration of pressure exceeds 2 hours. Moreover, ulcers might be quite small initially, but with continued pressure and ischemia, ulcers rapidly can get larger, deeper, and sometimes infected. Infectious complications of pressure ulcers include cellulitis, osteomyelitis, and

bacteremia. Factors that increase susceptibility for developing pressure ulcers include external and host factors. External factors include pressure, friction, shear force, and moisture; host factors include malnutrition, anemia, and vascular disease.

Most pressure ulcer infections are polymicrobial. Pathogens isolated frequently include *Staphylococci*, *Enterococci*, Enterobacteriaceae, and *Pseudomonas* spp. Anaerobic bacteria, such as *Bacteroides fragilis*, *Peptostreptococcus*, and *Clostridium* spp, also are common pathogens.

Risk factors, presentation, diagnosis, and classification

The most common sites of pressure ulcers are the sacrum and hips (67%), but other sites, such as the occiput, elbows, lower extremities, and heels, also are affected commonly. Populations at increased risk for pressure ulcers include persons who are older, incontinent, unconscious, or paralyzed. As a result of immobilization, the postoperative period is an important risk period for the development of pressure ulcers. Several comorbid conditions also are associated with pressure ulcers, including contractures, spasticity, PVD, diabetes mellitus, and autonomic regulatory dysfunction. Furthermore, medications that cause immobility and devices that cause excessive heat can predispose patients to pressure ulcers. In general, patients who develop pressure ulcers usually have impaired mobility, mental status, and sensation [10,11].

Most pressure ulcers are diagnosed when they are observed directly by health care providers, nursing home staff, or family members. Sometimes, patients present with systemic signs of infection, such as fever, bacteremia, or declining cognitive status.

The system used most commonly for classifying pressure ulcers is the National Pressure Ulcer Advisory Panel. This system has four classification stages based on the depth of the ulcer. Stage I represents intact skin with early signs of impending ulceration including erythema, warmth, and induration. Stage II lesions present as shallow ulcers, involving the epidermis and often the dermis. Often, pigmentation changes are present. Stage III ulcers involve a full-thickness loss of skin with extension into the subcutaneous tissue but sparing of the fascia and may present as foul-smelling ulcerative lesions with pigmentation changes. Stage IV ulcers present with complete, full-thickness skin and subcutaneous tissue loss. There usually is ulcer penetration into the deep fascia and involvement of the muscle, tendon, joint capsule, or bone [11]. When the bone is involved, osteomyelitis invariably is present.

Treatment and outcomes

The general treatment of pressure ulcers is based on four modalities: pressure reduction, surgical intervention, nutrition, and wound management. Empiric antibiotic regimens should be focused on common pathogens, include *Staphylococci*, *Pseudomonas*, Enterobacteriaceae, and anaerobes

when pressure ulcers are complicated by infection. Culture data can be helpful in guiding therapy, particularly if specimens are obtained from the blood or from operative tissue. In general, swab cultures do not provide useful information. Typically, 10- to 14-day antibiotic regimens are prescribed for the treatment of infected ulcers, although no studies have studied antibiotic duration systematically [12]. Infected stage III and stage IV ulcers and osteomyelitis usually require longer durations of treatment (eg, 4–6 weeks), and surgical débridement, often in conjunction with flap placement, frequently is required for definitive cure [11]. If definitive surgical cure cannot be attained, then patients frequently have infectious relapses. The role of chronic, suppressive antibiotics in the management of osteomyelitis complicating stage IV ulcers remains unclear. Frequently, providers treat acute infectious flares rather than chronically suppressing patients with systemic antibiotics.

Prevention

Several modalities exist for the prevention of pressure ulcers. Detailed review of recommended preventive practices is beyond the scope of this article. The basic modalities for prevention include using support surfaces, repositioning patients routinely, optimizing nutritional status, and maintaining moist sacral skin [10].

Surgical site infections

Epidemiology of surgical site infections in elderly

SSIs are a growing threat to the health of the aging population. SSIs are a common complication of hospitalization, occurring in 2% to 5% of all patients undergoing surgery in the United States [13]. Given the high number of surgical procedures performed in the United States, this translates into 300,000 to 500,000 SSIs each year [14]. From 1980 to 1998, the percentage of operations performed for patients ages 65 years and older increased from 19% to 43% of all surgical operations [15]. SSIs account for 11% of nosocomial infections in patients ages 65 years and older [16], and as the population of older persons increases over time, the number of SSIs in this population likely will increase. Many aspects of SSIs are similar in the elderly population and in the general population, including pathophysiology, risk factors, and associated poor outcomes. Several key differences and important similarities are reviewed here.

SSIs remain a leading cause of morbidity and mortality in all populations, leading to increased length of hospitalization [14], costs [17], and mortality [18]. Overall, SSI is believed to account for up to \$10 billion in health care expenditure annually [19] and 77% of deaths in patients who have SSIs are attributed directly to the SSI [20]. Outcomes for elderly patients are reported to be even worse. Compared with uninfected control patients ages 65 years and older, elderly patients who have SSI have 4 to 5 times higher mortality, longer length of hospitalization, and at least twofold greater

hospital costs [21,22]. Elderly patients who have infection have worse outcomes compared with younger infected patients [23], and this holds true also with SSI. Compared with younger patients who have SSI, elderly patients who have SSI are 3 times more likely to die, have 4 more days of hospitalization, and have more than \$40,000 extra attributable costs [22].

Definition and pathophysiology

The CDC has developed standardized surveillance criteria for defining SSIs that are used widely [24]; SSIs are classified as incisional or organ/space (Fig. 1). Incisional SSIs are classified further into superficial (involving only skin or subcutaneous tissue of the incision) or deep (involving fascia or muscular layers). Organ/space SSIs include infections occurring in any part of the body opened or manipulated during surgery. Definitions used for standardized surveillance criteria are described in Box 1.

The risk for developing a SSI is a balance between microbial contamination of the surgical wound and host immunity. Microbial contamination of surgical sites is universal. The period of greatest risk for infection is from the time of incision to the time of wound closure [19]. Pathogens that lead to SSI are acquired from a patient's endogenous flora or exogenously from an operating room environment. There is no evidence to suggest that wound

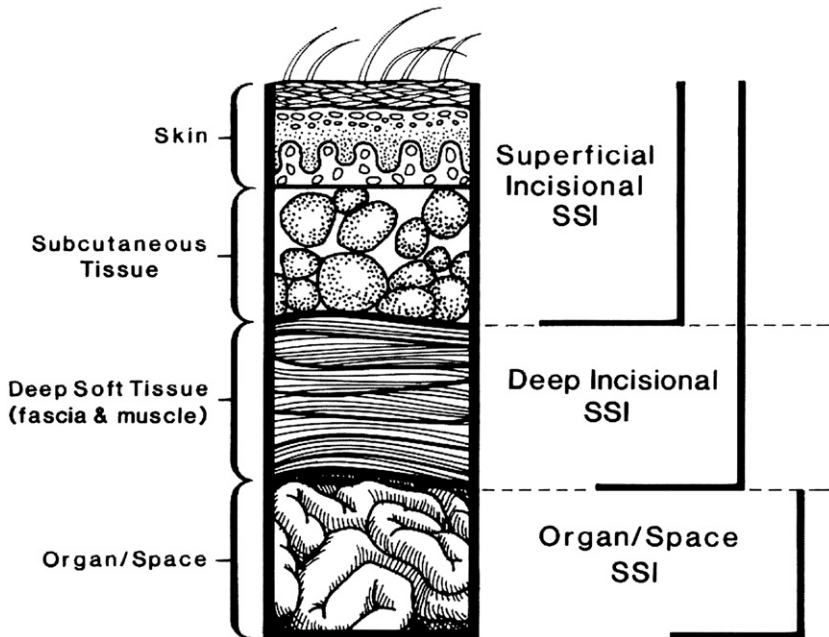


Fig. 1. Classification of SSIs. (From Horan TC, Gaynes RP, Martone WJ, et al. CDC definitions of nosocomial surgical site infections, 1992: a modification of CDC definitions of surgical wound infections. *Infect Control Hosp Epidemiol* 1992;13(10):606-8; with permission.)

Box 1. Criteria for defining a surgical site infection*

Incisional surgical site infection

Superficial infection involves skin or subcutaneous tissue of the incision and at least one of the following:

1. Purulent drainage, with or without laboratory confirmation, from the superficial incision
2. Organisms isolated from an aseptically obtained culture from the superficial incision
3. At least one of the following signs or symptoms: pain, localized swelling, erythema, or heat; *and* superficial incision is opened deliberately by surgeon, unless incision is culture negative.
4. Diagnosis of superficial incisional SSI by surgeon

Deep infection involves deep soft tissues (eg, fascial and muscle layers) of the incision and at least one of the following:

1. Purulent drainage from the deep incision, excluding organ/space^a
2. A deep incision that dehisces spontaneously or is opened deliberately by a surgeon when a patient has one or more of the following signs or symptoms: fever (>38°C) or localized pain, unless site is culture negative.
3. An abscess or other evidence of infection is found on direct examination, during repeat surgery, or by histopathologic or radiologic examination
4. Diagnosis of a deep incisional SSI by surgeon

* For all classifications, infection is defined as occurring within 30 days after the operation if no implant is placed or within 1 year if an implant is in place and the infection is related to the incision. For the sake of this classification, the CDC defines "implant" as a nonhuman-derived implantable foreign body (eg, prosthetic heart valve, nonhuman vascular graft, mechanical heart, or joint prosthesis) that is placed permanently in a patient.

^a Report infection that involves both superficial and deep incision sites as a deep incisional SSI.

From Horan TC, Gaynes RP, Martone WJ, et al. CDC definitions of nosocomial surgical site infections, 1992: a modification of CDC definitions of surgical wound infections. *Infect Control Hosp Epidemiol* 1992;13(10):606-8; with permission.

contamination is more frequent or more concentrated in patients age 65 years or older than in younger patients.

Gram-positive cocci, such as *Staphylococci*, from endogenous host flora located at or near an operative site, remain the leading cause of SSIs (Box 2)

Box 2. Ten most common pathogens in surgical site infections

Pathogen	Percent of infections
<i>S aureus</i>	20
Coagulase-negative staphylococci	14
<i>Enterococcus</i> spp	12
<i>P aeruginosa</i>	8
<i>Escherichia coli</i>	8
<i>Enterobacter</i> spp	7
<i>Proteus mirabilis</i>	3
<i>Streptococcus</i> spp	3
<i>K pneumoniae</i>	3
<i>Candida albicans</i>	2

Data from Mangram AJ, Horan TC, Pearson ML, et al. Guideline for prevention of surgical site infection, 1999. Hospital Infection Control Practices Advisory Committee. *Infect Control Hosp Epidemiol* 1999;20(4):250–78 [quiz: 279–80]; and National Nosocomial Infections Surveillance (NNIS) report, data summary from October 1986–April 1996, issued May 1996. A report from the National Nosocomial Infections Surveillance (NNIS) System. *Am J Infect Control* 1996; 24(5):380–8.

[20,25]. For surgeries involving the abdomen or genitourinary tract, gram-negative pathogens and anaerobes also are important pathogens. Pathogens that cause SSIs are similar in elderly and younger populations [26]. Modern methods of pre- and perioperative antisepsis can reduce but not eliminate the endogenous skin flora of surgical patients; 20% of bacterial skin flora reside in skin appendages, such as sebaceous glands, hair follicles, and sweat glands [27], and, thus, are difficult to eradicate completely. Rarely, inoculation of a surgical site with endogenous flora from remote sites of a patient may occur [28].

Exogenous sources of contamination, including surgical personnel, operating room environment, and surgical instruments, occasionally are implicated in SSIs. Infections resulting from exogenous sources most commonly occur sporadically, but several point source outbreaks are documented [29,30]. Finally, postsurgical inoculation of a surgical site secondary to a remote focus of infection, such as urinary tract infection or pneumonia, occurs rarely [31].

Risk factors for surgical site infection

Many risk factors for SSI are elucidated for the general surgical population (Box 3). Surprisingly few studies have examined specific risk factors specifically for patients age 65 years or older.

Box 3. Risk factors for surgical site infections

Perioperative characteristics

Age
 Diabetes mellitus
 and hyperglycemia
 Tobacco use
 Obesity
 Malnutrition
 Immunosuppression
 (steroids, HIV)
 Prolonged hospitalization
 Colonization with *S aureus*

Operative characteristics

Appropriate patient
 skin preparation
 Appropriate hair removal
 Surgical team
 Appropriate
 surgical scrub
 Operating room traffic
 Surgical technique

Procedural

Appropriate
 antimicrobial
 prophylaxis
 Hypothermia
 Oxygenation

From Mangram AJ, Horan TC, Pearson ML, et al. Guideline for prevention of surgical site infection, 1999. Hospital Infection Control Practices Advisory Committee. *Infect Control Hosp Epidemiol* 1999;20(4):250–78 [quiz: 279–80]; with permission.

Age is a complex but immutable risk factor for SSIs. Different groups of investigators report contradictory results concerning the relationship between increasing age and risk for SSIs. For example, several investigators conclude that increasing age is associated with a greater risk for all types for postoperative infections, including SSIs [32–34]. Some investigators speculate that factors indirectly related to age, such as increased prevalence of comorbid conditions, increased severity of acute illness, and decreased host response to bacterial invasion in older patients, are the reasons why older patients might have an increased risk for SSI [35,36]. In other studies, advanced age is associated with a decreased risk for SSI [33,37]. In a recent large cohort study of more than 144,000 surgical procedures, increasing age independently predicted an increased risk for deep and organ space SSI until age 65, but at ages 65 years and older, increasing age independently predicted a linear decrease in the risk for SSI [38]. The explanation for this finding of decreased risk after age 65 is unclear, and may be because of either selection bias (ie, frail elderly patients might be less likely to have surgical procedures) or a “hardy survivor” effect.

Many diseases and risk factors for SSI occur with increased frequency in older patients. Diabetes mellitus, which leads to 2 to 5 times higher rates of SSI than in patients who are not diabetic [39], is more prevalent with age.

Similarly, elderly patients have higher prevalence of PVD and resulting tissue ischemia. As with smoking, decreased tissue oxygenation increases the risk for wound infection and dehiscence by decreasing collagen synthesis [40] and affecting the oxidative killing mechanisms of host neutrophils [41]. Furthermore, advanced age is associated with poor nutritional intake, malnutrition, and hypoalbuminemia, known risk factors for SSI [42]. Studies concerning the usefulness of pre-, peri-, or postoperative total parenteral nutrition or total enteral nutrition for preventing postoperative complications and SSI, however, have provided inconsistent and generally unfavorable results [43–45].

Few specific risk factors for patients ages 65 years and older are described. One recent case-control study of 1158 patients ages 65 years and older undergoing surgical procedures shows that obesity (odds ratio [OR] 1.8) and chronic obstructive pulmonary disease (OR 1.7) were independent risk factors for SSI among elderly patients; elderly patients who had private insurance, perhaps a marker for higher socioeconomic status, had lower risk for SSI (OR 0.3) [46]. The investigators of another case-control study of 340 patients ages 65 years and older undergoing orthopedic surgery conclude that patients admitted from a health care facility (nursing home, outside hospital, or rehabilitation facility) were more than 4 times more likely to develop a SSI than patients admitted from home [21].

Although many other factors contribute to the risk for SSI, the burden of surgical wound inoculation remains one of the most well understood and accepted risks. That is, the higher the amount of surgical wound contamination, the higher the risk for infection. Even in the setting of appropriate antimicrobial prophylaxis, the risk for SSI increases as total bacterial burden of the surgical wound increases [47]. Generally, wound contamination with greater than 10^5 microorganisms is required to lead to SSI [48]. When foreign bodies are present, the inoculum may be much lower. When sutures are present, the required inoculum of organisms is decreased by 99.99% (from 10^6 to 10^2 organisms) [49]—as few as 10 colony-forming units of bacteria with polytetrafluoroethylene vascular grafts [50] or 1 colony-forming unit of bacteria with dextran beads is necessary to potentially cause SSI [51]. These findings are important particularly as orthopedic and vascular procedures with implants are common for patients age 65 years or older [15].

Prevention of surgical site infection in patients age 65 years or older

No studies have been performed to determine methods of preventing SSIs specifically in patients age 65 years or older. Thus, in addition to paying close attention to comorbid conditions (discussed previously) and encouraging glucose control (for patients who have diabetes mellitus) and cessation of tobacco use, standard techniques used in the general surgical population must be applied rigorously to elderly surgical patients. Several proved modalities to prevent SSIs in the general population exist and are incorporated into national quality improvement initiatives.

The Institute for Healthcare Improvement's 100,000 Lives Campaign, a nationwide campaign to improve patient outcomes by preventing medical errors, included prevention of SSI as one of their six major areas of focus [52]. Four specific interventions to prevent SSI have been targeted: appropriate selection, timing, and discontinuation of prophylactic antimicrobial agents; appropriate hair removal; postoperative glucose control; and maintaining postoperative normothermia.

The appropriate use of perioperative antimicrobial prophylaxis is accepted as a well-proved intervention to reduce the risk for SSI in elective procedures [20]. The Centers for Medicare and Medicaid Services created the Surgical Infection Prevention Project in 2002 to decrease the morbidity and mortality associated with postoperative SSI by promoting appropriate selection and timing of prophylactic antimicrobials. An expert panel identified proved performance measures for quality improvement: IV antimicrobial prophylaxis within 1 hour before incision (2 hours are allowed for the administration of vancomycin and fluoroquinolones) [53]; antimicrobial prophylactic agent consistent with guidelines [54]; and discontinued prophylactic antimicrobial agent within 24 hours after surgery end time. The Surgical Infection Prevention Project focuses on several procedures important for older populations: hip arthroplasty, knee arthroplasty, cardiothoracic surgery, vascular surgery, and colorectal surgery. When used together, these performance measures lead to decreased rates of SSI. A national collaborative of 56 hospitals participated in implementing these performance measures and, over a 1-year period, reported a mean reduction in the rate of SSI of 27% [55].

The use of razors for hair removal or hair removal the night before surgery leads to higher rates of SSI [56,57]. If hair removal is necessary, clippers or a depilatory method should be used on the day of surgery.

As discussed previously, diabetes and hyperglycemia are established as independent risk factors for SSI. In fact, elevated serum glucose in the pre- and postoperative periods are associated with increased risk for SSI [39,58]. Aggressive glycemic control, including postoperative IV insulin, can reduce the rate of SSI and the rate of death while in an intensive care unit [59]. A study of 1585 patients who had diabetes and underwent open heart surgery showed that aggressive postoperative glucose control with continuous IV insulin infusion reduced the rate of SSI from 2.4% to 1.5% ($P < .02$) [39].

Surgical patients may become hypothermic, defined as a core body temperature below 36°C, from exposure to cold operating room ambient temperatures, anesthesia, or changes in body heat distribution, or routinely during some types of cardiac surgeries [60]. Elderly patients, in particular, may become hypothermic more easily as a result of loss of fat with age. Hypothermia increases the risk for SSI through thermoregulatory vasoconstriction and impaired immunity. Vasoconstriction is universal in patients who have hypothermia [61] and leads to decreased partial pressure of oxygen in tissues [62], decreased microbial killing [63], impaired chemotaxis and phagocytosis of granulocytes, and decreased motility of macrophages [64].

A randomized, controlled trial evaluating 200 patients undergoing elective colorectal surgery demonstrated a threefold reduction in the rate of SSI by maintaining body temperature above 36°C [65].

Decreased tissue oxygenation leads to increased risk for SSI [66] by limiting the respiratory burst of neutrophils [67]. Increasing age may exacerbate this effect as aging leads to decreased levels of tissue oxygenation [68]. Thus far, three randomized, controlled trials on postoperative oxygenation have been published, with conflicting results [69–71]. Both studies in favor of supplemental oxygen included patients who underwent colorectal surgery, whereas the study reporting adverse effect of supplemental oxygen included patients undergoing various of types of surgery; when results of the three studies are pooled, the rate of SSI decreases from 15.2% in patients receiving 30% to 35% supplemental fraction of inspired oxygen (FIO₂) to 11.5% in patients who received 80% FIO₂ during and 6 hours after surgery (3.7% absolute risk reduction; $P = .10$) [72]. Given the low cost of supplemental oxygen, plausible biologic rationale, and potential benefit, supplemental oxygen therapy should be considered strongly as a strategy to reduce the rate of SSI, particularly in colorectal surgery.

Surveillance for surgical site infection

The majority of SSIs are diagnosable within 21 days of surgery [73,74]. Surgical procedures have been shifting to outpatient settings during the past 3 decades [75]. Thus, postdischarge and outpatient SSI surveillance increasingly are becoming important. Currently, no one method of surveillance is proved more beneficial than others for the geriatric population. In particular, the diagnosis of SSI in the setting of an implanted device is challenging for clinicians, as signs and symptoms not always are uniform and can occur long after a surgical procedure [76]. Thus, surveillance for SSI should continue for at least 12 months for procedures in which an implant is placed. A recent analysis of 756 patients who had undergone insertion of a hip or knee prosthesis confirmed that all SSIs were detected within 12 months of the procedure [77]. Similarly, geriatric patients may not manifest typical symptoms of infection (eg, fever or elevated white blood cell count), but might present with cognitive or functional decline; thus, vigilance is necessary when evaluating older patients after surgery.

Treatment of surgical site infection

No specific guidelines exist for the treatment of SSI, but the principles for treatment of SSI are the same in elderly and younger populations. Superficial SSIs, including mild wound drainage and simple cellulitis, can be treated in an outpatient setting, usually with oral antibiotics. Serious SSIs (those classified as deep or organ space), however, generally require readmission to a hospital for further surgical débridement and IV antibiotics. In both situations, the key to curing the infection is removal of dead, necrotic tissue,

with antibiotics used only as adjunctive therapy. Empiric antimicrobial choices should cover pathogens that cause SSI most commonly in a given anatomic site but should be tailored to culture results when available.

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