Chapter 4

# Procedure-Related Reduction of the Risk of Infection

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## Introduction

Surgical site infection (SSI) is the third most common type of nosocomial infection, accounting for 38% of all infections in the 27 million patients who undergo surgery in the United States each year.[1] These infections are associated with substantial morbidity, mortality, and expense. Critical bacterial burden is by far the most significant of the many factors that influence surgical wound healing and determine the potential for infection and its incidence.

## Classification and Monitoring

Since 1986, nosocomial infection rates have been identified as an indicator of quality of care and a reliable measure of institutional quality assurance. The 2006 Institute of Medicine report *Rewarding Provider Performance: Aligning Incentives in Medicare*[2] recommended pay-for-performance programs as a means of aligning performance improvement incentives. The US Centers for Medicare and Medicaid Services soon adopted the pay-for-performance approach for provider reimbursement under Medicare. More than half of all commercial health maintenance organizations now use a pay-for-performance system, despite widespread concern as to the choice and validity of the measures used to determine improvement.

The American public has come to believe that every surgical infection is preventable. An increasing number of health plans now include the rate of SSIs in the hospital and physician profiles available to patients, for the purpose of encouraging patients to seek care from providers with favorable profiles. As the number of provider categories, the number of measures, and the financial risks increase, clinicians increasingly are held accountable for their actions and omissions. Orthopaedic surgeons have come under particular scrutiny as the use of surgical implants, the complexity of surgical procedures, and the percentage of patients with a significant comorbidity have increased. However, the high cost of health care, increased patient awareness, legal issues, and pressure from insurance companies may cause medical practice to be defined less by patient safety and outcomes than by evidence-based or possibly unrealistic best practice scenarios.

### Classification Systems

In 1964, the US National Research Council introduced a system for classifying surgical wounds into four sequential categories (clean, clean contaminated, contaminated, and dirty) based on critical bacterial burden and increasing risk of SSI.[3] Subsequent improvements in treatment and outcomes led to a decreased risk of SSI[4,5] (Table 1). However, investigation revealed considerable variation within each wound class associated with the type, nature, and length of the surgical procedure[6] as well as extrinsic and intrinsic factors influencing the incidence of SSI.

The risk index of the Study on the Efficacy of Nosocomial Infection Control, known as the SENIC index, was introduced in 1985. The SENIC index considers patient and wound characteristics, including the presence of comorbidities, the anatomic site, and the duration of the procedure.[5] This index proved twice as effective in predicting the incidence of SSI as the US National Research Council's original classification. However, a wide range of infection risk once again was observed within each wound category.

Investigators at the US Centers for Disease Control and Prevention (CDC) currently use a risk index created as part of the National Nosocomial Infections Surveillance (NNIS) system.[6] The NNIS risk index is based on three independent and equally weighted variables, which are used to assign a three-point score.[6,7] One point is assigned to each of the following, if present: a contaminated or dirty wound; severe systemic disease, as indicated by a score of 3 or higher on the American Society of Anesthesiologists physical status classification[8-10] (Table 2); and excessive duration of surgery, defined as surgical time exceeding the 75th percentile for the specific procedure.[11] For total hip arthroplasty

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**Table 1**

**Surgical Wound Classification System of the US National Research Council, With Initial (1980) and Improved (1991) Risk of Infection**

| Category | Characteristics | Risk of Infection, 1980* | Risk of Infection, 1991† |
|---|---|---|---|
| Clean | No inflammation<br>Respiratory, alimentary, genitourinary tracts not entered<br>No break in aseptic surgical technique | 2%-5% | < 2.1% |
| Clean contaminated | Healed but previously infected wound<br>Respiratory, alimentary, genitourinary tracts entered with no significant spillage<br>Break in aseptic surgical technique | 6%-9% | 3.3% |
| Contaminated | Acute inflammation (no pus)<br>Open trauma wound < 4 hr old<br>Visible wound contamination (gross intrasurgical spillage from a hollow viscous | 13%-20% | 6.4% |
| Dirty | Acute inflammation (pus)<br>Open trauma wound ≥ 4 hr old<br>Previously perforated viscous | 40% | 7.1% |

*Data from Cruse PJ, Ford R: The epidemilogy of wound infection: A 10-year prospective study of 62,939 wounds. *Surg Clin North Am*1980;60:27-40.
† Data from Culver DH, Horan TC, Gaynes RP, et al: Surgical wound infection rates by wound class, operative procedure, and patient risk index: The National Nosocomial Infections Surveillance System. *Am J Med*1991;91:152S-157S.

**Table 2**

**American Society of Anesthesiologists Physical Status Classification**

| Score | Patient Physical Status |
|---|---|
| 1 | Normal, healthy |
| 2 | Mild systemic disease |
| 3 | Severe systemic disease |
| 4 | Severe systemic disease that is a constant threat to life |
| 5 | Moribund; not expected to survive without surgical procedure |
| 6 | Declared brain dead; organs being removed for donation |

(Adapted with permission from American Society of Anesthesiologists: ASA Physical Status Classification System. http://www.asahq.org/clinical/physicalstatus.htm. Accessed June 11, 2008.)

procedures requiring less than 2 hours, an NNIS risk index of 0, 1, or 2 to 3 was correlated with an SSI rate of 0.86%, 1.65%, or 2.5%, respectively.[12] Refinement of the NNIS risk index is needed to incorporate anatomic and site-specific outliers to the formula.[12,13]

## Monitoring

The CDC developed the NNIS system during the early 1970s to monitor the incidence of health care–associated infections as well as the associated risk factors and pathogens. The NNIS system helps professionals and hospitals stay abreast of the rapidly expanding science and practice of infection prevention and control and improve their management of endemic and epidemic episodes of nosocomial infection. In 1992, the CDC introduced the term "surgical site infection" to distinguish infection of a surgical incision from infection of a traumatic wound.[14] SSI is defined as an incisional (superficial) or deep infection occurring within 30 days of surgery or within 1 year of the introduction of a surgical implant.

## The Biology of Surgical Site Infection

Unacceptable SSI rates still sometimes occur, despite the best efforts of infection prevention practitioners and improvements in surgical technique, antibiotic prophylaxis, instrument sterilization methods, and operating room practices. Bacteria can gain access to a surgical wound from several sources. Endogenous contamination arises from the patient's skin or nares; the gastrointestinal, genitourinary, bronchial, or sinusoidal tract; or a concomitant remote-site infection. Exogenous contamination comes from airborne or transient pathogens transferred from instruments, implants, or personnel.

Most SSIs are caused by commensal organisms from the patient's skin or transient organisms disseminated from health care personnel or surgical instruments.[15] Normal resident skin flora inhabit even the deepest layers of the dermis and are difficult to remove; these commensal organisms include *Staphylococcus*, diphtheroid, *Pseudomonas*, and *Propionibacterium* species. Transient organisms are not consistently present but are easily exchanged between individuals. Studies have identified operating room personnel as the source of contamination in 98% of SSIs, with 30% of the contaminants airborne and 70% transferred through surgical instruments or other contact.[16-19]

Although microorganisms gain access to all surgical wounds, only a small percentage of surgical patients develop a clinical infection. The presence of organisms in a wound is less important than the level of bacterial growth during the hours or days after the surgery. Whether an infection develops depends on the number and virulence of the bacteria in the wound, the status or viability of the wound, and the ability of host defenses to eliminate the bacteria.

## Best Practices for Reducing Pathogen Load

Aseptic practice must include procedures to reduce the number of microorganisms present and prevent them from reaching the wound. In addition, operating room practices must prevent the accidental transfer of microorganisms from people, places, or equipment. Surgical best practices encompass both the principles of asepsis and measures to safeguard efficient wound healing using the host's natural defenses.

### Coexisting Remote-Site Infection

Any open or inflamed presurgical wound is a potential source of hematogenous or contiguous surgical wound contamination and should be treated and resolved before elective surgery is undertaken.[9,20,21] Because systemic disease can be caused by infectious oral microbes, a patient with an immunologic or nutritional deficiency should be in good dental health before an elective surgical procedure.[22]

### Colonization With Virulent Microflora

Presurgical nares colonization with *Staphylococcus aureus* is present in 20% to 30% of all healthy patients and is strongly associated with skin colonization.[23] However, some carriers may have negative nasal cultures. High-density nasal colonization is the single most important risk factor for developing SSI with *S aureus* after hip or knee replacement surgery.[24] Carriers of *S aureus* in the anterior nares were found to have five times the risk of developing SSI than patients without colonization.[25] Patients with nasal colonization who were treated with 2% mupirocin nasal ointment for 5 days before surgery had a rate of endogenous infection with *S aureus* two to nine times lower than comparable untreated patients.[26,27] Presurgical screening, colonization-directed prophylaxis, and decolonization protocols have led to significantly lower rates of SSI caused by *S aureus*.

### Presurgical Length of Stay

A patient may be exposed to transient strains of nosocomial pathogens during hospitalization, leading to a change in skin flora and an increased risk of infection.[4,28] Length of stay in a health care facility is significantly associated with SSI risk.[10,29,30] A patient who has had a prolonged stay in a health care facility should be evaluated for comorbid conditions, cultured for potential colonization with virulent pathogens, and given a prophylactic antibiotic tailored to the antibiogram of both sending and receiving facilities.[10]

### Antibiotic Prophylaxis

Routine use of prophylactic antibiotics has led to a dramatic reduction in infection rates for most types of wounds.[6] Prospective double-blind studies supported the use of antibiotic prophylaxis for clean and clean-contaminated orthopaedic procedures. The antibiotic should be carefully selected by considering current recommendations as well as timing, dosage, patient allergies, and possible pathogen resistance to antimicrobial agents. Prophylactic antibiotics should be administered over a short period of time to avoid opportunistic nosocomial infection, including late SSI caused by hematogenous seeding of implants or surgical hematomas[31-33] (see chapter 19).

### Hair Removal

The risk of SSI was found to be significantly higher when hair was removed before surgery using a razor than when hair was not removed or was removed using electric clippers immediately before the incision.[34] Razor shaving produces microscopic cuts in the epidermis, exposing deep-seated microorganisms and increasing the risk of bacteremia or direct wound contamination.

### Antimicrobial Skin Preparation

The CDC recommends showering with chlorhexidine gluconate before elective surgery as a means of reducing microbial colony counts on the skin. However, this practice has not been proved to decrease the incidence of SSI.[1,35]

For presurgical preparation, a 4% concentration of chlorhexidine gluconate has been shown to be more effective than povidone-iodine or triclocarban soap and water in reducing rates of intrasurgical wound contamination.[36] Povidone-iodine and 4% chlorhexidine gluconate are equally effective for decreasing the initial bacterial skin contamination of both the patient and the surgeon. However, 4% chlorhexidine gluconate is effective for a longer period of time ($P < 0.0001$) and has a greater cumulative effect with prolonged use;[37-39] it also is less toxic, more stable in an open wound, and less irritating to the skin. Repeated scrubbing with alcohol alone or with chlorhexidine gluconate and alcohol was found to be more effective in reducing bacteria than traditional povidone-iodine and chlorhexidine gluconate scrubbing regimens.[40]

## Barrier Draping

Although the use of either antiseptic-impregnated, adhesive surgical drapes or a postpreparation microbiologic sealant (InteguSeal, Kimberly-Clark, Roswell, GA) can reduce skin bacterial counts, no published controlled clinical study has documented a decreased incidence of SSI.[41]

Glove perforation routinely occurs during most orthopaedic procedures, and, therefore, double gloves should be used. The number of perforations is directly correlated with procedure duration; almost all gloves were found to be punctured after a procedure longer than 3 hours.[42]

## Ventilation and Clean Air System

The amount of airborne contamination can be significantly reduced by controlling movement into and within the operating room and by using personal isolation suits.[43] The design and efficiency of the operating room ventilation system, measured in air exchanges per hour, and staff compliance with air flow procedures also are important.[44] Although clean air technology has not been proved to independently decrease the incidence of deep SSI, it was found to significantly decrease rates of bacterial wound contamination, prolonged wound discharge, and superficial infection of the surgical site.[45,46] The CDC recommends that an orthopaedic implantation procedure be performed in an operating room equipped with an ultraclean air system, and the National Institutes of Health concluded that a laminar airflow system is the best option.

## Duration of Surgery

The risk of SSI is proportional to the duration of the surgical procedure and roughly doubles with every elapsed hour.[4,9,10,47] Increasing endogenous and exogenous contamination, increasing wound compromise, and the effect of intrinsic and extrinsic host defense factors are responsible. In addition, time-sensitive factors including prolonged patient immobility, patient positioning, and maintenance of sufficient padding for dependent parts and bony prominences can be affected by an unanticipated delay in completing the surgery.[48] Presurgical attention to patient positioning and padding as well as diligent postsurgical surveillance for injury can be helpful in avoiding ulceration, remote-site infection, or SSI caused by bacteremia or direct cross contamination.

## Earlier Same-Site Surgery

The risk of SSI is increased if the patient has undergone an earlier surgical procedure at the site.[49,50] The factors responsible for the increased risk include the additional time required to perform a revision procedure, the presence of ischemic scar tissue, and unsuspected microbial contamination of previously implanted hardware by dormant strains in an exopolysaccharide biofilm.[9,51,52] Subclinical infection must be anticipated and pursued with tissue cultures in any revision surgery. To decrease the risk of SSI, every effort should be made to avoid tissue devitalization, hasten wound closure, and intervene aggressively as soon as any sign of wound failure or infection appears.

## Suture Materials

The surgeon must consider the chemical composition of suture material, as the presence of any foreign material increases a wound's susceptibility to infection.[53] Absorbable synthetic monofilament suture is associated with a lower rate of infection than absorbable synthetic braided suture, which in turn is associated with a lower rate of infection than nonabsorbable braided suture or natural suture such as catgut or silk.[54] Antimicrobial-impregnated surgical suture materials are beneficial in preventing SSI in a critically contaminated wound.[55]

# Best Practices for Avoiding Wound Contamination

Intrinsic and extrinsic factors can contribute to wound contamination even after the surgery. Bacteremia often is associated with a remote-site nosocomial infection (such as pneumonia, a urinary tract infection, or primary bloodstream infection) resulting from the use of an invasive medical device (such as a ventilator, a urinary catheter, an indwelling drain, or a central intravascular line). These devices act as a pathway for environmental microorganisms, facilitate the transfer of pathogens from one part of the body to another, and provide surfaces on which pathogens can proliferate while protected from antimicrobial agents and host defenses; they should be avoided whenever possible.

## Postsurgical Drains

A retrospective analysis of more than 73,000 SSIs found that retention of a surgical drain for more than 24 hours was associated with a significantly increased risk of cross contamination with a resistant nosocomial pathogen.[31] The CDC recommends that clean procedures be performed without using a surgical drain; if a drain is necessary, a closed suction system is preferred. The drain should exit through a separate incision and, if possible, should be removed within 24 hours.[1]

## Wound Dressings

A surgical wound can be contaminated directly through closed wound margins until it seals.[31] A systematic review of 111 studies found that the rate of infection under an occlusive dressing was significantly lower than the rate of infection under a nonocclusive dressing.[56]

## Induced Bacteremia

A patient who is immunocompromised or immunosuppressed or has undergone a prosthetic joint replacement is at an increased risk of developing a prosthesis-related joint infection caused by the hematogenous spread of microorganisms. Patients with pins, plates, screws, or other orthopaedic hardware at sites other than a synovial joint are not at increased risk of hematogenous seeding. The American Academy of Orthopaedic Surgeons recommends that clinicians consider antibiotic prophylaxis for all patients with a joint replacement before any invasive procedure that could cause bacteremia.[57]

## Best Surgical Practices for Enhancing the Wound Environment

The local host defenses for controlling or eliminating the proliferation of contaminating pathogens include a physiologic responsiveness to injury fueled by adequate tissue perfusion. The primary defense against surgical pathogens is oxidative killing by neutrophils. Local factors that compromise tissue perfusion, cellular responsiveness, and wound closure are associated with wound-healing disturbances and an increased risk of SSI.[58] Before surgical intervention, every effort should be made to reverse or counter site-specific factors affecting outcomes (Table 3).

**Table 3**

**Local Wound Factors Associated With Increased Risk of Surgical Site Infection**

| |
|---|
| Anatomic site (superficial joint [elbow, knee], unprotected bone [tibia, sternum], area at risk of contamination [sacrum, coccyx]) |
| Chronic edema |
| Earlier infection |
| Earlier surgery |
| Excessive scarring |
| Ischemia |
| Obesity |
| Radiation injury |
| Soft-tissue deficit |
| Vascular disease |

### Wound Oxygenation

Transcutaneous measurement of tissue oxygen tension at the proposed surgical site can reveal ischemia predictive of a postsurgical healing disturbance.[59] A study of patients with diabetes and chronic ischemia found healing of major and minor amputations in 91% of patients whose tissue oxygen tension value was higher than 30 mm Hg; in comparison, the rate of healing was 50% for patients with a value lower than 30 mm Hg. Reversal of tissue hypoxia both restored the ability to heal normally and decreased the incidence of SSI.[60]

### Normothermia

Intrasurgical hypothermia causes vasoconstriction, reduction in blood flow to the surgical site, and increased susceptibility to SSI. The use of presurgical and intrasurgical warming decreases the rate of SSI in clean and clean-contaminated procedures.[61,62]

### Pain Control

Surgical and postsurgical pain evokes profound neuroendocrine cytokine activity, which in turn evokes a regional arteriolar vasoconstriction and thereby reduces tissue perfusion, tissue oxygen tension, collagen deposition, and wound tensile strength.[63] Poorly controlled surgical pain is associated with an increased risk of SSI.[64]

### Hyperbaric Oxygen Therapy

Hyperbaric oxygen therapy is the only method proved to reverse the effects of ionizing radiation. The sensitivity of endothelial cells and myofibroblasts to the effects of radiation causes progressive and permanent fibrosis, capillary loss, and regional ischemia resulting from obliterating endarteritis. In hyperbaric oxygen therapy, a steep oxygen gradient is created between normal and injured tissues, catalyzing the release of platelet- and macrophage-derived angiogenesis factors, which stimulates fibroblast proliferation and collagen synthesis to promote ingrowth of new blood vessels.[65,66] Presurgical treatment with hyperbaric oxygen enhances the potential for normal wound healing in previously irradiated tissues and reduces the incidence of SSI.[67,68]

### Surgical Wound Hematoma

A rapidly expanding hematoma in the surgical wound can be an important contributor to the development of SSI.[69] The lesion can block the influx of antibiotics into the surgical site, cause wound-healing disturbances through tissue devitalization, and lead to prolonged wound drainage.[70] The presence of an expansile hematoma mandates débridement and wound revision.

### Prolonged Wound Drainage

Serous drainage from a postsurgical wound initially can be treated with compressive and occlusive dressings to prevent retrograde introduction of bacteria. Prolonged drainage requires a biopsy for culturing as well as formal débridement and wound revision.[71] Drainage exceeding 5 days' duration is associated with a threefold to fourfold increase in the risk of developing a deep SSI.[70,72-74]

### Blood Transfusion

Several prospective randomized studies have associated intrasurgical or postsurgical blood transfusion with SSI.[75] However, presurgical blood transfusion was not associated with increased risk. This discrepancy suggests that the deleterious effect of intrasurgical or postsurgical transfusion may in part be a surrogate for risk factors such as hypovolemia, decreased tissue perfusion, low oxygen tension, and prolonged surgical time.

**Table 4**

### Systemic Factors Associated With Increased Risk of Surgical Site Infection

| |
|---|
| Advanced age |
| Alcoholism |
| Coagulopathies |
| Colonization with pathogens resistant to antimicrobial agents |
| Diabetes mellitus |
| Hemoglobinopathy |
| Hypoxia |
| Immune deficiency |
| Malignancy |
| Malnutrition |
| Nicotine use |
| Obesity |
| Organ failure |
| Parenteral drug abuse |
| Skin colonization with bacterial pathogens |

## Best Surgical Practices for Treating Patients With Comorbidities

A patient's predisposition to SSI is strongly influenced by risk factors inherent in underlying health conditions (Table 4). Compromised health is associated with a high incidence of skin or wound breakdown, frequent episodes of bacteremia, immune deficiencies, multiple-joint surgical procedures, poor nutritional status, and increased risk of bleeding. The overall effect is cumulative, and the baseline risk of developing SSI increases with each adverse medical condition.[58,76,77] The presence of three or more comorbidities in patients with a periprosthetic total joint infection of the hip or knee led to a 22% mortality rate and a 100% rate of treatment failure.[58] Elective orthopaedic reconstruction is contraindicated if the patient has a neutrophil count lower than 1,000, a CD4 (T cell) count lower than 200, or an active remote site of infection; or is an intravenous drug abuser.[76,77]

The treatment of patients with comorbidities requires specific precautions.[78] If surgical intervention is necessary, a low-risk procedure of short duration should be chosen. The exposure should be straightforward, with the use of foreign materials limited.[79] If an implant is required, concomitant use of an antibiotic-impregnated depot will help to clear any transient wound contamination (see chapter 9).[80] The outcome can be further safeguarded by postsurgical surveillance for conditions amenable to early intervention, including superficial skin necrosis, prolonged wound drainage, an expansile hematoma, and early signs of sepsis.

**Table 5**

### Methodology for Prevention of Surgical Site Infection

| |
|---|
| **Reduce Pathogen Load** |
| Avoidance of additional surgery |
| Avoidance of shaving |
| Clean air systems |
| Decolonization |
| Glove changes |
| Isolation suits |
| Limited operating room traffic |
| Limited surgical time |
| Limited use of postsurgical drains |
| Occlusive wound dressings |
| Pressure sore prevention |
| Prevention or treatment of remote infection |
| Prophylactic antibiotics |
| Refreshed instrument basins |
| Suction tip replacement |
| Surgical barriers |
| Surgical preparation |
| Surgical scrubs |
| Suture material selection |
| Wound irrigation |
| **Avoid Wound Contamination and Enhance the Wound Environment** |
| Early intervention for complications |
| Hematoma decompression |
| Hemostasis |
| Hyperbaric oxygen |
| Implant selection |
| Limited surgical time |
| Normothermia |
| Pain control |
| Soft-tissue flaps |
| Supplemental oxygen |
| Surgical technique |
| Vacuum-assisted wound closure |
| Vascular bypass |
| Wound surveillance |
| **Treat Patients With Comorbidities** |
| Antibiotic desensitization |
| Detoxification (alcohol, tobacco, drugs) |
| Glucose control |
| Low-risk procedures |
| Nutrition |
| Reverse coagulopathies |
| Systems maintenance |

### Glucose Control

In patients with diabetes who underwent a gastrointestinal or coronary artery bypass graft procedure, a glucose level above 220 mg/dL led to a fivefold increase in the SSI rate. Patients whose glucose level was kept within the normal range had the same SSI rates as patients who did not have diabetes.[81] An elevated intrasurgical or postsurgical glucose level is a risk factor for SSI.[1,9,81-83]

### Tobacco Use

Tobacco use is an important risk factor for serious postsurgical complications.[9] A study of 811 consecutive patients after hip or knee arthroplasty found that smoking tobacco was the most important risk factor for developing a postsurgical complication, especially a complication related to wound healing.[84,85] The CDC recommends that patients abstain from using all tobacco products for at least 30 days before elective surgery to reduce the risk of SSI.[1]

### Nutritional Status

The association of malnutrition and surgical morbidity is well recognized.[9,86-88] Malnourished patients are at increased risk of developing deep SSI after a major surgical procedure.[89] Perisurgical nutritional support decreases the risk of septic postsurgical complications.[90-92]

### Renal Status

Chronic renal failure increases the risk of SSI.[9,93] A patient who is dependent on hemodialysis should receive such a treatment the day before major surgery to minimize the effects of fluid and electrolyte shifts and to correct abnormalities inherent in renal disease.[94]

## Summary

To decrease the risk of SSI, caregivers must limit patient exposure to exogenous and endogenous pathogens; establish and maintain a vital, resilient wound; and work to prevent, accommodate, and minimize systemic factors affecting the overall immunity of the host. Infection occurs when the number and virulence of a pathogen are sufficient to overwhelm the wound and the physiologic capability of the host to respond (Table 5).

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