18. Stuart J, Cartwright K, Dawson A, et al. Risk factors for meningococcal disease: a case control study in south west England. Community Med 1988;10:139–46.
19. Caugant DA, Hoiby EA, Magnus P, et al. Asymptomatic carriage *of Neisseria meningitidis* in a randomly sampled population. J Clin Microbiol 1994;32:323–30.
20. Cartwright KAV, Stuart JM, Jones DM, Noah ND. The Stonehouse survey: nasopharyngeal carriage of meningococci and *Neisseria lactamica*. Epidemiol Infect 1987;99:591–601.
21. Broome CV. The carrier state: *Neisseria meningitidis*. J Antimicrob Chemother 1986;18(Suppl A):25–34.
22. Kaiser AB, Hennekens CH, Saslaw MS, Hayes PS, Bennett JV. Seroepidemiology and chemoprophylaxis of disease due to sulfonamide resistant *Neisseria meningitidis* in a civilian population. J Infect Dis 1974;130:217–24.
23. Statistics New Zealand. New Zealand now, housing. Wellington: Statistics New Zealand, 1998.
24. Denny FW. The clinical impact of human respiratory virus infections. Am J Respir Crit Care Med 1995;152:S4–12.
25. Aaby P, Bukh J, Lisse IM, Smits AJ. Overcrowding and intensive exposure as determinants of measles mortality. Am J Epidemiol 1984;120:49–63.
26. Brimblecombe FS, Cruickshank PL, Masters PL, Stewart GT, Sanderson D. Family studies of respiratory infections. Br Med J 1958;1:119–28.
27. Haneberg B, Tonjum T, Rodahl K, Gedde-Dahl TW. Factors preceding the onset of meningococcal disease with special emphasis on passive smoking, stressful events, physical fitness and general symptoms of ill health. NIPH Ann 1983;6:169–74.
28. Cartwright KAV, Jones DM, Smith AJ, et al. Influenza A and meningococcal disease. Lancet 1991;338:334–57.
29. Yusuf HR, Rochat RW, Baughman WS, et al. Maternal cigarette smoking and invasive meningococcal disease: a cohort study among young children in metropolitan Atlanta, 1989–96. Am J Public Health 1999;89:712–17.
30. Imrey PB, Jackson LA, Ludwinski PH, et al. Outbreak of serogroup C meningococcal disease associated with campus bar patronage. Am J Epidemiol 1996;143:624–30.
31. Munford RS, Taunay ADE, Morais JSD, Fraser DW, Feldman RA. Spread of meningococcal infections in households. Lancet 1974;1:1275.
32. Openshaw PJM. When we sneeze does the immune system catch a cold? BMJ 1991;303:935–6.

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Predictors of infectious complications after burn injuries in children

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Background. **Infections are the major life-threatening complication of burn injury and occur with the greatest frequency in children. Knowledge of their occurrence and management, however, is extrapolated from studies in adults. We performed a prospective study of infectious complications in burned children.**

Objective. **To delineate epidemiology, risk factors and microbiology of infections in burned children where burn care and surgical interventions are optimal.**

Methods. **Children hospitalized for burns were entered into prospective study. Characteristics of the burn injury were assessed, and active surveillance for infections was performed.**

Results. **Seventy patients were entered [mean age, 42 months; mean total body surface area (TBSA), burn 15%]. Twenty-seven percent of patients developed 39 infections: 13 involved the burn wound (burn wound sepsis, 6; graft loss, 5; and cellulitis, 2); 13 were catheter-associated septicemia; 13 involved other sites (i.e. pneumonia, 4; urinary tract infection, 3; bacteremia, 2; endocarditis, 1; myocardial abscess, 1; toxin-mediated syndrome, 1; and otitis media, 1). Twenty-three infections were caused by a single organism, 9 infections by more than 1 organism and in 7 infections defined by CDC criteria no organism was recovered. Organisms causing infection were: *Staphylococcus aureus,* 19; *Candida albicans*, 4; *Pseudomonas aeruginosa*, 4; coagulase-negative *Staphylococcus,* 4; *Enterococcus* sp., 3; *Escherichia coli,* 1; *Klebsiella oxytoca,* 1; *Serratia marcescens*, 1; *Streptococcus pneumoniae*, 1;**

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***Streptococcus pyogenes*, 1; *Aspergillus fumigatus*, 1; *and Candida parapsilosis*, 1. Burn mechanism (flame and inhalation), extent (TBSA >30%) and depth (full thickness) were risk factors for infection; young age and site of burn were not.**

Conclusion. **The most common infections occurring in burn children are burn wound infections and catheter-associated septicemia. Characteristics of burn injury predict risk of infection. Children with flame and inhalation injury, TBSA burned >30% and full thickness burns are at high risk of infectious complications.**

INTRODUCTION

The management of burn wounds and outcome of affected pediatric patients have improved dramatically in the past 20 years. Improved outcome is the result of many factors including the establishment of centers specializing in burn management, better knowledge of the pathophysiology and treatment of shock, nutritional advances, use of topical antimicrobial agents and the aggressive surgical care of wounds using early debridement and grafting. Today the extent of burn associated with a 50% survival in pediatric patients has increased from 55% total body surface area (TBSA) to 80% TBSA.[1] Despite these advances children, especially those <1 year of age, have a considerable disadvantage in comparison with older individuals. Although burn wound infection occurs with the greatest frequency in pediatric patients,[2] most information on infectious complications and their treatment has been extrapolated from studies in adults. We performed a prospective study to delineate epidemiology, risk factors and microbiology of infections in burned children.

METHODS

Patient population. All children with TBSA burns of 10% or more, full thickness burns, circumferential burns and/or burns involving face, ears, distal extremities or perineum are hospitalized and cared for in the Stuart Hulnick Burn Center, which is a free standing eight bed unit within St. Christopher's Hospital for Children. All children hospitalized for care of burns during a 12-month period (October, 1993, to September, 1994) were eligible and were invited to participate in the study. Children hospitalized solely during investigation of the home environment were excluded.

Patients were followed prospectively throughout their hospitalization for clinical signs of possible infection, at which time investigation was recommended. Pursuit of infectious complications was at the discretion of the attending physician, but in a highly collaborative way.

Clinical evidence of infection of the burn wound or graft was defined by local signs of infection (drainage of purulent fluid; focal, multifocal or generalized black, gray, green or dark brown discoloration; hemorrhage in unburned tissue; hemorrhagic discoloration and vascular thrombosis of underlying fat; erythema or edema of unburned skin at the wound margin; unexpected rapid eschar separation; conversion of partial thickness injury to full thickness necrosis; vesicular lesions in healing or healed burns; or nonadherence of grafts. Confirmatory histologic confirmation of infection consisted of: presence of microorganisms in unburned subeschar tissue; exaggerated inflammatory response in unburned tissue; dense microbial growth at interface of nonviable and viable tissue; or perivascular, perineural and perilymphatic proliferation of organisms. Semiquantitative skin cultures were performed by modification of the Maki method,[3, 4] whenever there was clinical evidence of infection of the burn wound or graft. Quantitative culture of tissue biopsy culture also was performed during surgical debridement when infection of the burn wound was suspected, by the method of Loebl et al.[5] Cultures of other sites, such as trachea, blood and urine, were performed as indicated clinically. Isolates were identified using standard laboratory procedures.

Definition on infectious complications. The major outcomes measured were occurrence of infection and the ability of risk factors (age, TBSA burned, depth and mechanism of injury), to predict occurrence of infection.

Definitions of burn wound infection and septicemia were modified from those of Pruitt and Yurt.[6] Burn wound infection was diagnosed when local and/or histologic signs were indicative of infection and there was growth of $>10^5$ colony-forming units/cm^2 or $>10^5$ colony-forming units/g of tissue in quantitative skin or biopsy cultures, respectively. Burn wound sepsis was defined[6] as evidence of burn wound infection (by criteria above) concurrent with organ dysfunction, not necessarily in association with bacteremia. Catheter-associated infection was defined as positive culture of the blood obtained through an intravascular catheter on two occasions, or once concurrent with organ dysfunction without another cause. Bacteremia was defined as isolation of a pathogenic organism from the bloodstream, in a patient without an intravascular catheter, without evidence of organ dysfunction or extravascular infection. Toxin-mediated syndrome was defined according to clinical definitions of staphylococcal and streptococcal toxic shock syndrome,[7, 8] to include episodes fulfilling published criteria but not necessarily in the presence of hypotension. Endocarditis, septicemia, urinary tract infection, pneumonia and cellulitis were defined strictly according to definitions for nosocomial infections published by the Centers for Disease Control and Prevention.[9]

Statistical analysis. When appropriate, statistical analyses were made with the use of multivariate regression, chi square analysis or Fisher's exact method. For all variables a two tailed *P* value of 0.05 or less was considered to indicate statistical significance.

RESULTS

Seventy patients comprised the study population. Forty-three (61%) were males. Their ages ranged from 5 to 168 months, with a mean of 42 months and a median of 25 months. The TBSA burned ranged from 1 to 70%, with a mean of 15% and a median of 11%. Scald burns were the predominant type of burn, affecting 61% of patients, followed by flame injuries (20%), flame and associated inhalation injuries (13%) and contact burns (6%). Forty-seven (67%) patients sustained full thickness burns. Skin sites burned were: head and/or neck, 40 (57%); upper extremities, 46 (66%); trunk, 54 (77%); perineum and/or buttocks, 11 (16%); and lower extremities, 53 (76%).

From the time of admission all patients had topical silver sulfadiazine (Silvadene) applied to burned areas except the face where bacitracin was used. Topical antimicrobial therapy was changed if neutropenia secondary to Silvadene was noted or if infection occurred. Selection of topical therapy was based on knowledge of patient's flora. Dressing changes were performed twice daily. There was no use of whirlpool baths or immersion showering. Routine bathing was performed once daily during a dressing change with sterile saline to burns and soap and water to unaffected areas. Patients were not routinely isolated. Isolation was done according to the hospital infection control guidelines for methicillin-resistant *Staphylococcus aureus*, vancomycin-resistant *Enterococcus* and presumed or proved viral infections. Systemic antibiotics were not administered routinely, because primary therapy of the burn wound consists of surgical management with debridement, early tangential excision and grafting and application of antimicrobial agents topically. Forty-seven patients required from 1 to 15 surgical debridements and grafting. In the majority of patients the first excision was done in the first 7 days of hospitalization.

On admission 5 patients were receiving oral antibiotics (4 amoxicillin and 1 trimethoprim-sulfamethoxazole), all for acute otitis media. Only 16 of the 70 patients (23%) received systemic antibiotic(s) during their hospitalization. Nutritional assessment, including caloric requirements, was done on admission and enteral feedings were initiated on stabilization of the patient. Central venous access, preferably not through a burned site, was placed in patients with severe burn and/or inhalation injury. Central venous catheters were changed 5 to 7 days after insertion or earlier for mechanical reasons. Intravenous tubing was changed every 72 h for fluids with the exception of parenteral alimentation tubing which was changed every 24 h. Intravenous catheter insertion sites were covered in sterile nontransparent dressing and changed every 72 h.

Infections. Thirty-nine infections developed in 19 patients. Thirteen infections directly involved the burn wound (burn wound sepsis, 6; graft loss, 5; and cellulitis, 2). Other infections included: catheter-associated septicemia, 13; pneumonia, 4; urinary tract infection, 3; bacteremia, 2; endocarditis, 1; myocardial abscess, 1; toxin-mediated syndrome, 1; and otitis media, 1. Nine patients had a single infection, 4 patients had 2 infections, 5 patients had 3 infections and 1 patient had 7 infections. Thirteen infections occurred in the first week, 12 in the second week and 14 after the second week. The most common early infections were cellulitis and pneumonia. Localized infection of the burn wound leading to graft loss and cardiac infections occurred late in the hospital course; the remainder of the infections were distributed in an even fashion throughout the hospitalization.

Two patients (2.9%) died, a 19-month-old girl with a 70% TBSA scald burn and a 60-month-old girl who sustained a 25% TBSA flame burn with severe inhalation injury. The hospital courses of these two patients were complicated by infections, which led to their death.

Risk factors. Patient characteristics as potential risk factor for infection are shown in Table 1. Age was not independently predictive of infection. Type of burn, percent TBSA involved and depth of injury were associated with infection. All nine patients who sustained a flame and inhalation injury developed infection(s). This rate was higher than that seen in other burn types ($P = < 0.0001$). All patients with TBSA of >30% developed infections compared with 15.6% of those with <30% TBSA burns ($P < 0.0001$). Thirty-eight

TABLE 1. Risk factors for infection

Risk Factors	No. of Patients	Infection	No Infection	*P*
Age (mo)				
0–24	36	6 (17)*	30	0.03†
25–48	16	7 (43)	9	0.08
>48	18	6 (33)	12	0.2
Type of burn				
Scald	43	8 (19)	35	0.04†
Flame	14	2 (14)	12	0.19
Flame + inhalation	9	9 (100)	0	<0.0001
Contact	4	0	4	0.27
% total body surface area burned				
0–10	34	1 (3)	33	<0.0001†
11–20	24	7 (29)	17	0.3
21–30	6	2 (33)	4	0.5
>30	6	6 (100)	0	<0.0001
Depth				
Full thickness	47	38%		<0.001
Partial thickness	23	4%		

* Numbers in parentheses, percent.

† *P* not significant in multivariate analysis.

percent of patients with full thickness burns developed infections, compared with 4% of patients with partial thickness burns ($P < 0.001$). All infections relating to the burn wound developed in patients with full thickness burns.

Seventy patients had 204 sites burned: legs, 53; arms, 46; chest, 29; face, 22; neck, 14; back, 14; abdomen, 11; buttocks, 7; perineum, 4; and scalp, 4. Thirteen infections occurred involving the burned site: arms, 5; chest, 3; lower extremity, 3; face, 1; and scalp, 1. No site was significantly associated with increased risk for infection. No infections occurred at donor sites.

Microbiology. Infections and their causative agents are shown in Table 2. Twenty-three infections were caused by a single organism, 9 infections by more than 1 organism and in 7 infections defined by CDC criteria no organism was recovered. Organisms causing infection were: *S. aureus*, 19; *Candida albicans*, 4; *Pseudomonas aeruginosa*, 4; coagulase-negative *Staphylococcus,* 4; *Enterococcus* sp., 3; *Escherichia coli*, 1; *Klebsiella oxytoca,* 1; *Serratia marcescens*, 1; *Streptococcus pneumoniae*, 1; *Streptococcus. pyogenes,* 1; *Aspergillus fumigatus*, 1; *and Candida parapsilosis,* 1.

DISCUSSION

Burn victims are susceptible to a wide variety of infectious complications, and such complications greatly increase the morbidity and mortality of burn victims. Increasing total body surface area and depth of burn correlate with excessive risk of infectious complications. Age has been suggested to be inversely proportional to risk of infection in children. In our patients age was not an independent risk factor for infection.

TABLE 2. Etiology of infectious complications

Infection Type	No.	Pathogen	No.
Catheter infections*	13 (33)†	*Staphylococcus aureus*	7
		Coagulase-negative *Staphylococcus*	4
		Enterococcus sp.	2
		Pseudomonas aeruginosa	1
		Candida albicans	1
		Klebsiella oxytoca	1
Burn wound infection			
Burn wound sepsis*	6 (15)	*S. aureus*	3
		P. aeruginosa	2
		C. albicans	1
		Enterococcus sp.	1
		Serratia marcescens	1
		Aspergillus sp.	1
Cellulitis	2 (5)	*S. aureus*	2
Graft loss‡	5 (13)	*S. aureus*	3
		C. albicans	1
Pneumonia‡	4 (10)	*Streptococcus pneumoniae*	1
Cardiac infections‡	2 (5)	*S. aureus* (endocarditis)	1
UTI*	3 (8)	*Escherichia coli*	1
		Pseudomonas aeruginosa	1
		C. albicans	1
		Candida parapsilosis	1
Bacteremia	2 (5)	*S. aureus*	2
Toxin-mediated disease*	1 (3)	*S. aureus*	1
		Streptococcus pyogenes	1
Otitis media‡	1 (3)	None isolated	

* More than one pathogen isolated in some cases.
† Numbers in parentheses, percent.
‡ Pathogen not identified in all cases.
UTI, urinary tract infection.

All of our patients with TBSA burns of >30% developed infections, which parallels observations in adults, in which 30% TBSA burn is a significant breakpoint with excessive risk for infection.[10, 11] This is also the percent TBSA at which immune dysfunction related to burn injury is has been demonstrated.[11, 12] Other risk factors for infection in our patients were sustaining a flame and inhalation injury and having a full thickness burn. Both are correlated with prolonged and complicated hospitalizations with need for critical care and invasive monitoring. Additionally the mucosal insult on the respiratory tract increases risk for secondary pneumonia.

In our series fatality was low. Eighty-three percent of children with TBSA burns of >30% survived. The only fatality occurring among patients with burns involving <30% TBSA was associated with severe inhalation injury.

No specific site of burn was significantly predisposed to infection. Although lower extremities were burned more commonly than upper extremities, the most common infected burn site was the upper extremity. This contradicts the prevailing tenet that lower extremity burns, particularly those of the feet are more prone to infection. An important factor may be that all of our patients studied were hospitalized, where meticulous care and reduction of weight bearing were emphasized.

S. aureus was the most common cause of infection in our patients. Although any organism is a potential pathogen in burned patients, predominant pathogens have changed over decades of observation in the United States. In the 1930s and 1940s *Streptococcus pyogenes* was the predominant pathogen, followed by *S. aureus*. In the 1950s and early 1960s *S. aureus* became the predominant cause of burn infections. Gram-negative bacilli, especially *P. aeruginosa*, emerged in the 1960s and early 1970s.[13] Since then *S. aureus* has reemerged, *Streptococcus pyogenes* has remained infrequent, the spectrum of Gram-negative bacilli involved has broadened and fungal and viral pathogens have become increasingly important. These changes are direct reflections of complications of aggressive therapy and survival of more severely affected patients as well as secular trends. The predominance of *S. aureus* infections in our patients reflects a high colonization rate of children on admission (56%) and its inherent pathogenic ability.

S. aureus was a causative agent for both early and late infections. Other Gram-positives caused infection in the first 2 weeks of hospitalization. Gram-negative rods were present early on, but *P. aeruginosa* was a cause of later infections. Yeasts and molds were etiologic agents of late occurring infections, particularly

after therapy of previous infections with broad spectrum antibiotics.

A wide range of infections was seen in our patients. The most common infections were burn wound-related and catheter-related. The major factor associated with burn wound infections is high density of microorganisms on the wound or graft bed. Cultures of biopsied burn specimens yielding growth in excess of 10^5 organisms per g of tissue are significantly associated with wound infection.[10, 11, 14–21] At this density invasion of surface bacteria into tissue under the burn wound is facilitated, thus causing burn wound infection. The goal of topical antimicrobial therapy is to reduce the number of colonizing organisms. Although not sterilizing the wound, decreased superficial contamination diminishes likelihood of deeper migration. Use of optimal topical therapy together with aggressive surgical debridement and nutritional support constitute the best prophylaxis for burn wound infections. Despite optimal topical antimicrobial therapy and early and aggressive surgical debridement and grafting in our patients, infections involving the burn wound (burn wound sepsis, cellulitis and graft loss) occurred in 16% (11 of 70) of our patients.

Many of our patients, especially those with large burns and inhalation injury, required placement of central venous catheters for fluid resuscitation, medications and nutrition. Franceschi et al.[22] found that the rate of catheter infection correlated inversely with the distance of the catheter insertion site from the burn wound, infections occurring more commonly with insertion sites <30 cm from the burn wound. Frequently in children the catheter must be inserted through burned areas, which are heavily colonized with microorganisms, increasing the likelihood for infection.

Risk factors for blood stream infection include wound manipulation and the presence of an intravenous catheter. In our patients dressing changes were preformed twice daily. Dressing changes and surgical wound debridement have been associated with bacteremia in 7.7 to 65% of episodes.[23–26] The greater the TBSA burned and thus manipulated, the greater is the risk of bacteremia. The use of prophylactic systemic antibiotics before burn wound manipulation, particularly debridement, has been shown to decrease the incidence of bacteremia but has not had a beneficial effect on subsequent clinical course or incidence of burn wound infection.[23, 25–27] Bacteremia can occur in the absence of wound manipulation or other identifiable risk factors, presumably from translocation of intestinal organisms.[28] Bacteremia secondary to burn wound manipulation or intestinal translocation is usually transient and does not result in infections at distant sites or interfere with graft adherence, as in our two patients who had "spontaneous" *S. aureus* bacteremia without evidence of wound infection or organ dysfunction.

Cardiac infections developed in two (2.9%) of our patients. This high incidence is attributable to the increase in bacteremia and the use of central venous catheters. One patient had endocarditis of the tricuspid valve, on the fifth week of hospitalization, after all burn areas had been grafted and were well-healed; *S. aureus* was the responsible organism. Although this patient had never had catheter-associated septicemia, his skin was colonized with *S. aureus* and he had multiple intravascular catheters during his prolonged hospitalization; thus possibly he had undetected bacteremias associated with debridement. A myocardial abscess was diagnosed postmortem, 2 weeks after burn injury, in another patient who died from cardiorespiratory failure. This child had two previous episodes of catheter-related septicemia. Both were polymicrobial with *S. aureus* and *Enterococcus* spp. isolated. No postmortem cultures were performed; therefore cause and infectivity of the infection could not be determined.

Pneumonia complicating burns is usually an early infection, occurring in the first week post-burn injury, as it was in 3 of our 4 cases. A causative pathogen was identified in only one patient, who had bacteremic *S. pneumoniae* pneumonia on the day of admission. One patient with inhalation injury developed an aspiration pneumonia in his third week of a complicated hospital course. Pneumonia is the leading infectious cause of death in burned adults. Thermal damage produced by inhalation injury predisposes the airway to infection by producing structural damage to the respiratory tract epithelium, impairing surfactant production, mucociliary transport and macrophage function and producing atelectasis.[29]

Urinary tract infections occurred in 4.3% of our patients. In burned patients urinary tract infections are commonly associated with the use of a urinary catheter, which frequently is perceived to be required for management of perineal burns and monitoring of fluid balance. In a retrospective review of nosocomial infections in pediatric burn patients, Schlager et al.[30] reported that 21% of infections were catheter-associated urinary tract infections. Our rate may have been lower due to our practice to limit the use of urinary catheters and early removal in cases where they are used. These infections occasionally can cause bacteremia and systemic illness. Removal of the urinary catheter frequently suffices for treatment, although systemic antibiotic therapy increases the rate of sterilization and decreases the risk of bacteremia.

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REFERENCES

1. Ryan CM, Schoenfeld DA, Thorpe WP, et al. Objective estimates of the probability of death from burn injuries. N Engl J Med 1998;338:362–6.
2. Pruitt BA Jr, Curreri PN. The burn wound and its care. Arch Surg 1971;103:461–8.
3. Maki D, McCormack KN. Defatting catheter insertion sites in total parenteral nutrition is of no value as an infection control measure. Am J Med 1987;83:833–40.
4. Bertone SA, Fisher MC, Mortensen JE. Quantitative skin cultures at potential catheter sites in neonates. Infect Control Hosp Epidemiol 1994;15:315–8.
5. Loebl EC, Marvin JA, Heck EL, et al. The method of quantitative burn-wound biopsy cultures and its routine use in the care of the burned patient. Am J Clin Pathol 1974;6:20–4.
6. Pruitt BA Jr, Yurt RW. Treating burn and soft tissue infections. Infect Surg 1983;2:626–50.
7. Todd J, Fishout M, Kapral F, Welsh T. Toxic-shock syndrome associated with phage group-I staphylococci. Lancet 1978;2: 1116–8.
8. Working Group on Severe Streptococcal Infections. Defining the group A streptococcal toxic shock syndrome. JAMA 1993; 269:390–1.
9. Garner JS, Jarvis WR, Emori TG, et al. CDC definitions for nosocomial infections, 1988. Am J Infect Control 988;16:128–40.
10. Pruitt BA Jr. The diagnosis and treatment of infection in the burn patient. Burns 1984;11:79–91.
11. Pruitt DA Jr, McManus AT. Opportunistic infection in severely burned patients. Am J Med 1984;76(3A):146–54.
12. McIrvine AJ, O'Mahony JB, Saporoschetz I, et al. Depressed immune response in burn patients: use of monoclonal antibodies and functional assays to define the role of suppressor cells. Ann Surg 1982;196:297–304.
13. Alexander JW, MacMillan BG. Infections of burn wounds: In: Bennett JV, Brachman PS, eds. Hospital infections. 1st ed. Boston: Little, Brown, 1979.
14. Pruitt BA Jr. Sepsis and survival in burn patients. J Intensive Care Med 1987;2:294–6.
15. Pruitt BA Jr, Foley FD. The use of biopsies in burn patient care. Surgery 1973;73:887–97.
16. McManus AT, Kim SH, McManus WF, et al. Comparison of quantitative microbiology and histopathology in divided burn wound biopsy specimens. Arch Surg 1987;122:74–6.
17. Woolfrey BF, Fox JM, Quall CO. An evaluation of burn wound quantitative microbiology: quantitative eschar cultures. Am J Clin Pathol 1981;75:532–7.
18. Herruzo-Cabrera R, Vizcaino-Alcaide MJ, Pinedo-Castillo C, et al. Diagnosis of local infection of a burn by semiquantitative culture of the eschar surface. J Burn Care Rehabil 1992;13:639–41.
19. Levine NS, Lindberg RB, Mason AD, et al. The quantitative swab culture and smear: a quick simple method for determining the number of viable aerobic bacteria on open wounds. J Trauma 1976;16:89–94.
20. Krizek TJ, Robson MC, Kho E. Bacterial growth and skin graft survival. Surg Forum 1967;18:518.
21. Bacchetta CA, Magee W, Rodeheaver G, et al. Biology of infections of split thickness skin grafts. Am J Surg 1975;130: 63–7.
22. Franceschi D, Gerding RL, Phillips G, et al. Risk factors associated with intravascular catheter infections in burned patients: a prospective, randomized study. J Trauma 1989; 29:811–15.
23. Sasaki TM, Major MC, Welsh GW, et al. Burn wound manipulation-induced bacteremia. J Trauma 1979;19:46–8.
24. Vindenes H, Bjerknes R. The frequency of bacteremia and fungemia following wound cleansing and excision in patients with large burns. J Trauma 1993;35:742–9.
25. Piel P, Scarnati S, Goldfarb W, et al. Antibiotic prophylaxis in patients undergoing burn wound excision. J Burn Care Rehabil 1985;6:422–4.
26. Alexander JW, MacMillan BG, Law EJ, et al. Lack of beneficial effects of restricted prophylactic antibiotics for debridement and/or grafting of seriously burned patients. Bull Clin Rev Burn Injuries 1984;4:20.
27. Rodgers GL, Fisher MC, Lo A, et al. Study of antibiotic prophylaxis during burn wound debridement in children. Burn Care Rehabil 1997;18:342–6.
28. Epstein MD, Banducci DR, Manders EK. The role of the gastrointestinal tract in the development of burn sepsis. Plast Reconstr Surg 1992;90:524–31.
29. Shirani KZ, Pruitt BA Jr, Mason AD. The influence of inhalation injury and pneumonia on burn mortality. Ann Surg 1987;205:82–7.
30. Schlager T, Sader J, Weber D, et al. Hospital-acquired infections in pediatric burn patients. South Med J 1994;87: 481–4.