Prevention of Surgical Site Infection

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Surgical site infection (SSI) is an important postoperative complication. It is second only to urinary tract infection as the most common nosocomial infection in hospitalized patients. Based on extensive epidemiologic surveys, it has been estimated that SSI develops in at least 2% of hospitalized patients undergoing operative procedures, although this is a likely underestimate because of incomplete post-discharge data. Other data indicate that SSI develops following 3% to 20% of certain procedures, and that the incidence is even higher in certain high-risk patients.

There seems to be a perception among some surgeons that SSI is a relatively trivial infection. However, based on survey data, there were over 290,000 infections in hospitalized patients in 2002, and SSI was estimated to be directly responsible for 8205 deaths of surgical patients that year. Thus, the mortality rate was 3% among patients who developed SSI. There is also significant morbidity associated with SSI; a large number of patients develop long-term disabilities as a result of poor wound healing and overt tissue destruction following these infections. Finally, the economic costs of SSI to both the patient and the health care delivery system are high.

Because of their frequency and clinical significance, SSI rates are of interest to regulatory agencies and to the public at large. Public reporting of SSI rates by health care entities is increasingly required, and this mandate is being extended to individual surgeons. Further, a number of regulatory programs have been implemented that apply both financial incentives for following best practices in preventing SSI and financial penalties when such infections occur. It can be anticipated that such programs will expand in the future.
DEFINITIONS

SSI is an infection that occurs somewhere in the operative field following a surgical intervention. The Centers for Disease Control and Prevention (CDC) considers SSI to include both incisional SSI and organ space SSI. Incisional SSI is subdivided into superficial and deep SSI, depending on whether the infection is limited to the skin and subcutaneous tissue only (superficial SSI) or extends into the deeper tissues, such as the fascial and muscular layers of the body wall (deep SSI). Organ/space SSI is an infection that occurs anywhere within the operative field other than where the body wall tissues were incised. Examples include intra-abdominal abscess developing after an abdominal operation, empyema developing after a thoracic operation, and osteomyelitis or joint infection developing after an orthopedic procedure.4

The National Healthcare Safety Network (NHSN) of the CDC has developed a series of criteria in an effort to objectively define SSI (Box 1). Although these criteria are relatively detailed, it is important to realize that the surgeon’s judgment ultimately determines whether an SSI is present in equivocal cases. Thus, when there are erythematous changes around a wound or it is draining material that is not clearly purulent, it is important that the surgeon’s opinion be clearly expressed as to whether or not an SSI is present.

RISK FACTORS FOR DEVELOPING SSI

The risk of developing SSI varies greatly according to the nature of the operative procedure and the specific clinical characteristics of the patient undergoing that procedure. Ultimately, it is necessary to consider a broad range of risk factors for developing preventative measures.

The CDC wound classification system5–7 is widely used to capture some of the risk of infection related to the type of operative procedure. This classification scheme focuses primarily on the degree of contamination likely to be present during the operation (Table 1). Thus, during Class I (clean) procedures, only microorganisms from the skin and external environment are likely to be introduced into the wound. With Class II (clean-contaminated) procedures, there is additional exposure to microorganisms colonizing the epithelial surfaces and lumen of structures of the respiratory, digestive, genital, and urinary tracts, although contamination should be limited in scope. With Class III (contaminated), and Class IV (dirty-infected) procedures, there is progressively greater exposure of the wound to potential pathogenic microorganisms.

Although the CDC wound classification scheme allows some stratification of risk, it does not take into account other risks related to the operative procedure or patient characteristics. Two large epidemiologic surveys performed by the CDC in the 1970s and 1980s established the importance of these other factors in developing SSI. In 1985, the Study on the Efficacy of Nosocomial Infection Control identified an abdominal operation, an operation of longer duration (2 hours or more), and a patient having three or more discharge diagnoses as being risk factors for the development of SSI in addition to wound classification (contaminated or dirty-infected versus clean or clean-contaminated).8 Subsequently, the National Nosocomial Infections Surveillance System (NNIS),9 the predecessor of the current NHSN, simplified risk stratification to three factors: (1) CDC wound classification (contaminated or dirty-infected); (2) a longer duration operation, defined as one that exceeded the 75th percentile for a given procedure; and (3) the medical characteristics of the patient, as determined by an American Society of Anesthesiology (ASA) score of III, IV, or V (presence of a severe systemic disease that results in functional limitations, is life threatening, or is expected to preclude survival from the operation) at the time of the operation.
With the widespread introduction of laparoscopic techniques into the surgical armamentarium, this three-point risk index has been further modified. A rule now calls for subtraction of one risk factor point when cholecystectomy or colon surgery is performed laparoscopically; however, for appendectomy and gastric surgery, subtraction of one point is done only if there are no other risk factors.

The impact of these risk factors can be seen in information provided by the NHSN about SSI rates for various operative procedures performed in 2006–2007. Selected data from this publication are summarized in Table 2. Looking at these figures, it is apparent that even with risk adjustment, there are intrinsic disparities in SSI rates with different procedures. For instance, among patients with no risk factors who underwent breast or colonic operations, the rate of SSI was fivefold higher with colonic surgery than it was with breast surgery. Nonetheless, with each procedure, there is a major impact of additional risk factors; SSI rates double to quadruple as the number of risk factors increase. Thus, it is clear that the risk of SSI is related to factors other than just wound classification.

The primary use of these analyses is in monitoring trends in SSI rates and in allowing individual institutions to benchmark their data against national averages. However, these broad-based risk adjustments do not easily lead to targeted interventions for the prevention of SSI. For this, knowledge of more specific risk factors is needed. Multivariate analyses have identified large numbers of specific risk factors which place the patient at higher risk of developing a SSI: (1) patient characteristics, such as increased age or the presence of a remote infection at the time of the operation; (2) aspects of preoperative, intraoperative, and postoperative management, such as delayed delivery of prophylactic antibiotics or flash sterilization of surgical instruments. One summary of risk factors, from the 1999 CDC guidelines on prevention of SSI, is reproduced in Box 2. Although these risk factors are not necessarily independent of each other, they do provide potential targets for developing preventative measures.

MICROBIOLOGY

SSI is caused by microorganisms introduced into the surgical wound at the time of the operative procedure. Most of these microorganisms come from the patient’s endogenous flora, but occasionally the pathogenic organisms are acquired from an exogenous source, such as the air in the operating room, surgical equipment, implants or gloves, or even medications administered during the operative procedure. When there is an unexplained local outbreak of SSI, investigations performed by infection control personnel may be useful in uncovering an exogenous source.

Large, cross-institutional surveys involving all surgical specialties have revealed that a small number of gram-positive cocci and gram-negative bacilli are responsible for most SSIs. The NNIS system categorized 17,671 isolates obtained from patients with SSI from 1986 to 1996. Over one half of the isolates were gram-positive cocci; Staphylococcus aureus was the most commonly isolated organism, followed by coagulase-negative staphylococci, and Enterococcus spp. Approximately one third of the isolates were gram-negative bacilli, with Escherichia coli, Pseudomonas aeruginosa, and Enterobacter spp being the most frequently encountered gram-negative organisms. About 5% of the isolates were anaerobic bacteria. More recent surveys involving multiple or single institutions have corroborated these general findings, although the specific distribution of organisms differs somewhat, probably reflecting different types of surgical practices at individual institutions.

This general pattern masks significant variability in the microbiology of SSI according to the type of operative procedure. For patients undergoing clean procedures,
Box 1
CDC criteria for defining an SSI

**Superficial incisional SSI**

Infection occurs within 30 days after the operative procedure

and

involves only skin or subcutaneous tissue of the incision

and

patient has at least one of the following:

- Purulent drainage from the superficial incision.
- Organisms isolated from an aseptically obtained culture of fluid or tissue from the superficial incision.
- At least one of the following signs or symptoms of infection: pain or tenderness; localized swelling, redness, or heat; and superficial incision is deliberately opened by surgeon and is culture-positive or not cultured. A culture-negative finding does not meet this criterion.

Diagnosis of superficial incisional SSI by the surgeon or attending physician.

**Deep incisional SSI**

Infection occurs within 30 days after the operation if no implant is left in place or within 1 year if implant is in place and the infection appears to be related to the operative procedure

and

involves deep soft tissues (eg, fascial and muscle layers) of the incision

and

patient has at least one of the following:

- Purulent drainage from the deep incision but not from the organ/space component of the surgical site.
- A deep incision spontaneously dehisces or is deliberately opened by a surgeon and is culture-positive or not cultured when the patient has at least one of the following signs or symptoms: fever (>38°C) or localized pain or tenderness. A culture-negative finding does not meet this criterion.
- An abscess or other evidence of infection involving the deep incision is found on direct examination, during reoperation, or by histopathologic or radiologic examination.

Diagnosis of a deep incisional SSI by a surgeon or attending physician.

**Organ/Space SSI**

Infection occurs within 30 days after the operation if no implant is left in place or within 1 year if implant is in place and the infection appears to be related to the operative procedure

and

infection involves any part of the body (excluding the skin incision, fascia, or muscle layers) that is opened or manipulated during the operative procedure

and

patient has at least one of the following:

- Purulent drainage from a drain that is placed through a stab wound into the organ/space.
Organisms isolated from an aseptically obtained culture of fluid or tissue in the organ/space.

An abscess or other evidence of infection involving the organ/space that is found on direct examination, during reoperation, or by histopathologic or radiologic examination.

Diagnosis of an organ/space SSI by a surgeon or attending physician.


staphylococci predominate as the cause of SSI, since these microorganisms are present on the skin at the site of most incisions. However, gram-negative and other enteric organisms colonize the skin at certain sites, including the axilla, perineum and groin; patients having incisions in those areas may have a SSI caused by gram-negative organisms. Thus, patients undergoing coronary artery bypass surgery are likely to have gram-positive organisms as the cause of a sternal wound infection, but are frequently found to have gram-negative organism as the cause of a leg wound infection.\(^\text{17}\) With clean-contaminated or contaminated wounds, bacteria from the respiratory, gastrointestinal, genital, or urinary tracts contribute to the infection. For instance, gram-negative bacilli and anaerobic organisms are frequent causes of SSI

<table>
<thead>
<tr>
<th>Class</th>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Clean</td>
<td>An uninfected operative wound in which no inflammation is encountered and the respiratory, alimentary, genital, or uninfected urinary tract is not entered. In addition, clean wounds are primarily closed and, if necessary, drained with closed drainage. Operative incisional wounds that follow nonpenetrating (blunt) trauma should be included in this category if they meet the criteria</td>
</tr>
<tr>
<td>II</td>
<td>Clean-contaminated</td>
<td>An operative wound in which the respiratory, alimentary, genital, or urinary tracts are entered under controlled conditions and without unusual contamination. Specifically, operations involving the biliary tract, appendix, vagina, and oropharynx are included in this category, provided no evidence of infection or major break in technique is encountered</td>
</tr>
<tr>
<td>III</td>
<td>Contaminated</td>
<td>Open, fresh, accidental wounds. In addition, operations with major breaks in sterile technique (eg, open cardiac massage), or gross spillage from the gastrointestinal tract, and incisions in which acute, nonpurulent inflammation is encountered are included in this category</td>
</tr>
<tr>
<td>IV</td>
<td>Dirty-infected</td>
<td>Old traumatic wounds with retained devitalized tissue and those that involve existing clinical infection or perforated viscera. This definition suggests that the organisms causing postoperative infection were present in the operative field before the operation</td>
</tr>
</tbody>
</table>

following procedures involving the lower gastrointestinal tract. Nonetheless, organisms derived from the skin may still contribute to these infections. In a recent trial of prophylactic antibiotics for subjects undergoing colorectal procedures, 11% of all isolates obtained from subjects with SSI were staphylococci, most of which were *S aureus*. With Class IV (dirty-infected) wounds, it is generally assumed that pathogenic organisms already present in the operative field will be responsible for a subsequent SSI. Finally, it should be noted that unique microbiological patterns may pertain to certain highly specialized procedures; for instance, enterococci are frequently found to be the pathogens causing SSI after liver transplantation.

The most significant change in the microbiology of SSI has been the increased involvement of resistant microorganisms in these infections. The number of SSI caused by methicillin-resistant *S aureus* (MRSA) has increased dramatically. Anderson and colleagues found that MRSA was responsible for 17% of all severe SSIs developing in 1010 patients at 26 community hospitals in the Southeast, and accounted for 53% of the infections due to *S aureus*. Naylor and colleagues documented MRSA in 40% of the severe postoperative SSIs developing in vascular surgery patients at 25 centers in Great Britain and Ireland. An increased occurrence of infections due to MRSA has also been recognized in studies of subjects undergoing cardiac, orthopedic, or plastic surgery procedures. The emergence of the USA300 clone of MRSA, commonly referred to as community-acquired MRSA, may further impact the microbiology of SSI. This strain is recognized as being responsible for significant numbers of serious hospital-acquired staphylococcal infections, a preliminary report also suggests its frequent involvement as a cause of SSI. The gram-negative bacilli isolated from patients with SSI also demonstrate increased resistance. These resistant organisms likely result from prior exposure of the patient to the health care environment or broad spectrum antimicrobial therapy. The increasing resistance of gram-negative organisms causing SSI parallels their increasing resistance when they cause other nosocomial infections.

Although infrequently identified in epidemiologic surveys, two infections, streptococcal gangrene due to Group A β-hemolytic streptococci and clostridial myonecrosis usually due to *Clostridium perfringens*, should be mentioned. These fulminant monomicrobial infections rarely develop following an operative procedure. The possibility of such an infection should be considered in a patient with clinical findings suggestive of

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**Table 2**

SSI rates (%) for selected procedures, according to risk index

<table>
<thead>
<tr>
<th>Procedure</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appendectomy</td>
<td>1.49</td>
<td>3.49</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bile duct, liver, or pancreatic surgery</td>
<td>8.77</td>
<td>16.34</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breast surgery</td>
<td>0.80</td>
<td>2.74</td>
<td>Not reported</td>
<td></td>
</tr>
<tr>
<td>Colon surgery</td>
<td>4.18</td>
<td>6.07</td>
<td>8.01</td>
<td>10.86</td>
</tr>
<tr>
<td>Gastric surgery</td>
<td>1.84</td>
<td></td>
<td>4.86</td>
<td></td>
</tr>
<tr>
<td>Herniorrhaphy (inpatient)</td>
<td>1.02</td>
<td>2.47</td>
<td>4.36</td>
<td></td>
</tr>
<tr>
<td>Peripheral vascular bypass surgery</td>
<td>2.00</td>
<td></td>
<td>6.69</td>
<td></td>
</tr>
<tr>
<td>Small bowel surgery</td>
<td>2.62</td>
<td></td>
<td>6.31</td>
<td></td>
</tr>
</tbody>
</table>

severe sepsis or septic shock out of proportion to those expected in a patient with a typical postoperative SSI. Typically, soft-tissue infections due to these organisms manifest themselves early after an operative procedure, sometimes within the first 24 hours. Because of their rapidly progressive nature, early surgical management coupled with appropriate antimicrobial therapy is mandatory.32

**PREVENTION OF SSI: GENERAL MEASURES**

Interventions to prevent SSI are based on knowledge of the various risk factors that predispose a patient to develop such an infection and an understanding of the microbiology of SSI. In this section, general measures to prevent SSI will be discussed; subsequent sections will focus on some of the issues related to antimicrobial prophylaxis and other interventions that target specific pathogens. The interventions discussed in this and subsequent sections are summarized in Table 3.
## Table 3
### Selected interventions for prevention of SSI

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Evidence^a</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Preoperative</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reduce hemoglobin A1c levels to &lt;7% before operation</td>
<td>Class II data</td>
<td>Anderson et al(^3)</td>
</tr>
<tr>
<td>Smoking cessation 30 d before operation</td>
<td>Class II data</td>
<td>Mangram et al, Anderson et al(^3)</td>
</tr>
<tr>
<td>Administer specialized nutritional supplements or enteral nutrition at severe nutritional risk for 7–14 d preoperatively; preoperative parenteral nutrition should not be routinely used, except selectively in patients with severe underlying malnutrition</td>
<td>Class I and Class II data with significant heterogeneity</td>
<td>Mangram et al, Anderson et al(^3) Weimann et al, Anonymous(^4)</td>
</tr>
<tr>
<td>Adequately treat preoperative infections, such as urinary tract infections</td>
<td>Class II data</td>
<td>Mangram et al, Anderson et al(^3)</td>
</tr>
<tr>
<td>Decolonization of unselected patients with mupirocin is not currently recommended</td>
<td>Class I data</td>
<td>Mangram et al, Anderson et al(^3), Kalmeijer et al, Perl et al, Konvalinka et al, Suzuki et al, Laupland and Conly</td>
</tr>
<tr>
<td>Identification and decolonization of <em>Staphylococcus aureus</em> carriers may be a potentially useful intervention, but requires further investigation</td>
<td>Limited Class I data</td>
<td>Rao et al, Hacek et al(^9)</td>
</tr>
<tr>
<td>Preoperative showering with chlorhexidine is not currently recommended</td>
<td>Class I data</td>
<td>Mangram et al, Anderson et al, Webster and Osborne(^4)</td>
</tr>
<tr>
<td><strong>Perioperative preparations</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Remove hair only if it will interfere with the operation; hair removal by clipping immediately before the operation or with depilatories; no pre- or perioperative shaving of surgical site(^b)</td>
<td>Class I data</td>
<td>Mangram et al, Anderson et al, Kjønniksen et al, Bratzler and Hunt, Springer(^7)</td>
</tr>
<tr>
<td>Use an antiseptic surgical scrub or alcohol-based hand antiseptic for preoperative cleansing of the operative team members’ hands and forearms</td>
<td>Class II data</td>
<td>Mangram et al, Anderson et al(^3)</td>
</tr>
<tr>
<td>Prepare the skin around the operative site with an appropriate antiseptic agent, including preparations based on alcohol, chlorhexidine, or iodine/iodophors</td>
<td>Class II data</td>
<td>Mangram et al, Anderson et al(^3), Digison(^4)</td>
</tr>
</tbody>
</table>
Administer prophylactic antibiotics for most clean-contaminated and contaminated procedures, and selected clean procedures; use antibiotics appropriate for the potential pathogens (Table 6)\(^b\)

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>Strength of Evidence</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Administer prophylactic antibiotics within 1 h before incision (2 h for vancomycin and fluoroquinolones)(^b)</td>
<td>Strong Class II data</td>
<td>Mangram et al,(^7) Bratzler and Hunt,(^44) Anonymous,(^67) Springer,(^70) Classen et al(^73)</td>
</tr>
<tr>
<td>Use higher dosages of prophylactic antibiotics for morbidly obese patients</td>
<td>Limited Class II data</td>
<td>Mangram et al,(^7) Forse et al(^38)</td>
</tr>
<tr>
<td>Use vancomycin as a prophylactic agent only when there is a significant risk of MRSA infection</td>
<td>Class I data</td>
<td>Mangram et al,(^7) Anderson et al,(^33) Anonymous,(^67) Bolon et al,(^96) Finkelstein et al(^97)</td>
</tr>
<tr>
<td>Operating room environment</td>
<td>Class II and Class III data</td>
<td>Mangram et al,(^7) Anderson et al(^33)</td>
</tr>
<tr>
<td>Provide adequate ventilation, minimize operating room traffic, and clean instruments and surfaces with approved disinfectants</td>
<td>Class II data</td>
<td>Mangram et al,(^7) Anderson et al(^33)</td>
</tr>
<tr>
<td>Avoid flash sterilization</td>
<td>Class II data</td>
<td>Mangram et al,(^7) Anderson et al(^33)</td>
</tr>
<tr>
<td>Use laminar airflow for orthopedic implant procedures. A common practice of uncertain utility</td>
<td>Contradictory Class II data</td>
<td>Mangram et al,(^7) Anderson et al,(^33) Brandt et al(^48)</td>
</tr>
<tr>
<td>Conduct of operation</td>
<td>Class III</td>
<td>Mangram et al,(^7) Anderson et al(^33)</td>
</tr>
<tr>
<td>Carefully handle tissue, eradicate dead space, and adhere to standard principles of asepsis</td>
<td>Class III</td>
<td>Mangram et al,(^7) Anderson et al(^33)</td>
</tr>
<tr>
<td>Avoid use of surgical drains unless absolutely necessary</td>
<td>Limited Class I, Class II data</td>
<td>Mangram et al,(^7) Barie(^49)</td>
</tr>
<tr>
<td>Leave contaminated or dirty-infected wounds open, with the possible exception of wounds following operations for perforated appendicitis</td>
<td>Limited Class I, Class II data</td>
<td>Mangram et al,(^7) Brasel et al,(^50) Cohn et al(^51)</td>
</tr>
<tr>
<td>Redose prophylactic antibiotics with short half-lives intraoperatively if operation is prolonged (for cefazolin if operation is &gt;3 h) or if there is extensive blood loss</td>
<td>Limited Class I, Class II data</td>
<td>Mangram et al,(^7) Scher,(^74) Swoboda et al(^75)</td>
</tr>
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### Table 3 (continued)

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Evidencea</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maintain intraoperative normothermia&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Class I; some contradictory Class II data</td>
<td>Mangram et al&lt;sup&gt;7&lt;/sup&gt;, Anderson et al&lt;sup&gt;33&lt;/sup&gt;, Bratzler and Hunt&lt;sup&gt;44&lt;/sup&gt;, Sessler and Akca&lt;sup&gt;53&lt;/sup&gt;, Kurz et al&lt;sup&gt;54&lt;/sup&gt;, Barone et al&lt;sup&gt;55&lt;/sup&gt;, Walz et al&lt;sup&gt;56&lt;/sup&gt;, Springer&lt;sup&gt;70&lt;/sup&gt;</td>
</tr>
<tr>
<td>Use 80% oxygen intraoperatively and immediately postoperatively. Not currently recommended, but a large clinical trial is evaluating the approach</td>
<td>Heterogeneous Class I data; meta-analysis supports use of this modality</td>
<td>Anderson et al&lt;sup&gt;33&lt;/sup&gt;, Greif et al&lt;sup&gt;57&lt;/sup&gt;, Pryor et al&lt;sup&gt;58&lt;/sup&gt;, Belda et al&lt;sup&gt;59&lt;/sup&gt;, Mayzler et al&lt;sup&gt;60&lt;/sup&gt;, Meyhoff et al&lt;sup&gt;62&lt;/sup&gt;</td>
</tr>
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</table>

### Postoperative management

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<thead>
<tr>
<th>Intervention</th>
<th>Evidencea</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Discontinue prophylactic antibiotics within 24 h after the procedure (48 h for cardiac surgery and liver transplant procedures); preferably, discontinue prophylactic antibiotics after skin closure&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Class I; meta-analyses support single dose regimens for prophylaxis</td>
<td>Mangram et al&lt;sup&gt;7&lt;/sup&gt;, Bratzler and Hunt&lt;sup&gt;44&lt;/sup&gt;, Anonymous&lt;sup&gt;67&lt;/sup&gt;, Springer&lt;sup&gt;70&lt;/sup&gt;, Barie&lt;sup&gt;76&lt;/sup&gt;, DiPiro et al&lt;sup&gt;77&lt;/sup&gt;, McDonald et al&lt;sup&gt;78&lt;/sup&gt;</td>
</tr>
<tr>
<td>Maintain serum glucose levels &lt;200 mg/dL on postoperative days 1 and 2&lt;sup&gt;d&lt;/sup&gt;</td>
<td>Class II data</td>
<td>Anderson et al&lt;sup&gt;33&lt;/sup&gt;, Bratzler and Hunt&lt;sup&gt;44&lt;/sup&gt;, Zerr et al&lt;sup&gt;63&lt;/sup&gt;, Furnary et al&lt;sup&gt;64&lt;/sup&gt;, Lazar et al&lt;sup&gt;65&lt;/sup&gt;, Carr et al&lt;sup&gt;66&lt;/sup&gt;, Springer&lt;sup&gt;70&lt;/sup&gt;</td>
</tr>
<tr>
<td>Monitor wound for the development of SSI</td>
<td>Class III data</td>
<td>Mangram et al&lt;sup&gt;7&lt;/sup&gt;, Anderson et al&lt;sup&gt;33&lt;/sup&gt;</td>
</tr>
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</table>

### Infection control and surveillance

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Evidencea</th>
<th>References</th>
</tr>
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<tbody>
<tr>
<td>Maintain an active surveillance system for monitoring incidence of SSI</td>
<td>Class II data</td>
<td>Mangram et al&lt;sup&gt;7&lt;/sup&gt;, Anderson et al&lt;sup&gt;33&lt;/sup&gt;</td>
</tr>
<tr>
<td>Provide feedback to practitioners regarding individual rates of SSI</td>
<td>Class II data</td>
<td>Mangram et al&lt;sup&gt;7&lt;/sup&gt;, Anderson et al&lt;sup&gt;33&lt;/sup&gt;</td>
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<sup>a</sup> Class I data from prospective, randomized, controlled trials or meta-analyses of such trials; Class II data from well-controlled prospective or retrospective studies with good study design; Class III data from uncontrolled studies, case series, or expert opinion. Evidence grades do not directly correspond to those provided in Mangram et al<sup>7</sup> and Anderson et al<sup>33</sup>.

<sup>b</sup> SCIP measure for cardiothoracic, vascular, colorectal surgical procedures, hip or knee arthroplasty, and hysterectomy.

<sup>c</sup> SCIP measure for colorectal procedures.

<sup>d</sup> SCIP measure for cardiac surgery procedures.
General measures to prevent SSI can be organized into those directed at the patient’s preoperative risk factors and those that relate to perioperative management of the patient. With respect to the latter, considerations include the patient’s and the operative team’s preparations for surgery, the operating room environment, intraoperative techniques, and other aspects of the patient’s intraoperative and postoperative cares.

As is typical with many medical therapies, there are varying degrees of scientific evidence supporting various interventions. Although some are supported by data from prospective randomized controlled trials or other high-quality studies, the evidence for many is based primarily on experience and expert opinion accumulated over the years, or even surgical dogma never subjected to rigorous evaluation. Practice guidelines summarizing recommendations and the evidence behind them for the prevention of SSI have been developed and updated by the CDC, most recently in 1999. Since then, no comprehensive set of guidelines for the prevention of SSI have appeared, although a recent publication from the Society for Healthcare Epidemiology of America and the Infectious Diseases Society of America summarizes the previous guidelines and provides some updates based on additional literature.

The patient’s pre-existing medical conditions are a major contributor to the risk of SSI. Significant numbers of patients undergoing operative procedures have one or more of the risk factors listed in Box 2. The preoperative history and physical examination will usually allow detection of these medical conditions. However, many of these risk factors are not readily amenable to intervention, even if a surgical procedure can be delayed. Age is obviously not a modifiable risk factor. Likewise, a prolonged preoperative hospital stay usually reflects the need for hospitalization of a seriously ill patient with a compromised physiologic state rather than an opportunity for intervention. Treating obesity or restoring immune competence to a patient who is immunosuppressed is generally not feasible in the short term. Generally accepted measures for preventing SSI include (1) optimizing preoperative glucose levels and lowering hemoglobin A1C concentrations in patients with diabetes; (2) encouraging patients to stop smoking at least 30 days before a procedure; and (3) treating any concomitant infection preoperatively. However, there are limited data indicating that these interventions successfully prevent SSI when applied to large populations. Small studies suggest that preoperative use of oral supplements or enteral nutrition for 7 to 14 days may reduce infectious complications such as SSI in patients at severe nutritional risk. However, use of preoperative parenteral nutrition has been associated with an increased risk of infectious complications, unless targeted at severely malnourished patients.

In contrast to interventions based on patients’ preoperative medical conditions, there are somewhat more complete data regarding certain perioperative approaches for prevention of SSI. Preoperative hair removal by shaving, particularly when performed the night before the procedure, has been consistently found to increase SSI rates. It is currently recommended that either hair not be removed or that it be removed by clipping immediately before the operation or by using non-caustic depilatories. Appropriate hair removal is one of the measures currently monitored as part of the Surgical Care Improvement Project (SCIP), an initiative developed by a partnership of nongovernmental and government organizations, including the American College of Surgeons, the CDC, and the Centers for Medicare and Medicaid Services (CMS).

Preoperative showering with antiseptic agents such as chlorhexidine has not been shown to have a beneficial impact on SSI rates. However, appropriate skin preparation at the time of the operative procedure with an antiseptic agent is
a well-established preventative measure. Acceptable antiseptic agents include alcohol, chlorhexidine, and iodine and iodophors, some of which now have been re-formulated to provide a longer duration of action.\textsuperscript{7,33,46} Use of chlorhexidine as a skin preparation has been recommended for prevention of catheter-related bloodstream infections;\textsuperscript{47} however, the available data have not conclusively shown that it, or any other surgical site preparation, is superior for the prevention of SSI.\textsuperscript{7,46} Similarly, although preparation of surgical team members' hands and forearms is a firm recommendation, the data are inadequate to indicate that any specific antiseptic agent or method is preferable.

The operating room environment may be the source of contamination leading to SSI in a limited number of cases. Generally accepted environmental measures to prevent SSI include maintaining adequate ventilation, minimizing operating room traffic, avoiding flash sterilization of operating room equipment, and cleaning surfaces and equipment with approved disinfectants.\textsuperscript{7,33} The use of laminar air flow in the operating room and respiratory isolation of the operating team have been suggested as additional measures to avoid infection, particularly during orthopedic implant procedures. However, high-quality data indicating that these interventions result in decreased infection rates are lacking,\textsuperscript{7} and a recent investigation questions whether use of laminar air flow has any efficacy whatsoever.\textsuperscript{48} Other aspects of the operating room environment, such as the types of surgical drapes or the attire of the surgical team, are of potential importance, but there is little information available indicating that any intervention related to these will directly impact the risk of SSI. Occasional outbreaks of SSI have been linked to the presence in the operating room of a team member with an active infection or colonization with a pathogenic organism; exclusion from the operating room is only recommended for individuals who have draining skin lesions or have been epidemiologically linked to patient infections.\textsuperscript{7}

The conduct of the operation by the surgeon and surgical team is another potential, although largely unproven, arena in which the risk of SSI might be altered. Traditionally, surgeons are taught that gentle handling of tissues, thorough irrigation of contamination, complete removal of devitalized or necrotic tissues, and avoidance of dead space are all important in avoiding infection.\textsuperscript{7,33} The use of drains has been associated with an increase rather than a decrease in the risk of SSI; in the absence of a clear indication, use of drains is strongly discouraged.\textsuperscript{49} Closure of a contaminated or dirty-infected wound remains a topic of debate. The universal rule that these wounds need to be left open has been challenged for some procedures. Using a decision analysis approach, Brasel and colleagues\textsuperscript{50} found that many wounds could be safely closed following operations for perforated appendicitis. However, a prospective randomized trial comparing primary closure with initial open management of dirty-infected wounds revealed that routine primary closure led to significantly more infections; nonetheless, hospital lengths of stay and costs of care did not differ between the two groups.\textsuperscript{51} Finally, there is little question that the use of minimally invasive approaches will decrease the risk of SSI; for instance, rates of SSI are significantly lower with laparoscopic appendectomy compared with open appendectomy.\textsuperscript{52}

With regard to management of the closed wound, various types of wound dressings, antibiotic ointments, and other adjuvants have been used. There are a number of new types of transparent, semipermeable, or antibacterial dressings available, some of which are marketed as being advantageous for the prevention of SSI. Nonetheless, there are almost no data indicating that any specific approach or method of postoperative wound management impacts SSI rates.

Several aspects of perioperative management, including avoidance of hypothermia, maintenance of high tissue oxygen concentrations, and treatment of hyperglycemia
have been investigated in some detail with respect to prevention of SSI. A frequent intraoperative problem is the development of hypothermia. A prospective trial of subjects undergoing colorectal operations found that subjects randomized to receive additional intraoperative warming to maintain normothermia (mean core temperature of 36.6°C) had a threefold reduction in SSI compared with subjects who did not receive supplemental warming (mean core temperature of 34.7°C). These positive results have been called into question somewhat by subsequent nonrandomized studies, which did not replicate this benefit. Nonetheless, maintenance of normothermia in patients undergoing colorectal procedures is one of the components currently monitored as part of the SCIP initiative.

More controversial is the use of increased inspired oxygen concentrations in the intraoperative and immediate postoperative periods. Four prospective randomized controlled trials compared use of 80% oxygen with 30% oxygen in subjects undergoing abdominal operations, primarily colorectal procedures. Two of these trials found significant reductions in the rates of SSI with the use of higher oxygen concentrations. One trial, which was underpowered, identified a trend toward fewer SSIs in subjects receiving 80% oxygen. However, one trial found an increase rather than a decrease in the SSI rates of subjects randomized to receive higher oxygen concentrations. A meta-analysis of these trials suggests the overall data favor use of higher oxygen concentrations, but given the heterogeneity of the results, this is still considered an unresolved issue. A large randomized clinical trial, currently underway in Denmark, will hopefully allow this controversy to be definitively resolved.

Avoidance of significant hyperglycemia in the intraoperative and postoperative period appears important in preventing SSI, particularly in patients undergoing cardiac surgical procedures. The risk of developing deep SSI and mediastinitis was found to be significantly reduced in cardiac surgery patients when frequent monitoring of blood glucose concentrations, coupled with use of insulin infusions as needed to control glucose concentrations was performed intraoperatively and postoperatively. This reduced risk applied to both diabetic and non-diabetic patients. Avoidance of serum glucose levels greater than 200 mg/dL at 6:00 AM on postoperative days 1 and 2 after cardiac surgery is one of the current performance measures of the SCIP initiative. Further, mediastinitis following coronary artery bypass surgery is a complication for which hospitals will receive no additional reimbursement from CMS, since it is considered a preventable infection.

With regard to other aspects of postoperative management, there are few interventions that have been recommended. Probably the most important detail is to monitor the surgical wound for the development of a SSI. It is generally accepted that early management of an infected wound helps avoid a more major subsequent complication. Unfortunately, some surgical practitioners are reluctant to intervene when there is a suspected SSI, which allows the infection to progress.

In addition to the efforts of individual surgeons, an effective infection control program is important in reducing institution-wide rates of SSI. Components of a successful infection control program include adequate surveillance for SSI, which is becoming increasingly difficult as hospital lengths of stay decrease and more patients develop SSIs as outpatients, and feedback to individual surgical practitioners so that practices can be modified.

PREVENTION OF SSI: ANTIMICROBIAL PROPHYLAXIS

Perioperative antimicrobial prophylaxis is widely used, and probably overused, for the prevention of SSI. In general, antimicrobial prophylaxis is recommended under two
circumstances: (1) when the risk of infection is relatively high, as it is for many clean-contaminated or contaminated operations, such as colorectal procedures; or (2) when the subsequent development of SSI could have disastrous consequences, such as with procedures involving implantation of a prosthetic vascular graft or orthopedic hardware.\(^7,67\) The use of antibiotic prophylaxis for certain clean procedures not meeting the second criteria, such as breast or hernia operations, remains controversial.\(^67,68\) As noted in Table 2, infection rates increase substantially for these operations in the presence of a single NNIS risk factor, one of which is a higher ASA score, indicating the patient has significant underlying medical comorbidities. However, whether or not a decision to use antimicrobial prophylaxis can be based on that risk assessment is unknown, because no definitive large-scale trials of antimicrobial prophylaxis have been performed in which subjects were stratified according to medical risk factors.\(^69\)

The general principles regarding antimicrobial prophylaxis include (1) selection of antimicrobial agents based on the likely pathogens responsible for a SSI with a particular operation; (2) administration of antibiotics shortly before the commencement of that operation such that serum and tissue levels are high at the time of incision and during the course of the operation; and (3) discontinuation of antimicrobial therapy at the end of the operation, or at most 24 to 48 hours after the procedure is completed.\(^37,67,69\) Compliance with these principles (appropriate selection, timing, and duration of antimicrobial prophylaxis) are monitored as part of the SCIP initiative,\(^44,70\) and are also included as measures in the Physician’s Quality Reporting Initiative of CMS, which provides financial incentives to practitioners who follow best practices.

Extensive guidelines regarding agents for surgical prophylaxis were published by the American Society of Health-System Pharmacists guidelines in 1999. The CDC guidelines also provide some general information about the subject.\(^7\) As part of the SCIP initiative, specific antimicrobial agents have been recommended for prophylaxis with certain operations: cardiothoracic, vascular or colorectal procedures, hip or knee arthroplasty, and hysterectomy.\(^44\) These recommendations are periodically updated.\(^70\) Prophylactic antibiotics for selected procedures are outlined in Table 4.

First and second generation cephalosporins are the preferred prophylactic agents for most surgical procedures.\(^7,44,67\) For clean procedures, the primary consideration is activity against staphylococci, although for clean-contaminated procedures, particularly upper gastrointestinal or gynecologic procedures, coverage of gram-negative *Enterobacteriaceae* is also a consideration. Both cefazolin and cefuroxime provide these antibacterial activities. Because of the large numbers of anaerobic bacteria in the lower gastrointestinal tract, anaerobic coverage is recommended for operations involving the distal small bowel, appendix, colon, and rectum. This can be provided by second generation cephalosporins with anti-anaerobic activity, such as cefoxitin or cefotetan, or by addition of an anti-anaerobic agent, such as clindamycin or metronidazole, to other first or second generation cephalosporins. For patients with significant β-lactam allergies, vancomycin or clindamycin is recommended for gram-positive coverage and aminoglycosides or fluoroquinolones are recommended when gram-negative activity is needed.\(^7,44,67\)

Much of the data supporting use of first and second generation cephalosporins for prophylaxis were derived from trials performed in the 1970s, 1980s, and early 1990s.\(^7,67\) Other than aminoglycoside-based regimens, few other agents were extensively tested in those trials. In the recent past, there have been very few trials focusing on the use of antimicrobial agents for surgical prophylaxis. This means that current recommendations are derived from data generated before the widespread development of resistance among gram-positive and gram-negative bacteria and that there
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<th>Recommended Agents</th>
<th>Potential Alternatives</th>
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<td>Mangram et al&lt;sup&gt;7&lt;/sup&gt;, Weimann et al&lt;sup&gt;40&lt;/sup&gt;, Anonymous&lt;sup&gt;67&lt;/sup&gt;, Springer&lt;sup&gt;70&lt;/sup&gt;</td>
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<td>Mangram et al&lt;sup&gt;7&lt;/sup&gt;, Weimann et al&lt;sup&gt;40&lt;/sup&gt;, Anonymous&lt;sup&gt;67&lt;/sup&gt;, Springer&lt;sup&gt;70&lt;/sup&gt;</td>
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<tr>
<td>Head and neck</td>
<td>Cefazolin, clindamycin</td>
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<td>Anonymous&lt;sup&gt;67&lt;/sup&gt;</td>
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<sup>a</sup> In the absence of a β-lactam allergy, vancomycin use is only recommended for prophylaxis when there is a high incidence of infections due to resistant staphylococci.
<sup>b</sup> An alternative for patients with significant allergies to β-lactam agents.
<sup>c</sup> Gentamicin, tobramycin, netilmicin, or amikacin, although gentamicin is the aminoglycoside generally recommended for use for prophylaxis.
<sup>d</sup> Ciprofloxacin, levofloxacin, or moxifloxacin. Not approved by the FDA for use in surgical prophylaxis. Because of its anti-anaerobic spectrum of activity, moxifloxacin could potentially be used without an additional anti-anaerobic agent.
<sup>e</sup> Clindamycin or metronidazole.
<sup>f</sup> Approved by the FDA for use as a prophylactic agent only for colorectal procedures.
is little data available regarding the efficacy of newer antimicrobial agents for surgical prophylaxis.

Only two newer antibiotics have been approved by the United States Food and Drug Administration (FDA) for surgical prophylaxis over the past decade or so. Alatrofloxacin, a fluoroquinolone, was found comparable to cefotetan for prophylaxis with colorectal procedures; however, this agent was subsequently withdrawn from the market. The other was ertapenem, which was evaluated against cefotetan in a prospective, randomized, controlled trial in subjects undergoing elective colorectal procedures. Overall, in the subset of evaluable subjects, 18% of those randomized to receive ertapenem developed a SSI compared with 31% of those who received cefotetan, a statistically significant difference. The difference was also significant in the modified intention-to-treat analysis. Thus, ertapenem can be used for prophylaxis for colorectal procedures and probably other operations involving the lower gastrointestinal tract where anaerobic coverage is needed, and is now included in the SCIP recommendations as an acceptable agent for colorectal procedures.

To achieve high concentrations in the tissues during an operative procedure, the timing of prophylactic antibiotics is critical. In experimental animal studies, infections were prevented only if antibiotics were administered immediately before or at the time a wound was made. This observation was supported by data from a large prospective observational trial by Classen and colleagues. In this study, subjects who received prophylactic antibiotics within a 2-hour period before the incision was made had the lowest incidence of SSI. Subjects who had antibiotics initiated more than 2 hours before the incision was made, and those whose antibiotics started more than 3 hours after the incision was made, had 6.7- and 5.8-fold increases in the risk of SSI, respectively. Even if antibiotics were started in the perioperative period, defined as 0 to 3 hours after the incision was made, the risk was still increased 2.4-fold, although this was not statistically significant. Thus, the general recommendation is that antibiotics should be administered within a 1-hour period before incision; however, a 2-hour time window is considered appropriate when vancomycin or fluoroquinolones are used, since these antibiotics need to be administered over a longer infusion time.

Adequate serum and tissue concentrations may not be maintained over the course of the operation, particularly with longer procedures or use of antibiotics with shorter half-lives. In addition, some patients may sustain rapid blood loss during a procedure, leading to inadequate concentrations of the prophylactic agent. Antimicrobial redosing is one solution to this problem. However, there are no firm guidelines with respect to this issue. Based on one study, it was recommended that cefazolin be redosed if the surgical procedure was longer than 3 hours. With use of an agent with a longer half-life, such as ertapenem, redosing would not generally be necessary.

Patients who are morbidly obese are another group of patients in whom achieving adequate antibiotic tissue levels can be challenging. One study noted low tissue levels of cefazolin when a 1-g dose was given preoperatively to morbidly obese subjects. This was overcome by using a higher 2-g dose. The use of the higher dose was associated with a decreased rate of SSI in these subjects. Although no definitive recommendation can be made, the use of higher doses of prophylactic agents would seem appropriate for patients who are morbidly obese.

When used for surgical prophylaxis, the duration of antibiotic therapy should be limited. With few exceptions, published guidelines recommend that antibiotics be discontinued within 24 hours of the operation. A maximum 48-hour duration of prophylactic therapy has been permitted for patients undergoing cardiovascular and
liver transplant procedures, although there is significant controversy regarding the need for longer therapy in those patients. Many authorities, in fact, question the utility of administering further antibiotics at all, once the incision is closed. Reviews of the available data suggest that single-dose regimens are as effective as multiple-dose regimens for surgical prophylaxis. Limiting the duration of antibiotic exposure should help curtail the development of resistant organisms and avoid other types of collateral damage, such as *Clostridium difficile*-associated disease. Nonetheless, it is routinely found that the principle of early discontinuation of prophylactic antimicrobial therapy is frequently violated by surgical practitioners, and is the SCIP measure that seems most refractory to change.

**PREVENTION OF SSI: SPECIAL CONSIDERATIONS REGARDING STAPHYLOCOCCAL INFECTIONS**

*S aureus* is responsible for more SSIs than any other microorganism. The incidence of SSI due to this organism appears to be increasing, as are the numbers of infections due to methicillin-resistant clones. Thus, there is considerable interest in approaches that could help prevent the development of SSI due to *S aureus*, including those due to MRSA.

Many, if not most, infections due to *S aureus* develop in patients colonized with this organism. Colonization of normal individuals with *S aureus* is quite common, and is a recognized risk factor for SSI. In epidemiologic surveys, approximately 25% to 30% of healthy individuals in the community were found to have their nares colonized with *S aureus*. In these healthy populations, nasal colonization with MRSA was uncommon, with only 1.0% to 2.6% of individuals found to carry this resistant pathogen. However, in a nationwide prevalence study, the number of individuals colonized with MRSA doubled from 0.8% in 2001–2002 to 1.5% in 2003–2004.

One potential approach to prevent SSI due to *S aureus* would be to preoperatively decolonize patients carrying this organism. Optimally, this approach would include preoperative screening of patients to detect those who were actually carriers of *S aureus*. This approach would be applicable both to patients colonized with methicillin-sensitive *S aureus* (MSSA) as well as those colonized with MRSA.

Preoperative decolonization of patients has been evaluated in a number of studies, although generally in unselected subjects rather than in confirmed carriers of *S aureus*. Topical mupirocin applied to the nares is the agent generally used for decolonization. Treatment with mupirocin eliminated nasal carriage of *S aureus* in 91% of colonized health care workers. In trials of preoperative decolonization, mupirocin was effective in 85%, 83%, and 82% of subjects colonized with *S aureus*. Data from nonrandomized trials suggested that decolonization of unselected preoperative subjects with mupirocin was effective in reducing the incidence of SSI due to *S aureus*; in some, the overall rate of SSI was also reduced. Recent studies of subjects undergoing orthopedic surgery, in which decolonization was applied only to subjects who were confirmed carriers of *S aureus*, also demonstrated improved outcomes with this approach. However, four prospective randomized controlled trials failed to demonstrate any benefit with use of preoperative mupirocin in unselected preoperative subjects. Recent meta-analyses of these randomized trials or of combined nonrandomized and randomized studies suggested that decolonization with mupirocin prevented SSI due to *S aureus*, but that an overall benefit in preventing SSI in general was less certain. The results appeared strongest for subjects undergoing cardiac or orthopedic procedures; the potential usefulness of this approach for patients undergoing general surgery was questionable. Given the variable results, the efficacy of decolonization is still considered an open question. Clearly, further
research is warranted, which may be facilitated by the increased availability of rapid screening techniques allowing more facile detection of \textit{S} \textit{aureus} carriage.

With respect to decreasing the risk of SSI specifically due to MRSA, a widely employed approach is to use prophylactic antibiotics effective against MRSA. Generally, vancomycin is the antibiotic used for this purpose in the United States; however, other glycopeptide antibiotics, such as teicoplanin, may be used elsewhere. Guidelines provide relatively little guidance as to when to use vancomycin. The CDC guidelines indicate that routine use of vancomycin is not recommended, although it may be the agent of choice when there is a cluster of SSI due to MRSA or coagulase-negative staphylococci. The ASHP guidelines suggest that vancomycin use should be restricted, although it is appropriate for surgical prophylaxis involving implantation of prosthetic materials at institutions where there is a high rate of infections due to MRSA or coagulase-negative staphylococci. However, neither guideline states a threshold for the incidence of infections due to resistant staphylococci that should lead to routine use of vancomycin for prophylaxis.

In part, this is due to the relatively poor results seen with the use of glycopeptides for prophylaxis. A meta-analysis by Bolon, and colleagues evaluated trials of subjects undergoing cardiac surgery randomized to receive prophylaxis with a glycopeptide or a \textit{\beta}-lactam antibiotic. No benefits were seen with the use of glycopeptides; the trend was actually toward better results with use of \textit{\beta}-lactam agents. In subgroup analyses, subjects who received glycopeptide prophylaxis were less likely to develop an infection due to a resistant gram-positive organism, but this advantage was more than offset by an overall increase in the numbers of gram-positive and total infections. A potential shortcoming of the meta-analysis is that the component studies took place before MRSA was widespread in the hospital setting; six of the seven studies were stated to occur in institutions where the prevalence of MRSA was low. Nonetheless, even in the institution where there was a high prevalence of MRSA, prophylaxis with vancomycin proved to be no better than prophylaxis with cefazolin.

There are several reasons why vancomycin may not be the ideal prophylactic agent, even in settings where there is a high prevalence of methicillin-resistant staphylococci. Vancomycin requires a prolonged infusion time to avoid development of the red man syndrome related to histamine release; this necessitates careful planning to ensure timely administration of the agent for prophylaxis. In addition, vancomycin distributes into tissues somewhat slowly; tissue concentrations may not be adequate to cover staphylococci in some patients. Further, vancomycin has no activity against gram-negative organisms. Thus, if vancomycin is used as the sole agent for prophylaxis, coverage of common gram-negative bacillary pathogens will be lacking; however, administration of a second agent to provide gram-negative coverage further increases the complexity of the prophylactic regimen. Finally, the therapeutic efficacy of vancomycin against staphylococci has been called into question recently. Vancomycin is generally considered less effective than \textit{\beta}-lactam agents when treating patients with infections due to MSSA, and there is some evidence that vancomycin is also less effective than other anti-MRSA agents for the treatment of infections due to MRSA. Additional research is urgently needed to determine optimal antibiotic prophylaxis in settings in which there is a high prevalence of MRSA, particularly since this high prevalence is increasingly becoming the rule rather than the exception.

**MANAGEMENT OF SSI**

SSI is suspected when there is erythema, drainage or fluctuance of the surgical incision, in the absence or presence of systemic signs of infection such as fever or
leukocytosis. Local signs of infection are usually apparent with superficial and deep SSI, although systemic signs are somewhat variable. In contrast, the presence of systemic signs of infection in the absence of local signs may indicate an organ/space infection or an infection originating from a source other than the surgical site.

The distinction between a superficial and a deep SSI may not be obvious on cursory examination; a necrotizing infection of the deeper tissues may progress if what was thought to be a superficial infection is neglected. Thus, the possibility of a necrotizing soft tissue infection should always be considered, especially when there is a particularly erythematous or painful wound, or patient appears more ill than would be expected with a relatively minor infection. The diagnosis of a necrotizing infection is best resolved by direct examination of the subcutaneous tissue and deeper layers.

Treatment of SSI nearly always involves opening the incision and establishing adequate drainage. The blind use of antibiotics to treat what appears to be cellulitis of the wound without adequately determining the need for drainage is to be discouraged. For most patients who have had their wounds opened and adequately drained, antibiotic therapy is unnecessary. One recommendation is to use antibiotics only when there are significant systemic signs of infection (temperature higher than 38.5°C or heart rate greater than 100 beats/min) or when erythema extends more than 5 cm from the incision. When antibiotics are used, selection should be based on the likely pathogens for a given operative procedure; thus, gram-positive organisms would be suspected following a clean orthopedic procedure, but involvement of gram-negative and anaerobic organisms would be expected if the infection followed a colorectal procedure. As with all soft tissue infections, the possibility that MRSA is involved in the infection needs to be kept in mind when choosing the empiric regimen. Although it has not necessarily been routine to culture most SSIs, this should be strongly considered in patients who will be treated with antibiotics, so that resistant microorganisms can be adequately treated.

For patients with complicated skin and soft tissue infections, antibiotic therapy is generally used. Thus, most patients with deep SSI who have elements of tissue necrosis should be treated with antibiotics. Antibiotic selection should follow the general guidelines established for the treatment of complicated skin and soft tissue infections. Patients who develop the rare early infections due to streptococci or clostridial organisms are usually treated with penicillin with or without clindamycin, and aggressive surgical debridement.

**SUMMARY**

SSI remains an important issue for surgeons, hospitals, and health care delivery systems. Despite encouraging trends in reduction of other nosocomial infections, there is little indication that much progress has been made in preventing SSI. In part, this may relate to the perceived trivial nature of this infection for many surgeons, despite the catastrophic consequences that occasionally follow the development of a SSI. A number of initiatives, some voluntary and others required by regulatory agencies, have been undertaken to improve surgical outcomes in recent years. The SCIP initiative and the National Surgical Quality Improvement Program include prevention of SSI as an important facet of their overall efforts to decrease surgical morbidity and mortality. These efforts to prevent SSI are handicapped, however, because many of the current recommendations regarding prevention are based on investigations performed several decades ago, when patient comorbidities were lower and pathogens were less resistant. To realize the full potential of these programs with
respect to prevention of SSI, new research needs to be performed, and investigators need to be reinvigorated to find better approaches to prevent this common complication of surgical therapy.

REFERENCES


