Surgical Site Infections

Jessica Seidelman, MD, MPH[a,b,*], Deverick J. Anderson, MD, MPH[a,b]

KEYWORDS

• Surgical site infection • Health care–associated infection • Risk • Prevention
• Outcome

KEY POINTS

- Surgical site infections (SSIs) are associated with prolonged hospitalizations, death, and overall poor patient outcomes.
- SSIs are typically caused by pathogens inoculated at the time of surgery from the patient's own flora.
- SSI acquisition depends on exposure to bacteria and the host's ability to control the inevitable bacterial contamination of the surgical wound.
- The 9 core best practices to reduce SSIs address preoperative bathing, antimicrobial prophylaxis, preoperative surgical site preparation, surgical hand preparation, normothermia, hyperoxygenation, wound protectors, glucose control, and perioperative checklist.
- SSI surveillance and feedback decrease SSI rates and are recommended by Centers for Disease Control and Prevention.

INTRODUCTION

Surgical site infections (SSIs) are a leading cause of health care–associated infections (HAIs). These infections can range in severity from nuisance to life threatening; overall, they contribute to substantial patient suffering. A large portion of SSIs are preventable, and SSI prevention is a key patient safety matter that requires teamwork among multiple health care personnel, including surgeons, nurses, anesthetists, and infection preventionists. This article provides updates to the epidemiology and diagnosis of SSIs with particular emphasis on risk factors, evidence-based prevention strategies, and surveillance.

[a] Division of Infectious Diseases and International Health, Department of Medicine, Duke University School of Medicine, Duke University, Durham, NC, USA; [b] Duke Center for Antimicrobial Stewardship and Infection Prevention, Duke University Medical Center, Durham, NC, USA
* Corresponding author. 315 Trent Dr Hanes House, Room 145, Durham, NC 27710.
E-mail address: jessica.seidelman@duke.edu

Infect Dis Clin N Am 35 (2021) 901–929
https://doi.org/10.1016/j.idc.2021.07.006 **id.theclinics.com**

EPIDEMIOLOGY

SSIs are the most common and most costly HAI in the United States, accounting for almost a quarter of all HAIs.[1,2] Although the risk of SSI is generally low, SSIs are common because of the volume of surgical procedures performed across the United States. In the United States in 2014, 17.2 million hospital visits (ambulatory or inpatient) included invasive, therapeutic surgeries.[3] SSIs occur in 1% to 5% of patients undergoing inpatient surgery.[4,5] Rates are generally lower among procedures performed in outpatient settings. Overall, approximately 300,000 SSIs occur each year, although this estimate likely underrepresents the true SSI burden because of limitations in surveillance and diagnosis.[4,5] SSIs are even more common in low-income and middle-income countries, occurring in 1% to 24% of procedures performed.[6]

Overall, rates of SSI are decreasing in the United States. Among 148 hospitals participating in serial point prevalence surveys performed by the Centers for Disease Control and Prevention (CDC), the rate of SSI decreased from 0.97 per 100 procedures in 2011 (n = 11,282 patients reviewed) to 0.56 per 100 procedures in 2015 (n = 12,299 patients reviewed; $P = .001$).[7] Similarly, the publicly reported rates of SSI following abdominal hysterectomy and colon surgery decreased approximately 10% in 2017 compared with the national baseline reported in 2016, although the decreases were not statistically significant.[8] However, decreases in SSI have been modest compared with decreases observed in several other HAIs, including central line–associated bloodstream infection, catheter-associated urinary tract infection, methicillin-resistant *Staphylococcus aureus* (MRSA) bacteremia, and *Clostridioides difficile* infection.

Rates of SSI vary by type of procedure and by setting. The CDC's National Healthcare Safety Network (NHSN) no longer routinely reports national rates of SSI following commonly performed procedures; therefore, most nationwide estimates currently used are from data reported almost 10 years ago. Clean-contaminated and dirty procedures, including procedures that enter a nonsterile viscera, have higher rates of SSI than clean procedures. For example, rates of SSI following colon, rectal, or other gastrointestinal (GI) procedures range from 4% to 25%, whereas the rate of SSI following coronary artery bypass grafting is approximately 3% and following hip or knee arthroplasty is 1% or lower.[9] In most cases, rates of SSI at surgical centers are inversely associated with surgical volume. That is, the more procedures performed, the lower the rate of SSI.[8] Although referral centers typically care for more complex patients and perform higher-risk procedures, these risks are often offset by high surgical volume and experience. Small community hospitals with low volume have higher rates of SSI than higher-volume community hospitals.[10]

CLINICAL OUTCOMES

SSIs lead to significant patient morbidity and mortality. Each SSI is associated with approximately 7 to 11 additional postoperative hospital days.[2,11] In total, patients with SSIs have 3.7 million excess hospital days each year.[12] Surgical patients who develop an SSI have a risk of death that is, between 2-fold and 11-fold higher than patients without an SSI[13]; 77% of deaths in patients with SSI are directly attributable to SSI.[14] Investigators from the Agency for Healthcare Research and Quality (AHRQ) estimated excess mortality related to SSI was 0.026 (95% confidence interval [CI], 0.009–0.059, meaning 26 deaths occur for every 1000 SSIs).[15] Each SSI leads to approximately $25,000 of additional costs, although attributable costs of SSI vary depending on the type of operative procedure and the type of infecting pathogen.[11,13,15] Overall,

SSIs are estimated to account for $3.5 billion to $10 billion annually in United States health care expenditures using the CPI (consumer price index for inpatient hospital services with all cost estimates adjusted for 2007 dollars).[5]

EVALUATION

Clinically, a surgical wound is considered infected when purulent drainage is present at the incision site or there is evidence of abscess involving the surgical bed. However, other presentations of surgical wound infections also occur, including wound dehiscence, nonpurulent drainage, local erythema, induration, pain, or systemic signs of infection. Because of the varied presentation of SSIs, many of which overlap with noninfectious causes, no single clinical definition of SSI exists. Instead, the diagnosis of SSI is typically made based on a constellation of clinical and examination features, laboratory and microbiologic data, and radiography results.

Despite the variability in clinical presentations of SSIs, specific definitions of SSI are used for epidemiologic and surveillance purposes. The NHSN provides the most commonly used SSI definitions. These definitions were designed to be objective and easy to apply but also flexible enough to identify clinically relevant SSIs with varied presentations.[16] NHSN categorizes SSIs into 3 groups: superficial incisional (involving the skin or subcutaneous tissue layers of the incision), deep incisional (involving muscle or connective tissue layers of the incision), and organ/space (involving structures, organs, or spaces deep to the incision) (**Fig. 1**, **Table 1**). Examples of organ/space infection include intra-abdominal abscess following colon surgery, periprosthetic joint infection following joint arthroplasty, and mediastinitis following cardiac surgery. Per NHSN definitions, surveillance for superficial-incisional infections is conducted for 30 days, whereas surveillance for deep-incisional and organ/space infections is conducted for 30 or 90 days, depending on the index surgical procedure.

Note that surveillance definitions have changed over time and that various epidemiologic surveys and studies of SSI prevention have used variable case-finding methods (eg, active vs passive surveillance), criteria for inclusion (eg, index procedure vs all procedures), depth of infection (eg, superficial-incisional, deep-incisional, or organ/space infection), and surveillance periods (eg, 30 vs 90 days). For example, 10% fewer SSIs were identified when, compared with previously used NHSN definitions, updated 2013 NHSN definitions that shortened surveillance periods were applied retrospectively to SSI surveillance data from a network of 35 hospitals and 2 ambulatory surgery centers.[17] Therefore, it is important to understand the surveillance criteria used when interpreting SSI data from different surveys or time periods, and how different criteria can affect reported rates.

CURRENT EVIDENCE

SSIs are typically caused by pathogens inoculated at the time of surgery. Most SSIs are caused by the patients' endogenous flora. However, exogenous sources of wound contamination are possible.[14,18] Most SSIs are caused by skin pathogens, although enteric pathogens are frequently seen in SSIs following GI procedures (**Table 2**).[19] Overall, *S aureus* is the most common cause of SSI. Although MRSA was previously a more frequent cause of SSI than methicillin-sensitive *S aureus* (MSSA), rates of MRSA SSI have declined; MSSA is now a more common cause of SSI than MRSA.[9] This trend is important because SSIs caused by resistant pathogens such as MRSA lead to worse clinical outcomes than SSIs caused by susceptible pathogens.[11] Outbreaks involving atypical organisms such as *Mycoplasma*, *Ureaplasma*, *Candida*

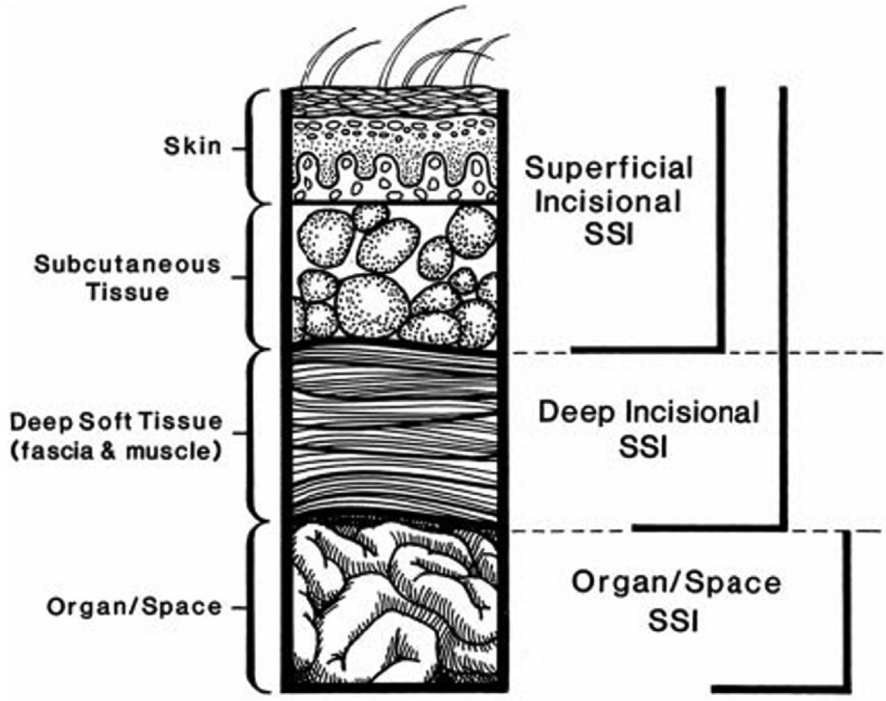

Fig. 1. NHSN categorization of SSIs. (*From* Horan TC, Gaynes RP, Martone WJ, Jarvis WR, Emori TG. CDC definitions of nosocomial surgical site infections, 1992: a modification of CDC definitions of surgical wound infections. Infect Control Hosp Epidemiol. 1992;13(10):606-608; with permission)

and other fungi, *Nocardia*, *Rhodococcus*, and nontuberculous mycobacteria (including rapidly growing mycobacteria) are uncommon, but have been described.[20–22]

Endogenous Contamination

Most SSIs are caused by the patients' endogenous flora contaminating the surgical site.[18] Even an inoculum as low as 100 colony-forming units into the wound can lead to an SSI.[23] Several factors may modify the risk of surgical wound contamination by endogenous flora.

Perioperative antibiotics

The absence of antibiotic prophylaxis also significantly increases the risk of SSI.[24] Contamination of operative sites, even clean ones, is unavoidable despite the best preparation and operative technique. The goal of antimicrobial prophylaxis is to reduce the risk of SSI by reducing the burden of microorganisms at the surgical site during the operative procedure.

Skin antisepsis

In addition, lack or inappropriate application of preoperative skin antiseptics increases the risk of SSI by failing to remove transient organisms from the skin where a surgical incision will be made. Effectiveness of incisional site preparation depends on correct application. Although skin disinfection before surgery drastically reduces the number

Table 1
Summary of National Healthcare Safety Network surveillance criteria for surgical site infection based on 2021 definitions

Superficial Incisional	Deep Incisional	Organ/Space
Any of the following: • Purulent drainage • Organism identified from aseptically obtained specimen or superficial incision deliberately opened by surgeon, attending physician, or designee • Diagnosis of superficial incisional SSI by surgeon, attending physician, or other designee culture not obtained	Any of the following: • Purulent drainage from the deep incision • A deep incision that spontaneously dehisces or is deliberately opened or aspirated by a surgeon, attending physician, or designee, and organism identified from deep soft tissues of the incision and 1 or more of the following symptoms: fever >38°C, localized pain or tenderness • Abscess or other evidence of infection involving the deep incision that is detected on gross anatomic or histopathologic examination, or imaging test	Any of the following: • Purulent drainage from a drain that is placed into the organ/space • Organism identified from fluid or tissue in the organ/space • Abscess or other evidence of infection involving the organ/space that is detected on gross anatomic or histopathologic examination, or imaging test evidence suggestive of infection

Deep incisional involves part of the incision deeper than the fascial layer. Organ/space involves any part of the body that is deeper than the muscle/fascial layers and was manipulated or entered during the operative procedure.

Modified from Centers for Disease Control and Prevention. Surgical Site Infection (SSI) Event Web site. https://www.cdc.gove/nhsn/pdfs/pscmanual/9pscssicurrent.pdf. Accessed 11/20/20.

of bacteria on the skin's surface, recolonization of the skin with bacteria from deeper skin layers and hair follicles may occur during the operation.[25]

Preoperative hair removal

Preoperative shaving of the surgical site is associated with a significantly higher SSI risk than either the use of depilatory agents or no hair removal.[14] One study found that SSI rates for patients who shaved before surgery was 5.6% compared with an SSI rate of 0.6% in patients who removed hair via depilatory agent or did not remove hair at all. The increased risk of SSI with shaving is attributed to microscopic cuts in the skin that later serve as niduses for bacteria to multiply. In addition, timing of hair removal is a key factor for development of SSI. One study found that patients who shaved immediately before the operation compared with patients who shaved within 24 hours preoperatively had lower rates of SSI (3.1% vs 7.1%).[26] Furthermore, if patients shaved more than 24 hours before operation, their SSI rates exceeded 20%.[26] Similarly, clipping hair immediately before an operation is associated with a lower risk of SSI than shaving or clipping the night before surgery (1.8% vs 4.0%).[27] Depilatory agents are associated with lower SSI risk compared with shaving or clipping[26]; however, these products can produce hypersensitivity reactions, which can compromise the integrity of the skin.[26]

Table 2
Common causes of surgical site infections

Organism	Clean Cardiac Bypass and Joint Arthoplasties[81,a] N = 6263 n (%); Rank	Community Hospitals[9,b] N = 3988 SSIs n (%); Rank
S aureus	2704 (41); 1	1357 (34); 1
MSSA	1520 (23)	683 (17)
MRSA	1184 (18)	674 (17)
Coagulase-negative staphylococci	1085 (17); 2	340 (9); 4
Enterococcus species	433 (7); 3	467 (12); 3
Pseudomonas aeruginosa	339 (5); 4	168 (4); 7
Escherichia coli	314 (5); 5	482 (12); 2
Streptococcus species	278 (4); 6	242 (6); 5

Abbreviation: MSSA, methicillin-sensitive *S aureus*.
[a] 6263 complex SSIs identified after 680,489 procedures performed in 880 hospitals over a 4-year period.
[b] 3,988 complex SSIs identified after 532,694 procedures performed in 29 community hospitals over a 5-year period.

Exogenous Contamination

Length of operation

Prolonged operative time may increase the risk of SSI. The increased risk may be related to increased wound contamination, increased tissue damage (eg, bleeding, cautery, suture), or some combination of these factors.[28]

Surgical technique

Poor surgical technique is widely thought to increase SSI risk.[14] Examples of poor surgical technique that may increase subsequent SSI risk include failure to maintain adequate blood supply, rough manipulation of tissues, accidental entry into a hollow viscus, leaving behind devitalized tissue, and inappropriate use of drains and sutures. In general, the impact of poor surgical technique is difficult to quantify or study.

Foreign material

Any foreign material, such as sutures or drains, promotes inflammation at the surgical site and increases the risk of SSI.[29] Furthermore, the presence of foreign material decreases the inoculum required to cause an SSI from 10^6 to 10^2 organisms.[30]

Wound contamination from operating room personnel

Health care personnel's hands and fingernails harbor bacteria that may be introduced into the surgical site.[31] Application of surgical gloves substantially reduces but does not prevent the potential passage of microorganisms to the patient. Gloves can become perforated during surgery and bacteria on gloved hands can multiply rapidly. Therefore, absence of appropriate hand antisepsis can expose patients to potential pathogens harbored on operating room personnel's hands. Furthermore, watches, long finger nails, artificial nails, and finger rings can increase bacterial counts on the hands of operating room personnel.[32,33] The number of SSIs caused by exogenous flora is unknown, although several outbreaks of SSI have been attributed to operating room personnel.

Airborne contamination

Transmission of bacteria from operating room personnel to patients can occur through several other routes, including shedding of bacteria from the personnel's hair, skin, and clothing. Actions that increase bacterial air counts are strongly associated with SSI risk.[34] Most of the airborne contamination comes from persons present in the operating room and their movements.[35] Several studies investigated the impact of operating room door openings on air quality, and the results were mixed.[36] However, some data suggest a positive correlation between high door opening rates and numbers of microorganisms in air samples.[37]

Wound care

Postoperative wound care practices are thought to modify risk of SSI.[38] Wounds that remain uncovered after surgery may be subject to environmental contamination or ongoing drainage that decreases the integrity of the surrounding skin.[39]

Intra-articular steroid injections

The relationship between intra-articular steroid injections and incidence of SSIs is under ongoing investigation. Infection may be introduced at the time of injection, especially if rigorous antisepsis is not applied.[40] In addition, the intra-articular steroid may decrease the host immune response to the introduction of such bacteria. Although some studies have shown an increase in SSI incidence in patients who receive intra-articular steroid injections,[41] other trials show no difference in SSI risk between patients who receive intra-articular steroid injections and those who do not.[42] Current clinical guidelines from the American Academy of Orthopedic Surgeons (AAOS) do not outline specific recommendations for intra-articular injection administration and SSI prevention.[43]

Periarticular injections

Recently, surgeons have incorporated local analgesia into pain management regimens for patients undergoing joint replacement surgery in order to improve postoperative pain control and promote early activity.[44] Physicians inject a wide variety of medications and often prepare them without the use of a sterile hood. Infection can be introduced when these injections are compounded or at the time of injection. Although no definitive evidence shows an increased risk of SSI associated with use of periarticular injections, medication cocktails should be compounded in a sterile fashion in the pharmacy and be administered with aseptic technique.[45]

Risk Factors

SSI acquisition depends on exposure to bacteria and the host's ability to control the inevitable bacterial contamination of a surgical wound. The likelihood of developing an SSI is a complex interaction among several variables, including overall host characteristics (ie, age, immunosuppression, obesity, diabetes), effectiveness of antimicrobial prophylaxis, surgical site tissue condition and presence of foreign material, and degree of wound contamination.

Table 3 provides a summary of known SSI risk factors, which are also discussed in detail later. Some risk factors that increase risk of SSI are nonmodifiable, such as gender and age. However, other risk factors are modifiable, and their optimization can decrease the likelihood of developing an SSI.

Patient Related, Nonmodifiable

Age

Several studies identify the extremes of age as a risk factor for SSI. The risk of SSI is higher in infants compared with older children and higher among older adults compared

Table 3
Summary of known surgical site infection risk factors

Risk Factor	Pathophysiology
Patient Related, Modifiable	
Diabetes	Hyperglycemia impairs innate immunity mechanism to fight bacteria. In addition, increased glucose level leads to glycosylation of proteins, which in turn slows wound healing[52]
Malnutrition	Poor nutrition leads to poor tissue healing, decreased collagen synthesis, and granuloma formation in surgical wounds. Low albumin level impairs macrophage activation and induces macrophage apoptosis, which decreases innate immunity response. Hypoalbuminemia can lead to tissue edema and leakage of interstitial fluid into the surgical wound[57]
Smoking tobacco	Tobacco smoke impairs wound healing by vasoconstriction, leading to relative ischemia, reduced inflammatory response, and alteration in collagen metabolism[61]
Obesity	Decreased blood flow in adipose tissue leads to less oxygen and antibiotic delivery[64,66,67]
Immunosuppressive medications and conditions	Immunosuppressive medications or clinical conditions blunt the inflammatory phase of wound healing[68,69]
Decreased tissue oxygenation	Decreased tissue oxygenation leads to diminished oxidative killing by neutrophils and impaired tissue healing caused by reduced collagen formation, neovascularization, and epithelialization. Low oxygen levels may decrease the efficacy of perioperative antibiotics[72,73]
Perioperative hypothermia	Perioperative hypothermia impairs host defenses against surgical wound contamination: vasoconstriction causing reduced tissue perfusion to wounded tissue with reduced access for key immune cells, decreased motility of key immune cells, and reduced scar formation[74]
Postoperative hyperglycemia	Cellular functions of leukocyte adherence, chemotaxis, phagocytosis, and bactericidal activity are improved by insulin and better glycemic control, suggesting a direct relation between cellular function deficits and increased blood glucose level[56]
Anticoagulation	Anticoagulants can cause of persistent oozing of the incision, slow wound healing[75]
Blood transfusions	Blood transfusions affect the risk of infection by modulating the immune system[76]

(*continued on next page*)

Table 3
(continued)

Risk Factor	Pathophysiology
Patient Related, Nonmodifiable	
Age	The skin's dermis and basement membrane thin with increasing age, and the skin loses its supply of cutaneous nerves and blood vessels, which can lead to poor wound healing[46]
History of radiation	Radiation therapy produces underlying tissue damage and contributes to poor wound healing
History of prior SSTI	A prior history of SSTIs may be related to differences in inherent immunity and susceptibility to infection[51]
Exogenous Sources	
Wound contamination from operating room personnel	Transition of skin flora on the hands of health care personnel to the patient and operating room from lack of appropriate hand washing or gloving to surgical sites[23] Movement of microorganisms from surgical staff's hair, mouths, bodies, or shoes to the operating room contaminates surgical wounds[23]
Airborne contamination	Increasing the number of microorganisms in the operating room environment increases opportunity for SSI. Most of the airborne contamination comes from persons present in the operating room and their movements[34,35]
Operation duration	Longer operative duration is associated with increased wound contamination, increased damage to wound cells, and the local environment[28]
Surgical technique	Not maintaining adequate blood supply, not gently handling tissue, inadvertent entry into hollo viscus, leaving behind devitalized tissue, inappropriate use of drains and sutures, and inappropriate postoperative wound management[14]
Foreign material	Foreign material promotes inflammation at the surgical site and increases the risk of SSI[29,30]
Intra-articular steroid injection	Infection may be introduced at the time of injection, especially if rigorous antisepsis is not applied. May decrease the host immune response to the introduction of such bacteria[81]
Periarticular joint injections	Injections are often prepared without the use of a sterile hood; infections can be introduced when these injections are compounded or at the time of infusion,

(*continued on next page*)

Table 3
(continued)

Risk Factor	Pathophysiology
	because catheters are commonly used to deliver the medications[45]
Wound care	Wounds that remain uncovered after surgery may be subject to environmental contamination or ongoing drainage that decreases the integrity of the surrounding skin[38,39]
Endogenous Sources	
Wound contamination from patient	Shaving creates microscopic cuts in the skin that later serve as niduses for bacteria to multiply[14] Absence of appropriate barrier devices and drapes allows bacteria from deeper skin layers and hair follicles to recolonize the surgical site during the operation Lack of or inappropriate administration of perioperative antibiotics does not prevent the inevitable burden of microorganisms at surgical site[24] Without appropriate surgical site preparation, soil and transient organisms are not removed[117] Wound classification delineates the degree of contamination of a surgical wound at the time of the operation[118]

with younger cohorts.[46] With increasing age, the skin's dermis and basement membrane thins. In addition, the skin loses its supply of cutaneous nerves and blood vessels. These physiologic changes contribute to slow or impaired wound healing.[47,48]

However, the risk of SSI may only increase up until a certain age. Kaye and colleagues[49] found that, after age 65 years, the risk of SSI decreased by 1.2% for each additional year of life. Therefore, the risk of SSI may be caused by comorbidities and immunosuppression and not directly by increasing age. Furthermore, this result may indicate a selection bias of healthier older patients for surgery.

History of radiation

History of prior radiation therapy at the site of surgery increases SSI risk because of the risk of underlying tissue damage.[50] Irradiated skin is hypovascular and easily injured with slight trauma. Given the damaged tissue and lack of perfusion, surgical incisions in locations with prior radiation treatment are more likely to develop a wound complication.

History of prior skin and soft tissue infection

History of a prior skin and soft tissue infection (SSTI) is another risk factor for SSI development.[51] Although not fully understood, the increased SSI risk among these patients may reflect differences in inherent immunity and susceptibility to infection.

Patient Related, Modifiable

Diabetes mellitus

Patients with diabetes mellitus are more likely to develop SSIs. In a meta-analysis including 14 prospective studies, patients diagnosed with diabetes were twice as

likely to develop an SSI compared with patients without a diagnosis of diabetes.[52] The increased risk of SSI among patients with diabetes is consistent across multiple surgical procedures and is likely multifactorial.[53] Patients with diabetes have a high incidence of small vessel disease, leading to impaired oxygen and nutrition delivery to peripheral tissues. Hypoxemia and lack of nutritional support reduce the systemic ability to prevent infection.[54] Dronge and colleagues[55] found that patients with a hemoglobin A1c level more than 7% were significantly more likely to develop infectious complications compared with patients with a hemoglobin A1c level less than 7%.

Postoperative hyperglycemia

Postoperative hyperglycemia may increase SSI risk more than a diagnosis of diabetes. Hyperglycemia impairs innate immunity mechanism to fight bacteria. In addition, increased glucose level leads to glycosylation of proteins, which in turn slows wound healing. Latham and colleagues[56] found that hyperglycemia during the immediate postoperative period was an independent risk factor for developing SSI even among patients without a history of diabetes, and the risk of infection correlated with the degree of glucose increase. Patients with blood glucose level of 200 mg/dL or higher within 48 hours after surgery had 2.5 times higher odds of developing an SSI than patients with glucose level less than 200 mg/dL.

Malnutrition

Malnutrition is prevalent among surgical patients. One of the most commonly used markers of malnutrition is albumin, and hypoalbuminemia increases the risk of SSI.[57] Hypoalbuminemia may lead to increased risk of SSI through several mechanisms. First, hypoalbuminemia can lead to poor tissue healing, decreased collagen synthesis, and granuloma formation in surgical wounds.[58] These factors can impair wound healing and predispose the tissue to infection. Second, low albumin level impairs macrophage activation and induces macrophage apoptosis, which decreases innate immunity response.[59] Lastly, hypoalbuminemia can lead to tissue edema and leakage of interstitial fluid into the surgical wound.[60] This fluid can serve as a medium for bacteria to proliferate and ultimately lead to infection.

Smoking

Smoking tobacco is associated with adverse outcomes following surgery, including SSI. Postoperative wound healing complications occur more often in smokers and former smokers compared with those who never smoked. A systematic review identified 4 randomized trials that assessed the effect of preoperative smoking cessation (4-week to 8-week interval of abstinence) on postoperative wound healing. Current or past smokers had an increased risk for postoperative infection (odds ratio [OR], 1.9; CI, 1.0–3.5).[61] Other studies have shown that abstinent smokers have a lower SSI risk than do current smokers.[62]

The many compounds that constitute tobacco smoke impair wound healing and increase SSI risk through several mechanisms.[63] The physiologic mechanisms include vasoconstriction, which causes relative ischemia of operated tissues. Tobacco smoke also leads to a reduced inflammatory response and impaired innate immune system response to bacteria.[63] Lastly, the elements in tobacco smoke can alter collagen metabolism, which is essential for skin and tissue integrity.

Obesity

Obesity is another risk factor associated with developing SSI. One meta-analysis included 20 studies that evaluated SSI outcomes in orthopedic surgeries. The investigators found that the risk of SSI for patients with obesity was almost 2 times the SSI

risk for patients without obesity (risk ratio [RR], 1.915; 95% CI, 1.53–2.40).[64] Studies including colorectal surgery patients (OR, 1.59; 95% CI, 1.32–1.91)[65] and coronary artery bypass surgery patients (OR, 1.8; 95% CI, 1.4–2.3)[66] have reported similar conclusions. An additional recent study also found a trend of increasing risk of SSI for almost all surgery types when body mass index increased from normal to morbidly obese.[67]

Obese patients may be at increased risk for SSI because of depth of adipose tissue, creation of dead space, and decreased blood flow in adipose tissue. Without adequate blood flow reaching the tissues, surgical wounds are less likely to heal. Decreased blood flow may also reduce antibiotic delivery and increase wound tension.[28]

Immunosuppressive medications and conditions

Patients with suppressed immune systems are at increased risk of SSI because the inflammatory phase of wound healing may be blunted. The increase in SSI risk is seen in patients with various levels of immunosuppression, including transplant recipients, patients undergoing chemotherapy, and other patients taking immunosuppressing medications.[68,69] In contrast, glucocorticoids may not affect SSI risk as strongly as other immunosuppressive therapies.[70] Some degree of antiinflammation may prevent wounds from becoming chronically inflamed, whereas significant suppression of inflammation can prevent wound healing.[71]

Decreased tissue oxygenation

Low oxygenation also increases the risk of SSIs. Oxygen tension is often low in wounds and in colorectal anastomoses at the end of surgery, which may reduce bacterial eradication, the body's defenses against bacteria, and tissue healing. Possible mechanisms include diminished oxidative killing by neutrophils and impaired tissue healing caused by reduced collagen formation, neovascularization, and epithelialization.[72] Further, many of the antibiotics used perioperatively for SSI prophylaxis are oxygen dependent in their effect,[73] and low oxygen levels may decrease their effectiveness.

Perioperative hypothermia

Maintaining normal body temperature is vital for the body to maintain its normal function. However, many factors that patients are exposed to in the operating room can cause hypothermia: anesthetic drugs, cold operating room, skin antisepsis, cold irrigation of a patient with the body uncovered, and the use of intravenous solutions. Most cellular functions are temperature dependent, and hypothermia also provokes systemic responses.[74] Several mechanisms help to explain why perioperative hypothermia impairs host defenses against surgical wound contamination, including vasoconstriction and subsequent diminished perfusion, decreased motility of key immune cells, and reduced scar formation, which is necessary to prevent wound dehiscence and recontamination.

Anticoagulation

Although postoperative anticoagulation is an evidence-based practice to prevent deep vein thrombosis in the postoperative period, anticoagulants may increase the risk of SSIs. Several studies have implicated anticoagulation therapy as a cause of persistent oozing of the incision, slow wound healing, and subsequent SSIs.[75]

Blood transfusions

Red blood cell transfusion also increases the risk of SSI.[76] Blood transfusions increase the risk of infection by impairing normal monocyte function and decreasing tumor necrosis α production in response to endotoxin (lipopolysaccharide).[77]

GUIDELINES FOR PREVENTION

Several detailed published guidelines for SSI prevention exist, including the following 4 published primary guideline documents:

1. "Strategies to Prevent Surgical Site Infections in Acute Care Hospitals: 2014 Update." [78] The Society for Healthcare Epidemiology of America (SHEA) sponsored this document, and members of numerous other organizations with SSI prevention expertise also contributed.
2. "Global Guidelines for the Prevention of Surgical Site Infection."[79] The World Health Organization (WHO) published this updated document in 2018.
3. "American College of Surgeons and Surgical Infection Society: Surgical Site Infection Guidelines, 2016 Update."[80]
4. "Centers for Disease Control and Prevention Guideline for the Prevention of Surgical Site Infection, 2017."[81]

Based on these guideline documents, available evidence, and our personal experience, the authors have categorized SSI prevention strategies into 3 categories: best practices that all acute care hospitals should follow, additional interventions with possible benefit, and unproven or controversial interventions.

Recommendations

The authors have identified 9 core best practices that are consistently recommended by SSI prevention experts and guideline documents (**Table 4**). The authors recommend that all acute care hospitals incorporate each of the following SSI prevention measures into surgical protocols:

Preoperative bathing

Preoperative bathing or showering on the night before or day of surgery is a simple, low-risk intervention that decreases bacterial load of the skin, including the surgical site.[82] Many experts strongly recommend this practice as a standard of care for SSI prevention,[81] but definitive reduction in SSI risk has not been proved. Furthermore, for routine preoperative bathing, the ideal soap or antiseptic agent is not known, and many hospitals implement bathing protocols based on local epidemiology and cost considerations (preoperative bathing with chlorhexidine gluconate [CHG] is discussed later).

Antimicrobial prophylaxis

Antimicrobial prophylaxis given shortly before surgical incision is indicated for most clean-contaminated procedures, as well as certain clean procedures with severe consequences of infection (eg, procedures involving implantation of prosthetic implants).[79] Multispecialty consensus guidelines recommend preferred antibiotic agents based on common pathogens known to cause SSI following particular procedures; however, local epidemiology of SSI and hospital antibiograms should also influence antibiotic selection.[83] Antimicrobial prophylaxis protocols should promote optimized timing and dose of antibiotics, including redosing of antibiotics during surgery for prolonged procedures, or when blood loss is excessive. For nonemergent colorectal procedures, oral antibiotics and mechanical bowel preparation should be provided on the day before surgery, in addition to intravenous antibiotic prophylaxis before skin incision.[78–80] Prophylactic antibiotics should be discontinued after closure of incision because of increased risk of adverse events and because no SSI reduction benefit associated with postoperative antibiotic prophylaxis has been shown.[79,81]

Table 4
Summary of surgical site infection primary prevention guidelines, risk factors, and best-practice recommendations

Prevention Guideline	Risk Factor Addressed	Recommendations and Supporting Evidence
Preoperative bathing	Endogenous bacterial contamination of surgical site	• Patients should bathe their full bodies with soap or an antiseptic agent on at least the night before or day of surgery[79,81]
Antimicrobial prophylaxis	Endogenous and exogenous bacterial contamination of surgical site	• Administer prophylactic antibiotics for indicated procedures according to evidence-based guidelines[83] • Select agent based on common pathogens that cause SSI for procedure being performed, published guidelines, and local antibiograms.[83] • Begin antibiotic infusion within 60 min before incision; however, for vancomycin and fluoroquinolones, begin infusion within 120 min before incision[78,80,83] • Adhere to guidelines for antibiotic doses, including weight-based vancomycin dosing and 3-g cefazolin doses for patients with weight >120 kg.[78,83] • Redose antibiotics during surgery for prolonged procedures or for procedures with excessive blood loss[78,83] • Provide oral antibiotics, intravenous antibiotics, and mechanical bowel preparation before nonemergent colorectal procedures[78–80] • Discontinue perioperative antibiotics after closure of surgical incision[79,81]
Perioperative preparation of operative site	Endogenous and exogenous bacterial contamination of surgical site	• Avoid hair removal at operative site unless hair interferes with surgery. If hair must be removed,

(*continued on next page*)

Table 4
(*continued*)

Prevention Guideline	Risk Factor Addressed	Recommendations and Supporting Evidence
		remove hair outside of operating room with a clipper or depilatory agent[78,79] • Use an alcohol-based antiseptic solution containing chlorhexidine for surgical skin preparation, unless a contraindication exists[79] • Use chlorhexidine or povidone-iodine solutions that contain low concentrations (eg, 4%) or no alcohol for vaginal antisepsis before hysterectomy and vaginal procedures, including cesarean section[87,88]
Surgical hand preparation	Exogenous bacterial contamination of surgical site	• Use an appropriate antiseptic agent to perform surgical hand scrub for the length of time recommended by the manufacturer[78,79]
Maintenance of normothermia	Hypothermia-induced vasoconstriction and tissue hypoxia at surgical site	• Use warming devices in the operating room before and during the surgical procedure to maintain body temperature of at least 36.0°C[78,79]
Hyperoxygenation	Tissue hypoxia at surgical site	• For patients undergoing general anesthesia and tracheal intubation and who have normal pulmonary function, provide 80% Fio_2 intraoperatively and, when possible, for 2–6 h after surgery[78,79,81]
Wound protectors	Endogenous and exogenous bacterial contamination of surgical site	• Use impervious plastic wound protectors for open abdominal surgery, particularly colorectal and biliary tract procedures[78,80]
Glucose control	Hyperglycemia	• Maintain immediate postoperative blood

(*continued on next page*)

Table 4
(*continued*)

Prevention Guideline	Risk Factor Addressed	Recommendations and Supporting Evidence
		glucose of ≤180 mg/dL for diabetic and nondiabetic patients[78–80]
Perioperative checklist	Potential for noncompliance with best practices	• Use a checklist based on the WHO Surgical Safety Checklist[90] to improve compliance with best practices, including SSI prevention strategies[78]

Abbreviation: Fio_2, fraction of inspired oxygen.

Perioperative preparation of operative site

Hair removal should be performed only if hair interferes with surgery.[78,84] When hair removal is necessary, a clipper or depilatory agent should be used outside of the operating room. Hair shaving should not occur. If a clipper is used, a disposable clipper head should be used and changed between patients.

Surgical skin preparation should be performed with an alcohol-containing antiseptic solution, unless a contraindication to the alcohol component exists (eg, procedures involving mucosa, cornea, or ear).[78] Alcohol-based skin preparation agents are flammable, and operating room personnel should take precautions to avoid fires in the operating room, especially for procedures using an electrosurgical device (eg, Bovie).[85] Personnel should allow the skin preparation solution to dry per manufacturer instructions for use and avoid pooling of the solution.

WHO guidelines preferentially recommend the use of CHG-alcohol solutions rather than povidone-iodine-alcohol solutions,[79] whereas other organizations do not make specific recommendations regarding type of alcohol-containing solution. Although few head-to-head studies have been performed to compare efficacy of CHG versus povidone-iodine–containing solutions, CHG alcohol was superior to povidone-iodine alcohol in a randomized controlled trial analyzing SSI prevention following cesarean section.[86] As a result, the authors preferentially recommend use of CHG alcohol for skin antisepsis. Vaginal cleansing with either 4% CHG or povidone-iodine should occur before hysterectomy and vaginal procedures, including cesarean section.[87,88]

Surgical hand preparation

Surgical hand preparation should be performed with either a suitable antimicrobial soap or alcohol-based hand rub.[78,79] Hand preparation should occur for the duration recommended by the manufacturer of the antiseptic agent chosen.

Maintenance of normothermia

Preoperative and intraoperative warming devices should be used in order to maintain temperature of at least 36.0°C.[74,78,84] In addition to decreasing SSI risk, maintenance of normothermia may also decrease blood loss and transfusion requirements.

Hyperoxygenation

Patients who undergo surgery with general anesthesia and tracheal intubation and who have normal pulmonary function should receive 80% fraction of inspired oxygen (Fio_2) intraoperatively and, when feasible, for 2 to 6 hours postoperatively.[78,79] The

benefit of hyperoxygenation may be greatest for open colorectal surgery. Updated WHO guidelines continue to recommend hyperoxygenation despite exclusion of 2 studies that showed benefit but have subsequently received scrutiny for possible data integrity breaches.[89]

Glucose control

Blood glucose should be monitored and controlled in the immediate postoperative time period for all surgical patients, including patients with and without a diagnosis of diabetes mellitus.[78–80] Protocols should be used to ensure that postoperative glucose monitoring occurs and that insulin is used to maintain glucose level less than or equal to 180 mg/dL for at least the first 24 hours after surgery. At a minimum, we recommend that all hospitalized postoperative patients without diabetes mellitus have 1 blood glucose value checked in the first 24 hours after surgery.

Perioperative checklist

Acute care hospitals should use surgical safety checklists to improve compliance with best practices for SSI prevention.[78] The authors recommend that hospitals modify the 19-item WHO Surgical Safety Checklist[90] to include additional SSI prevention practices detailed here, as well as other important SSI prevention measures applicable to the specific hospital or procedure.

Considerations

Numerous potentially effective SSI prevention strategies are not included in the 9 core best practices listed earlier. Compared with the core interventions, these additional strategies do not have the same strength of endorsement by SSI prevention guidelines or experts and have fewer data to support universal implementation. However, especially for hospitals, procedures, or patient populations with increased SSI rates despite adherence to core SSI prevention measures, implementation of additional strategies may help to decrease SSI risk.[78] This article discusses the following 4 commonly used auxiliary strategies for SSI prevention.

Staphylococcus aureus screening and decolonization

Patients undergoing high-risk cardiothoracic, orthopedic, or neurosurgical procedures known to be colonized with *S aureus* should receive preoperative intranasal mupirocin 2% ointment or an alternative intranasal agent with antistaphylococcal activity, as well as CHG bathing for up to 5 days before surgery.[78,84] However, no standardized approach exists for *S aureus* screening or decolonization, and guideline documents give conflicting recommendations regarding the practice of universal decolonization of patients undergoing high-risk procedures. For example, the WHO strongly recommends against routine decolonization of patients not known to be *S aureus* carriers, primarily to avoid spread of resistance to mupirocin.[79]

Although optimal strategies for *S aureus* screening and targeted decolonization versus universal decolonization before high-risk surgical procedures continue to be debated, experts generally agree that routine universal *S aureus* preoperative screening is not necessary before lower-risk procedures.[78,79] Nevertheless, some hospitals may consider screening and decolonization for certain patients and procedures depending on SSI rates caused by *S aureus*; patient-specific risk factors for *S aureus* SSI; and availability of resources to reliably screen, follow up screening results, and implement protocolized decolonization strategies for patients found to have nasal *S aureus* carriage. In addition, although decolonization protocols typically recommend preoperative application of 2% nasal mupirocin twice daily for several days close to the date of planned surgery,[80] application of intranasal povidone-

iodine swabs on the day of surgery shortly before incision may be an effective, more practical, and less expensive alternative that precludes concerns regarding resistance to mupirocin.[91,92]

Colorectal surgery bundles

Multiple studies have shown the potential for implementation of colorectal surgery best-practice bundles to improve outcomes, including SSI rates.[93] Colorectal surgery bundles commonly include elements of the 9 core best practices for SSI prevention detailed in this article, as well as additional, less-proven interventions. Based on success of bundles, theoretic benefit, and low risk of harm, the authors support implementation of additional strategies to be included for bundles for colorectal SSI prevention, such as gown and glove change before fascial and skin closure and use of a dedicated wound closure tray.

Operating room traffic control

Increased operating room foot traffic and door openings can disrupt air quality and flow, potentially increasing microbial concentrations at the surgical site and SSI risk.[36,37] The authors support interventions designed to decrease unnecessary operating room traffic and door openings.

Operating room disinfection

Attention must be paid to operating room maintenance and care, including air handling and disinfection of the environment and equipment.[14,78] Adjunctive disinfection practices, such as use of ultraviolet light in the operating room, might provide additional benefit but require further study.[94]

Controversies

Preoperative chlorhexidine gluconate bathing

Although all patients should receive a bath or shower the night before or evening of surgery, insufficient evidence exists to recommend universal preoperative use of CHG.[79] However, if not cost-prohibitive, CHG is a reasonable agent to use for preoperative bathing. Use of 2% CHG-impregnated cloths may provide greater reduction in SSI risk than use of CHG soap.[95]

Local antibiotics

Local and topical antibiotics are commonly used during surgery for the purpose of decreasing SSI risk. However, in general, high-quality data supporting these practices are sparse. For example, powdered vancomycin is commonly applied to surgical sites, but available data are insufficient to recommend its widespread use for SSI prevention. Furthermore, use of powdered vancomycin could be associated with local adverse effects.[96] Another example is use of gentamicin-collagen sponges to prevent SSI. Single-center trials suggested benefit when these sponges were used in colorectal surgery, but a multicenter randomized trial showed harm.[78,97] Meta-analyses have suggested that gentamicin-collagen sponges might be more successful in decreasing SSI risk following cardiothoracic surgery.[98]

Surgical wound irrigation

Incisional wounds are commonly irrigated with saline, antiseptics, or antibiotics for the purpose of SSI prevention. No consensus exists among best irrigation practices for SSI prevention, and high-quality data are not available to support routine irrigation with antibiotics. Incisional wound irrigation with povidone-iodine might be more effective in decreasing SSI risk than saline irrigation, but this topic requires further study.[79]

Antiseptic drapes

Some surgeons use adhesive plastic incise drapes, often impregnated with an antiseptic agent. These drapes are applied to patient skin after skin antisepsis is performed, and the surgeon cuts through the drape and skin. Available data do not support routine use of antiseptic drapes for SSI prevention.[78,84]

Antiseptic-impregnated sutures

Triclosan-coated sutures are commonly used to decrease SSI risk, but data evaluating this practice are mixed.[99] In addition, limited data have suggested possible negative effect on wound healing.[100] Guideline recommendations regarding triclosan-coated sutures are contradictory and range from a recommendation against routine use[78] to conditional recommendations supporting their use.[79,80]

Advanced dressings

Advanced dressings for primarily closed surgical wounds, such as silver-containing or antimicrobial-impregnated dressings, have not been proved to significantly decrease SSI rates compared with standard dressings.[80,84] Regardless of type of dressing used, dressings should be sterile and be placed and changed using aseptic technique.

Wound protectors

Plastic wound protectors can facilitate retraction of an incision without requiring additional mechanical retractors and can decrease SSI risk after open abdominal surgeries.[78,80] Benefit may be greatest for colorectal and biliary tract procedures.

Prophylactic negative pressure wound therapy

Routine use of prophylactic negative pressure wound therapy has not been shown to decrease SSIs. A recent, large, randomized clinical trial found that there was no significant difference in the risk of SSI after cesarean delivery in obese women with prophylactic negative pressure wound therapy (3.6%) versus standard wound dressing (3.4%).[101] Low-quality evidence suggests that prophylactic negative pressure wound therapy on primarily closed, high-risk surgical wounds decreases SSI risk compared with use of standard wound dressings.[79,80] Examples of high-risk wounds include wounds complicated by surrounding soft tissue damage, poor blood flow, hematoma, or intraoperative contamination. The pressure level or duration of negative pressure therapy needed to maximize SSI risk reduction is not known.

THERAPEUTIC OPTIONS

Surgical debridement and removal of the infected tissue is the most important facet of therapy for many SSIs, with antimicrobial therapy being an important adjunct component.[102] However, the type of debridement and duration of antimicrobial therapy depend on the depth and anatomic site of infection and the presence of prosthetic material. However, deep-incisional and organ/space infections almost universally require operative evacuation of the infected tissue.

Superficial-incisional SSIs can typically be treated with oral antibiotics and without surgical debridement. In contrast, patients with systemic symptoms (temperature >38.5°C, heart rate >110 beats/min) or with suspected deep-incisional or organ/space SSI generally require exploration of the surgical site in addition to antibiotics.[102] The specific antibiotic and duration of therapy are determined by the location of the infection, the depth of infection, the adequacy of surgical debridement, and resistance patterns of the causative pathogens. However, in general, clinicians should initiate systemic antibiotic therapy when a patient has systemic symptoms of infection or a clinician suspects a deep-incision or organ/space SSI. For example, 1 study found

that patients with mediastinitis who receive antibiotics active against the identified pathogen within 7 days of debridement had a 60% reduction in mortality compared with patients who did not receive effective antibiotic therapy.[103]

ASSESSMENT

The CDC conducted the Study of the Efficacy of Nosocomial Infection Control (SENIC) to determine the cost-effectiveness of infection prevention activities, including surveillance.[104] These and many subsequent studies concluded that infection surveillance programs and feedback of SSI rates to surgeons decrease overall SSI rates by 32% to 50%.[105,106] These data form the basis for the CDC's recommendation that hospitals routinely perform surveillance for SSIs and report the information back to surgeons.[14]

Surveillance Methodology

Strategies for targeting surveillance by procedure type

Given the significant resources required to perform SSI surveillance, many infection prevention programs focus their SSI surveillance efforts on a subset of patients. Hospitals may determine surgical populations for targeted surveillance in several ways. One strategy is to target high-volume surgical procedures for surveillance, because SSIs related to these procedures would pose risk to a large number of patients. Examples of high-volume procedures include colorectal procedures, abdominal hysterectomies, and hip and knee arthroscopies. Notably, US hospitals are required to publicly report rates of SSI following colorectal and abdominal hysterectomy procedures. Another strategy is to target high-risk procedures for surveillance, because SSIs after these procedures convey high risk of morbidity. Examples of high-risk procedures include spinal fusion and craniotomies. A third strategy is to focus surveillance efforts on surgical procedures that have rates of SSI that are higher than expected at that institution (based on historical comparisons). Typically, infection prevention programs use a combination of these targeting strategies.

Strategies for surgical site infection case finding

Direct prospective case finding through daily chart review and observation of the surgical site by infection prevention personnel is considered the most sensitive and rigorous method to identify patients with SSI.[106] However, direct surveillance is no longer practiced for several reasons. First, daily observation of the wound is resource intensive and impractical for modern infection prevention programs. Second, indirect methods that involve medical record review without direct wound observation have shown sensitivity of 84% to 89% and specificity of 99.8%, and they require fewer resources compared with direct observation.[107,108] In addition, most SSIs occur following discharge from the index hospitalization and can only be detected through postdischarge surveillance methods.[109]

Many infection prevention programs screen records for possible indicators of SSI and proceed with complete chart review only when an indicator is present. NHSN suggests several methods to identify patients with possible SSI, including review of medical records for signs and symptoms of SSI; review of admission, readmission, emergency room visits, and operating room logs; review of laboratory, imaging, or other diagnostic test reports; review of clinician notes; International Classification of Diseases-10 Clinical Modification (ICD-10-CM) Infection Diagnosis codes; surgeon surveys; and patient surveys.[16] However, NHSN states that facilities may use any combination of methods to identify potential patients with SSI. Therefore, considerable variability among case-finding methods across facilities exists.

Microbiology data

Many SSI surveillance programs use positive microbiology results as an indicator of possible SSI. However, microbiology data are not perfect indicators of SSI because cultures or other microbiology studies are not obtained in every case; superficial wound infections are frequently treated with local wound management with or without antibiotics. Studies have shown that surveillance systems that rely solely on microbiologic data only identify 33% to 65% of all SSIs.[110]

Antimicrobial administration

Antibiotic use beyond the expected number of postoperative days (used for perioperative prophylaxis) can be used to predict SSI.[110,111] However, postoperative antibiotic use alone is not sensitive or specific for SSI detection. Some patients continue to receive antibiotics following a surgical procedure for prophylaxis or treatment of a preexisting infection. In addition, antibiotics are not always required for management of superficial SSIs.[102] Therefore, antibiotics should only be used in combination with other parameters to screen patients for possible SSI.

Administrative claims data

Several studies have evaluated the utility of using administrative billing data as an indicator of SSI.[111–113] Using administrative claims data increases detection of SSIs relative to standard surveillance methods.[114] In addition, studies have found that using administrative data as a flag for further chart review by infection preventionists saves time and significantly decreases the burden of surveillance compared with standard methods.[113,114] In addition, US Centers for Medicare and Medicaid Services (CMS) Medicare claims data can be used to accurately distinguish between hospitals with high and low rates of infection following certain procedure types.

However, several limitations to using administrative data to facilitate surveillance exist. First, billing is performed retroactively. Therefore, surveillance systems that rely on administrative data as the first trigger to review charts for SSI cannot detect SSI in real time and intervene or provide feedback in a timely manner. Second, recent studies of administrative data for SSI surveillance have typically been performed on clean procedures, including cardiac, arthroplasty, vascular, and breast procedures. The utility of administrative billing data as an indicator of SSI following other procedure types is unclear.

Electronic surveillance

With the evolution of the electronic medical record, infection prevention programs and software vendors are exploring automated methods to detect patients with SSI. However, at this time, no single effective SSI prediction tool exists. For example, 1 study of an automated algorithm that used specified laboratory parameters and antimicrobial use to detect SSIs found the model performed poorly, with overall sensitivity of 37.8%, and had poor interhospital ability to generalize.[115] Development of electronic surveillance tools with improved accuracy is an area of ongoing research.

Surgical site infection metrics and data feedback

SSI rates are typically calculated per 100 procedures and may be stratified by procedure type and surgeon. SSI rates should be periodically assessed for trends and reported to surgeons, perioperative services staff, and hospital leaders. Peer-to-peer comparative SSI rate data may be helpful to identify surgeons whose rates are higher than those of peers; however, without adequate adjustment for difference in patient-level or procedure-level risk factors, peer-to-peer comparative data should be used for performance improvement purposes only.

Surveillance systems that detect SSIs in near real time allow for concurrent feedback to surgical and perioperative personnel as SSIs are identified. This type of surveillance allows performance improvement teams to investigate for potential lapses in patient care (eg, missed administration of perioperative antibiotic, failure to maintain postoperative glucose control) and take actions to improve compliance with recommended best practices. Because SSIs are low-frequency events, it can sometimes be difficult for infection prevention and perioperative teams to detect when there is a meaningful increase or cluster of infections over baseline. Use of statistical process control charts to analyze SSI surveillance data and alert staff when a potential cluster has occurred may promote earlier detection of important SSI rate increases.[116]

In summary, surveillance for SSI and data feedback are important functions of infection prevention teams. However, methods for conducting surveillance and the quality of surveillance vary across health care facilities and depend on availability of resources. Infection prevention programs should periodically evaluate their surveillance programs to ensure they are maximally effective and reflect the most recent recommended best practices for SSI prevention.

SUMMARY

SSIs are the most common and costly health care–associated infections in the United States[1,2,7] and lead to significant patient morbidity and mortality.[4,11] However, adhering to evidence-based preventive practices can decrease the rate of SSI. In general, aggressive surgical debridement in addition to appropriate antibiotic therapy is necessary for SSI treatment.

CLINICS CARE POINTS

- Before surgical incision, patients need to receive the appropriate type and dose of antibiotic within the appropriate time frame. Patients may also require additional antibiotic doses for prolonged procedures.
- Perioperative staff should only remove patient hair if it interferes with the surgery, and, even then, hair removal should be done with a clipper or depilatory agent outside of the operating room.
- The skin over the surgical site needs to be cleansed with an alcohol-containing antiseptic solution.
- Providers must perform hand preparation with a suitable antimicrobial soap or alcohol-based hand rub before entering the operating room.
- During the operation, every effort should be made to maintain normothermia, hyperoxygenation, and normal glucose levels.
- Hospitals should use surgical safety checklists to improve compliance with practices for SSI prevention.

DISCLOSURE

Dr Anderson reported grants from CDC Epicenter (U54CK000483) during the conduct of the study; grants from Agency for Healthcare Research and Quality, personal fees from UpToDate, and royalties for authorship outside the submitted work.

REFERENCES

1. Lewis SS, Moehring RW, Chen LF, et al. Assessing the relative burden of hospital-acquired infections in a network of community hospitals. Infect Control Hosp Epidemiol 2013;34(11):1229–30.
2. Zimlichman E, Henderson D, Tamir O, et al. Health care-associated infections: a meta-analysis of costs and financial impact on the US health care system. JAMA Intern Med 2013;173(22):2039–46.
3. Steiner CA, Karaca Z, Moore BJ, et al. STATISTICAL BRIEF# 223. 2017.
4. Agency for Healthcare Research and Quality. Healthcare cost and utilization project - statistics on hospital stays. 2013 web site. Available at: http://hcupnet.ahrq.gov/. Accessed November 17, 2020.
5. Scott RD. The direct medical costs of healthcare-associated infections in US hospitals and the benefits of prevention 2009. Available at: https://www.cdc.gov/hai/pdfs/hai/scott_costpaper.pdf.
6. Allegranzi B, Bagheri Nejad S, Combescure C, et al. Burden of endemic health-care-associated infection in developing countries: systematic review and meta-analysis. Lancet 2011;377(9761):228–41.
7. Magill SS, O'Leary E, Janelle SJ, et al. Changes in prevalence of health care–associated infections in US Hospitals. N Engl J Med 2018;379(18):1732–44.
8. Geubbels EL, Wille JC, Nagelkerke NJ, et al. Hospital-related determinants for surgical-site infection following hip arthroplasty. Infect Control Hosp Epidemiol 2005;26(5):435–41.
9. Baker AW, Dicks KV, Durkin MJ, et al. Epidemiology of surgical site infection in a community hospital network. Infect Control Hosp Epidemiol 2016;37(5):519–26.
10. Anderson DJ, Hartwig MG, Pappas T, et al. Surgical volume and the risk of surgical site infection in community hospitals: size matters. Ann Surg 2008;247(2):343–9.
11. Anderson DJ, Kaye KS, Chen LF, et al. Clinical and financial outcomes due to methicillin resistant Staphylococcus aureus surgical site infection: a multicenter matched outcomes study. PLoS One 2009;4(12):e8305.
12. Martone WJ, Nichols RL. Recognition, prevention, surveillance, and management of surgical site infections: introduction to the problem and symposium overview. Clin Infect Dis 2001;33(Suppl 2):S67–8.
13. Kirkland KB, Briggs JP, Trivette SL, et al. The impact of surgical-site infections in the 1990s: attributable mortality, excess length of hospitalization, and extra costs. Infect Control Hosp Epidemiol 1999;20(11):725–30.
14. Mangram AJ, Horan TC, Pearson ML, et al. Guideline for prevention of surgical site infection, 1999. Infect Control Hosp Epidemiol 1999;20(4):247–80.
15. Estimating the additional hospital inpatient cost and mortality associated with selected hospital-acquired conditions. Agency for healthcare research and quality. 2017. Available at: https://www.ahrq.gov/hai/pfp/haccost2017-results.html. Accessed October 20, 2019.
16. Centers for Disease Control and Prevention. Surgical Site Infection (SSI) event web site. Available at: https://www.cdc.gove/nhsn/pdfs/pscmanual/9pscssicurrent.pdf. Accessed November 20, 2020.
17. Dicks KV, Lewis SS, Durkin MJ, et al. Surveying the surveillance: surgical site infections excluded by the January 2013 updated surveillance definitions. Infect Control Hosp Epidemiol 2014;35(5):570–3.
18. Wenzel RP. Surgical site infections and the microbiome: an updated perspective. Infect Control Hosp Epidemiol 2019;40(5):590–6.

19. Seidelman JL, Baker AW, Ge M, et al. 905. Surgical site infections following colon surgery in a large network of community hospitals. Open Forum Infect Dis 2020;7(Supplement_1):S486–7.
20. Baker AW, Lewis SS, Alexander BD, et al. Two-phase hospital-associated outbreak of Mycobacterium abscessus: investigation and mitigation. Clin Infect Dis 2017;64(7):902–11.
21. Everett ED, Pearson S, Rogers W. Rhizopus surgical wound infection with elasticized adhesive tape dressings. Arch Surg 1979;114(6):738–9.
22. Wenger PN, Brown JM, McNeil MM, et al. Nocardia farcinica sternotomy site infections in patients following open heart surgery. J Infect Dis 1998;178(5):1539–43.
23. Leaper D, Edmiston C. World Health Organization: global guidelines for the prevention of surgical site infection. J Hosp Infect 2017;95(2):135–6.
24. Lee KY, Coleman K, Paech D, et al. The epidemiology and cost of surgical site infections in Korea: a systematic review. J Korean Surg Soc 2011;81(5):295–307.
25. Fleischmann W, Meyer H, Baer A. Bacterial recolonization of the skin under a Polyurethane drape in hip surgery. J Hosp Infect 1996;34(2):107–16.
26. Seropian R, Reynolds BM. Wound infections after preoperative depilatory versus razor preparation. Am J Surg 1971;121(3):251–4.
27. Ko W, Lazenby WD, Zelano JA, et al. Effects of shaving methods and intraoperative irrigation on suppurative mediastinitis after bypass operations. Ann Thorac Surg 1992;53(2):301–5.
28. Korol E, Johnston K, Waser N, et al. A systematic review of risk factors associated with surgical site infections among surgical patients. PLoS One 2013;8(12):e83743.
29. Berard F, Gandon J. Postoperative wound infections: the influence of ultraviolet irradiation of the operating room and of various other factors. Ann Surg 1964;160(Suppl 2):1–192.
30. Elek SD, Conen PE. The virulence of Staphylococcus pyogenes for man; a study of the problems of wound infection. Br J Exp Pathol 1957;38(6):573–86.
31. Davidson T, Lewandowski E, Smerecki M, et al. Taking your work home with you: potential risks of contaminated clothing and hair in the dental clinic and attitudes about infection control. Can J Infect Control 2017;32(3):137–42.
32. Fagernes M, Lingaas E. Factors interfering with the microflora on hands: a regression analysis of samples from 465 healthcare workers. J Adv Nurs 2011;67(2):297–307.
33. Parry MF, Grant B, Yukna M, et al. Candida osteomyelitis and diskitis after spinal surgery: an outbreak that implicates artificial nail use. Clin Infect Dis 2001;32(3):352–7.
34. Lidwell OM, Lowbury EJ, Whyte W, et al. Airborne contamination of wounds in joint replacement operations: the relationship to sepsis rates. J Hosp Infect 1983;4(2):111–31.
35. Edmiston CE Jr, Seabrook GR, Cambria RA, et al. Molecular epidemiology of microbial contamination in the operating room environment: Is there a risk for infection? Surgery 2005;138(4):573–9 [discussion: 579–582].
36. Roth JA, Juchler F, Dangel M, et al. Frequent door openings during cardiac surgery are associated with increased risk for surgical site infection: a prospective observational study. Clin Infect Dis 2019;69(2):290–4.

37. Andersson AE, Bergh I, Karlsson J, et al. Traffic flow in the operating room: an explorative and descriptive study on air quality during orthopedic trauma implant surgery. Am J Infect Control 2012;40(8):750–5.
38. Manian FA. The role of postoperative factors in surgical site infections: time to take notice. Clin Infect Dis 2014;59(9):1272–6.
39. Dumville JC, Gray TA, Walter CJ, et al. Dressings for the prevention of surgical site infection. Cochrane Database Syst Rev 2016;12:CD003091.
40. Charalambous CP, Tryfonidis M, Sadiq S, et al. Septic arthritis following intra-articular steroid injection of the knee–a survey of current practice regarding antiseptic technique used during intra-articular steroid injection of the knee. Clin Rheumatol 2003;22(6):386–90.
41. McIntosh AL, Hanssen AD, Wenger DE, et al. Recent intraarticular steroid injection may increase infection rates in primary THA. Clin Orthop Relat Res 2006; 451:50–4.
42. Desai A, Ramankutty S, Board T, et al. Does intraarticular steroid infiltration increase the rate of infection in subsequent total knee replacements? Knee 2009;16(4):262–4.
43. Jevsevar DS, Brown GA, Jones DL, et al. The American Academy of Orthopaedic Surgeons evidence-based guideline on: treatment of osteoarthritis of the knee. J Bone Joint Surg Am 2013;95(20):1885–6.
44. Yin JB, Cui GB, Mi MS, et al. Local infiltration analgesia for postoperative pain after hip arthroplasty: a systematic review and meta-analysis. J Pain 2014; 15(8):781–99.
45. Seidelman J, Baker AW, Anderson DJ, et al. Do periarticular joint infections present an increase in infection risk? Infect Control Hosp Epidemiol 2018;39(7): 890–1.
46. Kaye KS, Anderson DJ, Sloane R, et al. The effect of surgical site infection on older operative patients. J Am Geriatr Soc 2009;57(1):46–54.
47. Fore J. A review of skin and the effects of aging on skin structure and function. Ostomy Wound Manage 2006;52(9):24–35, quiz 36–37.
48. Reddy M. Skin and wound care: important considerations in the older adult. Adv Skin Wound Care 2008;21(9):424–36, quiz 437–438.
49. Kaye KS, Schmit K, Pieper C, et al. The effect of increasing age on the risk of surgical site infection. J Infect Dis 2005;191(7):1056–62.
50. Olsen MA, Lefta M, Dietz JR, et al. Risk factors for surgical site infection after major breast operation. J Am Coll Surg 2008;207(3):326–35.
51. Faraday N, Rock P, Lin EE, et al. Past history of skin infection and risk of surgical site infection after elective surgery. Ann Surg 2013;257(1):150–4.
52. Zhang Y, Zheng QJ, Wang S, et al. Diabetes mellitus is associated with increased risk of surgical site infections: a meta-analysis of prospective cohort studies. Am J Infect Control 2015;43(8):810–5.
53. Martin ET, Kaye KS, Knott C, et al. Diabetes and risk of surgical site infection: a systematic review and meta-analysis. Infect Control Hosp Epidemiol 2016;37(1): 88–99.
54. Turina M, Fry DE, Polk HC Jr. Acute hyperglycemia and the innate immune system: clinical, cellular, and molecular aspects. Crit Care Med 2005;33(7): 1624–33.
55. Dronge AS, Perkal MF, Kancir S, et al. Long-term glycemic control and postoperative infectious complications. Arch Surg 2006;141(4):375–80 [discussion: 380].

56. Latham R, Lancaster AD, Covington JF, et al. The association of diabetes and glucose control with surgical-site infections among cardiothoracic surgery patients. Infect Control Hosp Epidemiol 2001;22(10):607–12.
57. Hennessey DB, Burke JP, Ni-Dhonochu T, et al. Preoperative hypoalbuminemia is an independent risk factor for the development of surgical site infection following gastrointestinal surgery: a multi-institutional study. Ann Surg 2010; 252(2):325–9.
58. Testini M, Margari A, Amoruso M, et al. [The dehiscence of colorectal anastomoses: the risk factors]. Ann Ital Chir 2000;71(4):433–40.
59. Rivadeneira DE, Grobmyer SR, Naama HA, et al. Malnutrition-induced macrophage apoptosis. Surgery 2001;129(5):617–25.
60. Runyon BA. Low-protein-concentration ascitic fluid is predisposed to spontaneous bacterial peritonitis. Gastroenterology 1986;91(6):1343–6.
61. Wukich DK, McMillen RL, Lowery NJ, et al. Surgical site infections after foot and ankle surgery: a comparison of patients with and without diabetes. Diabetes Care 2011;34(10):2211–3.
62. Sorensen LT, Karlsmark T, Gottrup F. Abstinence from smoking reduces incisional wound infection: a randomized controlled trial. Ann Surg 2003;238(1):1.
63. Sorensen LT. Wound healing and infection in surgery: the pathophysiological impact of smoking, smoking cessation, and nicotine replacement therapy: a systematic review. Ann Surg 2012;255(6):1069–79.
64. Yuan K, Chen HL. Obesity and surgical site infections risk in orthopedics: a meta-analysis. Int J Surg 2013;11(5):383–8.
65. Wick EC, Hirose K, Shore AD, et al. Surgical site infections and cost in obese patients undergoing colorectal surgery. Arch Surg 2011;146(9):1068–72.
66. Harrington G, Russo P, Spelman D, et al. Surgical-site infection rates and risk factor analysis in coronary artery bypass graft surgery. Infect Control Hosp Epidemiol 2004;25(6):472–6.
67. Meijs AP, Koek MBG, Vos MC, et al. The effect of body mass index on the risk of surgical site infection. Infect Control Hosp Epidemiol 2019;40(9):991–6.
68. Filsoufi F, Rahmanian PB, Castillo JG, et al. Incidence, treatment strategies and outcome of deep sternal wound infection after orthotopic heart transplantation. J Heart Lung Transpl 2007;26(11):1084–90.
69. Payne WG, Naidu DK, Wheeler CK, et al. Wound healing in patients with cancer. Eplasty 2008;8:e9.
70. Corcoran TB, Myles PS, Forbes AB, et al. Dexamethasone and Surgical-Site Infection. N Engl J Med 2021;384(18):1731–41.
71. Bosanquet DC, Rangaraj A, Richards AJ, et al. Topical steroids for chronic wounds displaying abnormal inflammation. Ann R Coll Surg Engl 2013;95(4): 291–6.
72. Hopf HW, Holm J. Hyperoxia and infection. Best Pract Res Clin Anaesthesiol 2008;22(3):553–69.
73. Wetterslev J, Meyhoff CS, Jørgensen LN, et al. The effects of high perioperative inspiratory oxygen fraction for adult surgical patients. Cochrane Database Syst Rev 2015;(6). Available at: https://www.cochranelibrary.com/cdsr/doi/10.1002/14651858.CD008884.pub2/abstract?cookiesEnabled.
74. Kurz A, Sessler DI, Lenhardt R. Perioperative normothermia to reduce the incidence of surgical-wound infection and shorten hospitalization. Study of Wound Infection and Temperature Group. N Engl J Med 1996;334(19):1209–15.

75. Wang Z, Anderson FA Jr, Ward M, et al. Surgical site infections and other postoperative complications following prophylactic anticoagulation in total joint arthroplasty. PLoS One 2014;9(4):e91755.
76. Rohde JM, Dimcheff DE, Blumberg N, et al. Health care-associated infection after red blood cell transfusion: a systematic review and meta-analysis. JAMA 2014;311(13):1317–26.
77. Muszynski J, Nateri J, Nicol K, et al. Immunosuppressive effects of red blood cells on monocytes are related to both storage time and storage solution. Transfusion 2012;52(4):794–802.
78. Anderson DJ, Podgorny K, Berrios-Torres SI, et al. Strategies to prevent surgical site infections in acute care hospitals: 2014 update. Infect Control Hosp Epidemiol 2014;35(Suppl 2):S66–88.
79. Global guidelines for the prevention of surgical site infection, second edition. Geneva: World Health Organization; 2018. Licence: CC BY-NC-SA 3.0 IGO. p. 58–158.
80. Ban KA, Minei JP, Laronga C, et al. American college of surgeons and surgical infection society: surgical site infection guidelines, 2016 update. J Am Coll Surg 2017;224(1):59–74.
81. Berrios-Torres SI, Umscheid CA, Bratzler DW, et al. Centers for disease control and prevention guideline for the prevention of surgical site infection, 2017. JAMA Surg 2017;152(8):784–91.
82. Seal LA, Paul-Cheadle D. A systems approach to preoperative surgical patient skin preparation. Am J Infect Control 2004;32(2):57–62.
83. Bratzler DW, Dellinger EP, Olsen KM, et al. Clinical practice guidelines for antimicrobial prophylaxis in surgery. Am J Health Syst Pharm 2013;70(3):195–283.
84. Global guidelines for the prevention of surgical site infection. Geneva (Switzerland): World Health Organization; 2018. Available at: https://www.ncbi.nlm.nih.gov/books/NBK536404/.
85. Jones EL, Overbey DM, Chapman BC, et al. Operating room fires and surgical skin preparation. J Am Coll Surg 2017;225(1):160–5.
86. Tuuli MG, Liu J, Stout MJ, et al. A randomized trial comparing skin antiseptic agents at cesarean delivery. N Engl J Med 2016;374(7):647–55.
87. Prevention of infection after gynecologic procedures. The American College of Obstetricians and Gynecologists. ACOG practice bulletin. Number 195. 2018. Available at: https://www.acog.org/clinical/clinical-guidance/practice-bulletin/articles/2018/06/prevention-of-infection-after-gynecologic-procedures.
88. Haas DM, Morgan S, Contreras K, et al. Vaginal preparation with antiseptic solution before cesarean section for preventing postoperative infections. Cochrane Database Syst Rev 2018;7:Cd007892.
89. Myles PS, Carlisle JB, Scarr B. Evidence for compromised data integrity in studies of liberal peri-operative inspired oxygen. Anaesthesia 2019;74(5):573–84.
90. Haynes AB, Weiser TG, Berry WR, et al. A surgical safety checklist to reduce morbidity and mortality in a global population. N Engl J Med 2009;360(5):491–9.
91. Phillips M, Rosenberg A, Shopsin B, et al. Preventing surgical site infections: a randomized, open-label trial of nasal mupirocin ointment and nasal povidone-iodine solution. Infect Control Hosp Epidemiol 2014;35(7):826–32.
92. Rezapoor M, Nicholson T, Tabatabaee RM, et al. Povidone-iodine-based solutions for decolonization of nasal staphylococcus aureus: a randomized, prospective, placebo-controlled study. J Arthroplasty 2017;32(9):2815–9.

93. Keenan JE, Speicher PJ, Nussbaum DP, et al. Improving outcomes in colorectal surgery by sequential implementation of multiple standardized care programs. J Am Coll Surg 2015;221(2):404–14.e401.
94. Cook TM, Piatt CJ, Barnes S, et al. The impact of supplemental intraoperative air decontamination on the outcome of total joint arthroplasty: a pilot analysis. J Arthroplasty 2019;34(3):549–53.
95. Graling PR, Vasaly FW. Effectiveness of 2% CHG cloth bathing for reducing surgical site infections. AORN J 2013;97(5):547–51.
96. Baker AW, Chen LF. Letter to the editor regarding: "Effectiveness of local vancomycin powder to decrease surgical site infections: a meta-analysis" by Chiang et al. Spine J 2014;14(6):1092.
97. Bennett-Guerrero E, Pappas TN, Koltun WA, et al. Gentamicin-collagen sponge for infection prophylaxis in colorectal surgery. N Engl J Med 2010;363(11):1038–49.
98. Kowalewski M, Pawliszak W, Zaborowska K, et al. Gentamicin-collagen sponge reduces the risk of sternal wound infections after heart surgery: Meta-analysis. J Thorac Cardiovasc Surg 2015;149(6):1631–40.e1631.
99. Wu X, Kubilay NZ, Ren J, et al. Antimicrobial-coated sutures to decrease surgical site infections: a systematic review and meta-analysis. Eur J Clin Microbiol 2017;36(1):19–32.
100. Deliaert AE, Van den Kerckhove E, Tuinder S, et al. The effect of triclosan-coated sutures in wound healing. A double blind randomised prospective pilot study. J Plast Reconstr Aesthet Surg 2009;62(6):771–3.
101. Tuuli MG, Liu J, Tita ATN, et al. Effect of prophylactic negative pressure wound therapy vs standard wound dressing on surgical-site infection in obese women after cesarean delivery: a randomized clinical trial. JAMA 2020;324(12):1180–9.
102. Stevens DL, Bisno AL, Chambers HF, et al. Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014 update by the Infectious Diseases Society of America. Clin Infect Dis 2014;59(2):e10–52.
103. Karra R, McDermott L, Connelly S, et al. Risk factors for 1-year mortality after postoperative mediastinitis. J Thorac Cardiovasc Surg 2006;132(3):537–43.
104. Haley RW, Culver DH, White JW, et al. The efficacy of infection surveillance and control programs in preventing nosocomial infections in US hospitals. Am J Epidemiol 1985;121(2):182–205.
105. Cruse PJ, Foord R. The epidemiology of wound infection. A 10-year prospective study of 62,939 wounds. Surg Clin North Am 1980;60(1):27–40.
106. Olson MM, Lee JT Jr. Continuous, 10-year wound infection surveillance. Results, advantages, and unanswered questions. Arch Surg 1990;125(6):794–803.
107. Baker C, Luce J, Chenoweth C, et al. Comparison of case-finding methodologies for endometritis after cesarean section. Am J Infect Control 1995;23(1):27–33.
108. Cardo DM, Falk PS, Mayhall CG. Validation of surgical wound surveillance. Infect Control Hosp Epidemiol 1993;14(4):211–5.
109. Ming DY, Chen LF, Miller BA, et al. The impact of depth of infection and postdischarge surveillance on rate of surgical-site infections in a network of community hospitals. Infect Control Hosp Epidemiol 2012;33(3):276–82.
110. Wenzel RP, Osterman CA, Hunting KJ, et al. Hospital-acquired infections. I. Surveillance in a university hospital. Am J Epidemiol 1976;103(3):251–60.
111. Yokoe DS, Noskin GA, Cunningham SM, et al. Enhanced identification of postoperative infections among inpatients. Emerg Infect Dis 2004;10(11):1924–30.

112. Platt R, Kleinman K, Thompson K, et al. Using automated health plan data to assess infection risk from coronary artery bypass surgery. Emerg Infect Dis 2002;8(12):1433–41.
113. Inacio MC, Paxton EW, Chen Y, et al. Leveraging electronic medical records for surveillance of surgical site infection in a total joint replacement population. Infect Control Hosp Epidemiol 2011;32(4):351–9.
114. Bolon MK, Hooper D, Stevenson KB, et al. Improved surveillance for surgical site infections after orthopedic implantation procedures: extending applications for automated data. Clin Infect Dis 2009;48(9):1223–9.
115. Martin LA, Neighbors HW, Griffith DM. The experience of symptoms of depression in men vs women: analysis of the National Comorbidity Survey Replication. JAMA Psychiatry 2013;70(10):1100–6.
116. Baker AW, Haridy S, Salem J, et al. Performance of statistical process control methods for regional surgical site infection surveillance: a 10-year multicentre pilot study. BMJ Qual Saf 2018;27(8):600–10.
117. Dumville JC, McFarlane E, Edwards P, et al. Preoperative skin antiseptics for preventing surgical wound infections after clean surgery. Cochrane Database Syst Rev 2013;(3):CD003949.
118. McLaws ML, Murphy C, Whitby M. Standardising surveillance of nosocomial infections: the HISS program. Hospital infection standardised surveillance. J Qual Clin Pract 2000;20(1):6–11.