

Infection Control Resource

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Prevention Strategies for IC Practitioners and Professional Nurses

In this issue

Infection is one of the major complications of moderate to severe burns. The increasing antimicrobial resistance in microorganisms causing infection in hospitalized patients poses a challenge to burn care since it decreases the effectiveness of treatment and increases morbidity, mortality, and cost of care. As our authors, Drs. Khardori and Gulati, describe in their article, successful treatment of infection in the moderately to severely burned patient requires an understanding of common pathogens, mechanism of resistance to antimicrobial agents and infection control procedures. The outcome in burn patients can be optimized by a team approach including surgeons, ICU specialists, infectious disease clinicians, and infection control practitioners.

Intensive care units (ICUs) provide an environment to sustain life for severely ill patients. However, ICU patients are at substantial risk for acquiring healthcare-associated (nosocomial) infections. Of the approximately 2 million patients who develop nosocomial infections annually in the United States, 5% to 35% of these patients are in an ICU. Ventilator-associated pneumonia, intravascular catheter-related bloodstream infections, catheter-associated urinary tract infections, and surgical-site infections account for the majority of nosocomial infections. In her article, Ms. Hebden focuses on device-associated infections—specifically, risk factors for acquiring infections and prevention strategies.

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Management of infections in patients with moderate to severe burns

by Nancy Khardori, MD, Salil Gulati, MD

There is an estimated incidence of 1.25 million burns annually in the US.¹ Of these, 80% involve less than 20% of the total body surface area. Scalds are the most common cause and are responsible for more than 100,000 emergency room visits annually. Approximately 60,000 of the total burns require admission to the hospital. Children younger than 5 years and adults 65 or more years old are the most severely injured groups. The frequency of death after burn injury is determined by prior health condition, age, inhalation injury, and burn wound sepsis. In 1998 more than 3,000 burn-related deaths were reported in the U.S.A.¹

Severe or critical burns include third-degree burns involving more than 10% of body surfaces or critical body parts (e.g., face, hands, feet, or perineum) and burns complicated by respiratory tract injury, major soft-tissue injury, or fractures.² Moderate burns are defined as second-degree burns involving 15%–25% of body surface and third-degree burns involving 10% of body surface. Third-degree burns are full-thickness injuries; first- and second-degree burns are partial-thickness injuries. Partial-thickness burns can be superficial or deep.

Significant advances have been made in the general care of burn patients; however, infections remain a major cause of morbidity and mortality in patients with moderate to severe burns.³ It is estimated that up to 75% of deaths in patients with burn injury are related to infection.^{4,5} In addition, the resistance of various bacteria infecting burn patients decreases the effectiveness of treatment and increases morbidity, mortality, and cost of care.

Risk factors for infection

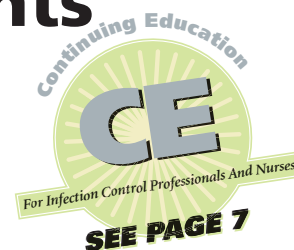
Thermal injury destroys the first line of defense against infection by disrupting the skin integrity. Initially the major organisms for burn wound infection are gram-positive skin flora that survive the burn.⁶ Over time, gram-negative bacteria colonize the eschar and become the pre-

Infections remain a major cause of morbidity and mortality in patients with moderate to severe burns.

dominant organisms by the end of the first week. Treatment with broad-spectrum antimicrobial agents leads to selection of multi-resistant organisms such as methicillin-resistant *Staphylococcus aureus* (MRSA), vancomycin resistant *Enterococcus* (VRE), and *Pseudomonas aeruginosa*. The indwelling catheters and monitoring devices that allow optimal management of fluids, electrolytes, and nutrition can become colonized and lead to systemic infections. Patients with severe burns often require intubation and respiratory support and are at high risk for ventilator-associated pneumonia (VAP). This is particularly common in burns complicated by respiratory-tract injury, due to the loss of mucosal barrier. The focus of immediate burn care is to prevent progression of injury and to maintain a viable interface at which both nonspecific and specific defenses against infection can be mounted.⁷

Patients with large burns are predisposed to systemic infection because of depression of nonspecific as well as specific (antibody-mediated and cellular) immune functions. The response of neutrophils to a site injury and antigen challenge, using the skin-window technique, was shown to be depressed in patients with 40% or greater burn.⁸ Depressed neutrophil response in vivo has been confirmed by quantifying the response to head-killed staphylococci in burn patients.⁹

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Infection control in the intensive care unit

by Joan N. Hebden, RN, MS, CIC

Intensive care units (ICUs) provide an environment that frequently sustains life for severely ill patients. However, ICU patients are at substantial risk for acquiring healthcare-associated (nosocomial) infections. Of the approximately 2 million patients who develop nosocomial infections annually in the United States, 5% to 35% of these patients are in an ICU.¹ The ICU patient population is more susceptible to infection for several reasons: acuteness of illness; immunocompromised state; the use of invasive devices for physiologic support and hemodynamic monitoring; the widespread use of antibiotics, which predispose to colonization with resistant bacteria such as methicillin-resistant *Staphylococcus aureus* (MRSA) and vancomycin-resistant *Enterococcus* (VRE); and the intensity of care provided by busy clinicians, which enhances the opportunity for cross-contamination. These infections lead to prolonged ICU and hospital length of stay, increased cost of care, increased resource utilization, and excess mortality.

Types of infections

Ventilator-associated pneumonia (VAP), intravascular catheter-related bloodstream infections (BSI), catheter-associated urinary tract infections (UTI), and surgical-site infections (SSI) account for the majority of nosocomial infections. Infection rates vary according to the type of ICU and the case mix of patients in the unit. The standard for many hospitals is the risk-adjusted infection rates published by the National Nosocomial Infections Surveillance System (NNIS) as shown in table 1.² It is important to note that using these rates as a comparative benchmark requires using the published Centers for Disease Control and Prevention (CDC) standardized infection definitions and device-day denominators.

When considering all ICU patients, data reported from Vincent et al³ in the European Prevalence of Infection in Intensive Care study show that pneumonia is the most common infection (46.9%), followed by lower respiratory tract infection other than pneumonia (17.8%), urinary tract infection (17.6%), and bloodstream infection (12%). Among surgical patients, surgical-site infections is the most common nosocomial infection and accounts for 38% of all such infections.⁴ (See the sidebar "Surgical-site Infections" for more information.)

This article focuses on device-associated infections: their impact on quality of care, risk factors for their acquisition, and prevention strategies.

Ventilator-associated pneumonia

Ventilator-associated pneumonia (VAP) generally refers to bacterial pneumonia that complicates the clinical course of 8% to 28% of patients requiring mechanical ventilation.⁵ The primary mode of entry of pathogenic bacteria into the lower respiratory tract is through aspiration of oropharyngeal secretions. The pathogens are predominantly gram-negative in origin and rapidly colonize the oropharynx after hospitalization. Invasive devices such as nasogastric and endotracheal tubes facilitate bacterial colonization of the tracheobronchial tree and predispose the patient to gastric reflux and aspiration. Early-onset VAP occurs within 72 hours after tracheal intubation and is most often due to community acquired pathogens such as *Haemophilus influenzae*, *Streptococcus pneumoniae*, and methicillin-sensitive *Staphylococcus aureus*. Late-onset VAP occurs after the first 3 days of mechanical ventilation and is frequently associated with *Pseudomonas aeruginosa*, other gram-negative pathogens, and MRSA. The attributable mortality rate for VAP when associated with late-onset pathogens has been suggested to be greater than 10%.⁵ In addition to the excess mortality, the economic ramifications are significant: it is estimated that an ICU patient on mechanical ventilation will have a prolonged hospital stay of 10 to 13 days, incurring approximately \$5,700 in extra charges.⁶

In light of the adverse clinical outcomes and increased costs associated with VAP, targeted reduction in the incidence should be a goal for all institutions that provide care to mechanically ventilated patients. The feedback of VAP rates to the ICU clinicians, as part of a continuous quality improvement program, is essential to achieve their endorsement and support of the initiative. Kollef⁵ proposes a program that includes nonpharmacologic and pharmacologic strategies to reduce the burden of colonization in the aerodigestive tract and to decrease the potential for aspiration:

- Wash hands between patient contacts.
- Place patients on mechanical ventilation in a semirecumbent position.
- Take measures to prevent unplanned extubation, such as appropriate use of restraints and securing the endotracheal tube to the patient.
- Avoid gastric overdistension.
- Avoid nasal intubation, as it predisposes the patient to sinusitis.
- Regularly remove condensate from ventilator circuits, as a high concentration of pathogenic bacteria is found in this fluid.

Surgical-site Infections

Surgical-site infection (SSI) is the most common nosocomial infection among surgical patients.⁴ The incidence and prevalence of postoperative infections vary depending on the definition implemented in the surveillance effort, the acuteness of the patients' illness, and the intensity of the surveillance.²² It is estimated that SSIs occur in 2% to 5% of patients after extra-abdominal surgery (e.g., orthopedic procedures) and in up to 20% of patients undergoing intra-abdominal procedures.²³⁻²⁷ Studies following patients into the post-discharge period have reported even higher rates of postoperative infection.^{28,29} These complications increase morbidity for patients and consume substantial additional resources.³⁰⁻³²

A major concern in SSIs—as in all infections—is the possibility that the pathogen is an antibiotic-resistant organism. There is, as a consequence, an increased interest in wound dressings that do not use antibiotics. Formulations with silver, iodine, and polyhexamethylene biguanide (PHMB) are increasingly popular. Examples of these newer dressings are Excilon AMD, Telfa AMD, and Kerlix AMD (Tyco Healthcare/Kendall), all of which contain PHMB. PHMB has shown efficacy against MRSA,³³ *P aeruginosa*, VRE, and *C albicans*.^{34,35}

(Information in this sidebar is supplemented by "Prevention and treatment of surgical-site infections" by Nancy Tomaselli in Infection Control Resource vol. 2 no. 1, available at <http://www.infectioncontrolresource.org>)

- Suction secretions pooled above the inflated endotracheal tube cuff.
- Maintain adequate pressure in the endotracheal tube cuff.
- Apply chlorhexidine oral rinses.
- Avoid unnecessary use of antibiotics to reduce the development of antibiotic-resistant nosocomial infections.
- Vaccinate against *S pneumoniae*, *H influenzae* type b, and influenza viruses.
- Choose stress-ulcer prophylaxis according to the patient's situation. The choice of sucralfate versus H₂ blockers is controversial.

Intravascular-catheter related bloodstream infections

Intravascular catheters are indispensable in critical care, being used for hemodynamic monitoring as well as administering fluids, electrolytes, drugs, blood products, and nutritional support. Central venous catheters (CVCs) pose the greatest risk of catheter-related bloodstream infections (CR-BSI). Data from the CDC show that, in the USA, approximately 80,000 CR-BSIs occur annually in ICUs. The attributable mortality from these infections has ranged from 12% to 25% with an estimated cost as high as \$29,000 and an

excess length of stay in the ICU of 6.5 days.⁷

CVCs become colonized with organisms from the skin surrounding the insertion site or from the catheter hub. Short-term noncuffed catheters, which are percutaneously inserted into the subclavian or internal jugular vein, are intended to remain in place for less than 30 days. The pathogenesis of infection for these catheters is primarily related to extraluminal colonization by organisms of cutaneous origin that invade at the time the catheter is inserted or in the days after insertion. The most common pathogens are coagulase-negative staphylococci (CNS), the predominant aerobic species on the human skin.

Long-term CVCs can be nontunneled (i.e., peripherally inserted) or tunneled (e.g., Hickman® or Broviac® (Bard) catheters that incorporate a Dacron cuff and implantable ports). These catheters generally remain in place longer than 30 days. The CR-BSI rates for tunneled catheters are substantially lower than the rates associated with short-term catheters. With long-term catheters, intraluminal colonization is the most common route of infection. The catheter hub, contaminated by the hands of health-care workers, is the major source colonizing the catheter lumen. Microorganisms migrate down the endoluminal surface of the catheter to the bloodstream. As with extraluminal colonization of the catheter, CNS are the leading pathogens responsible for these infections.

Although it occurs infrequently, a CVC can also become colonized hematogenously from a remote site of infection or from contaminated infusate. Other factors can contribute to microbial colonization of the catheter: post-insertion binding of organisms to the thrombin sheath that covers the internal and external surfaces of the catheter; adherence of organisms—such as CNS, *S aureus*, *Candida parapsilosis*—to the catheter surface (these organisms produce a biofilm that provides protection from antimicrobial agents); the material from which the catheters are made; and clinical interventions such as total paren-

teral nutrition and lipid emulsions.⁸

A definite diagnosis of CR-BSI usually requires removal of the catheter for semiquantitative culturing of the catheter tip. To avoid unnecessary removal of catheters, the diagnosis of CR-BSI should be made on the basis of both clinical and microbiologic findings. Accurate microbiologic data are essential to determine the causative agent and appropriate antimicrobial therapy; therefore, clinical staff must adhere to established procedural guidelines when obtaining blood samples. Recently published guidelines recommend that patients with suspected CR-BSI should have two separate blood samples drawn, with at least one drawn percutaneously.⁹ Using an existing intravascular line to draw the sample is discouraged because of the high risk for contamination with organisms that are colonizing the catheter hub.

Prevention strategies for CR-BSI published by the CDC¹⁰ focus on practices that reduce extraluminal and intraluminal contamination of the catheter. Various practices can help to minimize extraluminal contamination of the catheter:

- Provide proper skin antisepsis with 2% chlorhexidine before insertion.
- Make the subclavian vein the preferred site for insertion of CVCs. [Data show that insertion into the internal jugular vein leads to a higher risk of catheter colonization.]
- Maximize sterile barrier precautions during insertion; this includes sterile gown, gloves, cap, and mask, and full-body sterile drape.
- Formalize training in sterile techniques for physicians who will insert CVCs.
- Remove CVCs as soon as possible, and do not routinely replace them.
- Use appropriate dressings on CVCs. There is no recommendation for the use of a particular type of dressing.
- Maintain adequate nurse-to-patient ratios in the ICU.

To minimize intraluminal contamination of the catheter:

- Minimize the number of manipulations of the catheter hub(s).
- Perform hand hygiene before any manipulation of the catheter.
- Disinfect all catheter sampling ports with 70% alcohol before they are accessed.

Impregnating CVCs with antimicrobial agents is a new approach for preventing CR-BSI. Maki et al¹¹ reported that polyurethane CVCs coated on the external surface with chlorhexidine and silver sulfadiazine were twofold less likely to be colonized and fourfold less likely to produce bloodstream infection as compared with uncoated catheters. Catheters impregnated with minocycline and rifampin on their internal and external surfaces also were associated with a reduction in catheter colonization and CR-BSI.¹² The use of these catheters is recommended for institutions

Catheters: Silicone vs. Latex

- Latex is an allergen for many people, and it also has been shown to increase the incidence of urethritis (22%) as compared with silicone catheters (2%).³⁶⁻³⁸
- Use of all-silicone catheters prevents urethral strictures.^{39,40}
- Some studies suggest that all-silicone catheters may offer some resistance to bacterial adherence and biofilm formation, delaying onset of bacteriuria.^{41,42}
- All-silicone catheters resist encrustation longer than latex catheters.⁴³

(Information in this sidebar is abstracted from articles by T. Achmetov and M. Gray and by K.N. Moore in Infection Control Resource vol. 2 no. 2, available at <http://www.infectioncontrolresource.org>)

that have not achieved a targeted reduction in CR-BSI despite the implementation of other prevention strategies.

Catheter-associated urinary tract infections

Urinary tract infections are among the most common nosocomial infection, accounting for over 40% of the total.² These infections are almost exclusively related to the presence of a urinary catheter, with the risk of infection strongly associated with the duration of catheterization. The incidence of bacteriuria is approximately 5% per day of catheterization.¹³ Catheter-associated UTIs cause bacteremia in 0.5% to 4% of patients, are estimated to be directly responsible for 5% of deaths from nosocomial infections, and can prolong hospital length of stay by 1 to 3 days with excess cost ranging from \$589 to \$3,800.^{13,14}

The pathogenesis of UTI is multifactorial and includes introduction of bacteria colonizing the periurethral area at the time of insertion, migration of periurethral or perianal bacteria along the exterior of the catheter (extraluminal migration), and migration of bacteria from the drainage bag along the interior of the catheter (intraluminal migration).¹⁴ The organisms predominantly responsible for UTI are normal intestinal flora, with *Escherichia coli* the most common of the bacteria isolated. However, in the ICU, *candida* species have accounted for a larger proportion of UTIs (25%), followed by *E coli* (18%), enterococci (13%), *P aeruginosa* (11%), and enterobacter species (6%).²

Strategies to reduce the incidence of catheter-associated UTIs depend in part on novel closed urine drainage systems but fall primarily within the milieu of nursing interventions. Certain practices prevent extraluminal and intraluminal entry of pathogens into the urinary tract:¹⁴

- Avoid catheterization if possible.
- Minimize the duration of catheterization.

Table 1. Device-associated median infection rates stratified by intensive care unit (ICU) and anatomic site²

Type of ICU	UTI*	BSI*	VAP*
Burn	7.3	7.3	NA
Coronary	4.7	4.2	3.1
Cardiothoracic	2.5	2.2	5.1
Medical	5.5	5.0	3.6
Med-Surg (major teaching)	4.9	4.9	4.9
Med-Surg (non-major teaching)	3.5	3.3	5.6
Neurosurgical	6.7	4.1	9.6
Pediatric	4.3	5.9	2.2
Surgical	4.4	4.7	8.3
Trauma	6.7	6.6	NA

* Infections per 1,000 device-days
NA = not available for the entire referenced period

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Management of infections in patients with moderate to severe burns — continued

Circulating levels of immunoglobulins (antibodies) are decreased in proportion to the extent of injury, and persistently decreased levels of immunoglobulin G (IgG) have been related to mortality. Reversal of the ratio of T-helper (CD4) to T-suppressor (CD8) lymphocytes, and monocyte dysfunction, lead to suppression of cellular immune responses. Multiple cytokines including interleukin 1, interleukin 6, and tumor necrosis factor (TNF α) are elevated in severely burned patients. These increased levels are associated with increased rates of infection, morbidity, and mortality. Activation of coagulation pathways and generation of kinin have been implicated in the initiation of this exaggerated inflammatory response. Similar cytokine cascades are known to be activated in sepsis syndrome, a common complication of severe burns. This leads to significant difficulties in differentiating a systemic inflammatory response syndrome due to burns from that induced or augmented by sepsis syndrome.

Microbiology

The invasiveness of the organisms is related to degree of injury, disturbance of normal flora, virulence of the organism, extent of external contamination, and invasiveness of the patient's endogenous gastrointestinal and upper respiratory tract flora. Many studies have reported the prevalence of aerobic bacteria such as *P. aeruginosa*, *S. aureus*, *Escherichia coli*, *Klebsiella* subspecies, and *Enterococcus* subspecies. In studies using stringent microbiological techniques, anaerobic bacteria constituted 11%–31% of the burn wound isolates.¹⁰ *Peptostreptococcus* subspecies, *Bacteroides* subspecies, and *Propionibacterium acnes* were the predominant anaerobic organisms. *Bacteroides* subspecies were cultured from the wounds of 82% of patients who developed septic shock.¹¹

Over the past 10 years there has been an increase in fungal infections of burn wounds. Fungal infections are often preceded by bacterial infection and broad-spectrum antibacterial therapy. Infections caused by molds including *Aspergillus* subspecies can lead to local wound discoloration. Histologic section of wound biopsies is most reliable in determining invasion of viable tissue. Infections due to *Candida* subspecies are difficult to differentiate clinically from those caused by bacteria. Systemic infections due to fungi secondary to burn-wound infection may be difficult to document microbiologically. Systemic antifungal therapy is often used, even without positive blood cultures, based on clinical signs and tissue invasion.

Diagnosis

A microbiological workup of wounds involves various sampling methods. Swab and

Various types of topical agents and dressings are used to prevent wound sepsis and restore the barrier function.

wound-fluid samples are neither sensitive nor specific for the diagnosis of burn-wound infection. Deep-tissue biopsy following initial debridement of the superficial debris is considered most useful for determining the presence and the microbial loads of pathogens.¹² Tissue samples are cultured for both aerobic and anaerobic organisms. Because of the complex ecosystems in burn wounds, poor correlation between gram stain and culture results from deep-tissue biopsy specimens has been reported.¹³ The gold standard for diagnosing invasive fungal infections is histopathologic assessment of the tissue biopsy.

Patients with serious burns can have infections other than or in addition to the infection of the burn wounds. Most prominent among them are pulmonary infections. Nosocomial and VAPs occur in patients with and without respiratory tract injury. Microbiological sampling of the respiratory tract secretions may or may not be helpful in determining the exact cause of infection; however, absence of the growth of known respiratory pathogens points to noninfectious causes of pulmonary complications in burn patients, particularly adult respiratory distress syndrome (ARDS).

Urinary tract infections in burn patients are primarily due to the presence of indwelling catheters. Urine culture results are difficult to interpret because of a very high prevalence of colonization of the catheters.

Bloodstream infections (BSI) leading to sepsis syndrome and septic shock are responsible for the significant infection-related mortality in burn patients. BSI sources include burn wounds and the respiratory, urinary, and gastrointestinal tracts. In addition, indwelling vascular catheters entering through burned areas of the body are a predominant cause of bloodstream infection. Samples for culture drawn at the same time through an indwelling central venous catheter and the peripheral veins help in the decision for removing a central venous catheter. The diagnosis of suppurative thrombophlebitis is considered even in the presence of normal tissue, because local findings are seen in less than 35% of burned patients. The incidence of this complication is at least 5%. The addition of injured and necrotic tissue makes the clinical diagnosis even more difficult. The mortality rate in appropri-

ately treated patients reaches 60%.

Both positive and negative culture results in burn patients need to be interpreted in correlation with the clinical picture and prior/current antimicrobial therapy.

Prevention**Debridement and excision**

Nonviable burn tissue provides a growth medium for bacteria. Serial removal of such tissue prevents colonization and subsequent local and systemic infection. Historically, burns have been treated by waiting for the dead skin to separate from the deeper viable tissues by autolytic debridement. Septicemia leading to multi-organ dysfunction was common with this technique. In the past few decades, various proteolytic agents such as collagenase have been used to debride the nonviable tissues for smaller localized areas. There are concerns regarding the nonselective nature of some of these enzymatic agents that can lead to damage in unburned areas. In the 70s the eschar was tangentially excised until bleeding tissue was encountered then followed by split skin thickness grafting. Significant bleeding was a disadvantage with this technique, and a technique of fascial excision was used. Surgical excision is the standard of care in major burns, and autolytic and enzymatic methods are used adjunctively or in unstable patients.

Culture surveillance

The role of surveillance cultures of wounds in identifying and monitoring infectious complications in burn patients is not clear. Quantitative and semiquantitative culture techniques of burn biopsy have been used to predict colonization versus infection.¹⁴ The semiquantitative method, which is cost effective and time saving, offers reproducible and efficacious results. The breakpoint used in most diagnostic laboratories is 105 colony-forming units per gram of tissue.

Infection-control practices

Healthcare providers and visitors prevent cross-contamination of wounds by using gowns, gloves, and masks. Hand hygiene using conventional soap and water or waterless hand sanitizers is the single most important aspect of infection prevention. Hand hygiene procedures are performed before and after the use of gloves. Noncritical monitoring and diagnostic equipment are dedicated to each patient. Private rooms for patients aid in containment of organisms and facilitate isolation. To eliminate endemic or breakout infections, patients with the same infection can share rooms. Immunosuppressive therapy and designated burn units with special ventilation and restricted access enhance allograft acceptance in patients with extensive burns.¹⁵

In large burn centers, traditional Hubbard tanking of patients for debridement has the potential for cross-contamination, especially when proper decontamination of the tank between patients is erratic; a good alternative

is showering and debridement on a covered or readily disinfected plinth. HEPA (high-efficiency particulate air) filters in ventilation systems and monitoring of molds in the air may reduce the incidence of fungal wound infections.¹⁶

Barrier protection

The skin is a dynamic organ that protects against microbial environmental agents, among other functions. In the event of a thermal injury to the skin, the barrier function of the skin is lost, making the body vulnerable to multiple infectious agents. The immunological defenses of the body are compromised in severe burns.¹⁷ The insensible losses of body fluids are also increased after the skin loses its regulatory function of fluid conservation. Various types of topical agents and dressings are used to prevent wound sepsis and restore the barrier function.

Topical agents

- 1% silver sulfadiazine (Thermazene, Tyco Healthcare/Kendall; Silvadene, Monarch) is the topical agent most frequently used. It has moderate penetration into eschar and has in vitro action with several agents including *S aureus*, *P aeruginosa*, and *Candida albicans*. Its basic function is the slow release of silver into the superficial wound environment.¹⁸ A transient leukopenia is the most common complication, occurring in 5%–15% of cases, and resolves in a few days without an increase in the incidence of infection.¹⁹ Sulfadiazine cream is applied once or twice a day, is relatively painless, and does not stain fabrics.²⁰ It has a tendency to form a thick dry adherent layer over the burn wound that masquerades as a pseudo eschar.
- Cerium nitrate-silver sulfadiazine is not only available commercially in the U.S.A. but is available in Europe. Cerium is a rare earth element that has in-vitro microbicidal activity and is relatively nontoxic. Cerium nitrate-silver sulfadiazine is reported to be superior to silver sulfadiazine for the prophylaxis of moderate to severe burns.²¹
- Mafenide acetate (Sulfamylon, Bertek), a carbonic anhydrase inhibitor, is available as 11.1% water-soluble cream or as a 5% solution. Its application is reported to be painful but is controlled with analgesics. The cream is associated with hyperchloremic acidosis when it is used continuously for larger burns.²² However, the solution does not seem to cause acidosis, even when used on large areas for a prolonged period.²³ Excellent eschar penetration by the cream makes it an ideal candidate to treat deep helix burns because of the poor penetration of systemic antibiotics within the cartilage.²⁴ Mafenide acetate has excellent activity against gram-positive species except MRSA. It has good activity against gram-negative bacteria but has minimal antifungal activity.¹⁷ Nystatin is frequently

added to mafenide solution, enhancing its spectrum of antimicrobial activity to include fungal agents.^{25,26} Mafenide solution is rapidly absorbed through open wounds and requires frequent application.

- 0.5% silver nitrate solution is active against *Staphylococcus*, *Pseudomonas*, and many other gram-negative aerobes. The solution does not penetrate eschar, and it also leaches electrolytes from the wound, rapidly causing hyponatremia and hypokalemia in large burns. It stains all linen and dressing materials and can cause methemoglobinemia.
- Other topical agents such as polymyxin B and bacitracin are infrequently used on deeper burns but are commonly prescribed for more superficial burns. Bacitracin has efficacy against gram-positive bacteria.²⁷ Its use is limited by poor penetration of the ointment in dead eschar and it is not used for deeper burns.

Burn dressings

Burn dressings perform the following functions:¹⁷

- protective: They isolate burn wounds and minimize the growth of microorganisms on the surface by acting as a barrier.
- metabolic: They reduce evaporative heat loss and dehydration; this also prevents increased caloric requirements to maintain temperature.
- comforting: Exposed nerve endings in the wound bed are sensitive to air current. Bandages provide comfort by protecting from air; they also absorb drainage and act as a splint if enough bulk is incorporated into the dressing.

Burn dressings usually consist of three layers: an inner or contact layer, a middle absorbent layer, and an outer holding layer.

The inner or contact layer can be an antibiotic cream such as 1% silver sulfadiazine or mafenide acetate for deep burns. Partial-thickness burns or donor sites are usually dressed with a fine-mesh petrolatum gauze such as Adaptic (Johnson & Johnson) or Xeroform (Tyco Healthcare/Kendall). Adaptic is nonmedicinal; Xeroform is impregnated with 3% bismuth tribromophenate, has bacteriostatic action, comes in a sterile package, and conforms to body contour. Scarlet Red (Tyco Healthcare/Kendall) is o-tolylazo-o-tolylazo-(beta) naphthol 5% in white petroleum, lanolin, and olive oil on fine mesh gauze; it promotes re-epithelialization in donor sites,²⁸ comes as sterile strips, and conforms to all body parts.

In clean partial-thickness wounds, different occlusive dressings are commonly used as the inner layer. There are polyurethane-derived membranes and transparent films such as Polyskin II (Tyco Healthcare/Kendall), OpSite (Smith & Nephew), and Tegaderm (3M Nexcare). Semipermeable dressings with soluble collagen dressing or gelatin backing (Ultec, Tyco Healthcare/Kendall; DuoDerm,

ConvaTec; Biobrane, Bertek) along with hydrogels (Curagel, Tyco Healthcare/Kendall) are also used. These dressings might create an environment conducive for microbial growth if they are placed in contaminated wounds. A layer of wound exudate beneath these dressing may masquerade as pus.²⁹ Kerlix AMD (Tyco Healthcare/Kendall) applied as a first contact layer could be an important adjunct to decrease aerobic, anaerobic, and fungal polymicrobial bioburden of burn wounds.³⁰ This gauze dressing is impregnated with 0.2% polyhexamethylene biguanide (PHMB), an antimicrobial agent that is non-cytotoxic.^{31,32} PHMB as found in Kerlix AMD was cleared by the FDA for use as an effective barrier to bacteria and has shown efficacy against MRSA,³⁰ *P aeruginosa*,³³ VRE,³⁴ and *C albicans*. PHMB is safe and is commonly used as a substitute for chlorine in swimming pools and contact-lens cleanser. Unlike antibiotics, this antiseptic's mechanism of action does not involve efflux pumps; thus, at this time, it has not shown bacterial resistance.

In contrast to silver nitrate, Acticoat (Smith & Nephew) and Silverlon (Argentum Medical) give more controlled, prolonged release of silver to the wound area.³⁵ Skin may turn gray in response. Acticoat is a three-ply dressing with an absorbent rayon/polyester core between two layers of nanocrystalline silver-coated polyethylene mesh. This nanocrystalline coating allows for rapid exposure to water and subsequent silver ion and silver radical release.³⁵ Silverlon is a silver-plated three-dimensional polyamide fabric that utilizes a proprietary autocatalytic electroless chemical (reduction-oxidation) plating technology.

Aquacel hydrofiber (ConvaTec) is a moisture-retentive topical dressing. Recently, 1.2% by weight silver has been added to the product to create Aquacel Ag. Aquacel Ag releases silver within the dressing for up to two weeks, making it very convenient in the management of partial-thickness burns.³⁶

The absorbent layer consists of loose gauze fluff that absorbs exudates from the wound, keeping the wound moist for faster healing and acting as a barrier against bacteria. A bulky layer acts as a splint in extremities. For large areas, sterile absorbent pads such as Intersorb (Tyco Healthcare/Kendall) are frequently used. Intersorb consists of a fine-mesh lint-free gauze cover with stitched edges and a multi-layer cellulose filling. It comes in sterile packing and different sizes. Intersorb is also available as vests that secure the inner layer.

The outer layer holds the dressing in place. Kerlix rolls are commonly used for this purpose in the extremities. They are made of pre-washed, fluff-dried 100% woven gauze with a crinkle-weave pattern for bulk to cushion and protect wound areas. Kerlix is slightly elastic (permitting some swelling of the wound) with finished edges that reduce loose ends and lint. Kerlix AMD's broad-spectrum effectiveness provides protection against gram-negative, gram-positive, and fungal/yeast microorgan-

isms, and its use does not require a change of protocol. An added benefit is the possibility that it may help inhibit cross-contamination from patient to patient, patient to clinician, and patient to the environment.

Selective decontamination of the gastrointestinal tract

The burn patient's own gastrointestinal (GI) tract can act as a reservoir for potentially pathogenic microorganisms.³ Evidence suggests that the intestinal integrity of burn patients may be impaired. Bacterial translocation from an otherwise intact GI tract occurs in animals with thermal injury.^{37,38} In contrast, the importance of indigenous anaerobic bowel flora in preventing colonization of the GI tract by gram-negative bacilli has been established in experimental animals and human volunteers. Selective decontamination of the digestive tract using nonabsorbable antimicrobial agents that spare the anaerobic flora was shown to reduce colonization and infection rates in ICU patients, including trauma patients.³⁹ This approach was of value in the management of severe burns (>30% total body surface area) and inhalational injury.³ The regimen for adults consisted of tobramycin (80 mg), polymyxin E (100 mg), and amphotericin B (500 mg) given orally or through the nasogastric tube four times a day. Patients on mechanical ventilation also received these antibiotics as a 2% paste applied to the oral cavity four times daily.

Conclusion

The outcome in burn patients can be optimized by a multidisciplinary, holistic team approach that includes but is not limited to burn surgeons, critical-care specialists, infectious-disease clinicians, microbiologists, infection-control practitioners, nutrition experts, and other specialists as needed.⁶ Patients with severe burns remain hospitalized for prolonged periods, requiring multiple debridements, skin grafting, and various antibiotic regimens. While treating these patients aggressively, it is important to consider the selection pressure that antibiotics exert on the microbial flora and to remain vigilant of changes in antimicrobial susceptibility patterns.

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Infection control in the intensive care unit — continued

- Use staff trained in aseptic insertion of the catheter.
- Perform hand hygiene prior to insertion of the catheter.
- Perform meatal care with soap and water daily and as necessary.
- Maintain a closed drainage system.
- Obtain specimens in an aseptic fashion.
- Keep the drainage bag below the level of the bladder.

As mentioned with intravascular catheters, urinary catheters also develop biofilm encrustations, which contribute to infection. Numerous randomized trials have found that urinary catheters coated with silver hydrogel are effective in reducing the incidence of catheter-associated complications.^{14,15}

Factors contributing to the transmission of infection in ICUs

Hands of healthcare workers

It has long been recognized that the single most important procedure to prevent and control the transmission of infection is routine hand hygiene before and after patient contact. This practice is of particular concern in the ICU because of the number of interactions between healthcare workers and patients. Pittet et al¹⁶ found handwashing compliance to be poorer in ICUs (as opposed to internal medicine units) and during procedures associated with a high risk of contamination. They also found that the higher the intensity of care, the lower the compliance with handwashing. The lack of time to perform hand hygiene and the detrimental effects on the skin from frequent washing have been cited by healthcare workers as factors contributing to poor compliance. Recent studies have found that the introduction of alcohol-based handrub products in ICUs has resulted in a marked and sustained increase in compliance with hand-hygiene measures.¹⁷ The CDC has endorsed the use of these products in their recommendations for hand hygiene.¹⁸

Staffing in the ICU

Understaffing has been identified as a potential risk factor for the acquisition of nosocomial infections. Fridkin et al¹⁹ reported a high patient-to-nurse ratio as an independent risk factor for CR-BSI occurring in a surgical ICU. Needleman et al²⁰ found a positive association between the proportion of total hours of nursing care provided by registered nurses or the number of hours of care by registered nurses per day and 6 outcomes of care in medical patients (i.e., length of stay, UTI rates, pneumonia, upper gastrointestinal bleeding, and shock or cardiac arrest). Due to the need for isolation of patients with antibiotic-resistant bacteria, research to assess the impact on nurse workload is appearing in the literature. Saulnier and colleagues²¹ found a substantial increase in nurse workload for management

of patients with antibiotic-resistant bacteria based on 4 functional tasks required for their care. They concluded that this assessment is important in determining the number of nurse hours needed to comply with infection control recommendations.

Conclusion

The sophisticated technology used in ICUs to save lives is associated with a substantial risk of device-associated infection. These infections result in significant morbidity, mortality, and excess costs. Many of these infections are preventable. Therefore, an understanding of the pathogenesis underlying these infections and the recommended prevention strategies is essential for critical-care clinicians. Moreover, clinicians should have an understanding about device selection and maintenance. Hospital administrators need to carefully assess the burden of care in ICU's in order to maintain optimal staff-to-patient ratios.

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This continuing nursing education activity was approved by the Vermont State Nurses Association Inc. (VSNA) an accredited approver by the American Nurses Credentialing Center's Commission on Accreditation.

Upon completion of this offering the learner will be able to:

- Recognize the various types of topical agents and the dressing materials used in partial-thickness and deep burns along with the rationale for their use.
- List the risk factors, pathogenesis, and causes of infections in patients with moderate to severe burns.
- Discuss the role of various management strategies for infections in burn patients.
- Discuss the risk factors that contribute to the increased susceptibility to infection of ICU patients.
- Describe the pathogenesis of infection for each of the device-associated infections.
- Recognize three prevention strategies for each of the device-associated infections.

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- The most common side effect from the use of 1% silver sulfadiazine cream for burns is:
 - staining of tissues
 - hyponatremia
 - skin rash
 - transient leukopenia
- Which is NOT true about topical antimicrobial agents as first-line prophylaxis for deep burns with eschar (before surgical excision)?
 - 1% silver sulfadiazine has moderate penetration into eschar
 - Cerium nitrate-silver sulfadiazine has antimicrobial activity
 - Mafenide acetate is painless and has minimal penetration into eschar
 - Silver nitrate solution does not penetrate eschar
- The most common bacteria seen in infections of burn wounds include all except:
 - P aeruginosa*
 - S aureus*
 - Legionella pneumophila*
 - E coli*
- The growth of *Aspergillus* subspecies or other types of molds in burn wounds is generally treated with systemic antifungal therapy even without positive blood cultures.
 - True
 - False
- Essential infection prevention practices include:
 - hand hygiene between all patient contacts, and after glove use
 - semi-private room assignments
 - dedicating equipment to one patient whenever possible
 - a and c
- The sources for bloodstream infection in burn patients include:
 - gastrointestinal flora
 - urinary tract
 - central venous catheters
 - all of the above
- Which of the below is NOT a risk factor for infection in burn victims?
 - Destruction of the skin integrity
 - Worker shortage causes ill employees to care for burn victims
 - Depression of the immune system.
 - Indwelling catheters and monitoring devices becoming colonized and leading to systemic infection
- ICU patients are more susceptible to infection for which of the following reasons:
 - the use of invasive devices
 - the intensity of care
 - widespread use of antibiotics
 - all of the above
- Which healthcare-associated infection is associated with a prolonged hospital stay of 10-13 days?
 - urinary tract infection
 - intravascular-catheter related bloodstream infection
 - ventilator-associated pneumonia
 - surgical-site infections
- Intravascular catheter colonization appears to be inhibited by impregnating the surface with:
 - chlorhexidine
 - silver sulfadiazine
 - minocycline/rifampin
 - all of the above
- Which of the following are recommended infection prevention strategies for ventilator-associated pneumonia?
 - maintaining the patient in a semirecumbent position
 - routine replacement of the endotracheal tube
 - continuous subglottic suctioning
 - a & c
- The availability of alcohol-based handrubs in the ICU setting has been associated with:
 - poor acceptance by healthcare workers
 - a sustained increase in compliance with hand hygiene
 - skin irritation in healthcare workers' hands
 - skin irritation in patients
- The most important risk factor associated with the development of urinary tract infection in catheterized patients is:
 - performing meatal care with soap and water
 - traumatic insertion
 - duration of catheterization
 - lack of aseptic technique during insertion

Mark your answers clearly with an "X" in the box next to the correct answer. Please print clearly. Illegible writing will delay processing.

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Resource Volume 3, No. 1

Score

Participant's Evaluation

1. What is the highest degree you have earned (circle one)?	1. Diploma	2. Associate	3. Bachelor's	4. Master's	5. Doctorate	
2. Indicate to what degree you met the objectives of this program using 1= strongly agree to 6 = strongly disagree rating scale. Please circle the number that best reflects the extent of your agreement to each statement:						
a. Recognize the various types of topical agents and the dressing materials used in partial-thickness and deep burns along with the rationale for their use.	1	2	3	4	5	6
b. List the risk factors, pathogenesis, and causes of infections in patients with moderate to severe burns.	1	2	3	4	5	6
c. Discuss the role of various management strategies for infections in burn patients.	1	2	3	4	5	6
d. Discuss the risk factors that contribute to the increased susceptibility to infection of ICU patients.	1	2	3	4	5	6
e. Describe the pathogenesis of infection for each of the device-associated infections.	1	2	3	4	5	6
f. Recognize three prevention strategies for each of the device-associated infections.	1	2	3	4	5	6
3. How long did it take you to complete this home-study program? _____						
4. Have you used home study in the past? <input type="checkbox"/> Yes <input type="checkbox"/> No						
5. How many home-study courses do you typically use per year? _____						
6. What other areas would you like to cover through home study? _____						

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