

the number of breaks in the system, and teaching aseptic technique for suctioning). Although the benefits of selective decontamination of the oropharynx and gut with nonabsorbable antimicrobial agents and/or use of short-course postintubation systemic antibiotics have been controversial, a randomized multicenter trial demonstrated lowered ICU mortality rates among patients on mechanical ventilation who underwent oropharyngeal decontamination.

Among the logical preventive measures that require further investigation are placement of endotracheal tubes that provide channels for subglottic drainage of secretions, which has been associated with reduced infection risks during short-term postoperative use, and non-invasive mechanical ventilation whenever feasible. Use of silver-coated endotracheal tubes may lessen risk of VAP but is not considered routine. It is noteworthy that reducing the rate of VAP often has not reduced overall ICU mortality; this fact suggests that this infection is a marker for patients with an otherwise-heightened risk of death.

The most likely pathogens for nosocomial pneumonia and treatment options are discussed in **Chap. 153**. Several considerations regarding diagnosis and treatment are worth emphasizing. First, clinical criteria for diagnosis (e.g., fever, leukocytosis, development of purulent secretions, new or changing radiographic infiltrates, changes in oxygen requirement or ventilator settings) have high sensitivity but relatively low specificity. These criteria are most useful for selecting patients for bronchoscopic or nonbronchoscopic procedures that yield lower respiratory tract samples protected from upper-tract contamination; quantitative cultures of such specimens have diagnostic sensitivities in the range of 80%. Second, early-onset nosocomial pneumonia, which manifests within the first 4 days of hospitalization, is most often caused by community-acquired pathogens such as *Streptococcus pneumoniae* and *Haemophilus* species, although some studies have challenged this view. Late-onset pneumonias most commonly are due to *S. aureus*, *P. aeruginosa*, *Enterobacter* species, *Klebsiella pneumoniae*, or *Acinetobacter*. When invasive techniques are used to diagnose VAP, the proportion of isolates accounted for by gram-negative bacilli decreases from 50–70% to 35–45%. Infection is polymicrobial in as many as 20–40% of cases. The role of anaerobic bacteria in VAP is not well defined. Third, one multicenter study suggested that 8 days is an appropriate duration of therapy for nosocomial pneumonia, with a longer duration (15 days in that study) when the pathogen is *Acinetobacter* or *P. aeruginosa*. Finally, in febrile patients (particularly those who have endotracheal or gastric tubes inserted through the nares), occult respiratory tract infections, especially bacterial sinusitis and otitis media, should be considered.

SURGICAL WOUND INFECTIONS

Wound infections occur in ~500,000 patients each year, account for ~15–20% of nosocomial infections, contribute up to 7–10 extra postoperative hospital days, and result in \$3000 to \$29,000 in extra costs, depending on the operative procedure and pathogen(s). The average wound infection has an incubation period of 5–7 days—longer than many postoperative stays. For this reason and because many procedures are now performed on an outpatient basis, the incidence of wound infections has become more difficult to assess. These infections usually are caused by the patient's endogenous or hospital-acquired skin and mucosal flora and occasionally are due to airborne spread of skin squames that may be shed into the wound from members of the operating-room team. True airborne spread of infection through droplet nuclei is rare in operating rooms unless there is a “disseminator” (e.g., of group A streptococci or staphylococci) among the staff. In general, the common risks for postoperative wound infection are related to the surgeon's technical skill, the patient's underlying conditions (e.g., diabetes mellitus, obesity) or advanced age, and inappropriate timing of antibiotic prophylaxis. Additional risks include the presence of drains, prolonged preoperative hospital stays, shaving of operative sites by razor the day before surgery, long duration of surgery, and infection at remote sites (e.g., untreated UTI).

The substantial literature related to risk factors for surgical-site infections and the recognized morbidity and cost of these infections have led to national prevention efforts and to recommendations for

“bundling” preventive measures (Table 168-4). Additional measures include attention to technical surgical issues (e.g., avoiding open or prophylactic drains), operating-room asepsis, and preoperative therapy for active infection. Reporting surveillance results to surgeons has been associated with reductions in infection rates. Preoperative administration of intranasal mupirocin to patients colonized with *S. aureus*, preoperative antiseptic bathing, and intra- and postoperative oxygen supplementation have been controversial because of conflicting study results, but evidence seems mostly to favor these interventions.

The process of diagnosing and treating wound infections begins with a careful assessment of the surgical site in the febrile postoperative patient. Diagnosis of deeper organ-space infections or subphrenic abscesses requires a high index of suspicion and the use of CT or MRI. Diagnosis of infections of prosthetic devices, such as orthopedic implants, may be particularly difficult and often requires the use of interventional radiographic techniques to obtain periprosthetic specimens for culture. Cultures of periprosthetic joint tissue obtained at surgery may miss pathogens that are cloistered in prosthesis-adherent biofilms; cultures of sonicates from explanted prosthetic joints have been more sensitive, particularly for patients who have received antimicrobial agents within 2 weeks of surgery.

The most common pathogens in postoperative wound infections are *S. aureus*, coagulase-negative staphylococci, and enteric and anaerobic bacteria. In rapidly progressing postoperative infections manifesting within 24–48 h of a surgical procedure, the level of suspicion regarding group A streptococcal or clostridial infection (**Chaps. 173 and 179**) should be high. Treatment of postoperative wound infections requires drainage or surgical excision of infected or necrotic material and antibiotic therapy aimed at the most likely or laboratory-confirmed pathogens.

INFECTIONS RELATED TO VASCULAR ACCESS AND MONITORING

Intravascular device-related bacteremias cause ~10–15% of nosocomial infections; central vascular catheters (CVCs) account for most of these bloodstream infections. Past national estimates indicated that as many as 200,000 bloodstream infections associated with CVCs occurred each year in the United States, with attributable mortality rates of 12–25%, an excess mean length of hospital stay of 12 days, and an estimated cost of \$3700 to \$29,000 per episode; one-third to one-half of these episodes occurred in ICUs. However, infection rates have dropped steadily (Table 168-3) since the publication of guidelines by the Healthcare Infection Control Practices Advisory Committee (HICPAC) in 2002. With increasing care of seriously ill patients in the community, vascular catheter-associated bloodstream infections acquired in outpatient settings are becoming more frequent. Broader surveillance for infections—outside ICUs and even outside hospitals—will be needed.

Catheter-related bloodstream infections derive largely from the cutaneous microflora of the insertion site, with pathogens migrating extraluminally to the catheter tip, usually during the first week after insertion. In addition, contamination of the hubs of CVCs or of the ports of “needle-less” systems may lead to intraluminal infection over longer periods, particularly with surgically implanted or cuffed catheters. Intrinsic (during the manufacturing process) or extrinsic (on-site in a health care facility) contamination of infusate, although rare, is the most common cause of epidemic device-related bloodstream infection; extrinsic contamination may cause up to half of endemic bacteremias related to arterial infusions used for hemodynamic monitoring. The most common pathogens isolated from vascular device-associated bacteremias include coagulase-negative staphylococci, *S. aureus* (with ≥50% of isolates in the United States resistant to methicillin), enterococci, nosocomial gram-negative bacilli, and *Candida*. Many pathogens, especially staphylococci, produce extracellular polysaccharide biofilms that facilitate attachment to catheters and provide sanctuary from antimicrobial agents. “Quorum-sensing” proteins, a target for future interventions, help bacterial cells communicate during biofilm development.

Evidence-based bundles of control measures (Table 168-4) have been strikingly effective, eliminating almost all CVC-associated infections in one ICU study. Additional control measures for infections associated with vascular access include use of a chlorhexidine-impregnated