GUIDELINE FOR PREVENTION OF SURGICAL SITE INFECTION, 1999

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Special Report

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EXECUTIVE SUMMARY

The "Guideline for Prevention of Surgical Site Infection, 1999" presents the Centers for Disease Control and Prevention (CDC)'s recommendations for the prevention of surgical site infections (SSIs), formerly called surgical wound infections. This two-part guideline updates and replaces previous guidelines.[1,2]

Part I, "Surgical Site Infection: An Overview," describes the epidemiology, definitions, microbiology, pathogenesis, and surveillance of SSIs. Included is a detailed discussion of the pre-, intra-, and postoperative issues relevant to SSI genesis.

Part II, "Recommendations for Prevention of Surgical Site Infection," represents the consensus of the Hospital Infection Control Practices Advisory Committee (HICPAC) regarding strategies for the prevention of SSIs.[3] Whenever possible, the recommendations in Part II are based on data from well-designed scientific studies. However, there are a limited number of studies that clearly validate risk factors and prevention measures for SSI. By necessity, available studies have often been conducted in narrowly defined patient populations or for specific kinds of operations, making generalization of their findings to all specialties and types of operations potentially problematic. This is especially true regarding the implementation of SSI prevention measures. Finally, some of the infection control practices routinely used by surgical teams cannot be rigorously studied for ethical or logistical reasons (e.g., wearing vs not wearing gloves). Thus, some of the recommendations in Part II are based on a strong theoretical rationale and suggestive evidence in the absence of confirmatory scientific knowledge.

It has been estimated that approximately 75% of all operations in the United States will be performed in "ambulatory," "same-day," or "outpatient" operating rooms by the turn of the century.[4] In recommending various SSI prevention methods, this document makes no distinction between surgical care delivered in such settings and that provided in conventional inpatient operating rooms. This document is primarily intended for use by surgeons, operating room nurses, postoperative inpatient and clinic nurses, infection control professionals, anesthesiologists, healthcare epidemiologists, and other personnel directly responsible for the prevention of nosocomial infections.

This document does *not:*

- Specifically address issues unique to burns, trauma, transplant procedures, or transmission of bloodborne pathogens from healthcare worker to patient, nor does it specifically address details of SSI prevention in pediatric surgical practice. It has been recently shown in a multicenter study of pediatric surgical patients that characteristics related to the operations are more important than those related to the physiologic status of the patients.[5] In general, all SSI prevention measures effective in adult surgical care are indicated in pediatric surgical care.
- Specifically address procedures performed outside of the operating room (e.g., endoscopic procedures), nor does it provide guidance for infection prevention for invasive procedures such as cardiac catheterization or interventional radiology. Nonetheless, it is likely that many SSI prevention strategies also could be applied or adapted to reduce infectious complications associated with these procedures.
- Specifically recommend SSI prevention methods

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unique to minimally invasive operations (i.e., laparoscopic surgery). Available SSI surveillance data indicate that laparoscopic operations generally have a lower or comparable SSI risk when contrasted to open operations.[6-11] SSI prevention measures applicable in open operations (e.g., open cholecystectomy) are indicated for their laparoscopic counterparts (e.g., laparoscopic cholecystectomy).

- Recommend specific antiseptic agents for patient preoperative skin preparations or for healthcare worker hand/forearm antisepsis. Hospitals should choose from products recommended for these activities in the latest Food and Drug Administration (FDA) monograph.[12]

I. SURGICAL SITE INFECTION (SSI): AN OVERVIEW

A. INTRODUCTION

Before the mid-19th century, surgical patients commonly developed postoperative "irritative fever," followed by purulent drainage from their incisions, overwhelming sepsis, and often death. It was not until the late 1860s, after Joseph Lister introduced the principles of antisepsis, that postoperative infectious morbidity decreased substantially. Lister's work radically changed surgery from an activity associated with infection and death to a discipline that could eliminate suffering and prolong life.

Currently, in the United States alone, an estimated 27 million surgical procedures are performed each year.[13] The CDC's National Nosocomial Infections Surveillance (NNIS) system, established in 1970, monitors reported trends in nosocomial infections in U.S. acute-care hospitals. Based on NNIS system reports, SSIs are the third most frequently reported nosocomial infection, accounting for 14% to 16% of all nosocomial infections among hospitalized patients.[14] During 1986 to 1996, hospitals conducting SSI surveillance in the NNIS system reported 15,523 SSIs following 593,344 operations (CDC, unpublished data). Among surgical patients, SSIs were the most common nosocomial infection, accounting for 38% of all such infections. Of these SSIs, two thirds were confined to the incision, and one third involved organs or spaces accessed during the operation. When surgical patients with nosocomial SSI died, 77% of the deaths were reported to be related to the infection, and the majority (93%) were serious infections involving organs or spaces accessed during the operation.

In 1980, Cruse estimated that an SSI increased a patient's hospital stay by approximately 10 days and cost an additional $2,000.[15,16] A 1992 analysis showed that each SSI resulted in 7.3 additional postoperative hospital days, adding $3,152 in extra charges.[17] Other studies corroborate that increased length of hospital stay and cost are associated with SSIs.[18,19] Deep SSIs involving organs or spaces, as compared to SSIs confined to the incision, are associated with even greater increases in hospital stays and costs.[20,21]

Advances in infection control practices include improved operating room ventilation, sterilization methods, barriers, surgical technique, and availability of antimicrobial prophylaxis. Despite these activities, SSIs remain a substantial cause of morbidity and mortality among hospitalized patients. This may be partially explained by the emergence of antimicrobial-resistant pathogens and the increased numbers of surgical patients who are elderly and/or have a wide variety of chronic, debilitating, or immunocompromising underlying diseases. There also are increased numbers of prosthetic implant and organ transplant operations performed. Thus, to reduce the risk of SSI, a systematic but realistic approach must be applied with the awareness that this risk is influenced by characteristics of the patient, operation, personnel, and hospital.

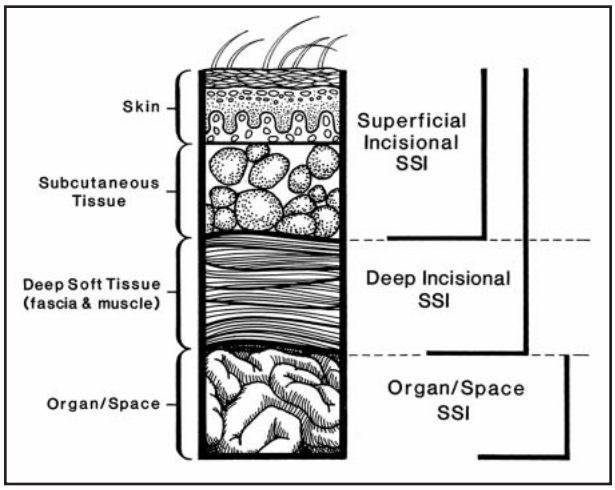

FIGURE. Cross-section of abdominal wall depicting CDC classifications of surgical site infection.[22]

B. KEY TERMS USED IN THE GUIDELINE

1. Criteria for Defining SSIs

The identification of SSI involves interpretation of clinical and laboratory findings, and it is crucial that a surveillance program use definitions that are consistent and standardized; otherwise, inaccurate or uninterpretable SSI rates will be computed and reported. The CDC's NNIS system has developed standardized surveillance criteria for defining SSIs (Table 1).[22] By these criteria, SSIs are classified as being either incisional or organ/space. Incisional SSIs are further divided into those involving only skin and subcutaneous tissue (superficial incisional SSI) and those involving deeper soft tissues of the incision (deep incisional SSI). Organ/space SSIs involve any part of the anatomy (e.g., organ or space) other than incised body wall layers, that was opened or manipulated during an operation (Figure). Table 2 lists site-specific classifications used to differentiate organ/space SSIs. For example, in a patient who had an appendectomy and subsequently developed an intra-abdominal abscess not draining through the incision, the infection would be reported as an organ/space SSI at the intra-abdominal site. Failure to use objective criteria to define SSIs has been shown to substantially affect reported SSI rates.[23,24] The CDC NNIS definitions of SSIs have been applied consistently by surveillance and surgical personnel in many settings and currently are a de facto national standard.[22,25]

TABLE 1
CRITERIA FOR DEFINING A SURGICAL SITE INFECTION (SSI)*

Superficial Incisional SSI

Infection occurs within 30 days after the operation
and
infection involves only skin or subcutaneous tissue of the incision
and at least *one* of the following:

1. Purulent drainage, with or without laboratory confirmation, from the superficial incision.
2. Organisms isolated from an aseptically obtained culture of fluid or tissue from the superficial incision.
3. 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, *unless* incision is culture-negative.
4. Diagnosis of superficial incisional SSI by the surgeon or attending physician.

Do *not* report the following conditions as SSI:

1. Stitch abscess (minimal inflammation and discharge confined to the points of suture penetration).
2. Infection of an episiotomy or newborn circumcision site.
3. Infected burn wound.
4. Incisional SSI that extends into the fascial and muscle layers (see deep incisional SSI).

Note: Specific criteria are used for identifying infected episiotomy and circumcision sites and burn wounds.[433]

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 operation
and
infection involves deep soft tissues (e.g., fascial and muscle layers) of the incision
and at least *one* of the following:

1. Purulent drainage from the deep incision but not from the organ/space component of the surgical site.
2. A deep incision spontaneously dehisces or is deliberately opened by a surgeon when the patient has at least one of the following signs or symptoms: fever (>38°C), localized pain, or tenderness, unless site is culture-negative.
3. An abscess or other evidence of infection involving the deep incision is found on direct examination, during reoperation, or by histopatholog ic or radiologic examination.
4. Diagnosis of a deep incisional SSI by a surgeon or attending physician.

Notes:

1. Report infection that involves both superficial and deep incision sites as deep incisional SSI.
2. Report an organ/space SSI that drains through the incision as a deep incisional SSI.

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 operation
and
infection involves any part of the anatomy (e.g., organs or spaces), other than the incision, which was opened or manipulated during an operation
and at least *one* of the following:

1. Purulent drainage from a drain that is placed through a stab wound‡ into the organ/space.
2. Organisms isolated from an aseptically obtained culture of fluid or tissue in the organ/space.
3. 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.
4. Diagnosis of an organ/space SSI by a surgeon or attending physician.

* Horan TC et al.[22]
† National Nosocomial Infection Surveillance definition: a nonhuman-derived implantable foreign body (e.g., prosthetic heart valve, nonhuman vascular graft, mechanical heart, or hip prosthesis) that is permanently placed in a patient during surgery.
‡ If the area around a stab wound becomes infected, it is not an SSI. It is considered a skin or soft tissue infection, depending on its depth.

2. Operating Suite

A physically separate area that comprises operating rooms and their interconnecting hallways and ancillary work areas such as scrub sink rooms. No distinction is made between operating suites located in conventional inpatient hospitals and those used for "same-day" surgical care, whether in a hospital or a free-standing facility.

3. Operating Room

A room in an operating suite where operations are performed.

4. Surgical Personnel

Any healthcare worker who provides care to surgical patients during the pre-, intra-, or postoperative periods.

5. Surgical Team Member

Any healthcare worker in an operating room during the operation who has a surgical care role. Members of the surgical team may be "scrubbed" or not; scrubbed members have direct contact with the sterile operating field or sterile instruments or supplies used in the field (refer to "Preoperative Hand/Forearm Antisepsis" section).

C. MICROBIOLOGY

According to data from the NNIS system, the distribution of pathogens isolated from SSIs has not changed markedly during the last decade (Table 3).[26,27] *Staphylococcus aureus,* coagulase-negative staphylococci, *Enterococcus* spp., and *Escherichia coli* remain the most frequently isolated pathogens. An increasing proportion of SSIs are caused by antimicrobial-resistant pathogens, such as methicillin-resistant *S. aureus* (MRSA),[28,29] or by *Candida albicans.*[30] From 1991 to 1995, the incidence of fungal SSIs among patients at NNIS hospitals increased from 0.1 to 0.3 per 1,000 discharges.[30] The increased proportion of SSIs caused by resistant pathogens and *Candida* spp. may reflect increasing numbers of severely ill and immunocompromised surgical patients and the impact of widespread use of broad-spectrum antimicrobial agents.

Outbreaks or clusters of SSIs have also been caused by unusual pathogens, such as *Rhizopus oryzae, Clostridium perfringens, Rhodococcus bronchialis, Nocardia farcinica, Legionella pneumophila* and *Legionella dumoffii,* and *Pseudomonas multivorans.* These rare outbreaks have been traced to contaminated adhesive dressings,[31] elastic bandages,[32] colonized surgical personnel,[33,34] tap water,[35] or contaminated disinfectant solutions.[36] When a cluster of SSIs involves an unusual organism, a formal epidemiologic investigation should be conducted.

D. PATHOGENESIS

Microbial contamination of the surgical site is a necessary precursor of SSI. The risk of SSI can be conceptualized according to the following relationship[37,38]:

$$\frac{\text{Dose of bacterial contamination} \times \text{virulence}}{\text{Resistance of the host patient}} = \text{Risk of surgical site infection}$$

Quantitatively, it has been shown that if a surgical site is contaminated with $>10^5$ microorganisms per gram of tissue, the risk of SSI is markedly increased.[39] However, the dose of contaminating microorganisms required to produce infection may be much lower when foreign material is present at the site (i.e., 100 staphylococci per gram of tissue introduced on silk sutures).[40-42]

Microorganisms may contain or produce toxins and other substances that increase their ability to invade a host, produce damage within the host, or survive on or in host tissue. For example, many gram-negative bacteria produce endotoxin, which stimulates cytokine production. In turn, cytokines can trigger the systemic inflammatory response syndrome that sometimes leads to multiple system organ failure.[43-45] One of the most common causes of multiple system organ failure in modern surgical care is intra-abdominal infection.[46,47] Some bacterial surface components, notably polysaccharide capsules, inhibit phagocytosis,[48] a critical and early host defense response to microbial contamination. Certain strains of clostridia and streptococci produce potent exotoxins that disrupt cell membranes or alter cellular metabolism.[49] A variety of microorganisms, including gram-positive bacteria such as coagulase-negative staphylococci, produce glycocalyx and an associated component called "slime,"[50-55] which physically shields bacteria from phagocytes or inhibits the binding or penetration of antimicrobial agents.[56] Although these and other virulence factors are well defined, their mechanistic relationship to SSI development has not been fully determined.

For most SSIs, the source of pathogens is the endogenous flora of the patient's skin, mucous membranes, or hollow viscera.[57] When mucous membranes or skin is incised, the exposed tissues are at risk for contamination with endogenous flora.[58] These organisms are usually aerobic gram-positive cocci (e.g., staphylococci), but may include fecal flora (e.g., anaerobic bacteria and gram-negative aerobes) when incisions are made near the perineum or groin. When a gastrointestinal organ is opened

TABLE 2
SITE-SPECIFIC CLASSIFICATIONS OF ORGAN/SPACE SURGICAL SITE INFECTION*

Arterial or venous infection
Breast abscess or mastitis
Disc space
Ear, mastoid
Endocarditis
Endometritis
Eye, other than conjunctivitis
Gastrointestinal tract
Intra-abdominal, not specified elsewhere
Intracranial, brain abscess or dura
Joint or bursa
Mediastinitis
Meningitis or ventriculitis
Myocarditis or pericarditis
Oral cavity (mouth, tongue, or gums)
Osteomyelitis
Other infections of the lower respiratory tract (e.g., abscess or empyema)
Other male or female reproductive tract
Sinusitis
Spinal abscess without meningitis
Upper respiratory tract
Vaginal cuff

* Horan TC et al.[22]

TABLE 3
DISTRIBUTION OF PATHOGENS ISOLATED* FROM SURGICAL SITE INFECTIONS, NATIONAL NOSOCOMIAL INFECTIONS SURVEILLANCE SYSTEM, 1986 TO 1996

	Percentage of Isolates	
Pathogen	**1986-1989[179] (N=16,727)**	**1990-1996[26] (N=17,671)**
Staphylococcus aureus	17	20
Coagulase-negative staphylococci	12	14
Enterococcus spp.	13	12
Escherichia coli	10	8
Pseudomonas aeruginosa	8	8
Enterobacter spp.	8	7
Proteus mirabilis	4	3
Klebsiella pneumoniae	3	3
Other *Streptococcus* spp.	3	3
Candida albicans	2	3
Group D streptococci (non-enterococci)	—	2
Other gram-positive aerobes	—	2
Bacteroides fragilis	—	2

*Pathogens representing less than 2% of isolates are excluded.

during an operation and is the source of pathogens, gram-negative bacilli (e.g., *E. coli),* gram-positive organisms (e.g., enterococci), and sometimes anaerobes (e.g., *Bacillus fragilis)* are the typical SSI isolates. Table 4 lists operations and the likely SSI pathogens associated with them. Seeding of the operative site from a distant focus of infection can be another source of SSI pathogens,[59-68] particularly in patients who have a prosthesis or other implant placed during the operation. Such devices provide a nidus for attachment of the organism.[50,69-73]

Exogenous sources of SSI pathogens include surgical personnel (especially members of the surgical team),[74-78] the operating room environment (including air), and all tools, instruments, and materials brought to the sterile field during an operation (refer to "Intraoperative Issues" section). Exogenous flora are primarily aerobes, especially gram-positive organisms (e.g., staphylococci and streptococci). Fungi from endogenous and exogenous sources rarely cause SSIs, and their pathogenesis is not well understood.[79]

E. RISK AND PREVENTION

The term *risk factor* has a particular meaning in epidemiology and, in the context of SSI pathophysiology and prevention, strictly refers to a variable that has a significant, independent association with the development of SSI after a specific operation. Risk factors are identified by multivariate analyses in epidemiologic studies. Unfortunately, the term risk factor often is used in the surgical literature in a broad sense to include patient or operation features which, although associated with SSI development in univariate analysis, are not necessarily independent predictors.[80] The literature cited in the sections that follow includes risk factors identified by both univariate and multivariate analyses.

Table 5 lists patient and operation characteristics that may influence the risk of SSI development. These characteristics are useful in two ways: (1) they allow stratification of operations, making surveillance data more comprehensible; and, (2) knowledge of risk factors before certain operations may allow for targeted prevention measures. For example, if it is known that a patient has a remote site infection, the surgical team may reduce SSI risk by scheduling an operation after the infection has resolved.

An SSI prevention measure can be defined as an action or set of actions intentionally taken to reduce the risk of an SSI. Many such techniques are directed at reducing opportunities for microbial contamination of the patient's tissues or sterile surgical instruments; others are adjunctive, such as using antimicrobial prophylaxis or avoiding unnecessary traumatic tissue dissection. Optimum application of SSI prevention measures requires that a variety of patient and operation characteristics be carefully considered.

1. Patient Characteristics

In certain kinds of operations, patient characteristics possibly associated with an increased risk of an SSI include coincident remote site infections[59-68] or colonization,[81-83] diabetes,[84-87] cigarette smoking,[85,88-92] systemic steroid use,[84,87,93] obesity (>20% ideal body weight),[85-87,94-97] extremes of age,[92,98-102] poor nutritional status,[85,94,98,103-105] and perioperative transfusion of certain blood products.[106-109]

a. Diabetes

The contribution of diabetes to SSI risk is controversial,[84-86,98,110] because the independent contribution of diabetes to SSI risk has not typically been assessed after controlling for potential confounding factors. Recent preliminary findings from a study of patients who underwent coronary artery bypass graft showed a significant relationship between increasing levels of HgA1c and SSI rates.[111] Also, increased glucose levels (>200 mg/dL) in the immediate postoperative period (≤48 hours) were associated with increased SSI risk.[112,113] More studies are needed to assess the efficacy of perioperative blood glucose control as a prevention measure.

b. Nicotine use

Nicotine use delays primary wound healing and may increase the risk of SSI.[85] In a large prospective study, current cigarette smoking was an independent risk factor for sternal and/or mediastinal SSI following cardiac surgery.[85] Other studies have corroborated cigarette smoking as an important SSI risk factor.[88-92] The limitation of these studies, however, is that terms like *current cigarette smoking* and *active smokers* are not always defined. To appropriately determine the contribution of tobacco use to SSI risk, standardized definitions of smoking history must be adopted and used in studies designed to control for confounding variables.

c. Steroid use

Patients who are receiving steroids or other immuno-

TABLE 4
OPERATIONS, LIKELY SURGICAL SITE INFECTION (SSI) PATHOGENS, AND REFERENCES ON USE OF ANTIMICROBIAL PROPHYLAXIS*

Operations	Likely Pathogens†‡	References
Placement of all grafts, prostheses, or implants	*Staphylococcus aureus;* coagulase-negative staphylococci	269,282-284,290
Cardiac	*S. aureus;* coagulase-negative staphylococci	251-253,462,463
Neurosurgery	*S. aureus;* coagulase-negative staphylococci	241,249,258,259,261,464,465
Breast	*S. aureus;* coagulase-negative staphylococci	242,248
Ophthalmic Limited data; however, commonly used in procedures such as anterior segment resection, vitrectomy, and scleral buckles	*S. aureus;* coagulase-negative staphylococci; streptococci; gram-negative bacilli	466
Orthopedic Total joint replacement Closed fractures/use of nails, bone plates, other internal fixation devices Functional repair without implant/device Trauma	*S. aureus;* coagulase-negative staphylococci; gram-negative bacilli	60,243-246,254,255,467-473
Noncardiac thoracic Thoracic (lobectomy, pneumonectomy, wedge resection, other noncardiac mediastinal procedures) Closed tube thoracostomy	*S. aureus;* coagulase-negative staphylococci; *Streptococcus pneumoniae;* gram-negative bacilli	240,247,474,475
Vascular	*S. aureus;* coagulase-negative staphylococci	250,463,476,477
Appendectomy	Gram-negative bacilli; anaerobes	263,452,478
Biliary tract	Gram-negative bacilli; anaerobes	260,262,479-484
Colorectal	Gram-negative bacilli; anaerobes	200,239,256,287-289,485-490
Gastroduodenal	Gram-negative bacilli; streptococci; oropharyngeal anaerobes (e.g., peptostreptococci)	256,257,491-493
Head and neck (major procedures with incision through oropharyngeal mucosa)	*S. aureus;* streptococci; oropharyngeal anaerobes (e.g., peptostreptococci)	494-497
Obstetric and gynecologic	Gram-negative bacilli; enterococci; group B streptococci; anaerobes	270-280,435
Urologic May not be beneficial if urine is sterile	Gram-negative bacilli	267

* Refer to "Antimicrobial prophylaxis in surgery," *The Medical Letter,* 1997,[266] for current recommendations of antimicrobial agents and doses.
† Likely pathogens from both endogenous and exogenous sources.
‡ Staphylococci will be associated with SSI following all types of operations.

suppressive drugs preoperatively may be predisposed to developing SSI,[84,87] but the data supporting this relationship are contradictory. In a study of long-term steroid use in patients with Crohn's disease, SSI developed significantly more often in patients receiving preoperative steroids (12.5%) than in patients without steroid use (6.7%).[93] In contrast, other investigations have not found a relationship between steroid use and SSI risk.[98,114,115]

d. Malnutrition

For some types of operations, severe protein-calorie malnutrition is crudely associated with postoperative nosocomial infections, impaired wound healing dynamics, or death.[116-124] The National Academy of Sciences/National Research Council (NAS/NRC),[94] Study on the Efficacy of Infection Control (SENIC),[125] and NNIS[126] schemes for SSI risk stratification do not explicitly incorporate nutritional status as a predictor variable, although it may be represented indirectly in the latter two. In a widely quoted 1987 study of 404 high-risk general surgery operations, Christou and coworkers derived an SSI probability index in which final predictor variables were patient age, operation duration, serum albumin level, delayed hypersensitivity test score, and intrinsic wound contamination level.[117] Although this index predicted SSI risk satisfactorily for 404 subsequent patients and was generally received as a significant advance in SSI risk stratification, it is not widely used in SSI surveillance data analysis, surgical infection research, or analytic epidemiology.

Theoretical arguments can be made for a belief that severe preoperative malnutrition should increase the risk of both incisional and organ/space SSI. However, an epidemiologic association between incisional SSI and malnutrition is difficult to demonstrate consistently for all surgical subspecialties.[118-120,124,127-131] Multivariate logistic regression modeling has shown that preoperative protein-calorie malnutrition is not an independent predictor of

TABLE 5
PATIENT AND OPERATION CHARACTERISTICS THAT MAY INFLUENCE THE RISK OF SURGICAL SITE INFECTION DEVELOPMENT

Patient
Age
Nutritional status
Diabetes
Smoking
Obesity
Coexistent infections at a remote body site
Colonization with microorganisms
Altered immune response
Length of preoperative stay
Operation
Duration of surgical scrub
Skin antisepsis
Preoperative shaving
Preoperative skin prep
Duration of operation
Antimicrobial prophylaxis
Operating room ventilation
Inadequate sterilization of instruments
Foreign material in the surgical site
Surgical drains
Surgical technique
Poor hemostasis
Failure to obliterate dead space
Tissue trauma

Adapted from references 25, 37.

mediastinitis after cardiac bypass operations.[85,132]

In the modern era, total parenteral nutrition (TPN) and total enteral alimentation (TEA) have enthusiastic acceptance by surgeons and critical care specialists.[118,133-137] However, the benefits of preoperative nutritional repletion of malnourished patients in reducing SSI risk are unproven. In two randomized clinical trials, preoperative "nutritional therapy" did not reduce incisional and organ/space SSI risk.[138-141] In a recent study of high-risk pancreatectomy patients with cancer, the provision of TPN preoperatively had no beneficial effect on SSI risk.[142] A randomized prospective trial involving 395 general and thoracic surgery patients compared outcomes for malnourished patients preoperatively receiving either a 7- to 15-day TPN regimen or a regular preoperative hospital diet. All patients were followed for 90 days postoperatively. There was no detectable benefit of TPN administration on the incidence of incisional or organ/space SSI.[143] Administering TPN or TEA may be indicated in a number of circumstances, but such repletion cannot be viewed narrowly as a prevention measure for organ/space or incisional SSI risk. When a major elective operation is necessary in a severely malnourished patient, experienced surgeons often use both pre- and postoperative nutritional support in consideration of the major morbidity associated with numerous potential complications, only one of which is organ/space SSI.[118,124,130,133,137,138,144-149] In addition, postoperative nutritional support is important for certain major oncologic operations,[135,136] after many operations on major trauma victims,[134] or in patients suffering a variety of catastrophic surgical complications that preclude eating or that trigger a hypermetabolic state. Randomized clinical trials will be necessary to determine if nutritional support alters SSI risk in specific patient-operation combinations.

e. Prolonged preoperative hospital stay

Prolonged preoperative hospital stay is frequently suggested as a patient characteristic associated with increased SSI risk. However, length of preoperative stay is likely a surrogate for severity of illness and co-morbid conditions requiring inpatient work-up and/or therapy before the operation.[16,26,65,85,94,100,150,151]

f. Preoperative nares colonization with *Staphylococcus aureus*

S. aureus is a frequent SSI isolate. This pathogen is carried in the nares of 20% to 30% of healthy humans.[81] It has been known for years that the development of SSI involving *S. aureus* is definitely associated with preoperative nares carriage of the organism in surgical patients.[81] A recent multivariate analysis demonstrated that such carriage was the most powerful independent risk factor for SSI following cardiothoracic operations.[82]

Mupirocin ointment is effective as a topical agent for eradicating *S. aureus* from the nares of colonized patients or healthcare workers. A recent report by Kluytmans and coworkers suggested that SSI risk was reduced in patients who had cardiothoracic operations when mupirocin was applied preoperatively to their nares, regardless of carrier status.[152] In this study, SSI rates for 752 mupirocin-treated patients were compared with those previously observed for an untreated group of 928 historical control patients, and the significant SSI rate reduction was attributed to the mupirocin treatment. Concerns have been raised regarding the comparability of the two patient groups.[153] Additionally, there is concern that mupirocin resistance may emerge, although this seems unlikely when treatment courses are brief.[81] A prospective, randomized clinical trial will be necessary to establish definitively that eradication of nasal carriage of *S. aureus* is an effective SSI prevention method in cardiac surgery. Such a trial has recently been completed on 3,909 patients in Iowa.[83] Five types of operations in two facilities were observed. Preliminary analysis showed a significant association between nasal carriage of *S. aureus* and subsequent SSI development. The effect of mupirocin on reducing SSI risk is yet to be determined.

g. Perioperative transfusion

It has been reported that perioperative transfusion of leukocyte-containing allogeneic blood components is an apparent risk factor for the development of postoperative bacterial infections, including SSI.[106] In three of five randomized trials conducted in patients undergoing elective colon resection for cancer, the risk of SSI was at least doubled in patients receiving blood transfusions.[107-109] However, on the basis of detailed epidemiologic reconsid-

TABLE 6
MECHANISM AND SPECTRUM OF ACTIVITY OF ANTISEPTIC AGENTS COMMONLY USED FOR PREOPERATIVE SKIN PREPARATION AND SURGICAL SCRUBS

Agent	Mechanism of Action	Gram-Positive Bacteria	Gram-Negative Bacteria	Mtb	Fungi	Virus	Rapidity of Action	Residual Activity	Toxicity	Uses
Alcohol	Denature proteins	E	E	G	G	G	Most rapid	None	Drying, volatile	SP, SS
Chlorhexidine	Disrupt cell membrane	E	G	P	F	G	Intermediate	E	Ototoxicity, keratitis	SP, SS
Iodine/Iodophors	Oxidation/substitution by free iodine	E	G	G	G	G	Intermediate	Minimal	Absorption from skin with possible toxicity, skin irritation	SP, SS
PCMX	Disrupt cell wall	G	F*	F	F	F	Intermediate	G	More data needed	SS
Triclosan	Disrupt cell wall	G	G	G	P	U	Intermediate	E	More data needed	SS

Abbreviations: E, excellent; F, fair; G, good; Mtb, *Mycobacterium tuberculosis;* P, poor; PCMX, para-chloro-meta-xylenol; SP, skin preparation; SS, surgical scrubs; U, unknown.
Data from Larson E.[176]
* Fair, except for *Pseudomonas* spp.; activity improved by addition of chelating agent such as EDTA.

erations, as many as 12 confounding variables may have influenced the reported association, and any effect of transfusion on SSI risk may be either small or nonexistent.[106] Because of methodologic problems, including the timing of transfusion, and use of nonstandardized SSI definitions, interpretation of the available data is limited. A meta-analysis of published trials will probably be required for resolution of the controversy.[154] There is currently no scientific basis for withholding necessary blood products from surgical patients as a means of either incisional or organ/space SSI risk reduction.

2. Operative Characteristics: Preoperative Issues

a. Preoperative antiseptic showering

A preoperative antiseptic shower or bath decreases skin microbial colony counts. In a study of >700 patients who received two preoperative antiseptic showers, chlorhexidine reduced bacterial colony counts ninefold (2.8×10^2 to 0.3), while povidone-iodine or triclocarban-medicated soap reduced colony counts by 1.3- and 1.9-fold, respectively.[155] Other studies corroborate these findings.[156,157] Chlorhexidine gluconate-containing products require several applications to attain maximum antimicrobial benefit, so repeated antiseptic showers are usually indicated.[158] Even though preoperative showers reduce the skin's microbial colony counts, they have not definitively been shown to reduce SSI rates.[159-165]

b. Preoperative hair removal

Preoperative shaving of the surgical site the night before an operation is associated with a significantly higher SSI risk than either the use of depilatory agents or no hair removal.[16,100,166-169] In one study, SSI rates were 5.6% in patients who had hair removed by razor shave compared to a 0.6% rate among those who had hair removed by depilatory or who had no hair removed.[166] The increased SSI risk associated with shaving has been attributed to microscopic cuts in the skin that later serve as foci for bacterial multiplication. Shaving immediately before the operation compared to shaving within 24 hours preoperatively was associated with decreased SSI rates (3.1% vs 7.1%); if shaving was performed >24 hours prior to operation, the SSI rate exceeded 20%.[166] Clipping hair immediately before an operation also has been associated with a lower risk of SSI than shaving or clipping the night before an operation (SSI rates immediately before = 1.8% vs night before = 4.0%).[170-173] Although the use of depilatories has been associated with a lower SSI risk than shaving or clipping,[166,167] depilatories sometimes produce hypersensitivity reactions.[166] Other studies showed that preoperative hair removal by any means was associated with increased SSI rates and suggested that no hair be removed.[100,174,175]

c. Patient skin preparation in the operating room

Several antiseptic agents are available for preoperative preparation of skin at the incision site (Table 6). The iodophors (e.g., povidone-iodine), alcohol-containing products, and chlorhexidine gluconate are the most commonly used agents. No studies have adequately assessed the comparative effects of these preoperative skin antiseptics on SSI risk in well-controlled, operation-specific studies.

Alcohol is defined by the FDA as having one of the following active ingredients: ethyl alcohol, 60% to 95% by volume in an aqueous solution, or isopropyl alcohol, 50% to 91.3% by volume in an aqueous solution.[12] Alcohol is readily available, inexpensive, and remains the most effective and rapid-acting skin antiseptic.[176] Aqueous 70% to 92% alcohol solutions have germicidal activity against bacteria, fungi, and viruses, but spores can be resistant.[176,177] One potential disadvantage of the use of alcohol in the operating room is its flammability.[176-178]

Both chlorhexidine gluconate and iodophors have broad spectra of antimicrobial activity.[177,179-181] In some comparisons of the two antiseptics when used as preoperative hand scrubs, chlorhexidine gluconate achieved greater reductions in skin microflora than did povidone-iodine and

also had greater residual activity after a single application.[182-184] Further, chlorhexidine gluconate is not inactivated by blood or serum proteins.[176,179,185,186] Iodophors may be inactivated by blood or serum proteins, but exert a bacteriostatic effect as long as they are present on the skin.[178,179]

Before the skin preparation of a patient is initiated, the skin should be free of gross contamination (i.e., dirt, soil, or any other debris).[187] The patient's skin is prepared by applying an antiseptic in concentric circles, beginning in the area of the proposed incision. The prepared area should be large enough to extend the incision or create new incisions or drain sites, if necessary.[1,177,187] The application of the skin preparation may need to be modified, depending on the condition of the skin (e.g., burns) or location of the incision site (e.g., face).

There are reports of modifications to the procedure for preoperative skin preparation which include: (1) removing or wiping off the skin preparation antiseptic agent after application, (2) using an antiseptic-impregnated adhesive drape, (3) merely painting the skin with an antiseptic in lieu of the skin preparation procedure described above, or (4) using a "clean" versus a "sterile" surgical skin preparation kit.[188-191] However, none of these modifications has been shown to represent an advantage.

d. Preoperative hand/forearm antisepsis

Members of the surgical team who have direct contact with the sterile operating field or sterile instruments or supplies used in the field wash their hands and forearms by performing a traditional procedure known as scrubbing (or the surgical scrub) immediately before donning sterile gowns and gloves. Ideally, the optimum antiseptic used for the scrub should have a broad spectrum of activity, be fast-acting, and have a persistent effect.[1,192,193] Antiseptic agents commercially available in the United States for this purpose contain alcohol, chlorhexidine, iodine/iodophors, parachloro-meta-xylenol, or triclosan (Table 6).[176,177,179,194,195] Alcohol is considered the gold standard for surgical hand preparation in several European countries.[196-199] Alcohol-containing products are used less frequently in the United States than in Europe, possibly because of concerns about flammability and skin irritation. Povidone-iodine and chlorhexidine gluconate are the current agents of choice for most U.S. surgical team members.[177] However, when 7.5% povidone-iodine or 4% chlorhexidine gluconate was compared to alcoholic chlorhexidine (60% isopropanol and 0.5% chlorhexidine gluconate in 70% isopropanol), alcoholic chlorhexidine was found to have greater residual antimicrobial activity.[200,201] No agent is ideal for every situation, and a major factor, aside from the efficacy of any product, is its acceptability by operating room personnel after repeated use. Unfortunately, most studies evaluating surgical scrub antiseptics have focused on measuring hand bacterial colony counts. No clinical trials have evaluated the impact of scrub agent choice on SSI risk.[195,202-206]

Factors other than the choice of antiseptic agent influence the effectiveness of the surgical scrub. Scrubbing technique, the duration of the scrub, the condition of the hands, or the techniques used for drying and gloving are examples of such factors. Recent studies suggest that scrubbing for at least 2 minutes is as effective as the traditional 10-minute scrub in reducing hand bacterial colony counts,[207-211] but the optimum duration of scrubbing is not known. The first scrub of the day should include a thorough cleaning underneath fingernails (usually with a brush).[180,194,212] It is not clear that such cleaning is a necessary part of subsequent scrubs during the day. After performing the surgical scrub, hands should be kept up and away from the body (elbows in flexed position) so that water runs from the tips of the fingers toward the elbows. Sterile towels should be used for drying the hands and forearms before the donning of a sterile gown and gloves.[212]

A surgical team member who wears artificial nails may have increased bacterial and fungal colonization of the hands despite performing an adequate hand scrub.[212,213] Hand carriage of gram-negative organisms has been shown to be greater among wearers of artificial nails than among non-wearers.[213] An outbreak of *Serratia marcescens* SSIs in cardiovascular surgery patients was found to be associated with a surgical nurse who wore artificial nails.[214] While the relationship between nail length and SSI risk is unknown, long nails—artificial or natural—may be associated with tears in surgical gloves.[177,180,212] The relationship between the wearing of nail polish or jewelry by surgical team members and SSI risk has not been adequately studied.[194,212,215-217]

e. Management of infected or colonized surgical personnel

Surgical personnel who have active infections or are colonized with certain microorganisms have been linked to outbreaks or clusters of SSIs.[33,34,76,218-237] Thus, it is important that healthcare organizations implement policies to prevent transmission of microorganisms from personnel to patients. These policies should address management of job-related illnesses, provision of postexposure prophylaxis after job-related exposures and, when necessary, exclusion of ill personnel from work or patient contact. While work exclusion policies should be enforceable and include a statement of authority to exclude ill personnel, they should also be designed to encourage personnel to report their illnesses and exposures and not penalize personnel with loss of wages, benefits, or job status.[238]

f. Antimicrobial prophylaxis

Surgical antimicrobial prophylaxis (AMP) refers to a very brief course of an antimicrobial agent initiated just before an operation begins.[239-265] AMP is not an attempt to sterilize tissues, but a critically timed adjunct used to reduce the microbial burden of intraoperative contamination to a level that cannot overwhelm host defenses. AMP does not pertain to prevention of SSI caused by postoperative contamination.[265] Intravenous infusion is the mode of AMP delivery used most often in modern surgical practice.[20,26,242,266-281] Essentially all confirmed AMP indications pertain to elective operations in which skin incisions are closed in the operating room.

TABLE 7
SURGICAL WOUND CLASSIFICATION

Class I/Clean: 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.

Class II/Clean-Contaminated: 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.

Class III/Contaminated: Open, fresh, accidental wounds. In addition, operations with major breaks in sterile technique (e.g., open cardiac massage) or gross spillage from the gastrointestinal tract, and incisions in which acute, nonpurulent inflammation is encountered are included in this category.

Class IV/Dirty-Infected: 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.

Garner JS[1] and Simmons BP.[2]

Four principles must be followed to maximize the benefits of AMP:

- Use an AMP agent for all operations or classes of operations in which its use has been shown to reduce SSI rates based on evidence from clinical trials or for those operations after which incisional or organ/space SSI would represent a catastrophe.[266,268,269,282-284]
- Use an AMP agent that is safe, inexpensive, and bactericidal with an in vitro spectrum that covers the most probable intraoperative contaminants for the operation.
- Time the infusion of the initial dose of antimicrobial agent so that a bactericidal concentration of the drug is established in serum and tissues by the time the skin is incised.[285]
- Maintain therapeutic levels of the antimicrobial agent in both serum and tissues throughout the operation and until, at most, a few hours after the incision is closed in the operating room.[179,266-268,282,284,286] Because clotted blood is present in all surgical wounds, therapeutic serum levels of AMP agents are logically important in addition to therapeutic tissue levels. Fibrin-enmeshed bacteria may be resistant to phagocytosis or to contact with antimicrobial agents that diffuse from the wound space.

Table 4 summarizes typical SSI pathogens according to operation type and cites studies that establish AMP efficacy for these operations. A simple way to organize AMP indications is based on using the surgical wound classification scheme shown in Table 7, which employs descriptive case features to *postoperatively* grade the degree of intraoperative microbial contamination. A surgeon makes the decision to use AMP by anticipating *preoperatively* the surgical wound class for a given operation.

AMP is indicated for all operations that entail entry into a hollow viscus under controlled conditions. The most frequent SSI pathogens for such clean-contaminated operations are listed in Table 4. Certain clean-contaminated operations, such as elective colon resection, low anterior resection of the rectum, and abdominoperineal resection of the rectum, also require an additional preoperative protective maneuver called "preparation of the colon," to empty the bowel of its contents and to reduce the levels of live microorganisms.[200,239,256,268,284,287] This maneuver includes the administration of enemas and cathartic agents followed by the oral administration of nonabsorbable antimicrobial agents in divided doses the day before the operation.[200,288,289]

AMP is sometimes indicated for operations that entail incisions through normal tissue and in which no viscus is entered and no inflammation or infection is encountered. Two well-recognized AMP indications for such clean operations are: (1) when any intravascular prosthetic material or a prosthetic joint will be inserted, and (2) for any operation in which an incisional or organ/space SSI would pose catastrophic risk. Examples are all cardiac operations, including cardiac pacemaker placement,[290] vascular operations involving prosthetic arterial graft placement at any site or the revascularization of the lower extremity, and most neurosurgical operations (Table 4). Some have advocated use of AMP during all operations on the breast.[80,242,264]

By definition, AMP is not indicated for an operation classified in Table 7 as contaminated or dirty. In such operations, patients are frequently receiving therapeutic antimicrobial agents perioperatively for established infections.

Cephalosporins are the most thoroughly studied AMP agents.[284] These drugs are effective against many gram-positive and gram-negative microorganisms. They also share the features of demonstrated safety, acceptable pharmacokinetics, and a reasonable cost per dose.[242] In particular, cefazolin is widely used and generally viewed as the AMP agent of first choice for clean operations.[266] If a patient is unable to receive a cephalosporin because of penicillin allergy, an alternative for gram-positive bacterial coverage is either clindamycin or vancomycin.

Cefazolin provides adequate coverage for many clean-contaminated operations,[268,291] but AMP for operations on the distal intestinal tract mandates use of an agent such as cefoxitin (or some other second-generation cephalosporin) that provides anaerobic coverage. If a patient cannot safely receive a cephalosporin because of allergy, a reasonable alternative for gram-negative cover-

TABLE 8
PARAMETERS FOR OPERATING ROOM VENTILATION, AMERICAN INSTITUTE OF ARCHITECTS, 1996

Temperature	68-73°F, depending on normal ambient temperatures
Relative humidity	30%-60%
Air movement	From "clean to less clean" areas
Air changes	Minimum 15 total air changes per hour Minimum 3 air changes of outdoor air per hour

American Institute of Architects.[299]

age is aztreonam. However, an agent such as clindamycin or metronidazole should also be included to ensure anaerobic coverage.

The aminoglycosides are seldom recommended as first choices for AMP, either as single drugs or as components of combination regimens.[242,264] References cited in Table 4 provide many details regarding AMP choices and dosages, antimicrobial spectra and properties, and other practical clinical information.

The routine use of vancomycin in AMP is not recommended for any kind of operation.[242,266,283,292] However, vancomycin may be the AMP agent of choice in certain clinical circumstances, such as when a cluster of MRSA mediastinitis or incisional SSI due to methicillin-resistant coagulase-negative staphylococci has been detected. A threshold has not been scientifically defined that can support the decision to use vancomycin in AMP. The decision should involve consideration of local frequencies of MRSA isolates, SSI rates for particular operations, review of infection prevention practices for compliance, and consultation between surgeons and infectious disease experts. An effective SSI surveillance program must be operational, with careful and timely culturing of SSI isolates to determine species and AMP agent susceptibilities.[80]

Agents most commonly used for AMP (i.e., cephalosporins) exhibit time-dependent bactericidal action. The therapeutic effects of such agents are probably maximized when their levels continuously exceed a threshold value best approximated by the minimal bactericidal concentration value observed for the target pathogens in vitro. When the duration of an operation is expected to exceed the time in which therapeutic levels of the AMP agent can be maintained, additional AMP agent should be infused. That time point for cefazolin is estimated as 3 to 4 hours. In general, the timing of a second (or third, etc.) dose of any AMP drug is estimated from three parameters: tissue levels achieved in normal patients by a standard therapeutic dose, the approximate serum half-life of the drug, and awareness of approximate MIC_{90} values for anticipated SSI pathogens. References in Table 6 should be consulted for these details and important properties of antimicrobial agents used for AMP in various specialties.

Basic "rules of thumb" guide decisions about AMP dose sizes and timing. For example, it is believed that a full therapeutic dose of cefazolin (1-2 g) should be given to adult patients no more than 30 minutes before the skin is incised.[242,285] There are a few exceptions to this basic guide. With respect to dosing, it has been demonstrated that larger doses of AMP agents are necessary to achieve optimum effect in morbidly obese patients.[293] With respect to timing, an exception occurs for patients undergoing cesarean section in whom AMP is indicated: the initial dose is administered immediately after the umbilical cord is clamped.[266,272,273] If vancomycin is used, an infusion period of approximately 1 hour is required for a typical dose. Clearly, the concept of "on-call" infusion of AMP is flawed simply because delays in transport or schedule changes can mean that suboptimal tissue and serum levels may be present when the operation starts.[242,294] Simple protocols of AMP timing and oversight responsibility should be locally designed to be practical and effective.

3. Operative characteristics: Intraoperative issues

a. Operating room environment

(1) Ventilation

Operating room air may contain microbial-laden dust, lint, skin squames, or respiratory droplets. The microbial level in operating room air is directly proportional to the number of people moving about in the room.[295] Therefore, efforts should be made to minimize personnel traffic during operations. Outbreaks of SSIs caused by group A beta-hemolytic streptococci have been traced to airborne transmission of the organism from colonized operating room personnel to patients.[233,237,296,297] In these outbreaks, the strain causing the outbreak was recovered from the air in the operating room.[237,296] It has been demonstrated that exercising and changing of clothing can lead to airborne dissemination of group A streptococci from vaginal or rectal carriage.[233,234,237,297]

Operating rooms should be maintained at positive pressure with respect to corridors and adjacent areas.[298] Positive pressure prevents airflow from less clean areas into more clean areas. All ventilation or air conditioning systems in hospitals, including those in operating rooms, should have two filter beds in series, with the efficiency of the first filter bed being $\geq$30% and that of the second filter bed being $\geq$90%.[299] Conventional operating room ventilation systems produce a minimum of about 15 air changes of filtered air per hour, three (20%) of which must be fresh air.[299,300] Air should be introduced at the ceiling and exhausted near the floor.[300,301] Detailed ventilation parameters for operating rooms have been published by the American Institute of Architects in collaboration with the U.S. Department of Health and Human Services (Table 8).[299]

Laminar airflow and use of UV radiation have been suggested as additional measures to reduce SSI risk for certain operations. Laminar airflow is designed to move particle-free air (called "ultraclean air") over the aseptic operating field at a uniform velocity (0.3 to 0.5 µm/sec), sweeping away particles in its path. Laminar airflow can be directed vertically or horizontally, and recirculated air is usually passed through a high efficiency particulate air (HEPA)

filter.[302,303] HEPA filters remove particles ≥0.3μm in diameter with an efficiency of 99.97%.[64,300,302,304] Most of the studies examining the efficacy of ultraclean air involve only orthopedic operations.[298,305-311] Charnley and Eftaknan studied vertical laminar airflow systems and exhaust-ventilated clothing and found that their use decreased the SSI rate from 9% to 1%.[305] However, other variables (i.e., surgeon experience and surgical technique) changed at the same time as the type of ventilation, which may have confounded the associations. In a multicenter study examining 8,000 total hip and knee replacements, Lidwell et al. compared the effects of ultraclean air alone, antimicrobial prophylaxis alone, and ultraclean air in combination with antimicrobial prophylaxis on the rate of deep SSIs.[307] The SSI rate following operations in which ultraclean air alone was used decreased from 3.4% to 1.6%, whereas the rate for those who received only antimicrobial prophylaxis decreased from 3.4% to 0.8%. When both interventions were used in combination, the SSI rate decreased from 3.4% to 0.7%. These findings suggest that both ultraclean air and antimicrobial prophylaxis can reduce the incidence of SSI following orthopedic implant operations, but antimicrobial prophylaxis is more beneficial than ultraclean air. Intraoperative UV radiation has not been shown to decrease overall SSI risk.[94,312]

(2) Environmental surfaces

Environmental surfaces in U.S. operating rooms (e.g., tables, floors, walls, ceilings, lights) are rarely implicated as the sources of pathogens important in the development of SSIs. Nevertheless, it is important to perform routine cleaning of these surfaces to reestablish a clean environment after each operation.[180,212,300,302] There are no data to support routine disinfecting of environmental surfaces or equipment between operations in the absence of contamination or visible soiling. When visible soiling of surfaces or equipment occurs during an operation, an Environmental Protection Agency (EPA)-approved hospital disinfectant should be used to decontaminate the affected areas before the next operation.[180,212,300-302,313-315] This is in keeping with the Occupational Safety and Health Administration (OSHA) requirement that all equipment and environmental surfaces be cleaned and decontaminated after contact with blood or other potentially infectious materials.[315] Wet-vacuuming of the floor with an EPA-approved hospital disinfectant is performed routinely after the last operation of the day or night. Care should be taken to ensure that medical equipment left in the operating room be covered so that solutions used during cleaning and disinfecting do not contact sterile devices or equipment.[316] There are no data to support special cleaning procedures or closing of an operating room after a contaminated or dirty operation has been performed.[300,301]

Tacky mats placed outside the entrance to an operating room/suite have not been shown to reduce the number of organisms on shoes or stretcher wheels, nor do they reduce the risk of SSI.[1,179,295,301]

(3) Microbiologic sampling

Because there are no standardized parameters by which to compare microbial levels obtained from cultures of ambient air or environmental surfaces in the operating room, routine microbiologic sampling cannot be justified. Such environmental sampling should only be performed as part of an epidemiologic investigation.

(4) Conventional sterilization of surgical instruments

Inadequate sterilization of surgical instruments has resulted in SSI outbreaks.[302,317,318] Surgical instruments can be sterilized by steam under pressure, dry heat, ethylene oxide, or other approved methods. The importance of routinely monitoring the quality of sterilization procedures has been established.[1,180,212,299] Microbial monitoring of steam autoclave performance is necessary and can be accomplished by use of a biological indicator.[212,314,319] Detailed recommendations for sterilization of surgical instruments have been published.[212,314,320,321]

(5) Flash sterilization of surgical instruments

The Association for the Advancement of Medical Instrumentation defines flash sterilization as "the process designated for the steam sterilization of patient care items for immediate use."[321] During any operation, the need for emergency sterilization of equipment may arise (e.g., to reprocess an inadvertently dropped instrument). However, flash sterilization is not intended to be used for either reasons of convenience or as an alternative to purchasing additional instrument sets or to save time. Also, flash sterilization is not recommended for implantable devices(*) because of the potential for serious infections.[314,320,321]

Flash sterilization is not recommended as a routine sterilization method because of the lack of timely biologic indicators to monitor performance, absence of protective packaging following sterilization, possibility for contamination of processed items during transportation to operating rooms, and use of minimal sterilization cycle parameters (i.e., time, temperature, pressure).[319] To address some of these concerns, many hospitals have placed equipment for flash sterilization in close proximity to operating rooms and new biologic indicators that provide results in 1 to 3 hours are now available for flash-sterilized items.[322-325] Nevertheless, flash sterilization should be restricted to its intended purpose until studies are performed that can demonstrate comparability with conventional sterilization methods regarding risk of SSI. Sterilization cycle parameters for flash sterilization are shown in Table 9.

b. Surgical attire and drapes

In this section the term *surgical attire* refers to scrub suits, caps/hoods, shoe covers, masks, gloves, and gowns. Although experimental data show that live microorganisms are shed from hair, exposed skin, and mucous membranes of operating room personnel,[75,181,326-330] few controlled clinical studies have evaluated the relationship between the use of surgical attire and SSI risk. Nevertheless, the use of barriers seems prudent to minimize a patient's exposure to the skin, mucous membranes, or hair of surgical team mem-

** According to the FDA, an implantable device is a "device that is placed into a surgically or naturally formed cavity of the human body if it is intended to remain there for a period of 30 days or more."[321]*

TABLE 9
PARAMETERS FOR FLASH STERILIZATION CYCLES, ASSOCIATION FOR THE ADVANCEMENT OF MEDICAL INSTRUMENTATION

Gravity-Displacement	**Minimum Exposure Time and Temperature**
Nonporous items	3 min at 132°C (270°F)
Nonporous and porous items	10 min at 132°C (270°F)
Prevacuum	**Minimum Exposure Time and Temperature**
Nonporous items	3 min at 132°C (270°F)
Nonporous and porous items	4 min at 132°C (270°F)

Association for the Advancement of Medical Instrumentation.[321]

bers, as well as to protect surgical team members from exposure to blood and bloodborne pathogens (e.g., human immunodeficiency virus and hepatitis viruses).

(1) Scrub suits

Surgical team members often wear a uniform called a "scrub suit" that consists of pants and a shirt. Policies for laundering, wearing, covering, and changing scrub suits vary greatly. Some policies restrict the laundering of scrub suits to the facility, while other facilities have policies that allow laundering by employees. There are no well-controlled studies evaluating scrub suit laundering as an SSI risk factor.[331] Some facilities have policies that restrict the wearing of scrub suits to the operating suite, while other facilities allow the wearing of cover gowns over scrub suits when personnel leave the suite. The Association of Operating Room Nurses recommends that scrub suits be changed after they become visibly soiled and that they be laundered only in an approved and monitored laundry facility.[212] Additionally, OSHA regulations require that "if a garment(s) is penetrated by blood or other potentially infectious materials, the garment(s) shall be removed immediately or as soon as feasible."[315]

(2) Masks

The wearing of surgical masks during operations to prevent potential microbial contamination of incisions is a longstanding surgical tradition. However, some studies have raised questions about the efficacy and cost-benefit of surgical masks in reducing SSI risk.[328,332-338] Nevertheless, wearing a mask can be beneficial since it protects the wearer's nose and mouth from inadvertent exposures (i.e., splashes) to blood and other body fluids. OSHA regulations require that masks in combination with protective eyewear, such as goggles or glasses with solid shields, or chin-length face shields be worn whenever splashes, spray, spatter, or droplets of blood or other potentially infectious material may be generated and eye, nose, or mouth contamination can be reasonably anticipated.[315] In addition, a respirator certified by the National Institute for Occupational Safety and Health with protection factor N95 or higher is required when the patient has or is suspected of having infectious tuberculosis.[339]

(3) Surgical caps/hoods and shoe covers

Surgical caps/hoods are inexpensive and reduce contamination of the surgical field by organisms shed from the hair and scalp. SSI outbreaks have occasionally been traced to organisms isolated from the hair or scalp (*S. aureus* and group A *Streptococcus)*,[75,76] even when caps were worn by personnel during the operation and in the operating suites.

The use of shoe covers has never been shown to decrease SSI risk or to decrease bacteria counts on the operating room floor.[340,341] Shoe covers may, however, protect surgical team members from exposure to blood and other body fluids during an operation. OSHA regulations require that surgical caps or hoods and shoe covers or boots be worn in situations when gross contamination can reasonably be anticipated (e.g., orthopedic operations, penetrating trauma cases).[315]

(4) Sterile gloves

Sterile gloves are put on after donning sterile gowns. A strong theoretical rationale supports the wearing of sterile gloves by all scrubbed members of the surgical team. Sterile gloves are worn to minimize transmission of microorganisms from the hands of team members to patients and to prevent contamination of team members' hands with patients' blood and body fluids. If the integrity of a glove is compromised (e.g., punctured), it should be changed as promptly as safety permits.[315,342,343] Wearing two pairs of gloves (double-gloving) has been shown to reduce hand contact with patients' blood and body fluids when compared to wearing only a single pair.[344,345]

(5) Gowns and drapes

Sterile surgical gowns and drapes are used to create a barrier between the surgical field and potential sources of bacteria. Gowns are worn by all scrubbed surgical team members and drapes are placed over the patient. There are limited data that can be used to understand the relationship of gown or drape characteristics with SSI risk. The wide variation in the products and study designs make interpretation of the literature difficult.[329,346-350]

Gowns and drapes are classified as disposable (single use) or reusable (multiple use). Regardless of the material used to manufacture gowns and drapes, these items should be impermeable to liquids and viruses.[351,352] In general, only gowns reinforced with films, coatings, or membranes appear to meet standards developed by the American Society for Testing and Materials.[351-353] However, such "liquid-proof" gowns may be uncomfortable because

they also inhibit heat loss and the evaporation of sweat from the wearer's body. These factors should be considered when selecting gowns.[353,354] A discussion of the role of gowns and drapes in preventing the transmission of bloodborne pathogens is beyond the scope of this document.[355]

c. Asepsis and surgical technique

(1) Asepsis

Rigorous adherence to the principles of asepsis by all scrubbed personnel is the foundation of surgical site infection prevention. Others who work in close proximity to the sterile surgical field, such as anesthesia personnel who are separated from the field only by a drape barrier, also must abide by these principles. SSIs have occurred in which anesthesia personnel were implicated as the source of the pathogen.[34,231,234,356-358] Anesthesiologists and nurse anesthetists perform a variety of invasive procedures such as placement of intravascular devices and endotracheal tubes, and administration of intravenous drugs and solutions. Lack of adherence to the principles of asepsis during such procedures,[359] including use of common syringes[360,361] and contaminated infusion pumps,[359,362-364] and the assembly of equipment and solutions in advance of procedures,[316,360] have been associated with outbreaks of postoperative infections, including SSI. Recommendations for infection control practices in anesthesiology have been published.[212,365-367]

(2) Surgical technique

Excellent surgical technique is widely believed to reduce the risk of SSI.[26,49,179,180,368,369] Such techniques include maintaining effective hemostasis while preserving adequate blood supply, preventing hypothermia, gently handling tissues, avoiding inadvertent entries into a hollow viscus, removing devitalized (e.g., necrotic or charred) tissues, using drains and suture material appropriately, eradicating dead space, and appropriately managing the postoperative incision.

Any foreign body, including suture material, a prosthesis, or drain, may promote inflammation at the surgical site[94] and may increase the probability of SSI after otherwise benign levels of tissue contamination. Extensive research compares different types of suture material and their presumed relationships to SSI risk.[370-379] In general, monofilament sutures appear to have the lowest infection-promoting effects.[3,94,179,180]

A discussion of appropriate surgical drain use and details of drain placement exceed the scope of this document, but general points should be briefly noted. Drains placed through an operative incision increase incisional SSI risk.[380] Many authorities suggest placing drains through a separate incision distant from the operative incision.[283,381] It appears that SSI risk also decreases when closed suction drains are used rather than open drains.[174] Closed suction drains can effectively evacuate postoperative hematomas or seromas, but timing of drain removal is important. Bacterial colonization of initially sterile drain tracts increases with the duration of time the drain is left in place.[382]

Hypothermia in surgical patients, defined as a core body temperature below 36°C, may result from general anesthesia, exposure to cold, or intentional cooling such as is done to protect the myocardium and central nervous system during cardiac operations.[302,383,384] In one study of patients undergoing colorectal operations, hypothermia was associated with an increased SSI risk.[385] Mild hypothermia appears to increase incisional SSI risk by causing vasoconstriction, decreased delivery of oxygen to the wound space, and subsequent impairment of function of phagocytic leukocytes (i.e., neutrophils).[386-390] In animal models, supplemental oxygen administration has been shown to reverse the dysfunction of phagocytes in fresh incisions.[391] In recent human experiments, controlled local heating of incisions with an electrically powered bandage has been shown to improve tissue oxygenation.[392] Randomized clinical trials are needed to establish that measures which improve wound space oxygenation can reduce SSI risk.

4. Operative Characteristics: Postoperative Issues

a. Incision care

The type of postoperative incision care is determined by whether the incision is closed primarily (i.e., the skin edges are re-approximated at the end of the operation), left open to be closed later, or left open to heal by second intention. When a surgical incision is closed primarily, as most are, the incision is usually covered with a sterile dressing for 24 to 48 hours.[393,394] Beyond 48 hours, it is unclear whether an incision must be covered by a dressing or whether showering or bathing is detrimental to healing. When a surgical incision is left open at the skin level for a few days before it is closed (delayed primary closure), a surgeon has determined that it is likely to be contaminated or that the patient's condition prevents primary closure (e.g., edema at the site). When such is the case, the incision is packed with a sterile dressing. When a surgical incision is left open to heal by second intention, it is also packed with sterile moist gauze and covered with a sterile dressing. The American College of Surgeons, CDC, and others have recommended using sterile gloves and equipment (sterile technique) when changing dressings on any type of surgical incision.[180,395-397]

b. Discharge planning

In current practice, many patients are discharged very soon after their operation, before surgical incisions have fully healed.[398] The lack of optimum protocols for home incision care dictates that much of what is done at home by the patient, family, or home care agency practitioners must be individualized. The intent of discharge planning is to maintain integrity of the healing incision, educate the patient about the signs and symptoms of infection, and advise the patient about whom to contact to report any problems.

F. SSI SURVEILLANCE

Surveillance of SSI with feedback of appropriate data to surgeons has been shown to be an important component of strategies to reduce SSI risk.[16,399,400] A successful surveillance program includes the use of epidemiologically sound infection definitions (Tables 1 and 2) and effective

TABLE 10
PHYSICAL STATUS CLASSIFICATION, AMERICAN SOCIETY OF ANESTHESIOLOGISTS*

Code	Patient's Preoperative Physical Status
1	Normally healthy patient
2	Patient with mild systemic disease
3	Patient with severe systemic disease that is not incapacitating
4	Patient with an incapacitating systemic disease that is a constant threat to life
5	Moribund patient who is not expected to survive for 24 hours with or without operation

* Reference 406.
Note: The above is the version of the ASA Physical Status Classification System that was current at the time of development of, and still is used in, the NNIS Risk Index. Meanwhile, the American Society of Anesthesiologists has revised their classification system; the most recent version is available at http://www.asahq.org/profinfo/physicalstatus.html.

surveillance methods, stratification of SSI rates according to risk factors associated with SSI development, and data feedback.[25]

1. SSI Risk Stratification

a. Concepts

Three categories of variables have proven to be reliable predictors of SSI risk: (1) those that estimate the intrinsic degree of microbial contamination of the surgical site, (2) those that measure the duration of an operation, and (3) those that serve as markers for host susceptibility.[25] A widely accepted scheme for classifying the degree of intrinsic microbial contamination of a surgical site was developed by the 1964 NAS/NRC Cooperative Research Study and modified in 1982 by CDC for use in SSI surveillance (Table 7).[2,94] In this scheme, a member of the surgical team classifies the patient's wound at the completion of the operation. Because of its ease of use and wide availability, the surgical wound classification has been used to predict SSI risk.[16,94,126,401-405] Some researchers have suggested that surgeons compare clean wound SSI rates with those of other surgeons.[16,399] However, two CDC efforts—the SENIC Project and the NNIS system—incorporated other predictor variables into SSI risk indices. These showed that even within the category of clean wounds, the SSI risk varied by risk category from 1.1% to 15.8% (SENIC) and from 1.0% to 5.4% (NNIS).[125,126] In addition, sometimes an incision is incorrectly classified by a surgical team member or not classified at all, calling into question the reliability of the classification. Therefore, reporting SSI rates stratified by wound class alone is not recommended.

Data on 10 variables collected in the SENIC Project were analyzed by using logistic regression modeling to develop a simple additive SSI risk index.[125] Four of these were found to be independently associated with SSI risk: (1) an abdominal operation, (2) an operation lasting >2 hours, (3) a surgical site with a wound classification of either contaminated or dirty/infected, and (4) an operation performed on a patient having ⩾3 discharge diagnoses. Each of these equally weighted factors contributes a point when present, such that the risk index values range from 0 to 4. By using these factors, the SENIC index predicted SSI risk twice as well as the traditional wound classification scheme alone.

The NNIS risk index is operation-specific and applied to prospectively collected surveillance data. The index values range from 0 to 3 points and are defined by three independent and equally weighted variables. One point is scored for each of the following when present: (1) American Society of Anesthesiologists (ASA) Physical Status Classification of >2 (Table 10), (2) either contaminated or dirty/infected wound classification (Table 7), and (3) length of operation >T hours, where T is the approximate 75th percentile of the duration of the specific operation being performed.[126] The ASA class replaced discharge diagnoses of the SENIC risk index as a surrogate for the patient's underlying severity of illness (host susceptibility)[406,407] and has the advantage of being readily available in the chart during the patient's hospital stay. Unlike SENIC's constant 2-hour cut-point for duration of operation, the operation-specific cut-points used in the NNIS risk index increase its discriminatory power compared to the SENIC index.[126]

b. Issues

Adjustment for variables known to confound rate estimates is critical if valid comparisons of SSI rates are to be made between surgeons or hospitals.[408] Risk stratification, as described above, has proven useful for this purpose, but relies on the ability of surveillance personnel to find and record data consistently and correctly. For the three variables used in the NNIS risk index, only one study has focused on how accurately any of them are recorded. Cardo et al. found that surgical team members' accuracy in assessing wound classification for general and trauma surgery was 88% (95% CI: 82%-94%).[409] However, there are sufficient ambiguities in the wound class definitions themselves to warrant concern about the reproducibility of Cardo's results. The accuracy of recording the duration of operation (i.e., time from skin incision to skin closure) and the ASA class has not been studied. In an unpublished report from the NNIS system, there was evidence that overreporting of high ASA class existed in some hospitals. Further validation of the reliability of the recorded risk index variables is needed.

Additionally, the NNIS risk index does not adequately discriminate the SSI risk for all types of operations.[27,410] It seems likely that a combination of risk factors specific to patients undergoing an operation will be more predictive. A

few studies have been performed to develop procedure-specific risk indices[218,411-414] and research in this area continues within CDC's NNIS system.

2. SSI Surveillance Methods

SSI surveillance methods used in both the SENIC Project and the NNIS system were designed for monitoring inpatients at acute-care hospitals. Over the past decade, the shift from inpatient to outpatient surgical care (also called ambulatory or day surgery) has been dramatic. It has been estimated that 75% of all operations in the United States will be performed in outpatient settings by the year 2000.[4] While it may be appropriate to use common definitions of SSI for inpatients and outpatients,[415] the types of operations monitored, the risk factors assessed, and the case-finding methods used may differ. New predictor variables may emerge from analyses of SSIs among outpatient surgery patients, which may lead to different ways of estimating SSI risk in this population.

The choice of which operations to monitor should be made jointly by surgeons and infection control personnel. Most hospitals do not have the resources to monitor all surgical patients all the time, nor is it likely that the same intensity of surveillance is necessary for certain low-risk procedures. Instead, hospitals should target surveillance efforts toward high-risk procedures.[416]

a. Inpatient SSI surveillance

Two methods, alone or together, have been used to identify inpatients with SSIs: (1) direct observation of the surgical site by the surgeon, trained nurse surveyor, or infection control personnel[16,97,399,402,409,417-420] and (2) indirect detection by infection control personnel through review of laboratory reports, patient records, and discussions with primary care providers.[15,84,399,402,404,409,418,421-427] The surgical literature suggests that direct observation of surgical sites is the most accurate method to detect SSIs, although sensitivity data are lacking.[16,399,402,417,418] Much of the SSI data reported in the infection control literature has been generated by indirect case-finding methods,[125,126,422,425,426,428-430] but some studies of direct methods also have been conducted.[97,409] Some studies use both methods of detection.[84,409,424,427,431] A study that focused solely on the sensitivity and specificity of SSIs detected by indirect methods found a sensitivity of 83.8% (95% CI: 75.7%-91.9%) and a specificity of 99.8% (95% CI: 99%-100%).[409] Another study showed that chart review triggered by a computer-generated report of antibiotic orders for post-cesarean section patients had a sensitivity of 89% for detecting endometritis.[432]

Indirect SSI detection can readily be performed by infection control personnel during surveillance rounds. The work includes gathering demographic, infection, surgical, and laboratory data on patients who have undergone operations of interest.[433] These data can be obtained from patients' medical records, including microbiology, histopathology, laboratory, and pharmacy data; radiology reports; and records from the operating room. Additionally, inpatient admissions, emergency room, and clinic visit records are sources of data for those postdischarge surgical patients who are readmitted or seek follow-up care.

The optimum frequency of SSI case-finding by either method is unknown and varies from daily to ⩽3 times per week, continuing until the patient is discharged from the hospital. Because duration of hospitalization is often very short, postdischarge SSI surveillance has become increasingly important to obtain accurate SSI rates (refer to "Postdischarge SSI Surveillance" section).

To calculate meaningful SSI rates, data must be collected on all patients undergoing the operations of interest (i.e., the population at risk). Because one of its purposes is to develop strategies for risk stratification, the NNIS system collects the following data on all surgical patients surveyed: operation date; NNIS operative procedure category;[434] surgeon identifier; patient identifier; age and sex; duration of operation; wound class; use of general anesthesia; ASA class; emergency; trauma; multiple procedures; endoscopic approach; and discharge date.[433] With the exception of discharge date, these data can be obtained manually from operating room logs or be electronically downloaded into surveillance software, thereby substantially reducing manual transcription and data entry errors.[433] Depending on the needs for risk-stratified SSI rates by personnel in infection control, surgery, and quality assurance, not all data elements may be pertinent for every type of operation. At minimum, however, variables found to be predictive of increased SSI risk should be collected (refer to "SSI Risk Stratification" section).

b. Postdischarge SSI surveillance

Between 12% and 84% of SSIs are detected after patients are discharged from the hospital.[98,337,402,428,435-454] At least two studies have shown that most SSIs become evident within 21 days after operation.[446,447] Since the length of postoperative hospitalization continues to decrease, many SSIs may not be detected for several weeks after discharge and may not require readmission to the operating hospital. Dependence solely on inpatient case-finding will result in underestimates of SSI rates for some operations (e.g., coronary artery bypass graft) (CDC/NNIS system, unpublished data, 1998). Any comparison of SSI rates must take into account whether case-finding included SSIs detected after discharge. For comparisons to be valid, even in the same institution over time, the postdischarge surveillance methods must be the same.

Postdischarge surveillance methods have been used with varying degrees of success for different procedures and among hospitals and include (1) direct examination of patients' wounds during follow-up visits to either surgery clinics or physicians' offices,[150,399,402,404,430,436,440,441,447,452,455] (2) review of medical records of surgery clinic patients,[404,430,439] (3) patient surveys by mail or telephone,[435,437,438,441,442,444,445,448,449,455-457] or (4) surgeon surveys by mail or telephone.[98,428,430,437-439,443,444,446,448,450,451,455] One study found that patients have difficulty assessing their own wounds for infection (52% specificity, 26% positive predictive value),[458] suggesting that data obtained by patient questionnaire may inaccurately represent actual SSI rates.

Recently, Sands et al. performed a computerized search of three databases to determine which best identified SSIs: ambulatory encounter records for diagnostic, testing, and treatment codes; pharmacy records for specific antimicrobial prescriptions; and administrative records for rehospitalizations and emergency room visits.[446] This study found that pharmacy records indicating a patient had received antimicrobial agents commonly used to treat soft tissue infections had the highest sensitivity (50%) and positive predictive value (19%), although even this approach alone was not very effective.

As integrated health information systems expand, tracking surgical patients through the entire course of care may become more feasible, practical, and effective. At this time, no consensus exists on which postdischarge surveillance methods are the most sensitive, specific, and practical. Methods chosen will necessarily reflect the hospital's unique mix of operations, personnel resources, and data needs.

c. Outpatient SSI surveillance

Both direct and indirect methods have been used to detect SSIs that complicate outpatient operations. One 8-year study of operations for hernia and varicose veins used home visits by district health nurses combined with a survey completed by the surgeon at the patient's 2-week postoperative clinic visit to identify SSIs.[459] While ascertainment was essentially 100%, this method is impractical for widespread implementation. High response rates have been obtained from questionnaires mailed to surgeons (72%->90%).[443,444,446,455,459-461] Response rates from telephone questionnaires administered to patients were more variable (38%,[444] 81%,[457] and 85%[455]), and response rates from questionnaires mailed to patients were quite low (15%[455] and 33%[446]). At this time, no single detection method can be recommended. Available resources and data needs determine which method(s) should be used and which operations should be monitored. Regardless of which detection method is used, it is recommended that the CDC NNIS definitions of SSI (Tables 1 and 2) be used without modification in the outpatient setting.

G. GUIDELINE EVALUATION PROCESS

The value of the HICPAC guidelines is determined by those who use them. To help assess that value, HICPAC is developing an evaluation tool to learn how guidelines meet user expectations, and how and when these guidelines are disseminated and implemented.

II. RECOMMENDATIONS FOR PREVENTION OF SURGICAL SITE INFECTION

A. RATIONALE

The Guideline for Prevention of Surgical Site Infection, 1999, provides recommendations concerning reduction of surgical site infection risk. Each recommendation is categorized on the basis of existing scientific data, theoretical rationale, and applicability. However, the previous CDC system for categorizing recommendations has been modified slightly.

Category I recommendations, including IA and IB, are those recommendations that are viewed as effective by HICPAC and experts in the fields of surgery, infectious diseases, and infection control. Both Category IA and IB recommendations are applicable for, and should be adopted by, all healthcare facilities; IA and IB recommendations differ only in the strength of the supporting scientific evidence.

Category II recommendations are supported by less scientific data than Category I recommendations; such recommendations may be appropriate for addressing specific nosocomial problems or specific patient populations.

No recommendation is offered for some practices, either because there is a lack of consensus regarding their efficacy or because the available scientific evidence is insufficient to support their adoption. For such unresolved issues, practitioners should use judgement to determine a policy regarding these practices within their organization. Recommendations that are based on federal regulation are denoted with an asterisk.

B. RANKINGS

Category IA. Strongly recommended for implementation and supported by well-designed experimental, clinical, or epidemiological studies.

Category IB. Strongly recommended for implementation and supported by some experimental, clinical, or epidemiological studies and strong theoretical rationale.

Category II. Suggested for implementation and supported by suggestive clinical or epidemiological studies or theoretical rationale.

No recommendation; unresolved issue. Practices for which insufficient evidence or no consensus regarding efficacy exists.

Practices required by federal regulation are denoted with an asterisk (*).

C. RECOMMENDATIONS

1. Preoperative

a. Preparation of the patient

1. Whenever possible, identify and treat all infections remote to the surgical site before elective operation and postpone elective operations on patients with remote site infections until the infection has resolved. *Category IA*

2. Do not remove hair preoperatively unless the hair at or around the incision site will interfere with the operation. *Category IA*

3. If hair is removed, remove immediately before the operation, preferably with electric clippers. *Category IA*

4. Adequately control serum blood glucose levels in all diabetic patients and particularly avoid hyperglycemia perioperatively. *Category IB*

5. Encourage tobacco cessation. At minimum, instruct patients to abstain for at least 30 days before elective operation from smoking cigarettes, cigars, pipes, or any other form of tobacco consumption (e.g., chewing/dipping). *Category IB*

6. Do not withhold necessary blood products from surgical patients as a means to prevent SSI. *Category IB*

7. Require patients to shower or bathe with an antiseptic agent on at least the night before the operative day. *Category IB*

8. Thoroughly wash and clean at and around the incision site to remove gross contamination before performing antiseptic skin preparation. *Category IB*

9. Use an appropriate antiseptic agent for skin preparation (Table 6). *Category IB*

10. Apply preoperative antiseptic skin preparation in concentric circles moving toward the periphery. The prepared area must be large enough to extend the incision or create new incisions or drain sites, if necessary. *Category II*

11. Keep preoperative hospital stay as short as possible while allowing for adequate preoperative preparation of the patient. *Category II*

12. No recommendation to taper or discontinue systemic steroid use (when medically permissible) before elective operation. *Unresolved issue*

13. No recommendation to enhance nutritional support for surgical patients solely as a means to prevent SSI. *Unresolved issue*

14. No recommendation to preoperatively apply mupirocin to nares to prevent SSI. *Unresolved issue*

15. No recommendation to provide measures that enhance wound space oxygenation to prevent SSI. *Unresolved issue*

b. Hand/forearm antisepsis for surgical team members

1. Keep nails short and do not wear artificial nails. *Category IB*

2. Perform a preoperative surgical scrub for at least 2 to 5 minutes using an appropriate antiseptic (Table 6). Scrub the hands and forearms up to the elbows. *Category IB*

3. After performing the surgical scrub, keep hands up and away from the body (elbows in flexed position) so that water runs from the tips of the fingers toward the elbows. Dry hands with a sterile towel and don a sterile gown and gloves. *Category IB*

4. Clean underneath each fingernail prior to performing the first surgical scrub of the day. *Category II*

5. Do not wear hand or arm jewelry. *Category II*

6. No recommendation on wearing nail polish. *Unresolved Issue*

c. Management of infected or colonized surgical personnel

1. Educate and encourage surgical personnel who have signs and symptoms of a transmissible infectious illness to report conditions promptly to their supervisory and occupational health service personnel. *Category IB*

2. Develop well-defined policies concerning patient-care responsibilities when personnel have potentially transmissible infectious conditions. These policies should govern (a) personnel responsibility in using the health service and reporting illness, (b) work restrictions, and (c) clearance to resume work after an illness that required work restriction. The policies also should identify persons who have the authority to remove personnel from duty. *Category IB*

3. Obtain appropriate cultures from, and exclude from duty, surgical personnel who have draining skin lesions until infection has been ruled out or personnel have received adequate therapy and infection has resolved. *Category IB*

4. Do not routinely exclude surgical personnel who are colonized with organisms such as *S. aureus* (nose, hands, or other body site) or group A *Streptococcus,* unless such personnel have been linked epidemiologically to dissemination of the organism in the healthcare setting. *Category IB*

d. Antimicrobial prophylaxis

1. Administer a prophylactic antimicrobial agent only when indicated, and select it based on its efficacy against the most common pathogens causing SSI for a specific operation (Table 4) and published recommendations.[266,268,269,282-284] *Category IA*

2. Administer by the intravenous route the initial dose of prophylactic antimicrobial agent, timed such that a bactericidal concentration of the drug is established in serum and tissues when the incision is made. Maintain therapeutic levels of the agent in serum and tissues throughout the operation and until, at most, a few hours after the incision is closed in the operating room. *Category IA*

3. Before elective colorectal operations in addition to d2 above, mechanically prepare the colon by use of enemas and cathartic agents. Administer nonabsorbable oral antimicrobial agents in divided doses on the day before the operation. *Category IA*

4. For high-risk cesarean section, administer the prophylactic antimicrobial agent immediately after the umbilical cord is clamped. *Category IA*

5. Do not routinely use vancomycin for antimicrobial prophylaxis. *Category IB*

2. Intraoperative

a. Ventilation

1. Maintain positive-pressure ventilation in the operating room with respect to the corridors and adjacent areas. *Category IB*

2. Maintain a minimum of 15 air changes per hour, of which at least 3 should be fresh air. *Category IB*

3. Filter all air, recirculated and fresh, through the appropriate filters per the American Institute of Architects' recommendations.[299] *Category IB*

4. Introduce all air at the ceiling, and exhaust near the floor. *Category IB*

5. Do not use UV radiation in the operating room to prevent SSI. *Category IB*

6. Keep operating room doors closed except as needed for passage of equipment, personnel, and the patient. *Category IB*

7. Consider performing orthopedic implant operations in operating rooms supplied with ultraclean air. *Category II*

8. Limit the number of personnel entering the operating room to necessary personnel. *Category II*

b. Cleaning and disinfection of environmental surfaces

1. When visible soiling or contamination with blood or other body fluids of surfaces or equipment occurs during an operation, use an EPA-approved hospital disinfectant to clean the affected areas before the next operation. *Category IB**

2. Do not perform special cleaning or closing of operating rooms after contaminated or dirty operations. *Category IB*

3. Do not use tacky mats at the entrance to the operating room suite or individual operating rooms for infection control. *Category IB*

4. Wet vacuum the operating room floor after the last operation of the day or night with an EPA-approved hospital disinfectant. *Category II*

5. No recommendation on disinfecting environmental surfaces or equipment used in operating rooms between operations in the absence of visible soiling. *Unresolved issue*

c. Microbiologic sampling

1. Do not perform routine environmental sampling of the operating room. Perform microbiologic sampling of operating room environmental surfaces or air only as part of an epidemiologic investigation. *Category IB*

d. Sterilization of surgical instruments

1. Sterilize all surgical instruments according to published guidelines.[212,299,314,321] *Category IB*

2. Perform flash sterilization only for patient care items that will be used immediately (e.g., to reprocess an inadvertently dropped instrument). Do not use flash sterilization for reasons of convenience, as an alternative to purchasing additional instrument sets, or to save time. *Category IB*

e. Surgical attire and drapes

1. Wear a surgical mask that fully covers the mouth and nose when entering the operating room if an operation is about to begin or already under way, or if sterile instruments are exposed. Wear the mask throughout the operation. *Category IB**

2. Wear a cap or hood to fully cover hair on the head and face when entering the operating room. *Category IB**

3. Do not wear shoe covers for the prevention of SSI. *Category IB**

4. Wear sterile gloves if a scrubbed surgical team member. Put on gloves after donning a sterile gown. *Category IB**

5. Use surgical gowns and drapes that are effective barriers when wet (i.e., materials that resist liquid penetration). *Category IB*

6. Change scrub suits that are visibly soiled, contaminated, and/or penetrated by blood or other potentially infectious materials. *Category IB**

7. No recommendations on how or where to launder scrub suits, on restricting use of scrub suits to the operating suite, or for covering scrub suits when out of the operating suite. *Unresolved issue*

f. Asepsis and surgical technique

1. Adhere to principles of asepsis when placing intravascular devices (e.g., central venous catheters), spinal or epidural anesthesia catheters, or when dispensing and administering intravenous drugs. *Category IA*

2. Assemble sterile equipment and solutions immediately prior to use. *Category II*

3. Handle tissue gently, maintain effective hemostasis, minimize devitalized tissue and foreign bodies (i.e., sutures, charred tissues, necrotic debris), and eradicate dead space at the surgical site. *Category IB*

4. Use delayed primary skin closure or leave an incision open to heal by second intention if the surgeon considers the surgical site to be heavily contaminated (e.g., Class III and Class IV). *Category IB*

5. If drainage is necessary, use a closed suction drain. Place a drain through a separate incision distant from the operative incision. Remove the drain as soon as possible. *Category IB*

3. Postoperative incision care

a. Protect with a sterile dressing for 24 to 48 hours postoperatively an incision that has been closed primarily. *Category IB*

b. Wash hands before and after dressing changes and any contact with the surgical site. *Category IB*

c. When an incision dressing must be changed, use sterile technique. *Category II*

d. Educate the patient and family regarding proper incision care, symptoms of SSI, and the need to report such symptoms. *Category II*

e. No recommendation to cover an incision closed primarily beyond 48 hours, nor on the appropriate time to shower or bathe with an uncovered incision. *Unresolved issue*

4. Surveillance

a. Use CDC definitions of SSI (Table 1) without modification for identifying SSI among surgical inpatients and outpatients. *Category IB*

b. For inpatient case-finding (including readmissions), use direct prospective observation, indirect prospective detection, or a combination of both direct and indirect methods for the duration of the patient's hospitalization. *Category IB*

c. When postdischarge surveillance is performed for detecting SSI following certain operations (e.g., coronary artery bypass graft), use a method that accommodates available resources and data needs. *Category II*

d. For outpatient case-finding, use a method that accommodates available resources and data needs. *Category IB*

e. Assign the surgical wound classification upon completion of an operation. A surgical team member should make the assignment. *Category II*

f. For each patient undergoing an operation chosen for surveillance, record those variables shown to be associated with increased SSI risk (e.g., surgical wound class, ASA class, and duration of operation). *Category IB*

g. Periodically calculate operation-specific SSI rates

** Federal regulation: OSHA.*

stratified by variables shown to be associated with increased SSI risk (e.g., NNIS risk index). *Category IB*

h. Report appropriately stratified, operation-specific SSI rates to surgical team members. The optimum frequency and format for such rate computations will be determined by stratified case-load sizes (denominators) and the objectives of local, continuous quality improvement initiatives. *Category IB*

i. No recommendation to make available to the infection control committee coded surgeon-specific data. *Unresolved issue*

REFERENCES

1. Garner JS. CDC guideline for prevention of surgical wound infections, 1985. Supercedes guideline for prevention of surgical wound infections published in 1982. (Originally published in 1995). Revised. *Infect Control* 1986;7(3):193-200.
2. Simmons BP. Guideline for prevention of surgical wound infections. *Infect Control* 1982;3:185-196.
3. Garner JS. The CDC Hospital Infection Control Practices Advisory Committee. *Am J Infect Control* 1993;21:160-2.
4. Hecht AD. Creating greater efficiency in ambulatory surgery. *J Clin Anesth* 1995;7:581-4.
5. Horwitz JR, Chwals WJ, Doski JJ, Suescun EA, Cheu HW, Lally KP. Pediatric wound infections: a prospective multicenter study. *Ann Surg* 1998;227:553-8.
6. Golub R, Siddiqui F, Pohl D. Laparoscopic versus open appendectomy: a metaanalysis. *J Am Coll Surg* 1998;186:545-53.
7. Mayol J, Garcia-Aguilar J, Ortiz-Oshiro E, De-Diego Carmona JA, Ferndandez-Represa JA. Risks of the minimal access approach for laparoscopic surgery: multivariate analysis of morbidity related to umbilical trocar insertion. *World J Surg* 1997;21:529-33.
8. Lacy AM, Garcia-Valdecasas JC, Delgado S, Grande L, Fuster J, Tabet J, et al. Postoperative complications of laparoscopic-assisted colectomy. *Surg Endosc* 1997;11:119-22.
9. Pagni S, Salloum EJ, Tobin GR, VanHimbergen DJ, Spence PA. Serious wound infections after minimally invasive coronary bypass procedures. *Ann Thorac Surg* 1998;66:92-4.
10. The Southern Surgeons Club. A prospective analysis of 1518 laparoscopic cholecystectomies. *N Engl J Med* 1991;324:1073-8.
11. Centers for Disease Control and Prevention. National Nosocomial Infections Surveillance (NNIS) report, data summary from October 1986-April 1997, issued May 1997. *Am J Infect Control* 1997;25:477-87.
12. Food and Drug Administration. Topical antimicrobial drug products for over-the-counter human use: tentative final monograph for health-care antiseptic drug products—proposed rule (21 CFR Parts 333 and 369). *Federal Register* 1994;59:31441-52.
13. Centers for Disease Control and Prevention, National Center for Health Statistics. *Vital and Health Statistics, Detailed Diagnoses and Procedures, National Hospital Discharge Survey, 1994.* Vol 127. Hyattsville, Maryland: DHHS Publication; 1997.
14. Emori TG, Gaynes RP. An overview of nosocomial infections, including the role of the microbiology laboratory. *Clin Microbiol Rev* 1993;6(4):428-42.
15. Cruse P. Wound infection surveillance. *Rev Infect Dis* 1981;4(3):734-7.
16. Cruse PJ, Foord R. The epidemiology of wound infection: a 10-year prospective study of 62,939 wounds. *Surg Clin North Am* 1980;60(1): 27-40.
17. Martone WJ, Jarvis WR, Culver DH, Haley RW. Incidence and nature of endemic and epidemic nosocomial infections. In: Bennett JV, Brachman PS, eds. *Hospital Infections.* 3rd ed. Boston: Little, Brown and Co; 1992. p. 577-96.
18. Boyce JM, Potter-Bynoe G, Dziobek L. Hospital reimbursement patterns among patients with surgical wound infections following open heart surgery. *Infect Control Hosp Epidemiol* 1990;11(2):89-93.
19. Poulsen KB, Bremmelgaard A, Sorensen AI, Raahave D, Petersen JV. Estimated costs of postoperative wound infections. A case-control study of marginal hospital and social security costs. *Epidemiol Infect* 1994;113(2):283-95.
20. Vegas AA, Jodra VM, Garcia ML. Nosocomial infection in surgery wards: a controlled study of increased duration of hospital stays and direct cost of hospitalization. *Eur J Epidemiol* 1993;9(5):504-10.
21. Albers BA, Patka P, Haarman HJ, Kostense PJ. Cost effectiveness of preventive antibiotic administration for lowering risk of infection by 0.25%. [German]. *Unfallchirurg* 1994;97(12):625-8.
22. Horan TC, Gaynes RP, Martone WJ, Jarvis WR, Emori TG. CDC definitions of nosocomial surgical site infections, 1992: a modification of CDC definitions of surgical wound infections. *Infect Control Hosp Epidemiol* 1992;13(10):606-8.
23. Ehrenkranz NJ, Richter EI, Phillips PM, Shultz JM. An apparent excess of operative site infections: analyses to evaluate false-positive diagnoses. *Infect Control Hosp Epidemiol* 1995;16(12):712-6.
24. Taylor G, McKenzie M, Kirkland T, Wiens R. Effect of surgeon's diagnosis on surgical wound infection rates. *Am J Infect Control* 1990;18(5):295-9.
25. SHEA, APIC, CDC, SIS. Consensus paper on the surveillance of surgical wound infections. *Infect Control Hosp Epidemiol* 1992;13(10): 599-605.
26. Nooyen SM, Overbeek BP, Brutel de la Riviere A, Storm AJ, Langemeyer JM. Prospective randomised comparison of single-dose versus multiple-dose cefuroxime for prophylaxis in coronary artery bypass grafting. *Eur J Clin Microbiol Infect Dis* 1994;13:1033-7.
27. Centers for Disease Control and Prevention. National Nosocomial Infections Surveillance (NNIS) report, data summary from October 1986-April 1996, issued May 1996. A report from the National Nosocomial Infections Surveillance (NNIS) System. *Am J Infect Control* 1996;24:380-8.
28. Schaberg DR. Resistant gram-positive organisms. *Ann Emerg Med* 1994;24(3):462-4.
29. Schaberg DR, Culver DH, Gaynes RP. Major trends in the microbial etiology of nosocomial infection. *Am J Med* 1991;91(3B):72S-5S.
30. Jarvis WR. Epidemiology of nosocomial fungal infections, with emphasis on *Candida* species. *Clin Infect Dis* 1995;20:1526-30.
31. Centers for Disease Control. Nosocomial outbreak of *Rhizopus* infections associated with Elastoplast wound dressings—Minnesota. *MMWR* 1978;27:33-4.
32. Pearson RD, Valenti WM, Steigbigel RT. *Clostridium perfringens* wound infections associated with elastic bandages. *JAMA* 1980;244:1128-30.
33. Richet HM, Craven PC, Brown JM, Lasker BA, Cox CD, McNeil MM, et al. A cluster of *Rhodococcus (Gordona) bronchialis* sternal-wound infections after coronary-artery bypass surgery. *N Engl J Med* 1991;324:104-9.
34. Wenger PN, Brown JM, McNeil MM, Jarvis WR. *Nocardia farcinica* sternotomy site infections in patients following open heart surgery. *J Infect Dis* 1998;178:1539-43.
35. Lowry PW, Blankenship RJ, Gridley W, Troup NJ, Tompkins LS. A cluster of *Legionella* sternal-wound infections due to postoperative topical exposure to contaminated tap water. *N Engl J Med* 1991;324: 109-13.
36. Bassett DC, Stokes KJ, Thomas WR. Wound infection with *Pseudomonas multivorans:* a water-borne contaminant of disinfectant solutions. *Lancet* 1970;1:1188-91.
37. Cruse PJ. Surgical wound infection. In: Wonsiewicz MJ, ed. *Infectious Diseases.* Philadelphia: W.B. Saunders Co; 1992. p. 758-64.
38. Altemeier WA, Culbertson WR. Surgical infection. In: Moyer CA, Rhoads JE, Allen JG, Harkins HN, eds. *Surgery, principles and practice.* 3rd ed. Philadelphia: JB Lippincott; 1965. p. 51-77.
39. Krizek TJ, Robson MC. Evolution of quantitative bacteriology in wound management. *Am J Surg* 1975;130:579-84.
40. Elek SD, Conen PE. The virulence of *Staphylococcus pyogenes* for man: a study of problems with wound infection. *Br J Exp Pathol* 1957;38: 573-86.
41. Noble WC. The production of subcutaneous staphylococcal skin lesions in mice. *Br J Exp Pathol* 1965;46:254-62.
42. James RC, MacLeod CJ. Induction of staphylococcal infections in mice with small inocula introduced on sutures. *Br J Exp Pathol* 1961;42: 266-77.
43. Henderson B, Poole S, Wilson M. Microbial/host interactions in health and disease: who controls the cytokine network? *Immunopharmacology* 1996;35:1-21.
44. Morrison DC, Ryan JL. Endotoxins and disease mechanisms. *Ann Rev Med* 1987;38:417-32.
45. Demling R, LaLonde C, Saldinger P, Knox J. Multiple-organ dysfunction in the surgical patient: pathophysiology, prevention, and treatment. *Curr Probl Surg* 1993;30:345-414.
46. Eiseman B, Beart R, Norton L. Multiple organ failure. *Surg Gynecol Obstet* 1977;14:323-6.
47. Fry DE, Pearlstein L, Fulton RL, Polk HC, Jr. Multiple system organ failure: the role of uncontrolled infection. *Arch Surg* 1980;115:136-40.
48. Kasper DL. Bacterial capsule—old dogmas and new tricks. *J Infect Dis* 1986;153:407-15.
49. Dellinger EP. Surgical infections and choice of antibiotics. In: Sabiston DC, ed. *Textbook of Surgery. The Biological Basis of Modern Surgical Practice.* 15th ed. Philadelphia: W.B. Saunders Co; 1997. p. 264-80.
50. Goeau-Brissonniere O, Leport C, Guidoin R, Lebrault C, Pechere JC,

Bacourt F. Experimental colonization of an expanded polytetrafluoroethylene vascular graft with *Staphylococcus aureus:* a quantitative and morphologic study. *J Vasc Surg* 1987;5(5):743-8.
51. Bergamini TM, Corpus RA Jr., Brittian KR, Peyton JC, Cheadle WG. The natural history of bacterial biofilm graft infection. *J Surg Res* 1994;56:393-6.
52. Baddour LM, Christensen GD, Hester MG, Bisno AL. Production of experimental endocarditis by coagulase-negative staphylococci: variability in species virulence. *J Infect Dis* 1984;150:721-7.
53. Christensen GD, Baddour LM, Simpson WA. Phenotypic variation of *Staphylococcus epidermidis* slime production in vitro and in vivo. *Infect Immun* 1987;55:2870-7.
54. Mayberry-Carson KJ, Tober-Meyer B, Smith JK, Lambe DW Jr., Costerton JW. Bacterial adherence and glycocalyx formation in osteomyelitis experimentally induced with *Staphylococcus aureus. Infect Immun* 1984;43:825-33.
55. Mills J, Pulliam L, Dall L, Marzouk J, Wilson W, Costerton JW. Exopolysaccharide production by *viridans* streptococci in experimental endocarditis. *Infect Immun* 1984;43:359-67.
56. Kaebnick HW, Bandyk DF, Bergamini TM, Towne JB. The microbiology of explanted vascular prostheses. *Surgery* 1987;102:756-61.
57. Altemeier WA, Culbertson WR, Hummel RP. Surgical considerations of endogenous infections—sources, types, and methods of control. *Surg Clin North Am* 1968;48:227-40.
58. Wiley AM, Ha'eri GB. Routes of infection: a study of using "tracer particles" in the orthopedic operating room. *Clin Orthop* 1979;139: 150-5.
59. Slaughter L, Morris JE, Starr A. Prosthetic valvular endocarditis. A 12-year review. *Circulation* 1973;47:1319-26.
60. Carlsson AK, Lidgren L, Lindberg L. Prophylactic antibiotics against early and late deep infections after total hip replacements. *Acta Orthop Scand* 1977;48:405-10.
61. Hunter JG, Padilla M, Cooper-Vastola S. Late *Clostridium perfringens* breast implant infection after dental treatment. *Ann Plast Surg* 1996;36(3):309-12.
62. Stuesse DC, Robinson JH, Durzinsky DS. A late sternal wound infection caused by hematogenous spread of bacteria. *Chest* 1995;108(6):1742-3.
63. Howe CW. Experimental wound sepsis from transient *Escherichia coli* bacteremia. *Surgery* 1969;66:570-4.
64. Velasco E, Thuler LC, Martins CA, Dias LM, Conalves VM. Risk factors for infectious complications after abdominal surgery for malignant disease. *Am J Infect Control* 1996;24(1):1-6.
65. Bruun JN. Post-operative wound infection. Predisposing factors and the effect of a reduction in the dissemination of staphylococci. *Acta Med Scand Suppl* 1970;514(Suppl):3-89.
66. Simchen E, Rozin R, Wax Y. The Israeli Study of Surgical Infection of drains and the risk of wound infection in operations for hernia. *Surg Gynecol Obstet* 1990;170:331-7.
67. Edwards LD. The epidemiology of 2056 remote site infections and 1966 surgical wound infections occurring in 1865 patients: a four year study of 40,923 operations at Rush-Presbyterian-St. Luke's Hospital, Chicago. *Ann Surg* 1976;184:758-66.
68. Valentine RJ, Weigelt JA, Dryer D, Rodgers C. Effect of remote infections on clean wound infection rates. *Am J Infect Control* 1986;14:64-7.
69. Cioffi GA, Terezhalmy GT, Taybos GM. Total joint replacement: a consideration for antimicrobial prophylaxis. *Oral Surg Oral Med Oral Pathol* 1988;66(1):124-9.
70. Heggeness MH, Esses SI, Errico T, Yuan HA. Late infection of spinal instrumentation by hematogenous seeding. *Spine* 1993;18(4):492-6.
71. Mont MA, Waldman B, Banerjee C, Pacheco IH, Hungerford DS. Multiple irrigation, debridement, and retention of components in infected total knee arthroplasty. *J Arthroplasty* 1997;12(4):426-33.
72. Ozuna RM, Delamarter RB. Pyogenic vertebral osteomyelitis and postsurgical disc space infections. *Ortho Clin North Am* 1996;27(1): 87-94.
73. Schmalzried TP, Amstutz HC, Au MK, Dorey FJ. Etiology of deep sepsis in total hip arthroplasty. The significance of hematogenous and recurrent infections. *Clin Orthop* 1992;280:200-7.
74. Calia FM, Wolinsky E, Mortimer EA Jr., Abrams JS, Rammelkamp CH Jr. Importance of the carrier state as a source of *Staphylococcus aureus* in wound sepsis. *J Hyg (Lond)* 1969;67:49-57.
75. Dineen P, Drusin L. Epidemics of postoperative wound infections associated with hair carriers. *Lancet* 1973;2(7839):1157-9.
76. Mastro TD, Farley TA, Elliott JA, Facklam RR, Perks JR, Hadler JL, et al. An outbreak of surgical-wound infections due to group A streptococcus carried on the scalp. *N Engl J Med* 1990;323:968-72.
77. Ford CR, Peterson DE, Mitchell CR. An appraisal of the role of surgical face masks. *Am J Surg* 1967;113:787-90.
78. Letts RM, Doermer E. Conversation in the operating theater as a cause of airborne bacterial contamination. *J Bone Joint Surg [Am]* 1983;65:357-62.
79. Giamarellou H, Antoniadou A. Epidemiology, diagnosis, and therapy of fungal infections in surgery. *Infect Control Hosp Epidemiol* 1996;17(8):558-64.
80. Lee JT. Surgical wound infections: surveillance for quality improvement. In: Fry DE, ed. *Surgical Infections.* Boston: Little, Brown and Co; 1995. p. 145-59.
81. Perl TM, Golub JE. New approaches to reduce *Staphylococcus aureus* nosocomial infection rates: treating *S. aureus* nasal carriage. *Ann Pharmacother* 1998;32:S7-S16.
82. Kluytmans JA, Mouton JW, Ijzerman EP, Vandenbroucke-Grauls CM, Maat AW, Wagenvoort JH, et al. Nasal carriage of *Staphylococcus aureus* as a major risk factor for wound infections after cardiac surgery. *J Infect Dis* 1995;171:216-9.
83. Perl TM, Cullen JJ, Pfaller MA, Wenzel RP, Herwaldt LA, The MARS Study Team. A randomized, double-blind, placebo-controlled clinical trial of intranasal mupirocin ointment (IM) for prevention of *S. aureus* surgical site infections (SSI) [abstract]. Abstracts of the IDSA 36th Annual Meeting 1998;91(88).
84. Gil-Egea MJ, Pi-Sunyer MT, Verdaguer A, Sanz F, Sitges-Serra A, Eleizegui LT. Surgical wound infections: prospective study of 4,486 clean wounds. *Infect Control* 1987;8(7):277-80.
85. Nagachinta T, Stephens M, Reitz B, Polk BF. Risk factors for surgical-wound infection following cardiac surgery. *J Infect Dis* 1987;156:967-73.
86. Lilienfeld DE, Vlahov D, Tenney JH, McLaughlin JS. Obesity and diabetes as risk factors for postoperative wound infections after cardiac surgery. *Am J Infect Control* 1988;16:3-6.
87. Slaughter MS, Olson MM, Lee JT Jr., Ward HB. A fifteen-year wound surveillance study after coronary artery bypass. *Ann Thorac Surg* 1993;56(5):1063-8.
88. Bryan AJ, Lamarra M, Angelini GD, West RR, Breckenridge IM. Median sternotomy wound dehiscence: a retrospective case control study of risk factors and outcome. *J R Coll Surg Edinb* 1992;37:305-8.
89. Jones JK, Triplett RG. The relationship of cigarette smoking to impaired intraoral wound healing: a review of evidence and implications for patient care. *J Oral Maxillofac Surg* 1992;50(3):237-9; discussion 239-40.
90. Vinton AL, Traverso LW, Jolly PC. Wound complications after modified radical mastectomy compared with tylectomy with axillary lymph node dissection. *Am J Surg* 1991;161(5):584-8.
91. Holley DT, Toursarkissian B, Vansconez HC, Wells MD, Kenady DE, Sloan DA. The ramifications of immediate reconstruction in the management of breast cancer. *Am Surg* 1995;61(1):60-5.
92. Beitsch P, Balch C. Operative morbidity and risk factor assessment in melanoma patients undergoing inguinal lymph node dissection. *Am J Surg* 1992;164(5):462-6; discussion 465-6.
93. Post S, Betzler M, vonDitfurth B, Schurmann G, Kuppers P, Herfarth C. Risks of intestinal anastomoses in Crohn's disease. *Ann Surg* 1991;213(1):37-42.
94. B'erard F, Gandon J. Postoperative wound infections: the influence of ultraviolet irradiation of the operating room and of various other factors. *Ann Surg* 1964;160(Suppl 1):1-192.
95. Nystrom PO, Jonstam A, Hojer H, Ling L. Incisional infection after colorectal surgery in obese patients. *Acta Chir Scand* 1987;153:225-7.
96. He GW, Ryan WH, Acuff TE, Bowman RT, Douthit MB, Yang CQ, et al. Risk factors for operative mortality and sternal wound infection in bilateral internal mammary artery grafting. *J Thorac Cardiovasc Surg* 1994;107(1):196-202.
97. Barber GR, Miransky J, Brown AE, Coit DG, Lewis FM, Thaler HT, et al. Direct observations of surgical wound infections at a comprehensive cancer center. *Arch Surg* 1995;130(10):1042-7.
98. Cruse PJ, Foord R. A five-year prospective study of 23,649 surgical wounds. *Arch Surg* 1973;107:206-10.
99. Claesson BE, Holmlund DE. Predictors of intraoperative bacterial contamination and postoperative infection in elective colorectal surgery. *J Hosp Infect* 1988;11:127-35.
100. Mishriki SF, Law DJ, Jeffery PJ. Factors affecting the incidence of postoperative wound infection. *J Hosp Infect* 1990;16:223-30.
101. Doig CM, Wilkinson AW. Wound infection in a children's hospital. *Br J Surg* 1976;63:647-50.
102. Sharma LK, Sharma PK. Postoperative wound infection in a pediatric surgical service. *J Pediatr Surg* 1986;21:889-91.
103. Casey J, Flinn WR, Yao JS, Fahey V, Pawlowski J, Bergan JJ. Correlation of immune and nutritional status with wound complications in patients undergoing vascular operations. *Surgery* 1983;93(6):822-7.
104. Greene KA, Wilde AH, Stulberg BN. Preoperative nutritional status of total joint patients. Relationship to postoperative wound complications. *J Arthroplasty* 1991;6(4):321-5.
105. Weber TR. A prospective analysis of factors influencing outcome after fundoplication. *J Pediatr Surg* 1995;30(7):1061-3; discussion 1063-4.
106. Vamvakas EC, Carven JH. Transfusion of white-cell-containing allogeneic blood components and postoperative wound infection: effect of

confounding factors. *Transfus Med* 1998;8:29-36.
107. Vamvakas EC, Carven JH, Hibberd PL. Blood transfusion and infection after colorectal cancer surgery. *Transfusion* 1996;36:1000-8.
108. Jensen LS, Kissmeyer-Nielsen P, Wolff B, Qvist N. Randomised comparison of leucocyte-depleted versus buffy-coat-poor blood transfusion and complications after colorectal surgery. *Lancet* 1996;348:841-5.
109. Heiss MM, Mempel W, Jauch KW, Delanoff C, Mayer G, Mempel M, et al. Beneficial effect of autologous blood transfusion on infectious complications after colorectal cancer surgery. *Lancet* 1993;342:1328-33.
110. Lidgren L. Postoperative orthopaedic infections in patients with diabetes mellitus. *Acta Orthop Scand* 1973;44:149-51.
111. Gordon SM, Serkey JM, Barr C, Cosgrove D, Potts W. The relationship between glycosylated hemoglobin (HgA1c) levels and postoperative infections in patients undergoing primary coronary artery bypass surgery (CABG) [abstract]. *Infect Control Hosp Epidemiol* 1997; 18(No. 5, Part 2):29(58).
112. Zerr KJ, Furnary AP, Grunkemeier GL, Bookin S, Kanhere V, Starr A. Glucose control lowers the risk of wound infection in diabetics after open heart operations. *Ann Thorac Surg* 1997;63(2):356-61.
113. Terranova A. The effects of diabetes mellitus on wound healing. *Plast Surg Nurs* 1991;11(1):20-5.
114. Ziv Y, Church JM, Fazio VW, King TM, Lavery IC. Effect of systemic steroids on ileal pouch-anal anastomosis in patients with ulcerative colitis. *Dis Colon Rectum* 1996;39(5):504-8.
115. Pons VG, Denlinger SL, Guglielmo BJ, Octavio J, Flaherty J, Derish PA, et al. Ceftizoxime versus vancomycin and gentamicin in neurosurgical prophylaxis: a randomized, prospective, blinded clinical study. *Neurosurgery* 1993;33(3):416-22; discussion 422-3.
116. Brown IW Jr, Moor GF, Hummel BW, Marshall WG Jr, Collins JP. Toward further reducing wound infections in cardiac operations. *Ann Thorac Surg* 1996;62(6):1783-9.
117. Christou NV, Nohr CW, Meakins JL. Assessing operative site infection in surgical patients. *Arch Surg* 1987;122:165-9.
118. Hu SS, Fontaine F, Kelly B, Bradford DS. Nutritional depletion in staged spinal reconstructive surgery. The effect of total parenteral nutrition. *Spine* 1998;23:1401-5.
119. Schackert HK, Betzler M, Zimmermann GF, Decker R, Geelhaar H, Edler L, et al. The predictive role of delayed cutaneous hypersensitivity testing in postoperative complications. *Surg Gynecol Obstet* 1986;162:563-8.
120. Katelaris PH, Bennett GB, Smith RC. Prediction of postoperative complications by clinical and nutritional assessment. *Aust N Z J Surg* 1986;56:743-7.
121. Leite JF, Antunes CF, Monteiro JC, Pereira BT. Value of nutritional parameters in the prediction of postoperative complications in elective gastrointestinal surgery. *Br J Surg* 1987;74:426-9.
122. Mullen JL, Gertner MH, Buzby GP, Goodhart GL, Rosato EF. Implications of malnutrition in the surgical patient. *Arch Surg* 1979;114:121-5.
123. Buzby GP, Mullen JL, Matthews DC, Hobbs CL, Rosato EF. Prognostic nutritional index in gastrointestinal surgery. *Am J Surg* 1980;139:160-7.
124. Ellis LM, Copeland EM 3rd, Souba WW. Perioperative nutritional support. *Surg Clin North Am* 1991;71:493-507.
125. Haley RW, Culver DH, Morgan WM, White JW, Emori TG, Hooton TM. Identifying patients at high risk of surgical wound infection. A simple multivariate index of patient susceptibility and wound contamination. *Am J Epidemiol* 1985;121:206-15.
126. Culver DH, Horan TC, Gaynes RP, Martone WJ, Jarvis WR, Emori TG, et al. Surgical wound infection rates by wound class, operative procedure, and patient risk index. National Nosocomial Infections Surveillance System. *Am J Med* 1991;91(Suppl 3B):152S-7S.
127. Windsor JA, Hill GL. Weight loss with physiologic impairment. A basic indicator of surgical risk. *Ann Surg* 1988;207:290-6.
128. Campos AC, Meguid MM. A critical appraisal of the usefulness of perioperative nutritional support. *Am J Clin Nutr* 1992;55:117-30.
129. McPhee IB, Williams RP, Swanson CE. Factors influencing wound healing after surgery for metastatic disease of the spine. *Spine* 1998;23: 726-33; discussion 732-3.
130. Mullen JL, Buzby GP, Matthews DC, Smale BF, Rosato EF. Reduction of operative morbidity and mortality by combined preoperative and postoperative nutritional support. *Ann Surg* 1980;192:604-13.
131. Windsor JA, Hill GL. Protein depletion and surgical risk. *Aust N Z J Surg* 1988;58:711-5.
132. Ulicny KS Jr, Hiratzka LF, Williams RB, Grunkemeier GL, Flege JB Jr, Wright CB, et al. Sternotomy infection: poor prediction by acute phase response and delayed hypersensitivity. *Ann Thorac Surg* 1990;50:949-58.
133. Shukla HS, Rao RR, Banu N, Gupta RM, Yadav RC. Enteral hyperalimentation in malnourished surgical patients. *Indian J Med Res* 1984;80:339-46.
134. Moore EE, Jones TN. Benefits of immediate jejunostomy feeding after major abdominal trauma—a prospective, randomized study. *J Trauma* 1986;26:874-81.
135. Daly JM, Lieberman MD, Goldfine J, Shou J, Weintraub F, Rosato EF, et al. Enteral nutrition with supplemental arginine, RNA, and omega-3 fatty acids in patients after operation: immunologic, metabolic, and clinical outcome. *Surgery* 1992;112:56-67.
136. Daly JM, Weintraub FN, Shou J, Rosato EF, Lucia M. Enteral nutrition during multimodality therapy in upper gastrointestinal cancer patients. *Ann Surg* 1995;221:327-38.
137. Moore FA, Feliciano DV, Andrassy RJ, McArdle AH, Booth FV, Morgenstein-Wagner TB, et al. Early enteral feeding, compared with parenteral, reduces postoperative septic complications. The results of a meta-analysis. *Ann Surg* 1992;216:172-83.
138. Starker PM, Lasala PA, Askanazi J, Gump FE, Forse RA, Kinney JM. The response to TPN: a form of nutritional assessment. *Ann Surg* 1983;198:720-4.
139. Muller JM, Brenner U, Dienst C, Pichlmaier H. Preoperative parenteral feeding in patients with gastrointestinal carcinoma. *Lancet* 1982;1: 68-71.
140. Holter AR, Fischer JE. The effects of perioperative hyperalimentation on complications in patients with carcinoma and weight loss. *J Surg Res* 1977;23:31-4.
141. Thompson BR, Julian TB, Stremple JF. Perioperative total parenteral nutrition in patients with gastrointestinal cancer. *J Surg Res* 1981;30: 497-500.
142. Brennan MF, Pisters PW, Posner M, Quesada O, Shike M. A prospective randomized trial of total parenteral nutrition after major pancreatic resection for malignancy. *Ann Surg* 1994;220:436-41; discussion 441-4.
143. The Veterans Affairs Total Parenteral Nutrition Cooperative Study Group. Perioperative total parenteral nutrition in surgical patients. *N Engl J Med* 1991;325:525-32.
144. Starker PM, LaSala PA, Askanazi J, Todd G, Hensle TW, Kinney JM. The influence of preoperative total parenteral nutrition upon morbidity and mortality. *Surg Gynecol Obstet* 1986;162:569-74.
145. Senkal M, Mumme A, Eickhoff U, Geier B, Spath G, Wulfert D, et al. Early postoperative enteral immunonutrition: clinical outcome and cost-comparison analysis in surgical patients. *Crit Care Med* 1997;25:1489-96.
146. Heatley RV, Williams RH, Lewis MH. Pre-operative intravenous feeding—a controlled trial. *Postgrad Med J* 1979;55:541-5.
147. Muller JM, Keller HW, Brenner U, Walter M, Holzmuller W. Indications and effects of preoperative parenteral nutrition. *World J Surg* 1986;10: 53-63.
148. Daly JM, Massar E, Giacco G, Frazier OH, Mountain CF, Dudrick SJ, et al. Parenteral nutrition in esophageal cancer patients. *Ann Surg* 1982;196:203-8.
149. Klein JD, Hey LA, Yu CS, Klein BB, Coufal FJ, Young EP, et al. Perioperative nutrition and postoperative complications in patients undergoing spinal surgery. *Spine* 1996;21:2676-82.
150. Garibaldi RA, Cushing D, Lerer T. Risk factors for postoperative infection. *Am J Med* 1991;91(Suppl 3B):158S-63S.
151. Lee JT. Operative complications and quality improvement. *Am J Surg* 1996;171:545-7.
152. Kluytmans JA, Mouton JW, VandenBergh MF, Manders MJ, Maat AP, et al. Reduction of surgical-site infections in cardiothoracic surgery by elimination of nasal carriage of *Staphylococcus aureus. Infect Control Hosp Epidemiol* 1996;17:780-5.
153. Boyce JM. Preventing staphylococcal infections by eradicating nasal carriage of *Staphylococcus aureus:* proceeding with caution. *Infect Control Hosp Epidemiol* 1996;17:775-9.
154. Blajchman MA. Allogeneic blood transfusions, immunomodulation, and postoperative bacterial infection: do we have the answers yet? *Transfusion* 1997;37:121-5.
155. Garibaldi RA. Prevention of intraoperative wound contamination with chlorhexidine shower and scrub. *J Hosp Infect* 1988;11(Suppl B):5-9.
156. Paulson DS. Efficacy evaluation of a 4% chlorhexidine gluconate as a full-body shower wash. *Am J Infect Control* 1993;21(4):205-9.
157. Hayek LJ, Emerson JM, Gardner AM. A placebo-controlled trial of the effect of two preoperative baths or showers with chlorhexidine detergent on postoperative wound infection rates. *J Hosp Infect* 1987;10: 165-72.
158. Kaiser AB, Kernodle DS, Barg NL, Petracek MR. Influence of preoperative showers on staphylococcal skin colonization: a comparative trial of antiseptic skin cleansers. *Ann Thorac Surg* 1988;45:35-8.
159. Rotter ML, Larsen SO, Cooke EM, Dankert J, Daschner F, Greco D, et al. A comparison of the effects of preoperative whole-body bathing with detergent alone and with detergent containing chlorhexidine gluconate on the frequency of wound infections after clean surgery. The European Working Party on Control of Hospital Infections. *J Hosp Infect* 1988;11:310-20.
160. Leigh DA, Stronge JL, Marriner J, Sedgwick J. Total body bathing with 'Hibiscrub' (chlorhexidine) in surgical patients: a controlled trial. *J Hosp Infect* 1983;4:229-35.

161. Ayliffe GA, Noy MF, Babb JR, Davies JG, Jackson J. A comparison of pre-operative bathing with chlorhexidine-detergent and non-medicated soap in the prevention of wound infection. *J Hosp Infect* 1983;4:237-44.
162. Lynch W, Davey PG, Malek M, Byrne DJ, Napier A. Cost-effectiveness analysis of the use of chlorhexidine detergent in preoperative whole-body disinfection in wound infection prophylaxis. *J Hosp Infect* 1992;21:179-91.
163. Brady LM, Thomson M, Palmer MA, Harkness JL. Successful control of endemic MRSA in a cardiothoracic surgical unit. *Med J Aust* 1990;152:240-5.
164. Tuffnell DJ, Croton RS, Hemingway DM, Hartley MN, Wake PN, Garvey RJ. Methicillin-resistant *Staphylococcus aureus;* the role of antisepsis in the control of an outbreak. *J Hosp Infect* 1987;10:255-9.
165. Bartzokas CA, Paton JH, Gibson MF, Graham F, McLoughlin GA, Croton RS. Control and eradication of methicillin-resistant *Staphylococcus aureus* on a surgical unit. *N Engl J Med* 1984;311:1422-5.
166. Seropian R, Reynolds BM. Wound infections after preoperative depilatory versus razor preparation. *Am J Surg* 1971;121:251-4.
167. Hamilton HW, Hamilton KR, Lone FJ. Preoperative hair removal. *Can J Surg* 1977;20:269-71, 274-5.
168. Olson MM, MacCallum J, McQuarrie DG. Preoperative hair removal with clippers does not increase infection rate in clean surgical wounds. *Surg Gynecol Obstet* 1986;162:181-2.
169. Mehta G, Prakash B, Karmoker S. Computer assisted analysis of wound infection in neurosurgery. *J Hosp Infect* 1988;11:244-52.
170. Alexander JW, Fischer JE, Boyajian M, Palmquist J, Morris MJ. The influence of hair-removal methods on wound infections. *Arch Surg* 1983;118(3):347-52.
171. Masterson TM, Rodeheaver GT, Morgan RF, Edlich RF. Bacteriologic evaluation of electric clippers for surgical hair removal. *Am J Surg* 1984;148:301-2.
172. Sellick JA Jr., Stelmach M, Mylotte JM. Surveillance of surgical wound infections following open heart surgery. *Infect Control Hosp Epidemiol* 1991;12(10):591-6.
173. Ko W, Lazenby WD, Zelano JA, Isom W, Krieger KH. Effects of shaving methods and intraoperative irrigation on suppurative mediastinitis after bypass operations. *Ann Thorac Surg* 1992;53:301-5.
174. Moro ML, Carrieri MP, Tozzi AE, Lana S, Greco D. Risk factors for surgical wound infections in clean surgery: a multicenter study. Italian PRINOS Study Group. *Ann Ital Chir* 1996;67:13-9.
175. Winston KR. Hair and neurosurgery. *Neurosurgery* 1992;31(2):320-9.
176. Larson E. Guideline for use of topical antimicrobial agents. *Am J Infect Control* 1988;16:253-66.
177. Hardin WD, Nichols RL. Handwashing and patient skin preparation. In: Malangoni MA, ed. *Critical Issues in Operating Room Management.* Philadelphia: Lippincott-Raven; 1997. p. 133-49.
178. Ritter MA, French ML, Eitzen HE, Gioe TJ. The antimicrobial effectiveness of operative-site preparative agents: a microbiological and clinical study. *J Bone Joint Surg Am* 1980;62(5):826-8.
179. Mayhall CG. Surgical infections including burns. In: Wenzel RP, ed. *Prevention and Control of Nosocomial Infections.* 2nd ed. Baltimore: Williams & Wilkins; 1993. p. 614-64.
180. Committee on Control of Surgical Infections of the Committee on Pre- and Postoperative Care, American College of Surgeons. *Manual on Control of Infection in Surgical Patients.* Philadelphia: J.B. Lippincott Co; 1984.
181. Hardin WD, Nichols RL. Aseptic technique in the operating room. In: Fry DE, ed. *Surgical Infections.* Boston: Little, Brown and Co; 1995. p. 109-18.
182. Lowbury EJ, Lilly HA. Use of 4 percent chlorhexidine detergent solution (Hibiscrub) and other methods of skin disinfection. *Br Med J* 1973;1:510-5.
183. Aly R, Maibach HI. Comparative antibacterial efficacy of a 2-minute surgical scrub with chlorhexidine gluconate, povidone-iodine, and chloroxylenol sponge-brushes. *Am J Infect Control* 1988;16:173-7.
184. Peterson AF, Rosenberg A, Alatary SD. Comparative evaluation of surgical scrub preparations. *Surg Gynecol Obstet* 1978;146:63-5.
185. Brown TR, Ehrlich CE, Stehman FB, Golichowski AM, Madura JA, Eitzen HE. A clinical evaluation of chlorhexidine gluconate spray as compared with iodophor scrub for preoperative skin preparation. *Surg Gynecol Obstet* 1984;158:363-6.
186. Lowbury EJ, Lilly HA. The effect of blood on disinfection of surgeons' hands. *Br J Surg* 1974;61:19-21.
187. Association of Operating Room Nurses. Recommended practices for skin preparation of patients. *AORN J* 1996;64(5):813-6.
188. Kutarski PW, Grundy HC. To dry or not to dry? An assessment of the possible degradation in efficiency of preoperative skin preparation caused by wiping skin dry. *Ann R Coll Surg Engl* 1993;75(3):181-5.
189. Gauthier DK, O'Fallon PT, Coppage D. Clean vs sterile surgical skin preparation kits. Cost, safety, effectiveness. *AORN J* 1993;58(3):486-95.
190. Hagen KS, Treston-Aurand J. A comparison of two skin preps used in cardiac surgical procedures. *AORN J* 1995;62(3):393-402.
191. Shirahatti RG, Joshi RM, Vishwanath YK, Shinkre N, Rao S, Sankpal JS, et al. Effect of pre-operative skin preparation on post-operative wound infection. *J Postgrad Med* 1993;39(3):134-6.
192. Larson EL, Butz AM, Gullette DL, Laughon BA. Alcohol for surgical scrubbing? *Infect Control Hosp Epidemiol* 1990;11(3):139-43.
193. Faoagali J, Fong J, George N, Mahoney P, O'Rouke V. Comparison of the immediate, residual, and cumulative antibacterial effects of Novaderm R*, Novascrub R*, Betadine Surgical Scrub, Hibiclens, and liquid soap. *Am J Infect Control* 1995;23(6):337-43.
194. Larson EL. APIC guideline for handwashing and hand antisepsis in health care settings. *Am J Infect Control* 1995;23:251-69.
195. Rubio PA. Septisol antiseptic foam: a sensible alternative to the conventional surgical scrub. *Int Surg* 1987;72(4):243-6.
196. Lowbury EJ, Lilly HA, Ayliffe GA. Preoperative disinfection of surgeons' hands: use of alcoholic solutions and effects of gloves on skin flora. *Br Med J* 1974;4:369-72.
197. Rotter ML. Hygienic hand disinfection. *Infect Control* 1984;5:18-22.
198. Ayliffe GA. Surgical scrub and skin disinfection. *Infect Control* 1984;5:23-7.
199. Lilly HA, Lowbury EJ, Wilkins MD, Zaggy A. Delayed antimicrobial effects of skin disinfection by alcohol. *J Hyg (Lond)* 1979;82:497-500.
200. Nichols RL, Smith JW, Garcia RY, Waterman RS, Holmes JW. Current practices of preoperative bowel preparation among North American colorectal surgeons. *Clin Infect Dis* 1997;24:609-19.
201. Wade JJ, Casewell MW. The evaluation of residual antimicrobial activity on hands and its clinical relevance. *J Hosp Infect* 1991;18(Suppl B):23-8.
202. Babb JR, Davies JG, Ayliffe GA. A test procedure for evaluating surgical hand disinfection. *J Hosp Infect* 1991;18(Suppl B):41-9.
203. Holloway PM, Platt JH, Reybrouck G, Lilly HA, Mehtar S, Drabu Y. A multi-centre evaluation of two chlorhexidine-containing formulations for surgical hand disinfection. *J Hosp Infect* 1990;16:151-9.
204. Kobayashi H. Evaluation of surgical scrubbing. *J Hosp Infect* 1991;18(Suppl B):29-34.
205. Nicoletti G, Boghossian V, Borland R. Hygienic hand disinfection: a comparative study with chlorhexidine detergents and soap. *J Hosp Infect* 1990;15:323-37.
206. Rotter ML, Koller W. Surgical hand disinfection: effect of sequential use of two chlorhexidine preparations. *J Hosp Infect* 1990;16:161-6.
207. O'Shaughnessy M, O'Malley VP, Corbett G, Given HF. Optimum duration of surgical scrub-time. *Br J Surg* 1991;78(6):685-6.
208. Hingst V, Juditzki I, Heeg P, Sonntag HG. Evaluation of the efficacy of surgical hand disinfection following a reduced application time of 3 instead of 5 min. *J Hosp Infect* 1992;20(2):79-86.
209. Wheelock SM, Lookinland S. Effect of surgical hand scrub time on subsequent bacterial growth. *AORN J* 1997;65:1087-92, 1094-8.
210. Deshmukh N, Kramer JW, Kjellberg SI. A comparison of 5-minute povidone-iodine scrub and 1-minute povidone-iodine scrub followed by alcohol foam. *Mil Med* 1998;163:145-7.
211. Masterson BJ. Cleansing the surgeon's hands. *Scientific American Surgeon* 1996;2:3-9.
212. Association of Operating Room Nurses. *Standards, Recommended Practices, Guidelines.* Denver: Association of Operating Room Nurses; 1999.
213. Pottinger J, Burns S, Manske C. Bacterial carriage by artificial versus natural nails. *Am J Infect Control* 1989;17:340-4.
214. Passaro DJ, Waring L, Armstrong R, Bolding F, Bouvier B, Rosenberg J, et al. Postoperative *Serratia marcescens* wound infections traced to an out-of-hospital source. *J Infect Dis* 1997;175(4):992-5.
215. Baumgardner CA, Maragos CS, Walz J, Larson E. Effects of nail polish on microbial growth of fingernails. Dispelling sacred cows. *AORN J* 1993;58:84-8.
216. Jacobson G, Thiele JE, McCune JH, Farrell LD. Handwashing: ring-wearing and number of microorganisms. *Nurs Res* 1985;34:186-8.
217. Hoffman PN, Cooke EM, McCarville MR, Emmerson AM. Microorganisms isolated from skin under wedding rings worn by hospital staff. *Br Med J (Clin Res Ed)* 1985;290:206-7.
218. Richet HM, Chidiac C, Prat A, Pol A, David M, Maccario M, et al. Analysis of risk factors for surgical wound infections following vascular surgery. *Am J Med* 1991;91(Suppl 3B):170S-172S.
219. Centers for Disease Control. Epidemic keratoconjunctivitis in an ophthalmology clinic—California. *MMWR* 1990;39:598-601.
220. Ford E, Nelson KE, Warren D. Epidemiology of epidemic keratoconjunctivitis. *Epidemiol Rev* 1987;9:244-61.
221. Birenbaum E, Linder N, Varsano N, Azar R, Kuint J, Spierer A, et al. Adenovirus type 8 conjunctivitis outbreak in a neonatal intensive care unit. *Arch Dis Child* 1993;68(5 Spec No):610-1.
222. Warren D, Nelson KE, Farrar JA, Hurwitz E, Hierholzer J, Ford E, et al. A large outbreak of epidemic keratoconjunctivitis: problems in controlling nosocomial spread. *J Infect Dis* 1989;160:938-43.
223. Jernigan JA, Lowry BS, Hayden FG, Kyger SA, Conway BP, Groschel

DH, et al. Adenovirus type 8 epidemic keratoconjunctivitis in an eye clinic: risk factors and control. *J Infect Dis* 1993;167:1307-13.
224. Boyce JM. Should we vigorously try to contain and control methicillin-resistant *Staphylococcus aureus? Infect Control Hosp Epidemiol* 1991;12:46-54.
225. Boyce JM, Opal SM, Potter-Bynoe G, Medeiros AA. Spread of methicillin-resistant *Staphylococcus aureus* in a hospital after exposure to a health care worker with chronic sinusitis. *Clin Infect Dis* 1993;17: 496-504.
226. Sheretz RJ, Reagan DR, Hampton KD, Robertson KL, Streed SA, Hoen HM, et al. A cloud adult: the *Staphylococcus aureus*-virus interaction revisited. *Ann Intern Med* 1996;124:539-47.
227. Belani A, Sherertz RJ, Sullivan ML, Russell BA, Reumen PD. Outbreak of staphylococcal infection in two hospital nurseries traced to a single nasal carrier. *Infect Control* 1986;7:487-90.
228. Kreiswirth BN, Kravitz GR, Schlievert PM, Novick RP. Nosocomial transmission of a strain of *Staphylococcus aureus* causing toxic shock syndrome. *Ann Intern Med* 1986;105:704-7.
229. Weber DJ, Rutala WA, Denny FW Jr. Management of healthcare workers with pharyngitis or suspected streptococcal infections. *Infect Control Hosp Epidemiol* 1996;17:753-61.
230. Viglionese A, Nottebart VF, Bodman HA, Platt R. Recurrent group A streptococcal carriage in a health care worker associated with widely separated nosocomial outbreaks. *Am J Med* 1991;91(3B):329S-33S.
231. Paul SM, Genese C, Spitalny K. Postoperative group A beta-hemolytic streptococcus outbreak with the pathogen traced to a member of a healthcare worker's household. *Infect Control Hosp Epidemiol* 1990;11:643-6.
232. Ridgway EJ, Allen KD. Clustering of group A streptococcal infections on a burns unit: important lessons in outbreak management. *J Hosp Infect* 1993;25:173-82.
233. Berkelman RL, Martin D, Graham DR, Mowry J, Freisem R, Weber JA, et al. Streptococcal wound infection caused by a vaginal carrier. *JAMA* 1982;247:2680-2.
234. Schaffner W, Lefkowitz LB Jr., Goodman JS, Koenig MG. Hospital outbreak of infections with group A streptococci traced to an asymptomatic anal carrier. *N Engl J Med* 1969;280:1224-5.
235. Richman DD, Breton SJ, Goldman DA. Scarlet fever and group A streptococcal surgical wound infection traced to an anal carrier. *J Pediatr* 1977;90:387-90.
236. Stromberg A, Schwan A, Cars O. Throat carrier rates of beta-hemolytic streptococci among healthy adults and children. *Scand J Infect Dis* 1988;20:411-7.
237. Stamm WE, Feeley JC, Facklam RR. Wound infection due to group A streptococcus traced to a vaginal carrier. *J Infect Dis* 1978;138:287-92.
238. Bolyard EA, Tablan OC, Williams WW, Pearson ML, Shapiro CN, Deitchman SD, et al. Guideline for infection control in healthcare personnel, 1998. Hospital Infection Control Practices Advisory Committee. *Am J Infect Control* 1998;26:289-354.
239. Nichols RL, Holmes JW. Prophylaxis in bowel surgery. *Curr Clin Top Infect Dis* 1995;15:76-96.
240. Nichols RL, Smith JW, Muzik AC, Love EJ, McSwain NE, Timberlake G, et al. Preventive antibiotic usage in traumatic thoracic injuries requiring closed tube thoracostomy. *Chest* 1994;106(5):1493-8.
241. Bullock R, van Dellen JR, Ketelbey W, Reinach SG. A double-blind placebo-controlled trial of perioperative prophylactic antibiotics for elective neurosurgery. *J Neurosurg* 1988;69(5):687-91.
242. Page CP, Bohnen JM, Fletcher JR, McManus AT, Solomkin JS, Wittmann DH. Antimicrobial prophylaxis for surgical wounds. Guidelines for clinical care. *Arch Surg* 1993;128(1):79-88.
243. McQueen M, Littlejohn A, Hughes SP. A comparison of systemic cefuroxime and cefuroxime loaded bone cement in the prevention of early infection after total joint replacement. *Int Orthop* 1987;11:241-3.
244. Henley MB, Jones RE, Wyatt RWB, Hofmann A, Cohen RL. Prophylaxis with cefamandole nafate in elective orthopedic surgery. *Clin Orthop* 1986;209:249-54.
245. Buckley R, Hughes GN, Snodgrass T, Huchcroft SA. Perioperative cefazolin prophylaxis in hip fracture surgery. *Can J Surg* 1990;33:122-7.
246. Boyd RJ, Burke JF, Colton T. A double-blind clinical trial of prophylactic antibiotic in hip fractures. *J Bone Joint Surg [Am]* 1973;55A:1251-8.
247. Bernard A, Pillet M, Goudet P, Viard H. Antibiotic prophylaxis in pulmonary surgery. A prospective randomized double-blind trial of flash cefuroxime versus forty-eight-hour cefuroxime. *J Thorac Cardiovasc Surg* 1994;107(3):896-900.
248. Platt R, Zucker JR, Zaleznik DF, Hopkins CC, Dellinger EP, Karchmer AW, et al. Perioperative antibiotic prophylaxis and wound infection following breast surgery. *J Antimicrob Chemother* 1993;31(Suppl B):43-8.
249. Rimoldi RL, Haye W. The use of antibiotics for wound prophylaxis in spinal surgery. *Orthop Clin North Am* 1996;27(1):47-52.
250. Bennion RS, Hiatt JR, Williams RA, Wilson SE. A randomized, prospective study of perioperative antimicrobial prophylaxis for vascular access surgery. *J Cardiovasc Surg (Torino)* 1985;26:270-4.
251. Kaiser AB, Petracek MR, Lea JW 4th, Kernodle DS, Roach AC, Alford WC Jr, et al. Efficacy of cefazolin, cefamandole, and gentamicin as prophylactic agents in cardiac surgery. Results of a prospective, randomized, double-blind trial in 1030 patients. *Ann Surg* 1987;206(6):791-7.
252. Miedzinski LJ, Callaghan JC, Fanning EA, Gelfand ET, Goldsand G, Modry D, et al. Antimicrobial prophylaxis for open heart operations. *Ann Thorac Surg* 1990;50:800-7.
253. Doebbeling BN, Pfaller MA, Kuhns KR, Massanari RM, Behrendt DM, Wenzel RP. Cardiovascular surgery prophylaxis. A randomized, controlled comparison of cefazolin and cefuroxime. *J Thorac Cardiovasc Surg* 1990;99:981-9.
254. Madsen MS, Neumann L, Andersen JA. Penicillin prophylaxis in complicated wounds of hands and feet: a randomized, double-blind trial. *Injury* 1996;27(4):275-8.
255. Burnett JW, Gustilo RB, Williams DN, Kind AC. Prophylactic antibiotics in hip fractures. A double-blind, prospective study. *J Bone Joint Surg [Am]*1980;62(3):457-62.
256. Nichols RL, Webb WR, Jones JW, Smith JW, LoCicero J 3rd. Efficacy of antibiotic prophylaxis in high risk gastroduodenal operations. *Am J Surg* 1982;143:94-8.
257. Lewis RT, Goodall RG, Marien B, Park M, Lloyd-Smith W, Wiegand FM. Efficacy and distribution of single-dose preoperative antibiotic prophylaxis in high-risk gastroduodenal surgery. *Can J Surg* 1991;34:117-22.
258. Young RF, Lawner PM. Perioperative antibiotic prophylaxis for prevention of postoperative neurosurgical infections. A randomized clinical trial. *J Neurosurg* 1987;66:701-5.
259. Djindjian M, Lepresle E, Homs JB. Antibiotic prophylaxis during prolonged clean neurosurgery. Results of a randomized double-blind study using oxacillin. *J Neurosurg* 1990;73:383-6.
260. Targarona EM, Garau J, Munoz-Ramos C, Roset F, Lite J, Matas E, et al. Single-dose antibiotic prophylaxis in patients at high risk for infection in biliary surgery: a prospective and randomized study comparing cefonicid with mezlocillin. *Surgery* 1990;107:327-34.
261. van Ek B, Dijkmans BA, van Dulken H, van Furth R. Antibiotic prophylaxis in craniotomy: a prospective double-blind placebo-controlled study. *Scand J Infect Dis* 1988;20:633-9.
262. Krige JE, Isaacs S, Stapleton GN, McNally J. Prospective, randomized study comparing amoxycillin-clavulanic acid and cefamandole for the prevention of wound infection in high-risk patients undergoing elective biliary surgery. *J Hosp Infect* 1992;22 (Suppl A):33-41.
263. Browder W, Smith JW, Vivoda LM, Nicholas RL. Nonperforative appendicitis: a continuing surgical dilemma. *J Infect Dis* 1989;159(6):1088-94.
264. Platt R. Guidelines for perioperative antibiotic prophylaxis. In: Abrutyn E, Goldmann DA, Scheckler WE, eds. *Saunders Infection Control Reference Service.* Philadelphia: W.B. Saunders Co; 1997. p. 229-34.
265. Sanderson PJ. Antimicrobial prophylaxis in surgery: microbiological factors. *J Antimicrob Chemother* 1993;31(Suppl B):1-9.
266. Anonymous. Antimicrobial prophylaxis in surgery. *Med Lett Drugs Ther* 1997;39(1012):97-102.
267. Scher KS. Studies on the duration of antibiotic administration for surgical prophylaxis. *Am Surg* 1997;63:59-62.
268. Nichols RL. Antibiotic prophylaxis in surgery. *J Chemother* 1989;1(3):170-8.
269. Ehrenkranz NJ. Antimicrobial prophylaxis in surgery: mechanisms, misconceptions, and mischief. *Infect Control Hosp Epidemiol* 1993;14(2):99-106.
270. Berkeley AS, Freedman KS, Ledger WJ, Orr JW, Benigno BB, Gordon SF, et al. Comparison of cefotetan and cefoxitin prophylaxis for abdominal and vaginal hysterectomy. *Am J Obstet Gynecol* 1988;158: 706-9.
271. Ehrenkranz NJ, Blackwelder WC, Pfaff SJ, Poppe D, Yerg DE, Kaslow RA. Infections complicating low-risk cesarean sections in community hospitals: efficacy of antimicrobial prophylaxis. *Am J Obstet Gynecol* 1990;162(2):337-43.
272. Soper DE. Infections following cesarean section. *Curr Opin Obstet Gynecol* 1993;5(4):517-20.
273. Enkin M, Enkin E, Chalmers I, Hemminki E. Prophylactic antibiotics in association with caesarean section. In: Chalmers I, Enkin M, Keirse MJ, eds. *Effective Care in Pregnancy and Childbirth.* London: Oxford University; 1989. p. 1246-69.
274. Allen JL, Rampon JF, Wheeless CR. Use of a prophylactic antibiotic in elective major gynecologic operations. *Obstet Gynecol* 1972;39:218-24.
275. The Multicenter Study Group. Single dose prophylaxis in patients undergoing vaginal hysterectomy: cefamandole versus cefotaxime. *Am J Obstet Gynecol* 1989;160:1198-201.
276. Roy S, Wilkins J, Galaif E, Azen C. Comparative efficacy and safety of cefmetazole or cefoxitin in the prevention of postoperative infection following vaginal and abdominal hysterectomy. *J Antimicrob Chemother* 1989;23(Suppl D):109-17.
277. Friese S, Willems FT, Loriaux SM, Meewis JM. Prophylaxis in gynae-

cological surgery: a prospective randomized comparison between single dose prophylaxis with amoxycillin/clavulanate and the combination of cefuroxime and metronidazole. *J Antimicrob Chemother* 1989;24(Suppl B):213-6.
278. Senior CC, Steigrad SJ. Are preoperative antibiotics helpful in abdominal hysterectomy? *Am J Obstet Gynecol* 1986;154:1004-8.
279. Hemsell DL, Martin JN Jr, Pastorek JG 2d, Nobles BJ, Hemsell PG, Helman N, et al. Single dose antimicrobial prophylaxis at abdominal hysterectomy. Cefamandole vs. cefotaxime. *J Reprod Med* 1988;33: 939-44.
280. Hemsell DL, Hemsell PG, Heard ML, Nobles BJ. Preoperative cefoxitin prophylaxis for elective abdominal hysterectomy. *Am J Obstet Gynecol* 1985;153:225-6.
281. DiPiro JT, Cheung RP, Bowden TA Jr, Mansberger JA. Single dose systemic antibiotic prophylaxis of surgical wound infections. *Am J Surg* 1986;152:552-9.
282. Trilla A, Mensa J. Perioperative antibiotic prophylaxis. In: Wenzel RP, ed. *Prevention and Control of Nosocomial Infections.* 2nd ed. Baltimore: Williams & Wilkins; 1993. p. 665-82.
283. Ehrenkranz NJ, Meakins JL. Surgical infections. In: Bennett JV, Brachman PS, eds. *Hospital Infections.* 3rd ed. Boston: Little, Brown and Co; 1992. p. 685-710.
284. Nichols RL. Surgical antibiotic prophylaxis. *Med Clin North Am* 1995;79(3):509-22.
285. Classen DC, Evans RS, Pestotnik SL, Horn SD, Menlove RL, Burke JP. The timing of prophylactic administration of antibiotics and the risk of surgical-wound infection. *N Engl J Med* 1992;326(5):281-6.
286. McDonald M, Grabsch E, Marshall C, Forbes A. Single- versus multiple-dose antimicrobial prophylaxis for major surgery: a systematic review. *Aust N Z J Surg* 1998;68:388-96.
287. Clarke JS, Condon RE, Bartlett JG, Gorbach SL, Nichols RL, Ochi S. Preoperative oral antibiotics reduce septic complications of colon operations: results of prospective, randomized, double-blind clinical study. *Ann Surg* 1977;186:251-9.
288. Condon RE, Bartlett JG, Greenlee H, Schulte WJ, Ochi S, Abbe R, et al. Efficacy of oral and systemic antibiotic prophylaxis in colorectal operations. *Arch Surg* 1983;118:496-502.
289. Bartlett JG, Condon RE, Gorbach SL, Clarke JS, Nichols RL, Ochi S. Veterans Administration Cooperative Study on bowel preparation for elective colorectal operation: impact of oral antibiotic regimen on colonic flora, wound irrigation cultures and bacteriology of septic complications. *Ann Surg* 1978;188:249-54.
290. Da Costa A, Kirkorian G, Cucherat M, Delahaye F, Chevalier P, Cerisier A, et al. Antibiotic prophylaxis for permanent pacemaker implantation: a meta-analysis. *Circulation* 1998;97:1796-801.
291. Bumpous JM, Johnson JT. The infected wound and its management. *Otolaryngol Clin North Am* 1995;28(5):987-1001.
292. Hospital Infection Control Practices Advisory Committee. Recommendations for preventing the spread of vancomycin resistance. *Infect Control Hosp Epidemiol* 1995;16(2):105-13.
293. Forse RA, Karam B, MacLean LD, Christou NV. Antibiotic prophylaxis for surgery in morbidly obese patients. *Surgery* 1989;106:750-7.
294. Polk HC, Wilson MA. Systemic antibiotic prophylaxis in surgery. In: Fry DE, ed. *Surgical Infections.* Boston: Little, Brown and Co; 1995. p. 127-33.
295. Ayliffe GA. Role of the environment of the operating suite in surgical wound infection. *Rev Infect Dis* 1991;13(Suppl 10):S800-4.
296. Gryska PF, O'Dea AE. Postoperative streptococcal wound infection. The anatomy of an epidemic. *JAMA* 1970;213:1189-91.
297. McIntyre DM. An epidemic of *Streptococcus pyogenes* puerperal and postoperative sepsis with an unusual carrier site—the anus. *Am J Obstet Gynecol* 1968;101:308-14.
298. Lidwell OM. Clean air at operation and subsequent sepsis in the joint. *Clin Orthop* 1986;211:91-102.
299. American Institute of Architects. *Guidelines for design and construction of hospital and health care facilities.* Washington (DC): American Institiute of Architects Press; 1996.
300. Nichols RL. The operating room. In: Bennett JV, Brachman PS, eds. *Hospital Infections.* 3rd ed. Boston: Little, Brown and Co; 1992. p. 461-73.
301. Laufman H. The operating room. In: Bennett JV, Brachman PS, eds. *Hospital Infections.* 2nd ed. Boston: Little, Brown and Co; 1986. p. 315-23.
302. Sessler DI, McGuire J, Hynson J, Moayeri A, Heier T. Thermoregulatory vasoconstriction during isoflurane anesthesia minimally decreases cutaneous heat loss. *Anesthesiology* 1992;76:670-5.
303. Hambraeus A. Aerobiology in the operating room—a review. *J Hosp Infect* 1988;11(Suppl A):68-76
304. Babb JR, Lynam P, Ayliffe GA. Risk of airborne transmission in an operating theatre containing four ultraclean air units. *J Hosp Infect* 1995;31(3):159-68.
305. Charnley J. Post-operative infection after total hip replacement with special reference to contamination in the operating room. Internal Publication 38, Centre for Hip Surgery, Wrightington Hospital, Wigan, Lancs., UK; 1972.
306. Friberg B. Ultraclean laminar airflow ORs. *AORN J* 1998;67:841-51.
307. Lidwell OM, Elson RA, Lowbury EJ, Whyte W, Blowers R, Stanley SJ, et al. Ultraclean air and antibiotics for prevention of postoperative infection. A multicenter study of 8,052 joint replacement operations. *Acta Orthop Scand* 1987;58:4-13.
308. Nelson JP. The operating room environment and its influence on deep wound infection. *J Bone Joint Surg* 1976;1-11.
309. Whyte W. Infection control in hospital operating rooms. *Cleanrooms 1993 Proceedings* 1993;157-66.
310. Charnley J. A clean-air operating enclosure. *Br J Surg* 1964;51:202-5.
311. Lidwell OM, Lowbury EJ, Whyte W, Blowers R, Stanley SJ, Lowe D. Effect of ultraclean air in operating rooms on deep sepsis in the joint after total hip or knee replacement: a randomised study. *Br Med J* 1982;285:10-4.
312. Taylor GJ, Bannister GC, Leeming JP. Wound disinfection with ultraviolet radiation. *J Hosp Infect* 1995;30(2):85-93.
313. Bueno Cavanillas A, Rodriguez-Contreras R, Delgado Rodriguez M, Moreno Abril O, Lopez Gigosos R, Guillen Solvas J, et al. Preoperative stay as a risk factor for nosocomial infection. *Eur J Epidemiol* 1991;7(6):670-6.
314. Favero MS, Bond W. Sterilization, disinfection, and antisepsis in the hospital. In: Balows A, Hausler WJ Jr, Herrmann KL, Isenberg HD, Shadomy HJ, eds. *Manual of Clinical Microbiology.* 5th ed. Washington (DC): American Society of Microbiology; 1991. p. 183-200.
315. U.S. Department of Labor, Occupational Safety and Health Administration. Occupational exposure to bloodborne pathogens; final rule (29 CFR Part 1910.1030). *Federal Register* 1991;56:64004-182.
316. Rudnick JR, Beck-Sague CM, Anderson RL, Schable B, Miller JM, Jarvis WR. Gram-negative bacteremia in open-heart-surgery patients traced to probable tap-water contamination of pressure-monitoring equipment. *Infect Control Hosp Epidemiol* 1996;17(5):281-5.
317. Centers for Disease Control. Postsurgical infections associated with nonsterile implantable devices. *MMWR Morb Mortal Wkly Rep* 1992;41(15):263.
318. Soto LE, Bobadilla M, Villalobos Y, Sifuentes J, Avelar J, Arrieta M, et al. Post-surgical nasal cellulitis outbreak due to *Mycobacterium chelonae. J Hosp Infect* 1991;19(2):99-106.
319. Favero MS, Manian FA. Is eliminating flash sterilization practical? *Infect Control Hosp Epidemiol* 1993;14(8):479-80.
320. American Society for Healthcare Central Service Personnel. Recommended practices for central service. Section one, continuous quality improvement. Chicago: American Hospital Association; 1993.
321. Association for the Advancement of Medical Instrumentation. Flash sterilization: steam sterilization of patient care items for immediate use (ANSI/AAMI ST37-1996). Arlington (VA): Association for the Advancement of Medical Instrumentation; 1996.
322. Rutala WA, Gergen MF, Weber DJ. Evaluation of a rapid readout biological indicator for flash sterilization with three biological indicators and three chemical indicators. *Infect Control Hosp Epidemiol* 1993;14(7):390-4.
323. Vesley D, Langholz AC, Rohlfing SR, Foltz WE. Fluorimetric detection of a *Bacillus stearothermophilus* spore-bound enzyme, alpha-D-glucosidase, for rapid indication of flash sterilization failure. *Appl Environ Microbiol* 1992;58:717-9.
324. Rutala WA, Jones SM, Weber DJ. Comparison of a rapid readout biological indicator for steam sterilization with four conventional biological indicators and five chemical indicators. *Infect Control Hosp Epidemiol* 1996;17:423-8.
325. Vesley D, Nellis MA, Allwood PB. Evaluation of a rapid readout biological indicator for 121 degrees C gravity and 132 degrees C vacuum-assisted steam sterilization cycles. *Infect Control Hosp Epidemiol* 1995;16:281-6.
326. Smith RF. What is the purpose of the scrub suit? [letter]. *AORN J* 1980;31(5):769.
327. Dineen P. The role of impervious drapes and gowns preventing surgical infection. *Clin Orthop* 1973;96:210-2.
328. Ha'eri GB, Wiley AM. The efficacy of standard surgical face masks: an investigation using "tracer particles." *Clin Orthop* 1980;148:160-2.
329. Moylan JA, Fitzpatrick KT, Davenport KE. Reducing wound infections. Improved gown and drape barrier performance. *Arch Surg* 1987;122:152-7.
330. Moylan JA, Balish E, Chan J. Intraoperative bacterial transmission. *Surg Forum* 1974;25:29-30.
331. Copp G, Mailhot CB, Zalar M, Slezak L, Copp AJ. Covergowns and the control of operating room contamination. *Nurs Res* 1986;35:263-8.
332. Beck WC. The surgical mask: another 'sacred cow'? *AORN J* 1992;55:955-7.

333. Mitchell NJ, Hunt S. Surgical face masks in modern operating rooms—a costly and unnecessary ritual? *J Hosp Infect* 1991;18:239-42.
334. Tunevall TG, Jorbeck H. Influence of wearing masks on the density of airborne bacteria in the vicinity of the surgical wound. *Eur J Surg* 1992;158(5):263-6.
335. Tunevall TG. Postoperative wound infections and surgical face masks: a controlled study. *World J Surg* 1991;15:383-8.
336. Orr NW. Is a mask necessary in the operating theatre? *Ann R Coll Surg Engl* 1981;63(6):390-2.
337. Lee JT. Making surgical care better: hard work, small gains [editorial]. *Infect Control Hosp Epidemiol* 1997;18:6-8.
338. Jarvis WR, Bolyard EA, Bozzi CJ, Burwen DR, Dooley SW, Martin LS, et al. Respirators, recommendations, and regulations: the controversy surrounding protection of health care workers from tuberculosis. *Ann Intern Med* 1995;122:142-6.
339. Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health. Protect yourself against tuberculosis: a respiratory protection guide for health care workers (Publication No. 96-102). Cincinnati: Department of Health and Human Services (NIOSH); 1995.
340. Humphreys H, Marshall RJ, Ricketts VE, Russell AJ, Reeves DS. Theatre over-shoes do not reduce operating theatre floor bacterial counts. *J Hosp Infect* 1991;17:117-23.
341. Weightman NC, Banfield KR. Protective over-shoes are unnecessary in a day surgery unit. *J Hosp Infect* 1994;28:1-3.
342. Whyte W, Hambraeus A, Laurell G, Hoborn J. The relative importance of routes and sources of wound contamination during general surgery. I. Non-airborne. *J Hosp Infect* 1991;18(2):93-107.
343. Dodds RD, Guy PJ, Peacock AM, Duffy SR, Barker SG, Thomas MH. Surgical glove perforation. *Br J Surg* 1988;75(10):966-8.
344. Tokars JI, Culver DH, Mendelson MH, Sloan EP, Farber BF, Fligner DJ, et al. Skin and mucous membrane contacts with blood during surgical procedures: risk and prevention. *Infect Control Hosp Epidemiol* 1995;16:703-11.
345. Short LJ, Bell DM. Risk of occupational infection with blood-borne pathogens in operating and delivery room settings. *Am J Infect Control* 1993;21:343-50.
346. Garibaldi RA, Maglio S, Lerer T, Becker D, Lyons R. Comparison of nonwoven and woven gown and drape fabric to prevent intraoperative wound contamination and postoperative infection. *Am J Surg* 1986;152(5):505-9.
347. Muller W, Jiru P, Mach R, Polaschek F, Fasching W. The use of disposable draping materials in the operating room and its effect on the postoperative wound infection rate. *Wein Klin Wochenschr* 1989;101:837-42.
348. Smith JW, Nichols RL. Barrier efficiency of surgical gowns. Are we really protected from our patients' pathogens? *Arch Surg* 1991;126(6):756-63.
349. Artz CP, Conn JH, Howard HS. Protection of the surgical wound with a new plastic film. *JAMA* 1960;174(14):1865-8.
350. Chiu KY, Lau SK, Fung B, Ng KH, Chow SP. Plastic adhesive drapes and wound infection after hip fracture surgery. *Aust N Z J Surg* 1993;63(10):798-801.
351. American Society for Testing Materials. *Standard Test Method for Resistance of Materials Used in Protective Clothing to Penetration by Synthetic Blood.* American Society for Testing Materials 1998;F1670-98.
352. American Society for Testing Materials. *Standard Test Method for Resistance of Materials Used in Protective Clothing to Penetration by Blood-borne Pathogens Using Phi-X174 Bacteriophage Penetration as a Test System.* American Society for Testing Materials 1997; F1671-976.
353. McCullough EA. Methods for determining the barrier efficacy of surgical gowns. *Am J Infect Control* 1993;21:368-74.
354. Lewis JA, Brown PL. Breaking the comfort barrier in impervious gowns. *Surgical Services Management* 1998;4(2):29-38.
355. Granzow JW, Smith JW, Nichols RL, Waterman RS, Muzik AC. Evaluation of the protective value of hospital gowns against blood strike-through and methicillin-resistant *Staphylococcus aureus* penetration *Am J Infect Control* 1998;26:85-93.
356. Walter CW, Kundsin RB, Harding AL, Page LK. The infector on the surgical team. *Clin Neurosurg* 1966;14:361-79.
357. Payne RW. Severe outbreak of surgical sepsis due to *Staphylococcus aureus* of unusual type and origin. *Br Med J* 1967;4:17-20.
358. Centers for Disease Control. Hospital outbreak of streptococcal wound infection—Utah. *MMWR Morb Mortal Wkly Rep* 1976;25:141.
359. Herwaldt LA, Pottinger J, Coffin SA. Nosocomial infections associated with anesthesia. In: Mayhall CG, ed. *Hospital Epidemiology and Infection Control.* Baltimore: Williams & Wilkins; 1996. p. 655-75.
360. Bennett SN, McNeil MM, Bland LA, Arduino MJ, Villarino ME, Perrotta DM, et al. Postoperative infections traced to contamination of an intravenous anesthetic, propofol. *N Engl J Med* 1995;333:147-54.
361. Froggatt JW, Dwyer DM, Stephens MA. Hospital outbreak of hepatitis B in patients undergoing electroconvulsive therapy [abstract]. Program and Abstracts of the 31st Interscience Conference on Antimicrobial Agents and Chemotherapy, Chicago 1991;157:(347).
362. Centers for Disease Control. Postsurgical infections associated with an extrinsically contaminated intravenous anesthetic agent—California, Illinois, Maine, and Michigan, 1990. *MMWR Morb Mortal Wkly Rep* 1990;39:426-7,433.
363. Daily MJ, Dickey JB, Packo KH. Endogenous *Candida* endophthalmitis after intravenous anesthsia with propofol. *Arch Ophthalmol* 1991;109:1081-4.
364. Villarino ME, McNeill MM, Hall WN. Postsurgical infections associated with an extrinsically contaminated intravenous anesthetic agent [abstract]. Program and Abstracts of the 31st Interscience Conference on Antimicrobial Agents and Chemotherapy, Chicago 1991;156:(346).
365. American Association of Nurse Anesthetists. *Infection Control Guide.* Park Ridge (IL): American Association of Nurse Anesthetists; 1993.
366. American Society of Anesthesiologists. *Recommendations for Infection Control for the Practice of Anesthesiology.* Park Ridge (IL): American Society of Anesthesiologists; 1992.
367. Garner JS, Favero MS. CDC guideline for handwashing and hospital environmental control, 1985. *Infect Control* 1986;7:231-43.
368. Zacharias A, Habib RH. Delayed primary closure of deep sternal wound infections. *Tex Heart Inst J* 1996;23(3):211-6.
369. Smilanich RP, Bonnet I, Kirkpactrick JR. Contaminated wounds: the effect of initial management on outcome. *Am Surg* 1995;61(5):427-30.
370. Blomstedt GC. Infections in neurosurgery: a randomized comparison between silk and polyglycolic acid. *Acta Neurochir (Wien)* 1985;76:90-3.
371. Scher KS, Bernstein JM, Jones CW. Infectivity of vascular sutures. *Am Surg* 1985;51:577-9.
372. Durdey P, Bucknall TE. Assessment of sutures for use in colonic surgery: an experimental study. *J R Soc Med* 1984;77:472-7.
373. Chu CC, Williams DF. Effects of physical configuration and chemical structure of suture materials on bacterial adhesion. A possible link to wound infection. *Am J Surg* 1984;147:197-204.
374. Askew AR. A comparison of upper abdominal wound closure with monofilament nylon and polyglycolic acid. *Aust N Z J Surg* 1983;53:353-6.
375. Kapadia CR, Mann JB, McGeehan D, Jose Biglin JE, Waxman BP, Dudley HA. Behaviour of synthetic absorbable sutures with and without synergistic enteric infection. *Eur Surg Res* 1983;15:67-72.
376. Bucknall TE, Teare L, Ellis H. The choice of a suture to close abdominal incisions. *Eur Surg Res* 1983;15:59-66.
377. Bucknall TE. Factors influencing wound complications: a clinical and experimental study. *Ann R Coll Surg Engl* 1983;65:71-7.
378. Varma S, Lumb WV, Johnson LW, Ferguson HL. Further studies with polyglycolic acid (Dexon) and other sutures in infected experimental wounds. *Am J Vet Res* 1981;42:571-4.
379. Bucknall TE, Ellis H. Abdominal wound closure—a comparison of monofilament nylon and polyglycolic acid. *Surgery* 1981;89:672-7.
380. Dougherty SH, Simmons RL. The biology and practice of surgical drains. Part II. *Curr Probl Surg* 1992;29(9):635-730.
381. Cruse PJE. Wound infections: epidemiology and clinical characteristics in surgical infectious disease. In: Howard RJ, Simmons RL, eds. *Surgical Infectious Diseases.* 2nd ed. Norwalk (CT): Appleton and Lange; 1988. p. 319-29.
382. Drinkwater CJ, Neil MJ. Optimal timing of wound drain removal following total joint arthroplasty. *J Arthroplasty* 1995;10(2):185-9.
383. Tollofsrud SG, Gundersen Y, Andersen R. Perioperative hypothermia. *Acta Anaesthesiol Scand* 1984;28:511-5.
384. Sessler DI. Mild perioperative hypothermia. *N Engl J Med* 1997;336(24):1730-7.
385. Kurz A, Sessler DI, Lenhardt R. Perioperative normothermia to reduce the incidence of surgical-wound infection and shorten hospitalization. Study of Wound Infection and Temperature Group. *N Engl J Med* 1996;334(19):1209-15.
386. Hunt TK, Hopf HW. Wound healing and wound infection. What surgeons and anesthesiologists can do. *Surg Clin North Am* 1997;77:587-606.
387. Wenisch C, Narzt E, Sessler DI, Parschalk B, Lenhardt R, Kurz A, et al. Mild intraoperative hypothermia reduces production of reactive oxygen intermediates by polymorphonuclear leukocytes. *Anesth Analg* 1996;82:810-6.
388. Jonsson K, Hunt TK, Mathes SJ. Oxygen as an isolated variable influences resistance to infection. *Ann Surg* 1988;208:783-7.
389. Jonsson K, Jensen JA, Goodson WH 3d, West JM, Hunt TK. Assessment of perfusion in postoperative patients using tissue oxygen measurements. *Br J Surg* 1987;74:263-7.
390. Chang N, Goodson WH 3d, Gottrup F, Hunt TK. Direct measurement of wound and tissue oxygen tension in postoperative patients. *Ann Surg* 1983;197:470-8.
391. Knighton DR, Halliday B, Hunt TK. Oxygen as an antibiotic. The effect of inspired oxygen on infection. *Arch Surg* 1984;119:199-204.

392. Ikeda T, Tayefeh F, Sessler DI, Kurz A, Plattner O, Petschnigg B, et al. Local radiant heating increases subcutaneous oxygen tension. *Am J Surg* 1998;175:33-7.
393. DuMortier JJ. The resistance of healing wounds to infection. *Surg Gynecol Obstet* 1933;56:762-6.
394. Morain WD, Colen LB. Wound healing in diabetes mellitus. *Clin Plast Surg* 1990;17:493-501.
395. American Hospital Association. *Infection Control in the Hospital.* Chicago: American Hospital Association; 1979. p. 161-2.
396. Castle M, Ajemian E. *Hospital Infection Control: Principles and Practice.* New York: John Wiley & Sons; 1987. p. 114-6.
397. Centers for Disease Control. *Isolation Techniques for Use in Hospitals.* Washington (DC): Public Health Service Publication; 1970.
398. Kravitz M. Outpatient wound care. *Crit Care Nurs Clin North Am* 1996;8(2):217-33.
399. Condon RE, Schulte WJ, Malangoni MA, Anderson-Teschendorf MJ. Effectiveness of a surgical wound surveillance program. *Arch Surg* 1983;118:303-7.
400. Haley RW, Culver DH, White JW, Morgan WM, Emori TG, Munn VP. The efficacy of infection surveillance and control programs in preventing nosocomial infections in US hospitals. *Am J Epidemiol* 1985;121:182-205.
401. Lennard ES, Hargiss CO, Schoenknecht FD. Postoperative wound infection surveillance by use of bacterial contamination catgeories. *Am J Infect Control* 1985;13:147-53.
402. Olson MM, Lee JT Jr. Continuous, 10-year wound infection surveillance. Results, advantages, and unanswered questions. *Arch Surg* 1990;125:794-803.
403. Olson M, O'Connor MO, Schwartz ML. Surgical wound infections. A 5-year prospective study of 20,193 wounds at the Minneapolis VA Medical Center. *Ann Surg* 1984;199:253-9.
404. Weigelt JA. Risk of wound infections in trauma patients. *Am J Surg* 1985;150:782-4.
405. Weigelt JA, Haley RW, Seibert B. Factors which influence the risk of wound infection in trauma patients. *J Trauma* 1987;27(7):774-81.
406. Anonymous. New classification of physical status. *Anesthesiology* 1963;24:-111.
407. Owens WD. ASA physical status classification: a study on consistency of ratings. *Anesthesiology* 1978;49:239-43.
408. National Nosocomial Infections Surveillance System. Nosocomial infection rates for interhospital comparison: limitations and possible solutions. A report from the National Nosocomial Infections Surveillance (NNIS) System. *Infect Control Hosp Epidemiol* 1991;12: 609-21.
409. Cardo DM, Falk PS, Mayhall CG. Validation of surgical wound surveillance. *Infect Control Hosp Epidemiol* 1993;14:211-5.
410. Horan T, Gaynes R, Culver D, National Nosocomial Infections Surveillance (NNIS) System, CDC. Development of predictive risk factors for nosocomial surgical site infections (SSI) [abstract]. *Infect Control Hosp Epidemiol* 1994;15(Suppl):P46(M72).
411. Nichols RL, Smith JW, Klein DB, Trunkey DD, Cooper RH, Adinolfi MF, et al. Risk of infection after penetrating abdominal trauma. *N Engl J Med* 1984;311:1065-70.
412. Nichols RL, Smith JW, Robertson GD, Muzik AC, Pearce P, Ozmen V, et al. Prospective alterations in therapy for penetrating abdominal trauma. *Arch Surg* 1993;128:55-64.
413. Horan TC, Culver DH, Gaynes RP, National Nosocomial Infections Surveillance (NNIS) System. Results of a multicenter study on risk factors for surgical site infections (SSI) following C-section (CSEC) [abstract]. *Am J Infect Control* 1996;24:84.
414. Roy MC, Herwaldt LA, Embrey R, Kuhns K, Wenzel RP, Perl TM. Does the NNIS risk index (NRI) predict which patients develop wound infection (SWI) after cardiothoracic (CT) surgery? [abstract]. 34th Interscience Conference on Antimicrobial Agents and Chemotherapy 1994; Orlando, FL:196.
415. Lee TB. Surveillance in acute care and nonacute care settings: current issues and concepts. *Am J Infect Control* 1997;25(2):121-4.
416. Lee JT. Wound infection surveillance. *Infect Dis Clin North Am* 1992;6(3):643-56.
417. Mead PB, Pories SE, Hall P, Vacek PM, Davis JH Jr, Gamelli RL. Decreasing the incidence of surgical wound infections. Validation of a surveillance-notification program. *Arch Surg* 1986;121:458-61.
418. Kerstein M, Flower M, Harkavy LM, Gross PA. Surveillance for postoperative wound infections: practical aspects. *Am Surg* 1978;44: 210-4.
419. Poulsen KB, Jepsen OB. Failure to detect a general reduction of surgical wound infections in Danish hospitals. *Dan Med Bull* 1995;42:485-8.
420. Haley RW, Schaberg DR, McClish DK, Quade D, Crossley KB, Culver DH, et al. The accuracy of retrospective chart review in measuring nosocomial infection rates. Results of validation studies in pilot hospitals. *Am J Epidemiol* 1980;111(5):516-33.
421. Mulholland SG, Creed J, Dierauf LA, Bruun JN, Blakemore WS. Analysis and significance of nosocomial infection rates. *Ann Surg* 1974;180:827-30.
422. Wenzel RP, Osterman CA, Hunting KJ, Gwaltney JM Jr. Hospital-acquired infections. I. Surveillance in a university hospital. *Am J Epidemiol* 1976;103:251-60.
423. Simchen E, Shapiro JM, Michel J, Sacks T. Multivariate analysis of determinants of postoperative wound infection: a possible basis for intervention. *Rev Infect Dis* 1981;3(4):678-82.
424. Collier C, Miller DP, Borst M. Community hospital surgeon-specific infection rates. *Infect Control* 1987;8(6):249-54.
425. Ehrenkranz NJ, Shultz JM, Richter EL. Recorded criteria as a "gold standard" for sensitivity and specificity estimates of surveillance of nosocomial infection: a novel method to measure job performance. *Infect Control Hosp Epidemiol* 1995;16:697-702.
426. Hirschhorn LR, Currier JS, Platt R. Electronic surveillance of antibiotic exposure and coded discharge diagnoses as indicators of postoperative infection and other quality assurance measures. *Infect Control Hosp Epidemiol* 1993;14:21-8.
427. Simchen E, Wax Y, Pevsner B, Erdal M, Michel J, Modan M, et al. The Israeli Study of Surgical Infections (ISSI): I. Methods for developing a standardized surveillance system for a multicenter study of surgical infections. *Infect Control Hosp Epidemiol* 1988;9(6):232-40.
428. Burns SJ, Dippe SE. Postoperative wound infections detected during hospitalization and after discharge in a community hospital. *Am J Infect Control* 1982;10(2):60-5.
429. Laxson LB, Blaser MJ, Parkhurst SM. Surveillance for the detection of nosocomial infections and the potential for nosocomial outbreaks. *Am J Infect Control* 1984;12(6):318-24.
430. Mertens R, Jans B, Kurz X. A computerized nationwide network for nosocomial infection surveillance in Belgium. *Infect Control Hosp Epidemiol* 1994;15:171-9.
431. Ehrenkranz NJ. Surgical wound infection occurrence in clean operations; risk stratification for interhospital comparisons. *Am J Med* 1981;(70):909-14.
432. Baker C, Luce J, Chenoweth C, Friedman C. Comparison of case-finding methodologies for endometritis after cesarean section. *Am J Infect Control* 1995;23:27-33.
433. Gaynes RP, Horan TC. Surveillance of nosocomial infections. In: Mayhall CG, ed. *Hospital Epidemiology and Infection Control.* Baltimore: Williams & Wilkins; 1996. p. 1017-31.
434. Horan TC, Emori TG. Definitions of key terms used in the NNIS system. *Am J Infect Control* 1997;25:112-6.
435. Polk BF, Tager IB, Shapiro M, Goren-White B, Goldstein P, Schoenbaum SC. Randomised clinical trial of perioperative cefazolin in preventing infection after hysterectomy. *Lancet* 1980;1:437-41.
436. Salem RJ, Johnson J, Devitt P. Short term metronidazole therapy contrasted with povidone-iodine spray in the prevention of wound infection after appendicectomy. *Br J Surg* 1979;66:430-1.
437. Walsh AL, Roberts FJ, Bryce EA. Post-discharge surveillance of surgical wound infections [letter]. *Can J Infect Control* 1996;11(1):29.
438. Brown RB, Bradley S, Opitz E, Cipriani D, Pieczarka R, Sands M. Surgical wound infections documented after hospital discharge. *Am J Infect Control* 1987;15:54-8.
439. Rosendorf LL, Octavio J, Estes JP. Effect of methods of postdischarge wound infection surveillance on reported infection rates. *Am J Infect Control* 1983;11(6):226-9.
440. Ferraz EM, Ferraz AA, Coelho HS, Pereira Viana V, Sobral SM, Vasconcelos MD, et al. Postdischarge surveillance for nosocomial wound infection: does judicious monitoring find cases? *Am J Infect Control* 1995;23:290-4.
441. Andenaes K, Amland PF, Lingaas E, Abyholm F, Samdal F, Giercksky KE. A prospective, randomized surveillance study of postoperative wound infections after plastic surgery: a study of incidence and surveillance methods. *Plast Reconstr Surg* 1995;96(4):948-56.
442. Keeling NJ, Morgan MW. Inpatient and post-discharge wound infections in general surgery. *Ann R Coll Surg Engl* 1995;77:245-7.
443. Manian FA, Meyer L. Adjunctive use of monthly physician questionnaires for surveillance of surgical site infections after hospital discharge and in ambulatory surgical patients: report of a seven-year experience. *Am J Infect Control* 1997;25:390-4.
444. Manian FA, Meyer L. Comparison of patient telephone survey with traditional surveillance and monthly physician questionnaires in monitoring surgical wound infections. *Infect Control Hosp Epidemiol* 1993;14:216-8.
445. Reimer K, Gleed C, Nicolle LE. The impact of postdischarge infection on surgical wound infection rates. *Infect Control* 1987;8(6):237-40.
446. Sands K, Vineyard G, Platt R. Surgical site infections occuring after hospital discharge. *J Infect Dis* 1996;173:963-70.
447. Weigelt JA, Dryer D, Haley RW. The necessity and efficiency of wound surveillance after discharge. *Arch Surg* 1992;127:77-82.
448. Gravel-Tropper D, Oxley C, Memish Z, Garber GE. Underestimation of

surgical site infection rates in obstetrics and gynecology. *Am J Infect Control* 1995;23:22-6.
449. Taylor S, Pearce P, McKenzie M, Taylor GD. Wound infection in total joint arthroplasty: effect of extended wound surveillance on wound infection rates. *Can J Surg* 1994;37(3):217-20.
450. Hulton LJ, Olmsted RN, Treston-Aurand J, Craig CP. Effect of postdischarge surveillance on rates of infectious complications after cesarean section. *Am J Infect Control* 1992;20:198-201.
451. Law DJ, Mishriki SF, Jeffery PJ. The importance of surveillance after discharge from hospital in the diagnosis of postoperative wound infection. *Ann R Coll Surg Engl* 1990;72:207-9.
452. Donovan IA, Ellis D, Gatehouse D, Little G, Grimley R, Armistead S, et al. One-dose antibiotic prophylaxis against wound infection after appendicectomy: a randomized trial of clindamycin, cefazolin sodium and a placebo. *Br J Surg* 1979;66:193-6.
453. Bates T, Down RH, Houghton MC, Lloyd GJ. Topical ampicillin in the prevention of wound infection after appendicectomy. *Br J Surg* 1974;61:489-92.
454. Centers for Disease Control and Prevention. Evaluation of blunt suture needles in preventing percutaneous injuries among health-care workers during gynecologic surgical procedures, New York City, March 1993-June 1994. *MMWR Morb Mortal Wkly Rep* 1997;46(2):25-9.
455. Fanning C, Johnston BL, MacDonald S, LeFort-Jost S, Dockerty E. Postdischarge surgical site infection surveillance. *Can J Infect Control* 1995;10(3):75-9.
456. Holbrook KF, Nottebart VF, Hameed SR, Platt R. Automated postdischarge surveillance for postpartum and neonatal nosocomial infections. *Am J Med* 1991;91(Suppl 3B):125S-30S.
457. Zoutman D, Pearce P, McKenzie M, Taylor G. Surgical wound infections occuring in day surgery patients. *Am J Infect Control* 1990;18: 277-82.
458. Seaman M, Lammers R. Inability of patients to self-diagnose wound infections. *J Emerg Med* 1991;9:215-9.
459. Goulbourne IA, Ruckley CV. Operations for hernia and varicose veins in a day-bed unit. *Br Med J* 1979;2:712-4.
460. Garvey JM, Buffenmyer C, Rycheck RR, Yee R, McVay J, Harger JH. Surveillance for postoperative infections in outpatient gynecologic surgery. *Infect Control* 1986;7(2):54-8.
461. Flanders E, Hinnant JR. Ambulatory surgery postoperative wound surveillance. *Am J Infect Control* 1990;18(5):336-9.
462. Gentry LO, Zeluff BJ, Cooley DA. Antibiotic prophylaxis in open-heart surgery: a comparison of cefamandole, cefuroxime, and cefazolin. *Ann Thorac Surg* 1988;46:167-71.
463. Maki DG, Bohn MJ, Stolz SM, Kroncke GM, Archer CW, Myerowitz PD. Comparative study of cefazolin, cefamandole, and vancomycin for surgical prophylaxis in cardiac and vascular operations. A double-blind randomized trial. *J Thorac Cardiovasc Surg* 1992;104:1423-34.
464. Haines SJ, Walters BC. Antibiotic prophylaxis for cerebrospinal fluid shunts: a metanalysis. *Neurosurgery* 1994;34(1):87-92.
465. Langley JM, LeBlanc JC, Drake J, Milner R. Efficacy of antimicrobial prophylaxis in placement of cerebrospinal fluid shunts: meta-analysis. *Clin Infect Dis* 1993;17:98-103.
466. Starr MB, Lally JM. Antimicrobial prophylaxis for ophthalmic surgery. *Surv Ophthalmol* 1995;39:485-501.
467. Gatell JM, Riba J, Lozano ML, Mana J, Ramon R, Garcia Sanmiguel J. Prophylactic cefamandole in orthopaedic surgery. *J Bone Joint Surg Am* 1984;66:1219-22.
468. Kent KC, Bartek S, Kuntz KM, Anninos E, Skillman JJ. Prospective study of wound complications in continuous infrainguinal incisions after lower limb arterial reconstruction: incidence, risk factors, and cost. *Surgery* 1996;119(4):378-83.
469. Wymenga AB, van Horn JR, Theeuwes A, Muytjens HL, Slooff TJ. Perioperative factors associated with septic arthritis after arthroplasty. Prospective multicenter study of 362 knee and 2,651 hip operations. *Acta Orthop Scand* 1992;63(6):665-71.
470. Stambough JL, Beringer D. Postoperative wound infections complicating adult spine surgery. *J Spinal Disord* 1992;5(3):277-85.
471. Trippel SB. Antibiotic-impregnated cement in total joint arthroplasty. *J Bone Joint Surg Am* 1986;68A:1297-302.
472. Nungu KS, Olerud C, Rehnberg L, Larsson S, Nordell P, Allvin I, et al. Prophylaxis with oral cefadroxil versus intravenous cefuroxime in trochanteric fracture surgery. A clinical multicentre study. *Arch Orthop Trauma Surg* 1995;114(6):303-7.
473. Norden CW. Antibiotic prophylaxis in orthopedic surgery. *Rev Infect Dis* 1991;13(Suppl 10):S842-6.
474. Aznar R, Mateu M, Miro JM, Gatell JM, Gimferrer JM, Aznar E, et al. Antibiotic prophylaxis in non-cardiac thoracic surgery: cefazolin versus placebo. *Eur J Cardiothorac Surg* 1991;5:515-8.
475. Cant PJ, Smyth S, Smart DO. Antibiotic prophylaxis is indicated for chest stab wounds requiring closed tube thoracostomy. *Br J Surg* 1993;80:464-6.
476. Pitt HA, Postier RG, MacGowan AW, Frank LW, Surmak AJ, Sitzman JV, et al. Prophylactic antibiotics in vascular surgery. Topical, systemic, or both? *Ann Surg* 1980;192:356-64.
477. Kaiser AB, Clayson KR, Mulherin JL Jr, Roach AC, Allen TR, Edwards WH, et al. Antibiotic prophylaxis in vascular surgery. *Ann Surg* 1978;188:283-9.
478. Bauer T, Vennits B, Holm B, Hahn-Pedersen J, Lysen D, Galatius H, et al. Antibiotic prophylaxis in acute nonperforated appendicitis. The Danish Multicenter Study Group II. *Ann Surg* 1989;209:307-11.
479. Skipper D, Corder AP, Karran SJ. A randomized prospective study to compare ceftizoxime with cephradine as single dose prophylaxis in elective cholecystectomy. *J Hosp Infect* 1991;17:303-6.
480. Kaufman Z, Engelberg M, Eliashiv A, Reiss R. Systemic prophylactic antibiotics in elective biliary surgery. *Arch Surg* 1984;119:1002-4.
481. Grant MD, Jones RC, Wilson SE, Bombeck CT, Flint LM, Jonasson O, et al. Single dose cephalosporin prophylaxis in high-risk patients undergoing surgical treatment of the biliary tract. *Surg Gynecol Obstet* 1992;174:347-54.
482. Lewis RT, Goodall RG, Marien B, Park M, Lloyd-Smith W, Wiegand FM. Biliary bacteria, antibiotic use, and wound infection in surgery of the gallbladder and common bile duct. *Arch Surg* 1987;122:44-7.
483. Saltzstein EC, Mercer LC, Peacock JB, Dougherty SH. Outpatient open cholecystectomy. *Surg Gynecol Obstet* 1992;174(3):173-5.
484. Meijer WS, Schmitz PI, Jeekel J. Meta-analysis of randomized, controlled clinical trials of antibiotic prophylaxis in biliary tract surgery. *Br J Surg* 1990;77:283-90.
485. Kaiser AB, Herrington JL Jr, Jacobs JK, Mulherin JL Jr, Roach AC, Sawyers JL. Cefoxitin versus erythromycin, neomycin, and cefazolin in colorectal operations. Importance of the duration of the surgical procedure. *Ann Surg* 1983;198:525-30.
486. Schoetz DJ Jr, Roberts PL, Murray JJ, Coller JA, Veidenheimer MC. Addition of parenteral cefoxitin to regimen of oral antibiotics for elective colorectal operations. A randomized prospective study. *Ann Surg* 1990;212:209-12.
487. Edmondson HT, Rissing JP. Prophylactic antibiotics in colon surgery. *Arch Surg* 1983;118:227-31.
488. Wapnick S, Guinto R, Reizis I, LeVeen HH. Reduction of postoperative infection in elective colon surgery with preoperative administration of kanamycin and erythromycin. *Surgery* 1979;85:317-21.
489. Washington JA 2d, Dearing WH, Judd ES, Elveback LR. Effect of preoperative antibiotic regimen on development of infection after intestinal surgery. Prospective, randomized, double-blind study. *Ann Surg* 1974;108:567-72.
490. Maki DG, Aughey DR. Comparative study of cefazolin, cefoxitin, and ceftizoxime for surgical prophylaxis in colo-rectal surgery. *J Antimicrob Chemother* 1982;10(Suppl C):281-7.
491. Rotman N, Hay JM, Lacaine F, Fagniez PL, The Association de Recherche en Chirurgie Cooperative Group. Prophylactic antibiotherapy in abdominal surgery. First- vs third-generation cephalosporins. *Arch Surg* 1989;124:323-7.
492. Lewis RT, Allan CM, Goodall RG, Marien B, Park M, Lloyd-Smith W, et al. Cefamandole in gastroduodenal surgery: a controlled, prospective, randomized, double-blind study. *Can J Surg* 1982;25(5):561-3.
493. McArdle CS, Morran CG, Anderson JR, Pettit L, Gemmell CG, Sleigh JD, et al. Oral ciprofloxacin as prophylaxis in gastroduodenal surgery. *J Hosp Infect* 1995;30(3):211-6.
494. Grandis JR, Vickers RM, Rihs JD, Yu VL, Johnson JT. Efficacy of topical amoxicillin plus clavulanate/ticarcillin plus clavulanate and clindamycin in contaminated head and neck surgery: effect of antibiotic spectra and duration of therapy. *J Infect Dis* 1994;170:729-32.
495. Johnson JT, Yu VL, Myers EN, Wagner RL. An assessment of the need for gram-negative bacterial coverage in antibiotic prophylaxis for oncological head and neck surgery. *J Infect Dis* 1987;155(2):331-3.
496. Elledge ES, Whiddon RG Jr, Fraker JT, Stambaugh KI. The effects of topical oral clindamycin antibiotic rinses on the bacterial content of saliva on healthy human subjects. *Otolaryngol Head Neck Surg* 1991;105:836-9.
497. Johnson JT, Yu VL, Myers EN, Wagner RL, Sigler BA. Cefazolin vs moxalactam? A double-blind randomzied trial of cephalosporins in head and neck surgery. *Arch Otolaryngol Head Neck Surg* 1986;112:151-3.

SELECTED READINGS

Association of Operating Room Nurses. *Standards, Recommended Practices, Guidelines.* Denver: Association of Operating Room Nurses; 1999.

Larson EL. APIC guideline for handwashing and hand antisepsis in health care settings. *Am J Infect Control* 1995;23:251-69.

Association of Operating Room Nurses. Recommended practices for skin preparation of patients. *AORN J* 1996;64:813-6.

Rutala WA. APIC guideline for selection and use of disinfectants. *Am J Infect Control* 1990;18:99-117.

Favero MS, Bond W. Sterilization, disinfection, and antisepsis in the hospital. In: Balows A, Hausler WJ Jr, Herrmann KL, Isenberg HD, Shadomy HJ, eds. *Manual of Clinical Microbiology.* 5th ed. Washington (DC): American Society for Microbiology; 1991. p. 183-200.

Association for the Advancement of Medical Instrumentation. Flash sterilization: steam sterilization of patient care items for immediate use (ANSI/AAMI ST37-1996). Arlington (VA): Association for the Advancement of Medical Instrumentation; 1996.

American Institute of Architects Committee. Guidelines for design and construction of hospital and health care facilities. Washington (DC): American Institute of Architects Press; 1996.

Association for the Advancement of Medical Instrumentation. Selection of surgical gowns and drapes in health care facilities (AAMI TIR No. 11-1994). Arlington (VA): Association for the Advancement of Medical Instrumentation; 1994.

Platt R. Guidelines for perioperative antibiotic prophylaxis. In: Abrutyn E, Goldmann DA, Scheckler WE, eds. *Saunders Infection Control Reference Service.* Philadelphia: W.B. Saunders Co; 1997. p. 229-34.

Classen DC, Evans RS, Pestotnik SL, Horn SD, Menlove RL, Burke JP. The timing of prophylactic administration of antibiotics and the risk of surgical-wound infection. *N Engl J Med* 1992;326:281-6.

Wong ES. Surgical site infections. In: Mayhall CG, ed. *Hospital Epidemiology and Infection Control.* Baltimore: Williams & Wilkins; 1996. p. 154-74.

Hochberg J, Murray GF. Principles of operative surgery: antisepsis, technique, sutures, and drains. In: Sabiston DC Jr, ed. *Textbook of Surgery: The Biological Basis of Modern Surgical Practice.* 15th ed. Philadelphia: W.B. Saunders Co; 1997. p. 253-63.

Roy MC. Surgical-site infections after coronary artery bypass graft surgery: discriminating site-specific risk factors to improve prevention efforts. *Infect Control Hosp Epidemiol* 1998;19:229-33.

Herwaldt LA, Pottinger J, Coffin SA. Nosocomial infections associated with anesthesia. In: Mayhall CG, ed. *Hospital Epidemiology and Infection Control.* Baltimore: Williams & Wilkins; 1996. p. 655-75.

Gaynes RP, Horan TC. Surveillance of nosocomial infections. In: Mayhall CG, ed. *Hospital Epidemiology and Infection Control.* Baltimore: Williams & Wilkins; 1996. p. 1017-31.

Roy MC, Perl TM. Basics of surgical-site infection surveillance. *Infect Control Hosp Epidemiol* 1997;18:659-68.

Lee JT. Surgical wound infections: surveillance for quality improvement. In: Fry DE, ed. *Surgical Infections.* Boston: Little, Brown and Co; 1995. p. 145-59.

Meier PA. Infection control issues in same-day surgery. In: Wenzel RP, ed. *Prevention and Control of Nosocomial Infections.* 3rd ed. Baltimore: Williams & Wilkins; 1997. p. 261-82.

Burke JP. Infections of cardiac and vascular prostheses. In: Bennett JV, Brachman PS, eds. *Hospital Infections.* 4th ed. Philadelphia: Lippincott-Raven; 1998. p. 599-612.

Lew DP, Waldvogel FA. Infections of skeletal prostheses. In: Bennett JV, Brachman PS, eds. *Hospital Infections.* 4th ed. Philadelphia: Lippincott-Raven; 1998. p. 613-20.

Nafziger DA, Saravolatz LD. Infection in implantable prosthetic devices. In: Wenzel RP, ed. *Prevention and Control of Nosocomial Infections.* 3rd ed. Baltimore: Williams & Wilkins; 1997. p. 889-923.