Review

SA guideline

The Challenge of Postoperative Infections: Does the Surgeon Make a Difference?

René G. Holzheimer, MD, PhD; Werner Haupt, MD;
Arnulf Thiede, MD, PhD; Andreas Schwarzkopf, MD, PhD

ABSTRACT

Postoperative infections remain a challenge in many surgical procedures despite improved surgical technique and powerful antibiotics. The number of sepsis cases has tripled from 1979 to 1992 due to increased invasive procedures in older and immune-suppressed patients. Increasingly, in recent years, outbreaks of resistant pathogens have been published, provoking the question of how postoperative infections and resistant pathogens should be dealt with.

Wound classification and risk stratification were developed to identify patients at risk for postoperative infection. However, other important intrinsic factors of the patient were not included, and further attempts have been made to increase sensitivity and specificity (eg, Study on the Efficacy of Nosocomial Infection Control project, National Nosocomial Infection Surveillance System score); the American Society of Anesthesiologists preoperative assessment score and the operation duration for specific procedures were introduced into the system as risk stratifiers.

Advances in immunology have identified new ways in which the surgeon can moderate the immune response (eg, hemorrhage and blood transfusion-induced immune suppression). The increased rate of resistance in enterococci and staphylococci has refocused attention on infection control in surgery. However, there are recent reports from both sides of the Atlantic indicating that guidelines for infection control and antibiotic policy have not become reflected in standard procedures in many hospitals.

New antibiotics may be developed, but resistance soon may follow. Sound techniques in surgery, with careful infection control and antibiotic policies, may be the only strategy to prevent further increases in resistance of pathogens in postoperative infections (*Infect Control Hosp Epidemiol* 1997;18:449-456).

Postoperative infections remain a challenge in many surgical procedures despite improved surgical techniques and perioperative antibiotic prophylaxis. According to the Centers for Disease Control and Prevention, the number of sepsis cases linked to microbial infections in hospital patients tripled from 1979 to 1992 (partly because of increased vulnerability of the patient population). Every year in the United States some 500,000 people acquire sepsis and 175,000 die.[1]

Wound infections remain a major source of postoperative morbidity, accounting for approximately one fourth of nosocomial infections. In a 12-month nationwide study in 1975 to 1976, it was estimated that wound infections accounted for approximately 24% of the nosocomial infections.[2] The incidence of wound infection varies from surgeon to surgeon, from hospital to hospital, and from one surgical procedure to another, and most importantly from one patient to another.

The most critical factors in the prevention of postoperative wound infection, although difficult to quantitate, appear to be the sound judgment and proper operative technique of the responsible surgeon and team, as well as the general health and stage of disease of the individual patient. Important factors in this respect are wound classification and risk stratification, including duration of surgery, the

From the Department of Surgery (Drs. Holzheimer, Thiede, and Schwarzkopf), Würzburg University, Germany; and the Department of Surgery (Dr. Haupt), Erlangen University, Germany.

Address reprint requests to PD Dr. René G. Holzheimer, Department of Surgery, Würzburg University, Josef-Schneider-Straße 2, D-97080 Würzburg, Germany.

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TABLE 1
POSSIBLE RISK FACTORS FOR POSTOPERATIVE WOUND INFECTIONS*

Host Factors	Surgery Factors
Age	Emergency vs elective procedure
Gender	Hair removal technique
Severity of disease	Service
ASA physical status classification	Surgeon
Immunocompromising diseases	Site of surgery
Diabetes mellitus	Procedure or procedures
Estimated prognosis	Intraoperative culture
Nutritional status	Perioperative antibiotics
Serum albumin	Duration of surgery
Weight	Drains
Presence of other infections	Packs
Duration of preoperative stay	Primary or secondary closure
	Drapes
	Irrigation
	Glove punctures

Abbreviations: ASA, American Society of Anesthesiologists.
*Modified from reference 19.

surgeon as immune modulator, the use and abuse of perioperative antibiotic prophylaxis and antibiotic treatment with effects on bacterial resistance, and infection control in the surgical department. It should be noted that the surgeon is involved in all factors.

Wound Classification and Risk Stratification

The idea of controlling for intrinsic risk in wound infection reporting is far from new. In 1895, Brewer reported wound infection rates in clean surgery to his surgeon colleagues and observed a 95% reduction in rates.[3] Although several excellent studies for identification of risk factors were performed[4,5]—Nichols published the first multivariate analyses in trauma surgery[6]—none of these included a classification scheme.

The first clinically used system for classification to identify the risk for postoperative infection was developed by Cruse and Foord[7] in 1973 with the four categories "clean," "clean-contaminated," "contaminated," and "dirty." It was supposed that clean operations without foreign body implant had a low risk for postoperative infection and that contaminated operations had a higher risk.[7] However, recent reports have challenged that view[8,9]; in clean operations, a high variation in postoperative infection rates was observed. The classification of Cruse and Foord also had the disadvantage that intrinsic factors for postoperative infections were not included. This led to a search for improved classifications.

The Study on the Efficacy of Nosocomial Infection Control (SENIC) developed an improved index for risk stratification (SENIC Index).[10,11] There were four independent risk factors identified: laparotomy, duration over 2 hours, contaminated or dirty operation, and three or more diagnoses at discharge.

A further modification of the SENIC Index is the National Nosocomial Infection Surveillance (NNIS) System Index,[12] which grades the risk for postoperative infection according to three factors: an American Society of Anesthesiologists preoperative assessment score (ASA)[13] of 3, 4, or 5; a contaminated or dirty operation; and an operation lasting longer than T, where T depends on the type of procedure.

It is supposed that increased operation time causes an increase in bacterial contamination. The long operation duration may be a marker for the complexity of the operation, the technical skill of the individual surgeon, and the reduced efficacy of antibiotic prophylaxis in certain operations after the longer duration. Culver stated that wound classification and operation duration can be considered indirect markers for quality of care. A surgeon with an operation time above the 75th percentile may have low infection rates; however, he is causing an increased risk for postoperative infections in his patients. It is necessary to analyze not only the distribution of patients according to risk categories but also in regard to wound infection rates within a category when looking at the individual surgeon's postoperative infections.

The significance of operation duration in regard to skill of surgeon, complexity of operation, and extent of tissue trauma as markers for postoperative complications also was reported by Hooton,[14] and differences among surgeons were mentioned by Conklin.[15] Two studies investigated risk factors in elective colorectal operations. Kaiser[16] reported a correlation with the duration of operation, and, in a more recent study, Coppa and Eng[17] identified the duration of operation and the localization of the procedure as risk factors. The duration of operation is a critical issue among surgeons. Contamination increases with time as wound edges dry out. However, speed associated with poor technique is not suitable to reduce the wound infection rate.[18]

Garibaldi et al[19] have identified a number of possible risk factors for postoperative wound infections (Table 1). As noted, intraoperative wound cultures were investigated as predictors of postoperative wound infections by Garibaldi et al.[19] However, only surgical-wound class, ASA physical status grouping, and duration of surgery were found to stratify risk;

the identification and quantification of organisms appears to be of limited value for prediction of outcome of infection.[20] Even though patients with positive intraoperative cultures had an increased rate of infection, this information had limited clinical utility. Patients in this study who received perioperative antibiotics and who developed infections frequently were infected with organisms that were resistant to the perioperative drug regimen, compared with patients who had not received antibiotics.

Meticulous hemostasis and gentle handling of tissue were associated with lower infection rates in reports by Kocher, von Bergmann, and Halsted.[21,22] The surgeon assumes responsibility for the operative procedure, including meticulous and gentle technique, and avoidance of hematomas, as well as the need for drains.[23,24] Poor tissue perfusion as a result of systemic hyperperfusion and local ischemia caused by inappropriate surgery also increased the risk of wound infection.[25] Although fastidious surgical technique is recognized easily, it is difficult to measure. The surgeon and the hospital where operations take place may be predictors of wound infection.[26-28]

Intraoperative decisions may influence the risk of postoperative infection. In a prospective study on nonperforated appendicitis, the risk of infection correlated with the decision of the surgeon to consider the appendix gangrenous and with the failure to start perioperative antibiotic prophylaxis.[29]

With progress in immunology, it has become more and more apparent that the patient's host defense is a major factor among the risks for postoperative infection, and it should be recorded in a risk-stratification system. Christou et al derived an index that included skin testing and blood studies to represent patients' nutritional status and host defense mechanisms more directly.[30] The significance of anergy in surgical patients has been demonstrated by Cainzos in several studies.[31]

The Surgeon as Immune Modulator

These findings have led to the conception that the surgeon himself may be an important immune modulator for the patient. Local and systemic host defense can be enhanced or suppressed by the surgeon.[32] Factors implicated in perioperative immune suppression that the surgeon can influence include hemorrhage, blood transfusion, and perioperative bacterial contamination. In humans, the effects of perioperative hemorrhage itself on the immune system cannot be separated easily from the magnitude of the stress response and blood transfusion; so, most information in this area has come from animal models in which bleeding is performed in controlled circumstances. In an experimental model of controlled hemorrhage, Chaudry et al[33] reported significant impairment of T-cell proliferation, interleukin-2 production, and class II major histocompatibility complex (MHC)-dependent antigen presentation. They also reported impaired Kupffer cell MHC class II antigen expression following hemorrhage, suggesting a reduced capacity to eliminate absorbed bacteria and endotoxin from portal blood.[34] This observation may have considerable importance, as the gut increasingly is recognized as an important source of sepsis following hemorrhage, both experimentally and in humans. Hypotension, even for short periods of time, permits bacterial translocation, and, in animals with shock, far fewer organisms are required to generate infection than in normal individuals.[35-38] Surgical technique and control of hemorrhage may influence the host immune response and the postoperative follow-up.

Infection Control

Infection control encompasses all aspects of surgical care, from entry to the hospital to adequate follow-up to ensure that wound infections are not missed if they occur shortly after discharge. Many, but not all, infection risk factors can be controlled, including theater discipline, sterility of instruments, handwashing, use of gloves and drapes, skin preparation, shaving, and aseptic technique.

Many important strides have been made in the prevention of nosocomial infections. Techniques for surveillance have been developed and utilized extensively. Surveillance has been defined as the systematic, active, ongoing observation of the occurrence and distribution of disease in a population and events or conditions that increase or decrease the risk of disease. It is important to recognize that the definition also includes analysis of data and dissemination of results, so that appropriate actions can be taken.[39]

Objectives for surveillance programs traditionally have included some or all of the following: determination of the magnitude of specific nosocomial infection problems, monitoring trends in specific infection rates, identification of patient groups at high risk for nosocomial infection, detection of nosocomial infection outbreaks, comparison with nosocomial infection experiences of other institutions, and satisfaction of requirements for accreditation.[40]

Landmark prevalence studies of nosocomial infections at all sites were conducted at Boston City Hospital in 1964 and 1967. Results of these studies indicated that the prevalence was similar in both years and that surgical patients were more likely to be infected than were medical patients.[41,42]

The Comprehensive Hospital Infections Project reported that the prevalence of nosocomial infections was higher on surgical than on medical services and that patients with nosocomial gram-negative urinary tract infections, surgical-wound infections, and pneumonia who had secondary bloodstream infection were more likely to die.[43,44]

Because of their frequency, morbidity, and cost of treatment, surgical-wound infections are an important prevention priority for infection control personnel in hospitals where surgical volume is high. The NNIS System, organized by the Centers for Disease Control, began operation in 1970. The importance of conducting surveillance of surgical-wound infection and reporting rates to surgeons was described in 1973 and subsequently emphasized by the results of the SENIC project.[7,45,46] The SENIC project, conducted between 1974 and 1983, was designed to evaluate the effectiveness of nationwide hospital infection prevention and control programs. The study found that 32% of nosocomial infections involving the four major sites (urinary tract, surgical wound, lower respiratory tract, and bloodstream) could be prevented by well-organized programs.[47,48]

In addition, the SENIC project results demonstrated that patient risk for surgical-wound infection varied dramatically with differences in the level of wound contamination by organisms during operative procedures, as previously reported, and in host susceptibility.[49,50] The experience of a number of other investigators has emphasized the importance of surgical-wound infection surveillance and the reporting of results to surgeons.[51,52]

What were the conclusions of these surveillance studies? How can wound infection rates be reduced?

Cruse argued for the strict compliance of 10 elements to reduce the infection rate of surgical wounds: (1) Short preoperative stay; (2) hexachlorophene shower before operation; (3) shaving kept to a minimum; (4) contamination eschewed; (5) punctilious surgical technique; (6) as expeditious an operation as was safe; (7) scrupulous care in operations on elderly, obese, malnourished, or diabetic patients; (8) no drains brought out through the operative wound; (9) meticulous coagulation technique using the electrosurgical unit; and (10) information to each surgeon of his or her own clean-wound infection rate and the average of his or her peers.

Application of this concept led to a steady fall in the clean-wound infection rate, to 0.6% in 1977.[53]

This led to the conclusion that the clean-wound infection rate is the measure of the institution and the surgeon. Wounds become infected during operations, and endogenous contamination has a larger impact than exogenous contamination on the development of postoperative infections.[54]

Some of Cruse's statements have been questioned subsequently. It has been argued that clean wounds should not get infected and that almost all wounds in higher classes do. The epidemiological evidence, however, does not support this view. Patients with clean operations had infection rates as high as 15%, whereas 70% of patients with contaminated or dirty operations had no infection.[55,56]

Implementation of standard practice for infection control in intensive-care units (ICUs) is far from satisfactory. Almost 17% of ICUs participating in a recent multicenter study routinely used 3 or more of 12 suboptimal practices.[57] In general, there is a large variation in the use of infection control practices; in many areas, consensus is missing.[57,58] Indeed, there are reports that infection control in some countries in surgery is nonexistent.[59]

Antibiotic Prophylaxis

Clinical surgery has witnessed a remarkable reduction in postoperative wound infection over the past 25 years, mainly because of the empirical, but rational, use of antibiotics as prophylactic agents. The realization that the efficacy of antibiotic prophylaxis depends on the timing of administration was based on the work of Miles and Burke.[60,61] Polk initiated the first fully controlled trial of antibiotic prophylaxis in alimentary tract surgery and found that systemic cephaloridine administered just before the operation and then again immediately after the operation in two additional doses produced a highly statistically significant reduction in incisional infection.[62,63] Then, in further studies, single-dose cephalosporin prophylaxis was advocated.[64] However, the timing of the initial administration, the appropriate choice of antibiotic agents, and the limitation of the duration of administration have been a subject of discussion among surgeons for the last 10 years. In general, antibiotic prophylaxis now is recommended in elective clean surgical procedures utilizing a foreign body and in clean-contaminated procedures. Second-generation cephalosporins are administered intravenously prior to incision, and additional doses are necessary only when the procedure is longer than 3-4 hours.[65] However, recent data suggest that prophylaxis may be beneficial also in clean operations without foreign body implant.[66]

According to Leaper,[68] not all studies support the efficacy of prophylactic antibiotics in reducing the occurrence of postoperative intra-abdominal infection or improving the healing of sutured experimental wounds (eg, anastomosis). However, a reduction in postoperative chest and urinary tract infections has been observed.[67,68] The most effective administration route remains a matter of discussion. Furthermore, the choice of antibiotic for prophylaxis remains controversial and may have a detrimental effect on resistance.[69-71]

Some recent reports on antibiotic prophylaxis are alarming. Despite indication for prophylaxis, antibiotic prophylaxis was started in 82.1% of patients undergoing elective operations and in only 72.1% of emergencies.[72] Sixty percent of appendectomies were performed without prophylaxis, despite new reports that antimicrobial prophylaxis may be beneficial. In colorectal surgery, only 79% received standard antibiotic prophylaxis; in ileocecal operations, the rate was even lower (56.5%).[73-75] The timing and duration of antibiotic prophylaxis have varied from 2 to 24 hours before the operation to up to 24 hours and more after the operation.[76]

Antimicrobial Resistance

Antimicrobial resistance is commonplace among bacteria involved in surgical infections. Resistance traits can be encoded on chromosomes or transmissible plasmids. The basic mechanisms of resistance are alteration of drug target, prevention of drug access to target, and drug inactivation. Examples include alteration of penicillin-binding proteins in resistance to penicillinase-resistant penicillins, ribosomal binding site protection in tetracycline resistance, and β-lactamase destruction of β-lactam compounds.[77]

Recent publications have indicated an increased resistance of certain pathogens (eg, enterococci) and that widespread use of certain antibiotics (eg, cephalosporins) may result in increased resistance of selected pathogens[78-82] (Table 2).

However, person-to-person contact also may be responsible for the spread of resistant hospital infections. In this respect, it should be noted that the ability to detect and monitor infectious disease is of utmost importance. False perceptions that such threats had dwindled or disappeared led to complacency and decreased vigilance regarding infectious diseases, resulting in the weakening of surveillance.[83]

We and others have demonstrated that enterococci are the third most common pathogens in surgical-wound infections.[84] Use of single agents, either for prophylaxis or treatment of intra-abdominal infections, has resulted in enterococcal superinfections.[85,86] The patterns of drug resistance are different on each side of the Atlantic, however, in part because of different patterns of antibiotic use. Even though some drug-resistance patterns vary, one serious worry can be shared equally on both sides of the Atlantic: the emergence of vancomycin-resistant strains of enterococci, which cause urinary tract and wound infections. Up to the mid-1980s, vancomycin resistance was negligible. Between 1989 and 1993, hospital-acquired vancomycin-resistant enterococci increased sharply.[87] Even more alarming is the possibility that enterococci will spread vancomycin resistance to other genera of bacteria.[88]

TABLE 2
BACTERIAL RESISTANCE IN SURGICAL INFECTIONS*

Site	Pathogens
Head/neck surgery	*Staphylococcus aureus, Bacteroides, Fusobacterium, Klebsiella*
Intra-abdominal infections	*Escherichia coli, Klebsiella, Bacteroides*
Necrotizing cellulitis	*S aureus, Bacteroides, E coli*
Chronic osteomyelitis	*S aureus*
Diabetic foot infections	*S aureus, Bacteroides, Proteus, E coli*
Surgical ICU infections	*Klebsiella, Pseudomonas, S aureus, Staphylococcus epidermidis, Xanthomas, Serratia, Acinetobacter*

Abbreviation: ICU, intensive-care unit.
*Modified from reference 94.

The use of hospital policy regarding antibiotic prescription as a method to control infection is not new. Dr. Maxwell Finland,[89] of Boston, was critical for many years of the indiscriminate use of antibiotics. In 1960, Barber, in England, showed the effects of a policy change on staphylococcal infections in a large hospital.[90] In a study evaluating risk factors associated with nosocomial infection in two university hospitals, Chavigny and Fischer reported that 65.3% of antibiotics were given before a nosocomial infection occurred and that antibiotic therapy is a relevant risk factor of concern in hospital-acquired infection.[91] Chavigny and Fischer also reported that antibiotic therapy, instrumentation, surgical operations, and age accounted for 95% of the variation in nosocomial infection rates.

The overall antibiotic-use policy, as well as individual judgment regarding antibiotic prescription, might be employed judiciously to decrease the risk of nosocomial infection in acute care (Table 3). Surgical prophylaxis extended beyond 48 hours causes selection of resistant bacteria, as does the excessive use of broad-spectrum antibiotics. Inappropriately low doses of antibiotics will promote resistance by selec-

TABLE 3
ANTIBIOTIC USE CONSIDERATIONS

Choice of antibiotic
Dosage
Indication
Route of administration
Disregard to antibiotic susceptibility test or clinical symptoms of resistance
Timing of antibiotic therapy or premature discontinuation
Combination of antibiotics
Overuse of antibiotics

tion of subpopulations that have the ability to grow in increasing concentrations of antibiotic.[92,93]

Is the ultimate answer to resistance to develop new compounds against resistant pathogens? Probably not! As each new antimicrobial agent has been introduced over the years, it has been met inevitably with the emergence of resistant organisms. Nonetheless, efforts to stay one step ahead of the organisms continue and focus on chemical modification of currently known agents, as well as the development of potentiators of known antimicrobials, inhibitors of new targets in bacterial cellular function, inhibitors of factors related to bacterial virulence or pathogenesis, and antisense antinucleotides.[94]

The answer to maintaining long-term effective use of therapeutic agents lies in better, more prudent use of antibiotics in human and animal health care, as has been advocated continually since the first discovery of bacteria resistant to antibiotics.[95] Guidelines for limitation of emergence and spread of antimicrobial resistance have been published recently.[96,97] The problem of resistance in surgical patients will persist, and knowledge of resistance patterns and the risks for development of resistance are essential to ensure successful operations. The development of inhibitor combinations and novel agents and modification of existing agents will provide some relief. However, the factors that promote the development and spread of resistance must be addressed. These include inadequate hygiene, inadequate wound care, handwashing, disposal of dressings, failure to isolate patients with large wounds or urinary tract infections who are colonized or infected with resistant organisms, extension of surgical prophylaxis beyond 48 hours, excessive use of broad-spectrum antibiotics, inappropriate dosing of antimicrobial agents, and failure to complete treatment.

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