Surgical Site Infections

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KEYWORDS

• Surgical site infection • Health care–acquired infection
• Outcome

Attempts at reducing the rate of surgical site infections (SSIs) date to the early nineteenth century with the study of the epidemiology and prevention of surgical fever by James Young Hamilton.[1] Thereafter, Joseph Lister pioneered his use of antiseptics for the prevention of orthopedic SSIs in 1865. Fortunately, many other advances have been made in surgery and infection control over the past 150 years. However, as medicine has advanced, new types of infection risks have developed. For example, over the past 50 years, the frequency of surgical procedures has increased, procedures have become more invasive, a greater proportion of operative procedures include insertion of foreign objects, and procedures are performed on an increasingly morbid patient population. As a result, SSIs remain a leading cause of morbidity and mortality in modern health care.

EPIDEMIOLOGY AND OUTCOMES

Epidemiology

SSIs are a devastating and common complication of hospitalization, occurring in 2% to 5% of patients undergoing surgery in the United States.[2] As many as 15 million procedures are annually performed in the United States; thus, approximately 300,000 to 500,000 SSIs occur each year.[3] SSI is the second most common type of health care–associated infection (HAI).[4] *Staphylococcus aureus* is the most common cause of SSI, occurring in 20% of SSIs among hospitals that report to the Centers for Disease Control and Prevention (CDC) (**Table 1**)[5] and causes as many as 37% of SSIs that occur in community hospitals.[6] In fact, methicillin-resistant *S aureus* (MRSA) is not only a common pathogen in tertiary care and academic institutions but is also the single most common SSI pathogen in community hospitals.[6]

Outcomes

SSIs lead to increased duration of hospitalization, cost, and risk of death. Each SSI leads to more than 1 week of additional postoperative hospital days.[3,7] The costs

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Table 1
The 10 most common pathogens causing SSIs in hospitals that report to the CDC

Pathogen	Percentage of Infections (%)
S aureus	20
Coagulase-negative staphylococci	14
Enterococci	12
Pseudomonas aeruginosa	8
Escherichia coli	8
Enterobacter species	7
Proteus mirabilis	3
Streptococci	3
Klebsiella pneumoniae	3
Candida albicans	2

Data from 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; and 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].

attributable to SSI range from $3000 to $29,000 per patient per SSI, depending on the type of procedure.[8] In total, SSIs cost the US health care system approximately $10 billion annually.[9] SSI increases mortality risk by 2 to 11 fold.[10] Moreover, 77% of deaths in patients with SSI are attributed directly to the SSIs.[11] SSIs caused by resistant organisms, such as MRSA, lead to even worse outcomes.[12,13]

DIAGNOSIS

Most SSIs that do not involve implants are diagnosed within 3 weeks of surgery.[14] The CDC's National Healthcare Surveillance Network (NHSN) has developed standardized criteria for defining an SSI (**Box 1**).[15] SSIs are classified as either incisional or organ/space (**Fig. 1**). Incisional SSIs are further classified into superficial (involving only skin or subcutaneous tissue of the incision) or deep (involving fascia and/or muscular layers). Organ/space SSIs include infections in a tissue deep to the fascia that was opened or manipulated during surgery. For all classifications, infection can occur within 30 days after the operation if no implant was placed or within 1 year if an implant was placed and the infection is related to the incision. The NHSN defines implant as a nonhuman-derived implantable foreign body (eg, prosthetic heart valve, nonhuman vascular graft, mechanical heart, or joint prosthesis) that is permanently placed in a patient.

Serum laboratory tests can be suggestive but none are specific for SSI. For example, basic hematologic abnormalities, including increasing white blood cell count and neutrophil concentration, are suggestive of infection. For example, leukocytosis of more than 15,000/mm^3 in the setting of hyponatremia (sodium<135 mEq/L) is predictive of necrotizing soft tissue infection.[16] However, many SSIs occur without any hematologic or serologic laboratory abnormalities. Culturing samples of all suspected cases of deep and organ/space infections should be done to guide therapy and determine the susceptibility of the infecting organism. Ideally, culture samples are obtained in the operative setting, and external wound swabs are avoided. Radiographic studies may be adjunctive for the diagnosis of SSI. Computed tomography is more reliable

Box 1
Criteria for defining an SSI[a]

Incisional SSI

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 deliberately opened by the surgeon (not applicable if culture-negative infection)
4. Diagnosis of superficial incisional SSI by the 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[b]
2. A deep incision that spontaneously dehisces or is deliberately opened by a surgeon when a patient has one or more of the following signs/symptoms, fever (>38°C), 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 radiological examination[c]
4. Diagnosis of a deep incisional SSI by the surgeon

Organ/space SSI:

Infection involves any part of the anatomy (eg, organs or organ spaces), which was opened or manipulated during an operation and at least one of the following:

1. Purulent drainage from a drain that is placed through the stab wound into the organ/space
2. Organisms isolated from an aseptically obtained culture from the organ/space
3. An abscess or other evidence of infection involving organ/space, which is found on examination (physical, histopathologic, or radiological) or during repeat surgery
4. Diagnosis of an organ/space SSI by the surgeon

[a] 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.
[b] Report infection that involves both superficial and deep incision sites as a deep incisional SSI.
[c] Report an organ/space SSI that drains through the incision as a deep incisional SSI.
Adapted 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.

than plain radiographs for the detection of free air in soft tissue and the presence of deep abscess.

Diagnosis of SSIs in the setting of an implant or prosthetic joint can be even more difficult. For example, radiographs are often difficult to interpret with the presence of prosthetic material or metal. Cultures directly from explanted material, however, may aid the diagnosis.[17] A recent trial comparing conventional tissue culture with culture of specimens after sonication of explanted joints demonstrated that sonicated specimens had a higher sensitivity for the diagnosis of prosthetic joint infection (PJI)

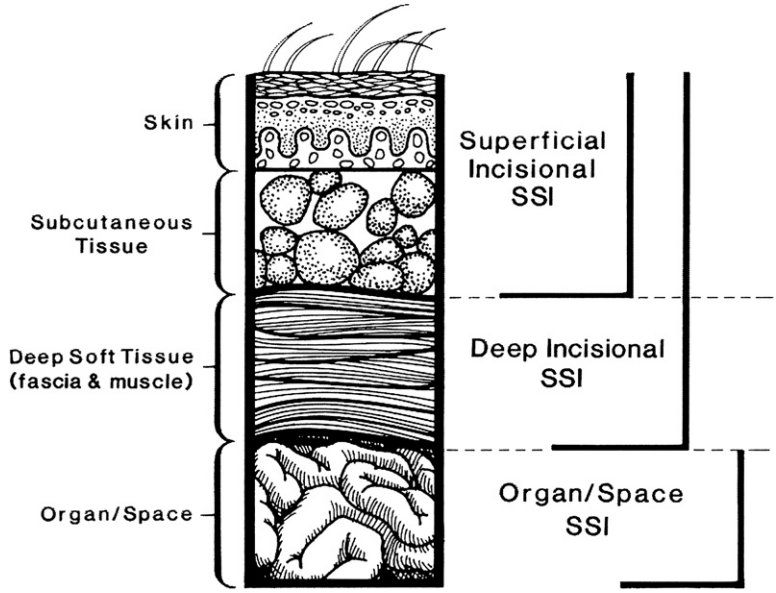

Fig. 1. CDC classification of surgical site infection (public domain). (*From* Horan TC, Gaynes RP, Martone WJ, et al. CDC definitions of nosocomial surgical site infections, 1992: a modification of CDC definitions of surgical wound infections. Infect Control Hosp Epidemiol 1992;13(10):606–8.)

than periprosthetic tissue culture (79% vs 61%, with an even wider difference in the subgroup of patients who had received antibiotics before explant) and similar specificity.[18] However, few microbiology labs have the capability to perform these cultures, and transport of specimens while avoiding contamination may be difficult.

PATHOGENESIS OF INFECTION

The likelihood that an SSI will occur is a complex relationship among (1) microbial characteristics (eg, degree of contamination, virulence of pathogen), (2) patient characteristics (eg, immune status, diabetes), and (3) surgical characteristics (eg, introduction of foreign material, amount of damage to tissues). Similar to taxes and death, microbial contamination of surgical sites is universal, despite the use of cutting-edge technology and expert technique. The pathogens that lead to SSI are acquired from the patient's endogenous flora or, less frequently, exogenously from the operating room (OR) environment.

Endogenous Contamination

The period of greatest risk for infection occurs while the surgical wound is open, that is, from the time of incision to the time of wound closure.[19] Twenty percent of bacterial skin flora resides in skin appendages, such as sebaceous glands, hair follicles, and sweat glands.[20] Thus, modern methods of pre- and perioperative antisepsis can reduce but not eliminate contamination of the surgical site by endogenous skin flora of the surgical patient. As a result, gram-positive cocci from patients' endogenous flora at or near the site of surgery remain the leading cause of SSI.[21]

Inoculation of the surgical site by endogenous flora from remote sites of the patient may also occur infrequently. Experiments using albumin microspheres as tracer particles revealed that 100% of surgical wounds are contaminated with particles (skin squames) from sites from the surgical patient (eg, head, groin), which are distant in location to the surgical wound.[22] Postsurgical inoculation of the surgical site secondary to a remote focus of infection (such as *S aureus* pneumonia) is an even less-frequent cause of SSI.[23]

Exogenous Contamination

Exogenous sources of contamination are occasionally implicated in the pathogenesis of SSI, including colonized or infected surgical personnel, the OR environment, and surgical instruments. Infections due to exogenous sources most commonly occur sporadically, but several exogenous point source outbreaks have been reported.[24–28] Surgical personnel colonized with *S aureus* are occasionally identified as sources of *S aureus* causing SSI.[29] Carriage of group A streptococci by OR personnel has been implicated as a cause of several SSI outbreaks.[24,30–33] It is important to remember, however, that most SSIs are not contracted from exogenous sources.

Unusual environmental pathogens are occasionally implicated in SSIs from sources in the OR. For example, *Rhodococcus bronchialis* was implicated in an outbreak of SSI after coronary artery bypass surgery because of colonization of an OR nurse by her dog.[25] Other unusual pathogens causing SSI include *Legionella pneumophila* after contamination of a prosthetic valve by tap water,[26,34,35] *Mycobacterium chelonae* and *Mycobacterium fortuitum* after breast augmentation,[27,36] *Rhizopus rhizopodiformis* after contamination of adhesive dressings,[28] *Clostridium perfringens* contamination of elastic bandages,[37] and *Pseudomonas multivorans* contamination of a disinfectant solution.[38]

Burden of Inoculation

Although many other factors contribute to the risk of SSI, the burden of pathogens inoculated into a surgical wound intraoperatively remains one of the most accepted risk factors. In fact, the greater the degree of surgical wound contamination, the higher the risk for infection. In the setting of appropriate antimicrobial prophylaxis, wound contamination with greater than 10^5 microorganisms is required to cause SSI.[39,40] However, the bacterial inoculum required to cause SSI may be much lower when foreign bodies are present.[41] For example, the presence of surgical sutures decreases the required inoculum for *S aureus* SSI by two-thirds (from 10^6 to 10^2 organisms).[42] Other models have demonstrated that the minimum inoculum for SSI due to virulent pathogens such as *S aureus* is as few as 10 colony-forming unit (CFU) in the presence of polytetrafluoroethylene vascular grafts[43] and 1 CFU in the proximity of dextran beads.[44]

Pathogen Virulence

Many potential SSI pathogens have intrinsic virulence factors or characteristics that contribute to their ability to cause infection. Several gram-positive organisms, including *S aureus*, coagulase-negative staphylococcus, and *Enterococcus faecalis*, possess microbial surface components recognizing adhesive matrix molecules that allow better adhesion to collagen, fibrin, fibronectin, and other extracellular matrix proteins.[44–47] Most of these same organisms also have the ability to produce a glycocalyx-rich biofilm, which shields the organisms from both the immune system and most antimicrobial agents.[48–50] In addition, once in the wound, some Staphylococci and Streptococci produce exotoxins that lead to host tissue damage,[51] interfere

with phagocytosis,[52] and alter cellular metabolism.[53] Many gram-negative pathogens produce endotoxins that stimulate cytokine production and, often, systemic inflammatory response syndrome.[54] Several bacteria possess polysaccharide capsules or other surface components that additionally inhibit opsonization and phagocytosis.[55]

RISK FACTORS

Risk factors for SSI are typically separated into patient-related (preoperative), procedure-related (perioperative), and postoperative categories (**Table 2**). In general, patient-related risk factors for the development of SSI can be categorized as either unmodifiable or modifiable. The most prominent unmodifiable risk factor is age. In a cohort study of more than 144,000 patients, increasing age independently predicted an increased risk of SSI until age 65 years, but at ages 65 years or more, increasing age independently predicted a decreased risk of SSI.[56] Modifiable patient-related risk factors include poorly controlled diabetes mellitus,[57] obesity,[58] tobacco use,[59,60] use of immunosuppressive medications,[61] and length of preoperative hospitalization.[19]

Procedure-related perioperative risk factors include wound class,[62] length of surgery,[63] shaving of hair,[64,65] hypoxia,[66,67] and hypothermia.[68] Of note, the act of surgery itself leads to increased risk of infection. The microbicidal activity of neutrophils harvested after surgery is 25% less than neutrophils harvested before surgery[69]; surgery leads to decreased levels of circulating HLA-DR antigens[70] and a decrease in T-cell proliferation and response[71]; neutrophils exhibit reduced chemotaxis and diminished superoxide production in the setting of perioperative hypothermia.[72] Specific recommendations are available regarding traffic in the OR and OR parameters, such as ventilation, to reduce the risk of exogenous seeding of the surgical wound as a result of personnel in the OR.[11,73] As a rule of thumb, the degree of microbial contamination of the OR air is directly proportional to the number of people in the room[74]; thus, traffic in and out of the room should be limited as much as possible.

Several risk factors that occur during the perioperative period, including hyperglycemia and diabetes mellitus,[57,75] remain important during the immediate postoperative period. Two additional risk variables that are present exclusively in the postoperative period are wound care and postoperative blood transfusions. Postoperative wound care is determined by the technique used for closure of the surgical site. Most wounds are closed primarily (ie, skin edges are approximated with sutures or staples) and these wounds should be kept clean by covering with a sterile dressing for 24 to 48 hours after surgery.[76] A meta-analysis of 20 studies of the associated risk of SSI after receipt of blood products demonstrated that patients who received even a single unit of blood in the immediate postoperative period were at increased risk for SSI (odds ratio, 3.5).[77]

PREVENTION

Methods to prevent SSI were recently summarized in the Society for Healthcare Epidemiology of America/Infectious Disease Society of America Compendium of Strategies to Prevent HAI in Acute Care Hospitals.[78] In particular, emphasis was placed on the importance of perioperative antimicrobial prophylaxis, avoiding shaving, glucose control for cardiac surgery, and measurement and feedback of rates of SSI to surgeons. If feasible, modifiable risk factors for SSI should be addressed. **Table 2** summarizes important risk factors and current guidelines for addressing each risk factor to decrease the risk of SSI.

Table 2
Risk factors for SSI and current recommendations to decrease the risk of SSI

Risk Factors	Recommendations
Intrinsic, patient-related (Preoperative)	
Age	No formal recommendation, relationship to increased risk of SSI may be secondary to comorbidities or immunosenescence[56,63,132]
Glucose control, diabetes mellitus	Control serum blood glucose levels[133]
Obesity	Increase dosing of perioperative antimicrobial agent for morbidly obese patients[134]
Smoking cessation	Encourage smoking cessation within 30 d of procedure[133]
Immunosuppressive medications	No formal recommendation[133]; in general, avoid immunosuppressive medications in the perioperative period if possible
Nutrition	Do not delay surgery to enhance nutritional support[133]
Remote sites of infection	Identify and treat all remote infections before elective procedures[133]
Preoperative hospitalization	Keep preoperative stay as short as possible[133]
Extrinsic, procedure-related (Perioperative)	
Preparation of the patient	
Hair Removal	Do not remove unless presence of hair interferes with the operation[133]; if hair removal is necessary, remove by clipping and do not shave immediately before surgery
Skin preparation	Wash and clean skin around incision site, using approved surgical preparations[133]
Chlorhexidine nasal and oropharyngeal rinse	No formal recommendation in most recent guidelines[133]; Recent RCT of cardiac surgeries showed decreased incidence of postoperative nosocomial infections[116]
Surgical scrub (surgeon's hands and forearms)	Use appropriate antiseptic agent to perform 2–5 min preoperative surgical scrub[133]
Incision site	Use appropriate antiseptic agent[133]
Antimicrobial prophylaxis	Administer only when indicated[133]
Timing	Administer within 1 h of incision to maximize tissue concentration[133]
Choice	Select appropriate agents based on surgical procedure, most common pathogens causing SSI for a specific procedure, and published recommendations[133]
Duration of therapy	Stop agent within 24 h after the procedure[133,135]
Surgeon skill/technique	Handle tissue carefully and eradicate dead space[133]
Incision time	No formal recommendation in most recent guidelines[133]; minimize as much as possible[136]
Maintain oxygenation with supplemental O_2	No formal recommendation in most recent guidelines,[133] RCTs have reported conflicting results in colorectal procedures[66,67,125]

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Table 2
(*continued*)

Risk Factors	Recommendations
Maintain normothermia	Avoid hypothermia in surgical patients whenever possible by actively warming the patient to >36°C, particularly in colorectal surgery[137]
OR characteristics	
Ventilation	Follow the American Institute of Architects' recommendations[73,133]
Traffic	Minimize OR traffic[133]
Environmental surfaces	Use an EPA-approved hospital disinfectant to clean visibly soiled or contaminated surfaces and equipment[133]

Abbreviations: EPA, Environmental Protection Agency; O_2, oxygen; RCT, randomized controlled trial.

Perioperative Antimicrobial Prophylaxis

The appropriate use of perioperative antimicrobial prophylaxis is a well-proven intervention to reduce the risk of SSI in elective procedures.[11,40,79] The goal of surgical antimicrobial prophylaxis is to reduce the concentration of potential pathogens at or in close proximity to the surgical incision. Four main principles dictate prophylactic antimicrobial use: (1) use antimicrobial prophylaxis for all elective operations that require entry into a hollow viscus, operations that involve insertion of an intravascular prosthetic device or prosthetic joint, or operations in which an SSI would pose catastrophic risk[80–83]; (2) use antimicrobial agents that are safe, cost-effective, and bactericidal against expected pathogens for specific surgical procedures[11]; (3) time the infusion so that a bactericidal concentration of the agent is present in tissue and serum at the time of incision[79] and; (4) maintain therapeutic levels of the agent in tissue and serum throughout the entire operation (ie, until wound closure).[81,84,85] Thus, the 2 major components of appropriate perioperative antimicrobial prophylaxis are using the appropriate agent at the appropriate dose and giving the agent at the appropriate time.

Administering antimicrobial prophylaxis shortly before incision reduces the rate of SSI.[79] The chosen agent should be given at a time that allows for maximum tissue concentration at the time of incision. The optimal administration time typically occurs within 2 hours before surgery.[86] In one retrospective study of approximately 3000 patients undergoing various elective inpatient procedures, the lowest rates of SSI occurred in the group of patients who received antimicrobial prophylaxis within 1 hour before incision.[79]

Current regulatory process measures state that starting infusion of antimicrobial prophylaxis within 1 hour before incision maximizes benefit (and for vancomycin and fluoroquinolones, within 2 hours before incision).[87] Thus, prophylaxis may be started as soon as 1 minute before incision and the OR team still "gets credit" from a regulatory perspective. Although this practice may follow the letter of the law, it certainly does not follow the spirit. For most agents, antimicrobial prophylaxis is most effective if infusion is started between 60 and 30 minutes before surgery. For example, in one prospective observational study of 3836 surgical patients, antimicrobial prophylaxis given 0 to 29 minutes before surgery was less effective than comparable therapy administered between 30 to 59 minutes before surgery, even after statistical adjustment for other confounding risk factors such as the American Society of Anesthesiologists score, duration of surgery, and wound class.[88] In another study involving 2048 patients undergoing cardiac bypass surgery, patients who received

vancomycin 0 to 15 minutes before the beginning of surgery had higher rates of postoperative infection than those who received vancomycin 16 to 60 minutes preoperatively.[89]

If a procedure is expected to last several hours, prophylactic agents should be redosed intraoperatively.[11] For example, cefazolin should be reinfused if a procedure lasts longer than 3 to 4 hours. One retrospective study of 1548 patients undergoing prolonged cardiac procedures (>400 minutes) demonstrated that patients who received intraoperative redosing of cefazolin had significantly fewer SSIs than those who did not receive redosing, even after adjusting for baseline risk (7.7% vs 16.0%; adjusted odds ratio, 0.44; 95% confidence interval [CI], 0.23–0.86).[90]

Although not directly related to the prevention of SSI, an additional measure related to perioperative surgical prophylaxis is the number of doses administered. Single-dose antimicrobial prophylaxis is equivalent to multiple perioperative doses for the prevention of SSI. A meta-analysis of more than 40 studies comparing single doses of parenteral antimicrobials with placebo or multiple doses in hysterectomies; cesarean sections; colorectal procedures; gastric, biliary, transurethral operations; and cardiothoracic procedures demonstrated that administering multiple doses of antibiotics provided no benefit for SSI prevention over a single dose.[91] Similarly, a more recent systematic review of 28 prospective randomized studies comparing single versus multiple doses of perioperative antimicrobials also concluded that there was no additional benefit of more than a single prophylactic dose.[84] Thus, current recommendations state that prophylactic antibiotics should not be given for longer than 24 hours after surgery or longer than 48 hours after cardiothoracic surgery.

Hospitals that improve compliance with the different components of appropriate antimicrobial prophylaxis decrease the rates of SSI. For example, the Center for Medicare and Medicaid Service created the Surgical Infection Prevention Project and performed a large study on the impact of improved antimicrobial prophylaxis process measures. The study, which included 34,133 procedures performed at 56 hospitals, led to an improvement of 27% in antibiotic timing, an improvement of 6% in antibiotic choice, an improvement of 27% in stopping prophylaxis within 24 hours of incision, and, most importantly, a reduction of 27% in the average rate of SSI.[92] A recent study included these 3 antimicrobial prophylaxis process measures as part of a global checklist to improve outcomes after surgical procedures.[93] This prospective quasi-experimental study of approximately 8000 operative patients demonstrated that implementation of a 19-item checklist in 8 institutions throughout the world led to lower rates of postoperative complications and death. Furthermore, the rate of appropriately administered antimicrobial prophylaxis improved by 60% and the rate of SSI decreased by half.

Avoid Shaving

Preoperative shaving leads to increased rates of SSI by causing microscopic abrasions of the skin, which become foci for bacterial growth.[7,64,65] Some studies, however, suggest that any form of hair removal, shaving, depilatory, or clipping, leads to increased rates of SSI and should thus be avoided when possible.[64,94,95] Thus, current recommendations state that hair should not be removed from the surgical site unless the hair interferes with the procedure.[11] If hair removal is necessary, the hair should be removed with electric clippers immediately before surgery.[96,97]

Glucose Control

As described earlier, diabetes mellitus is clearly associated with an increased risk of SSI.[57,75] Elevated serum glucose levels in both the pre- and postoperative periods

have been associated with an increased risk of SSI.[57,98] For example, in one study of 8910 patients undergoing cardiac surgery, rates of SSI decreased substantially after implementing an intravenous insulin regimen to maintain postoperative glucose levels lesser than 200 mg/dL for the first 48 hours after surgery.[57] In contrast, strict glucose level control in the intraoperative period has not been shown to decrease the risk of SSI and may actually lead to harm.[99] Thus, current recommendations state that (1) every effort should be made to improve control of diabetes mellitus before surgery and (2) postoperative serum glucose concentration should be maintained lesser than 200 mg/dL for the first 48 hours after surgery.

Surveillance and Feedback to Surgeons

Surveillance and reporting of infection rates to surgeons reduce the rate of SSI for all procedure classes.[7,100–102] Two main methods can be used to perform surveillance for SSIs, the direct method and the indirect method. The direct method with daily observation of the surgical site by the surgeon, a trained nurse, or infection control professional is the most accurate method of surveillance.[7,101,103,104] The indirect method of SSI surveillance consists of a combination of the following: review of microbiology reports, surgeon and/or patient surveys, and screening for readmission of surgical patients. The indirect method of SSI surveillance is less time consuming, can be readily performed by infection control personnel during surveillance rounds, and is both reliable (sensitivity, 84%–89%) and specific (specificity, 99.8%) compared with the gold standard of direct surveillance.[105,106] Automated data systems that use hospital databases with administrative claims data, antibiotic days, readmission to the hospital, and return to the OR and/or implementation of a system that imports automated microbiological culture data, surgical procedure data, and general demographic information can broaden indirect SSI surveillance and may obviate direct surveillance.[107–109]

The landmark study on the efficacy of nosocomial infection control study by Haley and colleagues[100] showed that establishing an infection control program that includes the feedback of SSI rates to surgeons can lower the overall rate of SSI by as much as 35% and remains one of the studies on which modern infection control programs are based. No studies have yet revealed the exact mechanism by which feedback to surgeons reduces the rate of SSI. Possible explanations for this reduction from feedback include (1) increased awareness of the problem of SSIs, (2) anxiety created by awareness that patient outcomes are being monitored, or (3) introspection concerning possible systematic, procedural, or technical errors.[19]

Simple rates of SSI provide minimal information for surgeons. Instead, rates of SSI should first be risk stratified using the National Nosocomial Infections Surveillance risk index. Then, rates of SSI for a surgeon and a specific procedure should be benchmarked against internal and external standards. That is, risk-stratified rates of SSI for a specific procedure can be compared with the surgeon's previous rates, rates of other surgeons at the institution, and national rates published by the CDC.

Unresolved issues in the prevention of SSI

Preoperative bathing Showering or bathing with an antiseptic agent, such as chlorhexidine gluconate, povidone-iodine, or triclocarban-medicated soap, decreases the amount of endogenous microbial flora on the skin.[110,111] However, this intervention has not yet been clearly demonstrated to lower rates of SSI in clinical trials.[112–114] In fact, a prospective, randomized, controlled, double-blind trial comparing preoperative showers with soap containing chlorhexidine gluconate with preoperative showers with nonmedicated soap in 1400 patients found no significant

difference in infection rates between the 2 groups.[112] Most likely, the lack of benefit is related to the method of application of the antiseptic; for example, chlorhexidine gluconate typically requires several applications for maximum microbial-reducing benefit.[115]

Decolonization of *S aureus* carriage Studies examining the utility of preoperative *S aureus* nasal decolonization with antimicrobial agents have produced inconsistent results. A randomized controlled trial examined the utility of oral and nasal rinses with chlorhexidine gluconate (0.12%) before cardiothoracic surgery for the prevention of postoperative nosocomial infections.[116] Although the overall number of SSIs was not different in the 2 groups, the number of deep SSIs was significantly decreased in the group that received chlorhexidine (1.9% vs 5.2%, $P = .002$). Given the proven benefit, low toxicity, and lack of emerging resistance in long-term clinical studies of chlorhexidine,[117] preoperative treatment with chlorhexidine represents a promising intervention for the prevention of SSIs.

Studies examining the efficacy of decolonization of *S aureus* with mupirocin have generally shown that mupirocin is effective at decolonization, but the impact on SSI remains unclear. One study with historical controls reported that the preoperative application of mupirocin to the nares of operative patients led to a decrease of 67% in the rate of SSI after cardiothoracic surgery (from 7.3% to 2.8%), regardless of *S aureus* carrier status.[118] In addition, a separate nonrandomized analysis of almost 1900 consecutive cardiothoracic procedures showed that the overall rate of SSI was lower in patients who received decolonization with mupirocin (2.7% vs 0.9%, $P = .005$).[117] These findings, however, were not corroborated in a double-blind randomized controlled trial in which 1933 surgical patients randomized to receive preoperative mupirocin were compared with 1931 patients randomized to placebo.[119] Treatment with mupirocin led to lower rates of *S aureus* colonization and lower rates of overall postoperative hospital-acquired infections caused by *S aureus* (3.8% of patients who received mupirocin vs 7.6% of patients who received placebo, $P = .02$). The intervention, however, did not lead to a significant decrease in the rate of SSI caused by *S aureus* (3.6% of patients who received mupirocin vs 5.8% of patients who received placebo; odds ratio, 2.9; 95% CI, 0.8–3.4).[119]

However, *S aureus* resistance to mupirocin has rapidly emerged in some institutions.[120] For example, extensive use of mupirocin correlated with an increase in resistance to mupirocin among *S aureus* strains from 3% to 65% over a 4-year period in one European institution.[121] The emergence of mupirocin resistance is concerning and clearly negates the efficacy of preoperative decolonization. Thus, many experts recommend that decolonization be limited to specific high-risk populations and not administered universally.

Perioperative oxygen supplementation Six randomized controlled trials have evaluated the utility of high inspired oxygen fraction in the perioperative setting.[66,67,122–125] Four studies demonstrated a reduction in the rate of SSI after administration of 80% FiO_2 during and after surgery,[66,67,122,124] one study demonstrated no difference,[123] and one study actually concluded that administration of 80% FiO_2 led to higher rates of SSI.[125] The investigators of a recent meta-analysis that included 5 of the earlier-mentioned trials (including the negative study) concluded that high inspired oxygen decreased the risk of SSI, although significant methodological differences were noted among the studies.[126] No increased risk of harm was noted in the randomized controlled trial that rigorously evaluated adverse outcomes.[123] Thus, supplemental oxygen seems to reduce the risk for SSI in certain surgeries such as colorectal and abdominal surgeries.

TREATMENT

Surgical opening of the incision with removal of necrotic tissue is the primary and most important aspect of therapy for many SSIs.[127] Antimicrobial therapy is an important adjunct to surgical debridement. The type of debridement and duration of the postoperative antimicrobial therapy depend on the anatomic site of infection and invasiveness of the SSI, although deep incisional and organ/space infections almost universally require operative drainage of accumulated pus. A key consideration for both the need for surgical debridement and duration of antimicrobial therapy is whether prosthetic material is present and infected.

Superficial incisional SSI can usually be treated without debridement, with oral antibiotics. Postoperative patients with suspected deep or organ/space SSI, fever (temperature>38.5°C), or tachycardia (heart rate, 110 beats/min) generally require antibiotics in addition to opening of the suture line.[127] Few published data exist to support the use of specific antimicrobial agents or specific therapeutic durations for the treatment of SSI. Decisions regarding the antimicrobial agent and length of therapy for SSI are influenced by the location of the infection (eg, mediastinum, abdominal cavity, joint), depth of infection, adequacy or completeness of surgical debridement, and resistance patterns of the pathogen. As a rule of thumb, effective systemic antimicrobial therapy should be started as soon as a deep incisional or organ/space SSI is suspected. For example, in one study, patients with mediastinitis who received antimicrobial therapy active against the infecting pathogen within 7 days of debridement had a reduction of 60% in mortality rates compared with patients who did not receive effective antimicrobial therapy.[128]

PJI is a unique problem that usually requires surgical debridement and prolonged antimicrobial therapy. Surgical treatment of PJI includes the following different strategies: debridement with retention of the prosthesis, 1- or 2-stage exchange with reimplantation, resection arthroplasty, and amputation.[129] Removal of foreign materials, such as wires, bone wax, and devitalized tissues, greatly improves the likelihood of cure.[130] Traditionally, a 2-stage exchange has been the standard treatment modality used to cure an infected prosthesis. The 2-stage exchange involves debridement and removal of the infected prosthesis followed by prolonged antimicrobial therapy (often up to 6–8 weeks) and subsequent reimplantation of a new prosthesis. The 1-stage exchange involves debridement, removal of the prosthetic joint, and immediate reimplantation of a new prosthesis. In some cases of SSI, the infected prosthesis can be salvaged through early surgical debridement in combination with effective antimicrobial therapy.[131] The likelihood of successful salvage of an infected prosthesis is improved if the following conditions are met: signs and symptoms of PJI are detected within 3 weeks of implantation, the implant remains stable and functional, the surrounding soft tissue remains in good condition, and the patient is treated with appropriate systemic antimicrobials.[129] Most patients with PJI should receive 6 to 8 weeks of intravenous antimicrobial therapy.[129]

SUMMARY

SSIs lead to an excess of health care resource expenditure, patient suffering, and death. Improved adherence to evidence-based preventative measures, particularly those related to appropriate antimicrobial prophylaxis, can decrease the rate of SSI. Diagnosis is difficult, particularly in the setting of a procedure that involved prosthetic material. In general, aggressive surgical debridement in combination with effective antimicrobial therapy are needed to optimize the treatment of SSIs.

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