Introduction to the Centers for Disease Control and Prevention and the Healthcare Infection Control Practices Advisory Committee guideline for the prevention of surgical site infections

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Introduction to the Centers for Disease Control and Prevention and the Healthcare Infection Control Practices Advisory Committee Guideline for the Prevention of Surgical Site Infections

Joseph S. Solomkin,1 John Mazuski,2 Joan C. Blanchard,3 Kamal M.F. Itani,4 Philip Ricks,5 E. Patchen Dellinger,6 George Allen,7 Rachel Kelz,8 Caroline E. Reinke,8 and Sandra I. Berrios-Torres5,*

Abstract

Surgical site infection (SSI) is a common type of health-care–associated infection (HAI) and adds considerably to the individual, social, and economic costs of surgical treatment. This document serves to introduce the updated Guideline for the Prevention of SSI from the Centers for Disease Control and Prevention (CDC) and the Healthcare Infection Control Practices Advisory Committee (HICPAC). The Core section of the guideline addresses issues relevant to multiple surgical specialties and procedures. The second procedure-specific section focuses on a high-volume, high-burden procedure: Prosthetic joint arthroplasty. While many elements of the 1999 guideline remain current, others warrant updating to incorporate new knowledge and changes in the patient population, operative techniques, emerging pathogens, and guideline development methodology.

Keywords: infection; prevention; surgical site infection

Surgical site infections (SSIs), defined as infections anatomically associated with a surgical procedure performed in an operating room and not present before operation, remain an important problem for both patients and the healthcare system. Among an estimated 27 million surgical procedures performed each year in the United States, SSIs occur at a rate of 2 per 100 procedures, or approximately 500,000 per year [1,2]. These infections lead to increased duration of hospitalization, costs, morbidity, and risk of death. The average SSI, whether detected during the initial hospitalization or post-discharge and resulting in readmission, is associated with approximately one additional week of hospitalization and increases risk of death two to 11-fold compared with uninfected surgical patients [3,4]. SSIs caused by resistant organisms such as methicillin-resistant Staphylococcus aureus (MRSA) lead to even worse outcomes [5]. Each SSI costs approximately $12,000–$35,000 (2007 US dollars) to manage, with an annual total cost to the US healthcare system of approximately $10 billion [6]. As part of ongoing efforts to reduce the incidence and burden of SSIs, the Centers for Disease Control and Prevention (CDC) and the Healthcare Infection Control Practices Advisory Committee (HICPAC) have updated recommendations for the prevention of these infections [6–9].

Historical perspective: Is there a need for a new guideline?

Infection control activities became commonplace in the 1960s, with hospitals constructing individualized programs...
in the absence of standardized definitions of events such as SSIs [10]. In 1964, the National Research Council (NRC) sponsored a study evaluating the efficacy of ultraviolet irradiation that provided data validating a wound classification scheme that described the risk of SSI in relation to the extent of wound contamination. [11]. A clear connection between the contaminating flora at various surgical sites and subsequent infecting pathogens was established. That landmark document’s classification scheme remains in use today.

In 1983, CDC published the Guideline for Prevention of Surgical Wound Infections [8,9]. This guideline addressed only incisional wound infections; recommendations were based primarily on expert opinion. The 1985 revision clarified ambiguities in the previous guideline and provided new information on pre-operative hair removal and operating room ventilation [12]. The 1999 guideline adopted the term “surgical site infection” [6,7]. This guideline, cited by more than 2,500 publications, has served as the foundation for individual professional society guidelines, hospital infection control teams, and for the generation of national quality metrics by key organizations such as the Institute for Healthcare Improvement (IHI) and the National Quality Forum.

While many elements of the 1999 guideline remain current, others warrant updating to incorporate new knowledge as well as changes in patient populations, operative techniques, emerging pathogens, and guideline development methodology. The background health of the US population clearly has changed, with increasing incidences of obesity, diabetes mellitus, and other metabolic diseases [13–18]. The dramatic increase in the use of minimally invasive surgical techniques and implants may require a different approach to SSI prevention for those procedures [19–23]. The emergence of MRSA and other antimicrobial resistant pathogens may impact the rate and severity of SSI as well as potential prevention strategies [24]. Finally, more rigorous methodological approaches to guideline development have been established, calling for significant input from stakeholders in addition to experts in the field. These approaches are important in securing the confidence of professional organizations and providers, especially because evidence-based practice guidelines are increasingly applied to monitor the quality of healthcare delivery.

A national action plan to prevent health-care–associated infections (HAIs) has been developed [25]. The updated SSI guideline addresses concerns raised in the Health and Human Services plan. Beginning in 2012, hospitals participating in the Centers for Medicare & Medicaid Services (CMS) Inpatient Prospective Payment System were required to report SSI data related to two procedures—total abdominal hysterectomy and colon operations—through CDC’s National Healthcare Safety Network (NHSN) surveillance system. These data are included in the Inpatient Quality Reporting data publicly reported by CMS through the Hospital Compare Web site [26].

Care bundles

Care bundles are a collection of standardized clinical practices that individually have been shown to improve patient outcome. When implemented together, they are believed to result in a superior outcome compared with implementation of individual measures. The concept of care bundles was developed by the IHI to improve the reliability of delivery of essential healthcare processes [27,28]. Based on the IHI definition, a bundle is composed of three to five level 1 evidence elements. The expressed purpose of care bundles was to reduce practice variation and simultaneously improve overall quality of care and outcomes [29].

Currently, certain initiatives make implementation of specific care bundles mandatory or nearly so. Payers, such as CMS, increasingly are monitoring compliance with care bundles, such as process measures from the Surgical Care Improvement Project, both as a publicly reportable quality measure and as a tool to determine remuneration for certain medical services [26,30].

**New approach to the guideline**

In addition to the Core section addressing topics and recommendations applicable across surgical procedures, the guideline includes a new, procedure-specific section, focused on a single high-volume, high-burden procedure. The new structure is meant to serve as a targeted and effective way to provide timely guideline development, updates, and responses to emerging needs in addressing key clinical questions without requiring an update of the full guideline [31].

The procedure-specific section focuses on prosthetic joint arthroplasties. Approximately 1.2 million arthroplasties are performed in the United States annually [32]. In upcoming years, it is projected that there will be significant increases in the number of prosthetic joint arthroplasties performed, as well as their related SSIs and cost of treatment [32–36].

**Methodology**

Since 2009, CDC and HICPAC have utilized a new, evidence-based guideline development methodology [31]. The methodology includes generating key questions based on external expert opinion, performing targeted systematic reviews of the best available evidence, and providing an explicit link between the evidence and the resultant recommendations using a modified Grading of Recommendations Assessment, Development, and Evaluation (GRADE) method [37]. The GRADE system determines the strength of a recommendation based on the rigor of the individual studies. The largest weight is provided to high-quality, randomized studies but can include observational studies. In the case of meta-analyses, the component studies are analyzed to assure proper inclusion and analysis methodology.

There are several benefits to applying the level of rigor characteristic of the GRADE methodology to the development of recommendations. Most important, it is a transparent, systematic process that provides confidence for making clear policy statements regarding high-level recommendations. In addition, the GRADE system identifies specific recommendations in which there is a possibility of future change based on further data, and allows for the easy identification of evidence gaps and elaboration of future research priorities. Potential shortcomings of the GRADE system relate to whether the bar for high-level evidence is set realistically to infection control strategies that may be difficult to study and are therefore rarely subjected to rigorous randomized controlled trials. Some core practices of infection prevention—for example, practices that confer greater likelihood of asepsis—may be unethical to study via randomized controlled trials; recommendations in support of these practices...
must be made on a different basis than other recommendations, thereby giving the appearance of a less objective, systematic approach of applying GRADE criteria in fields of infection prevention and public health.

**Participants**

In addition to CDC, HICPAC, its non-voting liaisons, and ex-officio members from professional organizations and other federal agencies, a multi-disciplinary team of 35 SSI prevention experts have contributed to the process. There is official representation by the American College of Surgeons, the American Academy of Orthopaedic Surgeons, the Surgical Infection Society—North America, the Musculoskeletal Infection Society, and the Association of periOperative Registered Nurses. Additional national and international experts provided expertise in general and orthopedic surgery, *S. aureus* colonization, biofilm, and environmental sciences. The University of Pennsylvania Health System’s Center for Evidence-based Practices provided expertise in evidence-based methodology and together with CDC and HICPAC leads, comprised the core writing group.

**Dissemination**

Previous CDC and HICPAC SSI guidelines were published in infection control journals [7,12,38] followed by summary statements in the surgical literature [8,39]. In 1983, Simmons stated: “...to prevent surgical wound infections, personnel who perform the operation must take the lead in instituting prevention measures” [9]. To engage further the surgical community and capitalize on the multi-disciplinary collaboration already established in the guideline development process, publishing in the general and orthopedic surgery literature will be important. In addition, the full guideline with recommendations, evidence, and GRADE tables will be available for free download on the CDC website.

This is one of two additional articles being published with the guideline, authored by leaders in SSI prevention who served as content experts on the guideline. These documents complement the updated guideline structure, now focused on the evidence-based recommendations and GRADE tables. This introductory article describes relevant changes in nomenclature, epidemiology, risk factors, pathogenesis, and the rationale for use of the GRADE system to generate recommendations. The “Priority Surgical Site Infection Prevention Research Opportunities” article compiles potential research questions based on evidence gaps identified. These articles reinforce the collaboration between clinical and public health in the guideline development process and additional questions derived by the content experts in defining research priorities.

**Epidemiology of SSI**

**Definition**

The first formal definition of a wound infection was “pus in the wound” [11]. In 1992, the CDC definition of infection at the surgical site expanded from “wound infection” to “surgical site infection” [40]. The change in nomenclature was introduced to provide a single term to include both incisional (superficial or deep) and organ/space infections. These SSI definitions were originally developed for use by hospitals reporting data to CDC’s National Nosocomial Infection Surveillance system, a precursor to the NHSN Patient Safety Component [40]. NHSN launched in 2005.

The impact of an SSI on both the patient and healthcare system varies by infection type. Several studies have shown that the number of infections detected increases the longer that surveillance is continued after discharge [41]. Superficial incisional SSIs often do not require hospitalization and are inconsistently diagnosed by post-discharge surveillance [42,43]. Collectively, deep incisional and organ space infections are considered “complex” SSIs. In most series, complex SSIs represent about one-third to one-half of SSIs, although this varies according to procedure (Table 1). Complex SSIs typically require re-hospitalization, drainage or debridement, and systemic antimicrobial therapy. These infections generate considerable morbidity, cost, and even death. Unfortunately, many surveys examining incidence, cost, morbidity, or death do not make a distinction between complex and superficial SSIs.

**Incidence**

Systematic study of the incidence of SSIs dates to the 1950s [44,45]. Much of this work focused on *S. aureus*, including investigations into the role of nasal colonization as a risk factor for infection [46]. Cruse and Foor [47,48] performed a 10-year, prospective study of wound infections after operation across multiple surgical services at a large teaching hospital. Using the NRC wound classification schemes, they noted that infection rates were <2% for “clean wounds,” 6%–9% for “clean-contaminated wounds,” 13%–20% for “contaminated wounds,” and approximately 40% for “dirty wounds.” Their work also supported the notion that reporting surgeon-specific SSI rates was associated with reduced infection rates [48–51]. Other single institution studies confirmed these findings [51–53].

Surveillance is a tool to ensure that hospital systems are working properly and that interventions taken to reduce the risk of HAIs are working [54]. National public reporting through NHSN focuses on procedure risk adjusted, category-specific, and combined deep incisional and organ/space SSIs (Table 1). The NRC “clean, clean-contaminated, contaminated, dirty” classification scheme is not used in public reporting.

**Risk factors**

Although debate remains over best methods, it is a well accepted that risk adjustment should be based on patient-specific factors and be consistently applied [55]. Risk factors for the development of SSI are typically divided into patient-related (pre-operative), procedure-related (peri-operative), and post-operative categories (Table 2). In general, patient-related risk factors for SSI can be categorized as either modifiable (e.g., poorly controlled diabetes mellitus, obesity, tobacco use, length of pre-operative hospitalization, and colonization) or non-modifiable (e.g., age). Procedure-related risk factors include, but are not limited to: wound class, organ site, and length of operation. Post-operative risk factors can include wound care, post-operative blood transfusions, and hyperglycemia in both diabetic and non-diabetic patients. Efforts to improve risk modeling are ongoing [2,56,57].
Pathogenesis and Microbiology

Understanding of the pathogenesis of SSI has changed substantially since the 1999 guideline. The concept of a "microbiome," the hypothesis that we live within and interact with cohorts of micro-organisms in a commensal relationship, has been well established [57]. Several different bacterial and fungal commensals can become pathogenic when they create biofilms, structured communities within three-dimensional matrices of extracellular polymeric substances [58]. Biofilms have significantly increased resistance to key mechanisms of innate host defense and antimicrobial agents [59]. Biofilm formation, for which there is currently no preventive strategy, is a key variable in infections with implanted devices and may remain under-recognized as a factor in all infections.

Recent efforts have been undertaken to understand the molecular basis of biofilm formation in staphylococci, which cause frequent biofilm-associated infections [60]. The development of a bacterial biofilm involves an initial microbial attachment and a subsequent maturation phase. A final detachment phase, which involves the separation of single

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Procedures, n</th>
<th>Infections</th>
<th>Rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abdominal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bile duct, liver, or pancreatic operation</td>
<td>10,228</td>
<td>328</td>
<td>3.2</td>
</tr>
<tr>
<td>Colon operation</td>
<td>300,526</td>
<td>8,952</td>
<td>3.0</td>
</tr>
<tr>
<td>Small bowel operation</td>
<td>22,058</td>
<td>453</td>
<td>2.1</td>
</tr>
<tr>
<td>Rectal operation</td>
<td>6,561</td>
<td>112</td>
<td>1.7</td>
</tr>
<tr>
<td>Herniorrhaphy</td>
<td>16,134</td>
<td>140</td>
<td>0.87</td>
</tr>
<tr>
<td>Gastric operation</td>
<td>31,494</td>
<td>228</td>
<td>0.72</td>
</tr>
<tr>
<td>Cholecyctectomy</td>
<td>65,079</td>
<td>272</td>
<td>0.41</td>
</tr>
<tr>
<td>Neurologic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ventricular shunt</td>
<td>7,399</td>
<td>143</td>
<td>1.9</td>
</tr>
<tr>
<td>Vascular</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peripheral vascular bypass surgery</td>
<td>8,755</td>
<td>198</td>
<td>2.2</td>
</tr>
<tr>
<td>Spine</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Refusion of spine</td>
<td>5,740</td>
<td>85</td>
<td>1.4</td>
</tr>
<tr>
<td>Spinal fusion</td>
<td>110,975</td>
<td>793</td>
<td>0.71</td>
</tr>
<tr>
<td>Laminectomy</td>
<td>100,750</td>
<td>361</td>
<td>0.35</td>
</tr>
<tr>
<td>Cardiac</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coronary artery bypass graft</td>
<td>117,972</td>
<td>796</td>
<td>0.67</td>
</tr>
<tr>
<td>Breast</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breast operation</td>
<td>13,801</td>
<td>148</td>
<td>1.1</td>
</tr>
<tr>
<td>Arthroplasty procedures</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hip prosthesis</td>
<td>291,628</td>
<td>2,006</td>
<td>0.69</td>
</tr>
<tr>
<td>Knee prosthesis</td>
<td>417,937</td>
<td>1,547</td>
<td>0.38</td>
</tr>
<tr>
<td>Obstetric and gynecologic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abdominal hysterectomy</td>
<td>307,648</td>
<td>2,020</td>
<td>0.66</td>
</tr>
<tr>
<td>Cesarean section</td>
<td>211,468</td>
<td>329</td>
<td>0.16</td>
</tr>
<tr>
<td>Ovarian operation</td>
<td>32,082</td>
<td>26</td>
<td>0.08</td>
</tr>
</tbody>
</table>

*Coronary artery bypass graft includes procedures with either SSI of chest only or chest and donor site incisions.

Table 2. Established Risk Factors for Surgical Site Infections

<table>
<thead>
<tr>
<th>Patient-related factors</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Age [79–80]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obesity or malnutrition [55,80,81]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes or hyperglycemia [82–84]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking [85]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Colonization [86,87]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Remote infection [88]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-operative hospital stay [89]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Procedure-related factors</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Instrument cleaning, decontamination, and sterilization</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Operating room ventilation [90]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-operative shaving [91]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Surgical skin preparation [92,93]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Surgical scrub [94]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Surgical technique [95–97]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Surgical drains [98]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tourniquet time [99]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Revision arthroplasty [71]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Procedure duration [100,101]</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Pathogenesis and Microbiology

Understanding of the pathogenesis of SSI has changed substantially since the 1999 guideline. The concept of a "microbiome," the hypothesis that we live within and interact with cohorts of micro-organisms in a commensal relationship, has been well established [57]. Several different bacterial and fungal commensals can become pathogenic when they create biofilms, structured communities within three-dimensional matrices of extracellular polymeric substances [58]. Biofilms have significantly increased resistance to key mechanisms of innate host defense and antimicrobial agents [59]. Biofilm formation, for which there is currently no preventive strategy, is a key variable in infections with implanted devices and may remain under-recognized as a factor in all infections.

Recent efforts have been undertaken to understand the molecular basis of biofilm formation in staphylococci, which cause frequent biofilm-associated infections [60]. The development of a bacterial biofilm involves an initial microbial attachment and a subsequent maturation phase. A final detachment phase, which involves the separation of single
cells or cell clusters by various mechanisms, is considered to be crucial for the dissemination of bacteria to secondary sites of infections. Detached biofilm bacteria may establish secondary biofilm infections elsewhere, which can possibly be of greater severity.

S. aureus: High-risk pathogen

The micro-organisms causing SSIs are well known [61]. Staphylococcal species are the predominant pathogens in all but gastrointestinal procedures (Table 3). In those procedures, particularly those involving the small and large intestines, *Escherichia coli*, **Pseudomonas aeruginosa**, Klebsiella spp., and other facultative aerobes predominate.

*Staphylococcus aureus* is an extraordinarily versatile pathogen that can survive in hostile environmental conditions, colonize mucous membranes and skin, and cause severe, non-purulent, toxin-mediated disease or invasive pyogenic infections. There has been rapid appearance and spread of a particular genotype of *S. aureus*, which in the United States is predominately the USA300 strain [62]. A variety of surface protein adhesins, excreted toxins, and complex translational and transcriptional regulatory systems allow this strain to be a highly virulent and common pathogen in skin and soft tissue infections, SSIs, pneumonia, bacteremia, and osteomyelitis [63–65]. The incidence of soft tissue and pulmonary infections from community-associated MRSA has increased dramatically, particularly in children and young, immunocompetent adults [66]. Although initially described as community-associated or community-onset MRSA, this strain is now quite common in healthcare settings and is replacing other strains of MRSA as a cause of HAIs [63–65]. The USA300 strain also has become a common pathogen for SSIs; it is the most common form of *S. aureus* found in SSIs in hospitalized patients [67,68]. Methods to reduce the risk of infection because of this organism are an important focus for clinical research.

**Hyperglycemia as a key risk factor for SSIs**

The increase in obesity and decrease in physical activity underlies the alarming rise in the incidence of diabetes mellitus [69]. The increasing incidence of this disease, and its hallmark, hyperglycemia, is associated with increased infection rates [70,71].

The mechanisms by which hyperglycemia produces end-organ damage are now well understood [72]. Hyperglycemia has been known since the early 1960s to cause oxidative stress [73]. The term “oxidative stress” refers to an imbalance between the production of reactive oxygen species and their resultant damage and a biologic system’s ability to detoxify these reactive intermediates and correct the resulting injury [74]. Certain cell types, such as capillary endothelial cells in the retina, mesangial cells in the renal glomerulus, and neurons and Schwann cells in peripheral nerves, are particularly vulnerable to these reactive oxygen species, explaining the organ-specific damage seen in diabetes mellitus.

### Table 3. Percent of Surgical Site Infections with Select Pathogen, by Procedure Category, Deep Incision and Organ Space Infections, Detected on Admission or Re-Admission, National Healthcare Safety Network, 2011**

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Procedure category</th>
<th>All</th>
<th>Orthopedic</th>
<th>Abdominal</th>
<th>Cardiac</th>
<th>Ob/gyn</th>
<th>Neurologic</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Staphylococcus aureus</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MSSA*</td>
<td></td>
<td>1,656</td>
<td>14.2</td>
<td>1,112</td>
<td>24.9</td>
<td>131</td>
<td>3.1</td>
</tr>
<tr>
<td>MRSA</td>
<td></td>
<td>1,199</td>
<td>10.3</td>
<td>779</td>
<td>17.4</td>
<td>141</td>
<td>3.3</td>
</tr>
<tr>
<td><em>Escherichia coli</em></td>
<td></td>
<td>1,184</td>
<td>10.2</td>
<td>203</td>
<td>4.5</td>
<td>773</td>
<td>18.1</td>
</tr>
<tr>
<td>Coagulase-negative staphylococci</td>
<td></td>
<td>1,084</td>
<td>9.3</td>
<td>601</td>
<td>13.5</td>
<td>128</td>
<td>3.0</td>
</tr>
<tr>
<td><em>Enterococcus faecalis</em></td>
<td></td>
<td>691</td>
<td>5.9</td>
<td>174</td>
<td>3.9</td>
<td>383</td>
<td>9.0</td>
</tr>
<tr>
<td><em>Pseudomonas aeruginosa</em></td>
<td></td>
<td>561</td>
<td>4.8</td>
<td>169</td>
<td>3.8</td>
<td>210</td>
<td>4.9</td>
</tr>
<tr>
<td>Klebsiella pneumoniae (and K. oxytoca)</td>
<td></td>
<td>491</td>
<td>4.2</td>
<td>92</td>
<td>2.1</td>
<td>285</td>
<td>6.7</td>
</tr>
<tr>
<td><em>Enterobacter</em> spp.</td>
<td></td>
<td>483</td>
<td>4.1</td>
<td>168</td>
<td>3.8</td>
<td>185</td>
<td>4.3</td>
</tr>
<tr>
<td><em>Enterococcus</em> spp.</td>
<td></td>
<td>410</td>
<td>3.5</td>
<td>73</td>
<td>1.6</td>
<td>256</td>
<td>6.0</td>
</tr>
<tr>
<td><em>Enterococcus faecium</em></td>
<td></td>
<td>290</td>
<td>2.5</td>
<td>39</td>
<td>0.9</td>
<td>201</td>
<td>4.7</td>
</tr>
<tr>
<td><em>Candida albicans</em></td>
<td></td>
<td>218</td>
<td>1.9</td>
<td>12</td>
<td>0.3</td>
<td>157</td>
<td>3.7</td>
</tr>
<tr>
<td>Other <em>Candida</em> spp. or NOS</td>
<td></td>
<td>124</td>
<td>1.1</td>
<td>17</td>
<td>0.4</td>
<td>82</td>
<td>1.9</td>
</tr>
<tr>
<td><em>Acinetobacter baumannii</em></td>
<td></td>
<td>40</td>
<td>0.3</td>
<td>21</td>
<td>0.5</td>
<td>6</td>
<td>0.1</td>
</tr>
<tr>
<td>Other</td>
<td></td>
<td>3,122</td>
<td>26.8</td>
<td>941</td>
<td>21.1</td>
<td>1,320</td>
<td>31.0</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td></td>
<td>11,650</td>
<td>100</td>
<td>4,468</td>
<td>100</td>
<td>4,264</td>
<td>100</td>
</tr>
</tbody>
</table>

*Preliminary analysis based on data available in 9/2012.

**The types of surgery included in each category are as follows: Orthopedic: open reduction of fracture, hip prosthesis, knee prosthesis, limb amputation, spinal fusion, refusion of spine, and laminctomy; abdominal: appendectomy, bile duct, liver, or pancreatic operation, gallbladder operation, colon operation, gastric operation, herniorrhaphy, small-bowel operation, spleen operation, abdominal operation, and rectal operation; cardiac: cardiac operation, coronary artery bypass graft with chest incision with or without donor incision, pacemaker operation, and thoracic operation; obstetric/gynecologic (Ob/gyn): cesarean section, abdominal hysterectomy, ovarian operation, and vaginal hysterectomy; neurologic: craniotomy and ventricular shunt.

*MRSA denotes methicillin resistant *S. aureus*, defined as resistant to methicillin, oxacillin, or cefoxitin; MSSA denotes not resistant to these antibiotics.
In addition, oxidative stress leads to the production of advanced glycation end-products, which are sensed as a "damage" signal by endothelial cells. These trigger an inflammatory reaction [76]. Sterile inflammation has a well-recognized role as an adjuvant for the establishment of tissue invasive infection at low organism densities, leading to an overall increased susceptibility to infection in patients with diabetes mellitus.

Can We Get to Zero SSI?

We are now in an era of zero tolerance for HAIs. Even though SSI rates are unlikely to ever be zero, this is an important agreed upon aspirational goal for improving patient safety. Despite current movement by CMS to link SSI reductions to payment incentives, we are likely to face many challenges in reducing SSIs, including the increasing proportion of infections caused by antimicrobial-resistant pathogens such as MRSA. This may, in turn, reflect increasing numbers of severely ill and immunocompromised surgical patients as well as the impact of poor antimicrobial stewardship through unnecessary widespread use of broad-spectrum antimicrobial agents. Even if it is not possible currently to conceive of a time when there will be zero SSIs, this remains a useful goal for the future. The goal of zero SSIs promotes not only prevention of the SSIs currently preventable using existing strategies, but also the research necessary to discover future strategies that can prevent the remaining fraction of infections.

Summary

There have been important changes in the epidemiology of SSI in the United States and elsewhere in the world since publication of the previous guideline in 1999. These include changes in risk factors for SSI, particularly the increased resistance and virulence of infecting organisms and the increasing use of surgical procedures in vulnerable patients at risk for infection because of underlying diseases such as obesity and diabetes mellitus. There have also been new insights into microbial mechanisms of infection as well as the mechanisms by which some disease processes lead to increased rates of infection. These developments warrant review, addition to, and, in some places, revision of previous recommendations for the prevention of SSI.

Author Disclosure Statement

No competing financial interests exist.

The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention. The contents of this publication do not necessarily reflect the views or policies of the Department of Health and Human Services, nor does mention of trade names, commercial products, or organizations imply endorsement by the United States government. The authors assume full responsibility of the accuracy and completeness of the ideas presented.

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