Prospective Analysis of Nosocomial Infection Rates, Antibiotic Use, and Patterns of Resistance in a Burn Population

Lucy Wibbenmeyer, MD,* Roy Danks, MD,† Lee Faucher, MD,‡
Marge Amelon, NP,§ Barbara Latenser, MD,* G. Patrick Kealey, MD,*
Loreen A. Herwaldt, MD§¶‖

Despite significant advances in burn care, infection remains a major cause of morbidity and mortality in burn patients. We sought to determine accurate infection rates, risk factors for infection, and the percentage of infections caused by resistant organisms. In addition, we attempted to identify interventions to decrease the use of antimicrobial drugs. Data were collected prospectively from 157 burn patients admitted to the University of Iowa Carver College of Medicine burn treatment center from October 2001 to October 2002. A research assistant reviewed the medical record for each patient identified by burn surgeons as being infected to determine whether these episodes met the infection control criteria for nosocomial infections. The infection control assessment agreed with the surgeon's assessment for 16.7% of the pneumonias, 70.0% of the burn wound infections, 57.1% of the urinary tract infections, and 70.0% of the bloodstream infections. By multiple logistic regression analysis, body surface area burned, comorbidities, and use of invasive devices were significantly related to acquisition of nosocomial infections as identified by both the burn surgeons and the infection control criteria. *Staphylococcus aureus* and *Pseudomonas* were the most common resistant organisms identified. In our population, surgeons could decrease antimicrobial use by using explicit criteria for identifying patients with hospital-acquired infections, limiting perioperative prophylaxis to patients at highest risk of infection, and decreasing the incidence of nosocomial infection with reduced use of devices and strict adherence to aseptic technique. (J Burn Care Res 2006;27:152–160)

Despite significant advances in burn care, infection remains a major cause of morbidity and mortality in patients with burns. On the basis of data submitted to the National Nosocomial Infections Surveillance System (NNIS) between 2002 and 2004, patients with burns had the highest rates of urinary tract infections and the second highest rates of ventilator-associated pneumonia and central venous catheter-associated bloodstream infections (trauma units had the highest rates).[1] The high infection rates occurred despite the fact that rates of device use in the burn units surveyed were lower than those in other intensive care units. The burn wound itself and the accompanying immunosuppression are two major factors that predispose burn patients to infection.

Given their propensity to acquire infections, patients with burns are exposed frequently to antimicrobial agents, thereby increasing their risk for colonization or infection with resistant organisms, particularly methicillin-resistant *Staphylococcus aureus* (MRSA) and vancomycin-resistant enterococci (VRE). In part because the burn patients' skin barrier is disrupted, the environment in burn units can become contaminated with resistant organisms, and these organisms can be transmitted easily from one

patient to another. Thus, burn units can be the site of explosive and prolonged outbreaks caused by resistant organisms.[2–6]

Surveying for nosocomial infections, assessing the antibiograms of infecting organisms, and reporting these results to clinicians are important mechanisms for reducing of the incidence of resistant organisms, including MRSA and VRE, in burn units.[7] We compared the burn physicians' assessment of which patients were infected with that of a research assistant trained in surveillance methods, who applied the infection control program's criteria for infection. Our aims were to compare our infection rates those of NNIS and to determine whether we could identify interventions that might decrease unnecessary use of these drugs. Additionally, we sought to determine how frequently antimicrobial resistant microorganisms cause infections in our burn unit, Iowa City.

METHODS

Burn Treatment and Unit Policies

All patients were resuscitated according to the Parkland Formula adjusted to achieve adequate perfusion endpoints. Inhalation injury was diagnosed by either laryngoscopy or bronchoscopy. Routine burn wound care consisted of daily cleansing and the twice-daily application of a topical antimicrobial ointment (silver sulfadiazine was used initially on all burns except those of the ears and nose, which were treated with mafenide acetate cream). Enteral feeding and early operative débridmentbridment were instituted for patients whose burn wounds were greater than 15% and for those whose wounds were not expected to heal within 3 weeks. Patients undergoing operative intervention received either allografts or autografts; only one patient received Integra™ (Integra Life Sciences Corp., Plainsboro, NJ) during the study period. Antimicrobial agents were given routinely for perioperative prophylaxis; prophylactic antibiotics were otherwise not administered.

The unit's policies direct staff to obtain blood, sputum, and urine cultures when patients without obvious sources of infection have fever and other signs of sepsis. Bronchoscopy was not performed routinely to diagnose pneumonia. Surface wound cultures were obtained on admission and biweekly; quantitative cultures were performed if wound sepsis was suspected. Physicians used data from routine surveillance cultures to guide their choice of preoperative antimicrobial agents. Since April 2001, perirectal swabs have been obtained from each patient on admission and weekly to identify patients colonized with VRE.

Beginning in February 2002, nasal swabs also were obtained on admission to identify patients colonized with MRSA. Each patient who carried VRE or MRSA was placed in a single room, and healthcare workers were required to wear gowns and gloves to enter the room. Patients were not isolated simply because they had burn wounds.

Data Collection

A burn research nurse collected data prospectively from 157 burn patients admitted to the burn treatment center from October 2001 to October 2002. The nurse collected demographic data, comorbidity data (confirmed coronary artery disease by previous myocardial infarction, treatment for angina or cardiac catheterization, congestive heart failure confirmed by echocardiography documenting decreased ejection fraction requiring treatment, chronic obstructive pulmonary disease requiring broncodilator therapy, asthma requiring broncodilators, peripheral vascular disease as defined by a history of claudication or previous revascularization, diabetes requiring diet modifications or treatment, and cancer), the type and extent of injury, hospital course, devices used (ventilator, central line, and urinary catheter days), the sites the burn surgeons thought were infected (blood, wound, respiratory, urine, or gastrointestinal tract), the possible etiologic agents and their antibiograms, the antimicrobial agents the patient received, white blood cell counts with differentials, vital signs 24 hours before and after the burn surgeon diagnosed an infection, and the burn surgeon's reasons for using antimicrobial agents.[8]

Definitions

Attending burn physicians identified infections based on their clinical judgment. Only episodes that occurred 48 hours after the patient's admission and that were treated with antimicrobial agents were included. Infectious episodes separated by at least 7 days were considered to be new infections. Burn surgeons did not use standardized criteria to diagnose infections. However, patients diagnosed with infections usually had at least one sign of systemic inflammatory response.[9] In general, surgeons sought to apply Peck's criteria when diagnosing burn wound infections.[10] They usually diagnosed urinary infections if the patient had a positive urinalysis or a positive urine culture and pneumonia if the patient's lung assessment changed, the sputum became purulent, the chest radiograph changed, or for the patient need increased oxygen concentrations. Surgeons considered patients who had positive blood cultures to

have bloodstream infections. Surgical research personnel later used the Centers for Disease Control and Prevention's (CDC's) definitions to determine which of these bloodstream infections were central line related.

A research assistant trained to identify nosocomial infections using the definitions developed by the CDC and by Peck and Garner et al[10,11] reviewed the medical record for each patient identified by burn surgeons as being infected to determine whether these episodes met the infection control criteria for nosocomial infections.

Data Analysis

The overall rate of nosocomial infection was calculated by dividing the number of nosocomial infections as identified by the burn physicians by the number of patients or by the number of patient days during the study period. Rates of device-related infections were calculated by dividing the number of device-related infections by the total number of days that the device was used in the study population as described by NNIS.[12] Statistical analysis was performed using SAS software (SAS version 9, SAS Institute Inc., Cary, NC). Wilcoxon rank-sum and Fisher's exact probability test were used to test whether clinical variables, that is, age, body surface area burned (BSAB), device use, operative interventions, and comorbidities, were associated with nosocomial infections. The variables that were found to have a significant association ($P < .05$) with nosocomial infection in the single factor analyses were included in a multiple logistic regression model. Using the backward elimination method, with $P > .05$ as the criterion for removal, variables were dropped from the model in order of decreasing P value. Collinearity among variables was assessed by examining the variance inflation factor. Variables with a variance inflation factor of greater than 2.5 were removed from the equation. Poisson regression was used to test for variation in the incidence density of nosocomial infections (infection per exposure days) between months and to assess the effect of early surgical intervention. Kaplan-Meier analysis was done to estimate the probability over time that a burn patient would acquire a nosocomial infection at specific time points in time after admission.

Human Subjects

Our University's Institutional Review Board approved the study.

Table 1. Comparison of the entire population and the populations with and without nosocomial infections

Patients	Age, Years	Burn Size, Percent	Length of Stay, Days*	Mortality, No. (%)	No. Patients With Comorbidities (%)	No. Operative Interventions (%)	No. Devices (%)
All patients (n = 157)	38.4 ± 22.1	13.4 ± 15.4	11.8 ± 16.3	9 (5.7)	84 (53.5)	84 (53.5)	4 (53.5)
Patients with NIs, per burn surgeons (n = 51)	45.7 ± 20.1†	21.7 ± 18.1‡	12.8 ± 16.2§	3 (5.9)	38 (74.5)‖	45 (88.4)¶	48 (94.1)
Patients without NIs per burn surgeons (n = 106)	34.9 ± 22.2†	9.4 ± 12.1‡	5.3 ± 5.6§	6 (5.7)	46 (43.4)‖	39 (36.8)¶	39 (36.8)
Patients with NIs, per ICC (n = 42)	43.7 ± 20.1**	22.8 ± 19.4††	10.8 ± 15.7‡‡	3 (7.1)	31 (73.8)§§	37 (88.1)‖‖	40 (95.2)¶¶
Patients without NIs per ICC (n = 115)	36.5 ± 22.5**	9.9 ± 12.0††	6.2 ± 6.7‡‡	6 (5.2)	53 (46.1)§§	47 (40.9)‖‖	44 (38.3)¶¶

ICC, infection control criteria; NIs, nosocomial infection. *Days from admission to the first nosocomial infection was used as the length of stay for patients with nosocomial infections; total length of stay was used for patients without nosocomial infections. †$P = .004$. ‡$P < .0001$. §$P = .0003$. ‖$P < .0001$. ¶$P < .0001$. **$P = .068$. ††$P < .0001$. ‡‡$P = .0001$. §§$P = .06$. ‖‖$P = .002$. ¶¶$P < .0001$.
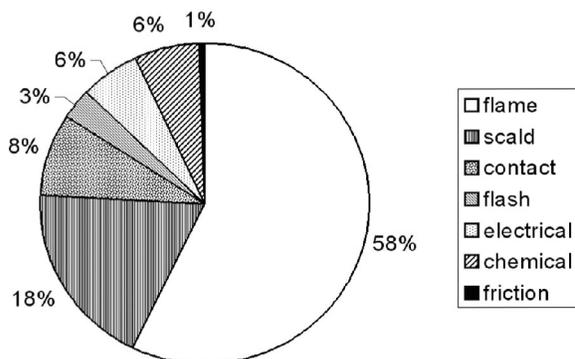
Figure 1. Percentages and types of burn injuries presented by patients.

RESULTS

Entire Study Population

One hundred fifty-seven burned patients were admitted during the study period (Table 1); 129 (82.2%) were admitted immediately after their burn injury and 9 (5.7%) were admitted more than 24 hours but less than 72 hours after their injuries. Most patients sustained flame burns (Figure 1). The mean age of patients during the study period was 38.4 ± 22.1 years. Eighty-four patients (53.5%) had invasive devices inserted; 79 patients (50.3%) had urinary catheters, 73 (46.5%) were intubated, and 27 (17.2%) had central venous catheters. Device use ratios were 0.49 for urinary catheters, 0.25 for ventilators, and 0.28 for central venous catheters. Device use was associated with comorbidities; 52 of 84 (61.9%) patients with devices had comorbidities compared with 32 of 73 (43.8%) of those who did not have devices (P = .026).

Patients with Nosocomial Infections as Assessed by Burn Surgeons

Of the 157 patients, 51 (32.5%) had 99 infections that were designated as nosocomial infections by the burn surgeons (Table 2). Thirty-four of these 51 patients (66.7%) had flame burns compared with 56 (52.8%) of the patients the surgeons felt were not infected (P = .08). All 10 patients diagnosed with inhalation injury had infections identified by the surgeons (P < .0001). The presence of diabetes did not predispose patients to infections per the surgeon assessment (6/10 with diabetes vs 45/147 without diabetes, P = .079). The incidence of nosocomial infections as identified by the surgeons was 53.4 (95% confidence interval 42.6–66.9) per 1000 patient days and it did not vary by month during the study period. Overall, 43.4% of these episodes occurred in the first week after the patients were hospitalized; 35 (56.7%) of the episodes assessed as burn wound infections and 6 (50%) of those assessed as pneumonia occurred in the first week of hospitalization. In contrast, only one (7.1%) of the episodes surgeons assessed as urinary tract infections and two (20.0%) of those assessed as bloodstream infections occurred during the first week. A Kaplan–Meier analysis using the burn surgeons' assessment of infection estimated that 36.9% ± 5.0% of all patients would acquire a nosocomial infection by day 7 of hospitalization, 55.3 ± 6.5 by day 14, and 72.3 ± 9.2 by day 21.

On the basis of the burn surgeons' assessments, burn wound infections (n = 60) were the most common nosocomial infection (Table 2). The median time to the first burn wound infection identified by the burn surgeons was 17 days (range, 4–43 days). More than 50% of the infected wounds were treated for cellulitis (n = 33); only four (6.7%) were treated for burn wound sepsis.[10]

Ten patients were treated for 14 catheter-associated urinary tract infections based on the burn surgeon's assessments. The catheter-associated urinary tract infection rate was 15.5 per 1000 urinary catheter days. Twelve patients were treated for pneumonia; 10 episodes were considered to be ventilator associated, for a rate of 22.1 per 1000 ventilator days. The burn surgeons treated eight patients for 10 episodes of bacteremia. One episode had no obvious source. The surgeons felt that four episodes were secondary to other infections (two wound infections, one urinary tract infection, and one pneumonia) and that five

Table 2. The number of infections by site: burn surgeons' assessment compared with infection control assessment

Infection	Surgeon's Assessment	Infection Control Assessment	Agreement between the Assessments, %
Any nosocomial infection	99 (51)*	60 (42)*	60.6
Burn wound infection	60 (47)	42 (38)	70.0
Pneumonia	12 (12)	2 (2)	16.7
Urinary tract infection	14 (10)	8 (7)	57.1
Bloodstream infection	10 (8)	7 (6)	70.0

*Number of infections followed by the number of patients in parentheses.

were associated with central venous catheter infections, for a central venous catheter-associated bloodstream infection rate of 7.8 per 1000 catheter days. The rate of burn wound infections was 32.4 per 1000 patient days.

Patients with Nosocomial Infections as Assessed by the Infection Control Criteria

Forty-two (26.8%) patients were identified as having 60 nosocomial infections as assessed by the infection control criteria (Table 2). Twenty-six of these patients (61.9%) had flame burns compared with 64 (55.7%) of the patients who were assessed as noninfected by the infection control criteria ($P = .11$). Ninety percent of patients identified as having inhalation injury had infections per the infection control staff ($P < .0001$). Patients with the diagnosis of diabetes were also more likely to have an infection (6/10 with diabetes vs 45/147 without diabetes, $P = .023$). The incidence of infection based on the infection control criteria was 32.4 (95% confidence interval 25.7–40.8) per 1000 patient days. The incidence of infection as identified by the infection control criteria did not vary substantially by month. Overall, 32 (53.3%) of the episodes identified by the infection control criteria occurred during the first week of hospitalization. Twenty-eight (66.7%) of the wound infections and one (50%) of the pneumonias identified by the infection control criteria occurred during the first week compared with only one (12.5%) of the urinary tract infections and two (28.6%) of the bloodstream infections. A Kaplan-Meier analysis using the infection control assessment of infection estimated that 30.5% ± 4.8% of all patients would acquire a nosocomial infection by day 7 of hospitalization, 39.2 ± 5.6 by day 14, and 65.1 ± 9.0 by day 21.

The infection control assessment agreed with the surgeons' assessment for 16.7% of the pneumonias, 70.0% of the burn wound infections, 57.1% of the urinary tract infections, and 70.0% of the bloodstream infections (Table 3a and b). Most of the burn

Table 3a. Independent risk factors for nosocomial infections identified by the burn surgeons

Variable	Odds Ratio	95% Confidence Intervals	P Value
BSAB (5% increment)	1.20	1.04–1.39	.012
Comorbidities	2.87	1.06–7.72	.037
Operative debridment	4.03	1.17–13.89	.023
Invasive device use	11.45	2.54–51.55	.002

BSAB, body surface area burned.

Table 3b. Independent risk factors for nosocomial infections identified using infection control criteria

Variable	Odds Ratio	95% Confidence Intervals	P Value
BSAB (5% increment)	1.21	1.0.6, 1.39	.007
Comorbidities	2.74	1.10, 6.78	.030
Invasive device use	26.57	5.63, 125.3	<.0001

BSAB, body surface area burned.

wound infections identified by the burn surgeons that were not identified by the infection control criteria did not meet Peck's criteria for burn wound infections (n = 11; the appearance of the wound did not change [n = 9] or the drainage was not cultured [n = 2]).[10] Five wounds that were identified as infected by burn surgeons but not by infection control criteria occurred in patients who had previous episodes of burn wound infection identified by both criteria. The infection control criteria did not identify the subsequent episodes as infections because the medical records did not document that these episodes occurred at separate sites and, thus, were distinct infections. Finally, two surgical site infections identified by surgeons as hospital acquired met the infection control definition of community acquired. On the basis of the infection control assessment, the rate of burn wound infections was 22.7 per 1000 patient days.

Of the 14 episodes treated as urinary tract infections by the surgeons, 6 did not meet the infection control criteria (3 episodes did not have urine cultures and 3 were not associated with symptoms of urinary tract infections). On the basis of the infection control assessment, the urinary catheter-associated infection rate was 8.8 per 1000 urinary catheter days. The surgeons treated 8 patients for 10 episodes of bloodstream infections. Infection control criteria identified three of these episodes as contamination, two as secondary bloodstream infections (one caused by *Serratia marcescens* from an abdominal abscess and one caused by *S. marcescens* from an infected burn wound), and three as primary bloodstream infections. On the basis of the infection control assessment, the central venous catheter-associated bloodstream infection rate was 5.8 per 1000 catheter days. Ten of the episodes identified by the burn surgeons as pneumonia did not meet the infection control criteria for this infection (four did not have radiological evidence of a pneumonia or did not have a chest radiograph, four episodes were not associated with a change in the sputum, and two episodes were not associated with signs or symptoms of pneumonia). On the basis, of the infection control assessment, the ventilator-asso-

ciated pneumonia rate was 2.3 per 1000 ventilator days.

Risk Factors for Nosocomial Infection

By multiple logistic regression analysis, invasive device use was the factor most strongly associated with nosocomial infection regardless of the criteria used to identify these infections (Table 3a and Table 3b). BSAB, comorbidities, operative débridmentbridment, and use of an invasive device were significantly associated with acquisition of nosocomial infections as identified by the burn surgeons (Table 3a). BSAB, comorbidities, and invasive device use were significantly associated with nosocomial infections as defined by the infection control criteria (Table 3b). All 10 patients with diabetes were represented in the larger group of patients with comorbidities. Likewise, the 10 patients with inhalation injury were included in the larger group of patients that had devices inserted as all 10 had received devices.

Use of Antimicrobial Agents and Antimicrobial Resistance

Ninety-five (60.5%) patients received antimicrobial agents; 52 patients were treated for one or more nosocomial infections as assessed by the surgeons and 43 were given antimicrobial agents solely for surgical prophylaxis. The median duration of a course of treatment for any purpose was 5 days (range, 1.0–46.0).

Of the 52 patients treated for nosocomial infections based on the burn surgeons' assessments, 46 also received antimicrobial agents prophylactically. This group of patients was treated for "nosocomial infection" for a median of 7 (range, 1.0–46.0) days. Forty-two (80.8%) of these 52 patients received a first-generation cephalosporin, 20 (38.5%) received fluoroquinolones, 16 (30.8%) received vancomycin, 13 (25.0%) received antipseudomonal penicillins, 5 (9.6%) received antistaphylococcal penicillins, and 9 (17.3%) received antifungal agents. Only four patients were treated with cefipime, three with clindamycin, and one each with cefotetan, ceftriaxone, meropenem, trimethoprim-sulfamethoxazole, and amoxicillin clavulanic acid. Of the 43 patients receiving antimicrobial agents for perioperative prophylaxis only, 39 (90.7%) received a first generation cephalosporin, 7 (16.3%) received fluoroquinolones, 5 (11.6%) received vancomycin, 4 (9.3%) received antipseudomonal penicillins, 2 each (4.7%) received meropenem, cefipime, and doxycline, 1 each (2.3%) received ceftazidime, clindamycin, and antistaphylococcal penicillins, and 5 (11.6%) received antifungal agents.

The spectrum of antimicrobial agents given to the subgroup of 42 patients identified by the infection control criteria was similar to that used to treat the larger group of 52 patients identified by the burn surgeons as having a nosocomial infection. Forty-two (100%) patients were treated with first generation cephalosporins, 20 (47.6%) with fluoroquinolones, 16 (38.1%) with vancomycin, 13 (30.1%) with an antipseudomonal penicillin, 2 (6.5%) with antistaphylococcal penicillin, 5 (11.9%) with penicillin, and 9 (21.4%) with antifungal agents. In addition, four (9.5%) patients received cefepime, three (7.1%) received clindamycin, two (4.8%) received metronidazole, and one patient each received cefotetan, ceftriaxone, meropenem, and bactrim single strength.

Twenty-six (46.2%) *S. aureus* isolates were resistant to methicillin. Ten of 25 (40%) *Pseudomonas* isolates were resistant to fluoroquinolones, 5 (16%) were resistant to third-generation cephalosporins, and 4 (20%) were resistant to gentamicin. *Enterobacter* spp., *Klebsiella pneumoniae*, and *Escherichia coli* that were resistant to third-generation cephalosporin were not identified in this patient population.

DISCUSSION

Surveillance for surgical site infections and reporting these rates to surgeons has been shown to reduce the rates of infection.[8,13] In addition, following nosocomial infection rates over the course of time allows surgeons and infection control staff to evaluate current practices such as hydrotherapy, isolation, and utility of surveillance cultures and to determine whether changes in practice change infection rates. Moreover, infection rates reported by the CDC[1] or other centers can serve as benchmark data against which programs can compare their own rates. We performed a prospective study to determine our rates of infection. We then compared our rates to those reported by CDC and by investigators who have done prospective studies of nosocomial infections among burn patients.

CDC recently reported pooled mean rates of device-associated infections for burn units reporting to NNIS between 2002 and 2004: catheter-associated urinary tract infections equaled 6.7 per 1000 urinary catheter days, catheter-associated bloodstream infections equaled 7.0 per 1000 central venous catheter days, and ventilator-associated pneumonia equaled 12.0 per 1000 ventilator days.[1] However, the CDC does not report burn wound infection rates. Thus, other prospective studies among burn patients provide the only benchmark data for these infections.

On the basis of the surgeons' assessments, which were much less specific than the definitions used by CDC, our rates of catheter-associated urinary tract

infection, catheter-associated bloodstream infection, and ventilator-associated pneumonia were greater than the pooled means reported by the CDC for burn units. However, on the basis of the infection control assessment, which used the CDC's definitions, our rates were similar to or less than the pooled means.

We identified three other studies published between 1992 and 2002 that also were prospective studies.[14–16] Of these studies, ours is the largest and only one to use multivariable analysis to assess independent risk factors for infections and to compare the burn surgeons' clinical assessments with those obtained when the strict criteria developed by the CDC and by Peck were applied.[9,10] The other studies did not specify their definitions[14] or were published before Peck[10] published the criteria for burn wound infections,[10,15,16] making it difficult to compare reported nosocomial infection rates.

We found an incidence of 26.8 infections per 100 patients by infection control assessment compared to 32.5 infections per 100 patients by surgeons' assessment. In the study by Taylor et al., the incidence of infection was 78 per 100 patients, which was substantially higher than of both of our estimates, or 32.8 per 1000 patient days, which was lower than the rate based on our surgeons' assessments but identical to that based on the infection control assessment.[14] Taylor et al[15] considered more sites of infection than we did during their 12-month study. In addition, the number of burn wound infections, pneumonias, and urinary tract infections were essentially equal in their population, which is considerably different than the distribution we noted. The mean age of the patients in this study was similar to ours (32 years) and BSAB was 1% to 20% for 61.2% of the patients.

Wurtz et al[16] conducted prospective surveillance for nosocomial infections among burn patients for 6 months. The average age of their patients was similar to ours (37 years) but the burns were larger (average burn size, 30.7%). These investigators found an incidence of 90 infections per 100 discharges or deaths, which was considerably higher than either of our estimates, or 32.3 infections per 1000 patient days, which was lower than our rate based on the surgeon's assessment and identical to that based on the infection control assessment. In addition, most of the infections were pneumonias and not burn wound infections.[15]

Of the prospective studies we identified, the one by Applegren et al. is the most recent and had the longest duration.[14] However, 20 of 83 (24%) patients with infections had community-acquired infections. The authors did not indicate how many community-acquired infections they included; therefore, the reader

cannot remove these infections from the reported incidence of 76.5 infections per 100 patients or 48 infections per 1000 patient days. The median age of these patients was slightly higher than ours (44) and the median BSAB (5%) was smaller than ours.

Our results indicate that the incidence of infections among burn patients depends on who is assessing the patient for infection. The incidence of infection based on the burn surgeons' clinical assessment was more than forty percent higher than that based on the criteria developed by CDC and by Peck et al and Garner et al.[10,11] The largest discrepancy occurred with the diagnosis of pneumonia followed by, urinary tract infections, wound infections and bloodstream infections. The infection control assessment, based on CDC and Peck et al criteria, may have underestimated the infection rate because the research assistant reviewed the medical records retrospectively. Therefore, the reseach assistant may not have had access to clinical observations and information that caused the surgeons to treat particular patients for infection. Thus, we cannot say for sure whether patients were undertreated or overtreated for infection.

However, we believe the surgeons are more likely to have overestimated the infection rate because they did not use standardized, written definitions. The infection control literature indicates that precise, written definitions are essential to accurately identify hospital-acquired infections correctly[17] and that clinical assessments of infection may substantially over estimate[18] or under estimate[19] the frequency of surgical infections. Thus, it is not surprising that the surgeons' assessment did not always coincide with that based on definitions developed by CDC and by Peck.[9,10]

We believe the discrepancy between the surgeons' assessment and the infection control assessment may suggest that burn patients are over-treated with antimicrobial agents and that antimicrobial use could possibly be decreased if more precise definitions of infection were used in clinical practice. For example, the burn surgeons treated 12 patients for pneumonia, but only 2 of those patients met the infection control definition of pneumonia. Most of the "pneumonias" that did not meet the CDC's definition occurred in patients whose radiographic infiltrates were explained by other etiologies (inhalation injury, congestive heart failure, atelectasis, and adult respiratory distress syndrome) and who did not meet the CDC's other criteria for pneumonia. Similarly, only 57% to 70.0% of infections as assessed by the surgeons at other sites met the strict criteria for infections. If the burn surgeons used more precise definitions of infection, such as those published by CDC and Peck[9,10] and if they limited antimicrobial therapy to patients who met

those definitions of infection, antimicrobial use could be reduced substantially in our burn unit.

Antimicrobial use in our burn unit could be further reduced if only perioperative prophylaxis was given to patients whose burn wounds were larger than 30% TBSA. Mozingo demonstrated that bacteremia secondary to surgical débridmentbridment is related to the size (greater than 45%) and age of the burn (greater than 10 days old) and that prophylaxis is not needed for patients with small, acute burns.[20] In our study, 69 (82%) of those receiving perioperative antimicrobial agents had burn wounds of less than 30% of the body surface area.

BSAB, the number of comorbidities, and invasive device use were significantly associated with nosocomial infection in the logistic regression model of risk factors for infection as identified by either set of criteria. Thus, decreased use of invasive devices, and improved aseptic technique when inserting devices could decrease the rates of nosocomial infections on burn units, thereby, decreasing use of antimicrobial agents and, in turn, decreasing the risk of selecting antimicrobial resistant organisms.

Our device use ratios were less than those reported by NNIS for burn units.[1] However, device use was by far the strongest predictor of nosocomial infections among our burn patients. CDC has developed evidence-based guidelines for preventing central venous catheter-associated bloodstream infections[21,22] and ventilator-associated pneumonia[21] and studies have demonstrated that implementing the guidelines lowers the incidence of these infections.[22,23] Thus, when possible, use of indwelling devices should be minimized and these devices should be removed when no longer needed. Moreover, healthcare workers must adhere strictly to aseptic technique when inserting and caring for these devices.

Despite limited use of nafcillin or broad-spectrum antimicrobial agents, antimicrobial resistant isolates were common among our patients. Nearly half (46.2%) of the *S. aureus* isolates causing clinical infections in our patients were resistant to oxacillin (or methicillin). Comparative data do not exist for burn units. However, NNIS recently reported that 52.9% of *S. aureus* isolates from patients in intensive care units are resistant to oxacillin, and *Pseudomonas* isolates often are resistant to levofloxacin (35.3%), ceftazidime (13.9%), and ciprofloxacin (34.8%). However, molecular typing data (not shown) from the infection control program indicate that during the study period patient-to-patient transmission of MRSA (11 patients and 3 healthcare workers) and VRE (11 patients) occurred on the burn unit. Thus, during the study period, patient-to-patient transmis-

sion may have been as important as over use of antimicrobial agents in maintaining a relatively high incidence of infections caused by resistant organisms. Thus, contact precautions, when caring for colonized and infected patients, and good hand hygiene, when caring for all patients, are important measures for decreasing transmission of resistant organisms and other important pathogens on burn units.[11,23–26]

Further study is needed to determine the role of prophylactic antimicrobial agents in thermally injured patients. However to date, there are no data demonstrating the efficacy of prophylaxis among burn patients. Neither prophylactic penicillin nor nystatin have decreased infection rates in burn patients.[27,28] In the study by Taylor et al,[15] a total of 11 of 36 (31%) burn patients who were colonized on admission with *S. aureus* subsequently acquired infections caused by this organism compared with 13% of the patients who were not colonized on admission. Eight of these patients became infected with their own strains. On the basis of their data, Taylor et al[15] suggested that burn patients who carry *S. aureus* should be treated to eradicate carriage. We believe this is a potentially useful intervention. We also believe that it should be tested in a clinical trial to determine whether it prevents *S. aureus* infections without increasing the incidence of infections caused by other more resistant organisms or increasing the incidence of *S. aureus* isolates that are resistant to the decolonizing agent.[29]

Investigators have studied several other methods for restricting use of antimicrobial agents as a means of decreasing the incidence of resistant organisms. Antimicrobial rotation and antibiotic restriction have had limited success curbing resistance in other critically ill patient populations.[7,30] Formulary restriction has been the technique that has reduced spread of resistant organisms among critically ill patients most successfully.[7,30–32] To date, there are no reports describing the use of formulary restriction in populations of burn patients.

CONCLUSION

Antimicrobial resistance is increasing in all patient populations, including patients with burns,[34,35] and nosocomial infections continue to be a serious complication of burn treatment. To limit use of antimicrobial agents and, thereby, reduce the incidence of antimicrobial resistance, burn surgeons should minimize use of prophylactic antimicrobial agents and apply standardized, written criteria, like those developed by the CDC and by Garner et al[11] and Peck et al,[9,10] when diagnosing and treating nosocomial infections. Moreover, adherence to infection control

precautions when caring for burn patients and their invasive devices, are critical for reducing the incidence of infections in this high-risk population.

ACKNOWLEDGMENTS

REFERENCES

1. Anonymous. National Nosocomial Infections Surveillance (NNIS) System Report, Data Summary from January 1992-June 2001, issued August 2001. Am J Infect Control 2001; 29:404–21.
2. Falk PS, Winnike J, Woodmansee C, Desai M, Mayhall 2nd. CG Outbreak of vancomycin-resistant enterococci in a burn unit. Infection Control Hospital Epidemiol 2000;21:575–82.
3. Meier PA, Carter CD, Wallace SE, Hollis RJ, Pfaller MA, Herwaldt LA. A prolonged outbreak of methicillin-resistant *Staphylococcus aureus* in the burn unit of a tertiary medical center. Infection Control Hospital Epidemiol 1996;17:798–802.
4. Roberts RB, Tennenberg AM, Eisner W, et al. Outbreak in a New York City teaching hospital burn center caused by the Iberian epidemic clone of MRSA. Microbial Drug Resistance-Mechanisms Epidemiol Disease 1998;4:175–83.
5. Roberts SA, Findlay R, Lang SD. Investigation of an outbreak of multi-drug resistant *Acinetobacter baumannii* in an intensive care burns unit. J Hospital Infect 2001;48:228–32.
6. Simor AE, Lee M, Vearncombe M, et al. An outbreak due to multiresistant *Acinetobacter baumannii* in a burn unit: risk factors for acquisition and management. Infection Control Hospital Epidemiol 2002;23:261–7.
7. Cunha BA. Strategies to control antibiotic resistance. Semin Respir Infect 2002;17:250–8.
8. Condon RE, Schulte WJ, Malangoni MA, Anderson-Teschendorf MJ. Effectiveness of a surgical wound surveillance program. Arch Surg 1983;118:303–7.
9. Bone RC, Balk RA, Cerra FB, et al. Definitions for sepsis and organ failure and guidelines for the use of innovative therapies in sepsis. The ACCP/SCCM Consensus Conference Committee. American College of Chest Physicians/Society of Critical Care Medicine [see comment]. Chest 1992;101:1644–55.
10. Peck MD, Weber J, McManus A, Sheridan R, Heimbach D. Surveillance of burn wound infections: a proposal for definitions. J Burn Care Rehabil 1998;19:386–9.
11. Garner JS, Jarvis WR, Emori TG, Horan TC, Huges JM. CDC definitions for nosocomial infections, 1988. Am J Infect Control 1988;16:128–40.
12. Anonymous. National Nosocomial Infections Surveillance (NNIS) report, data summary from October 1986-April 1996, issued May 1996. A report from the National Nosocomial Infections Surveillance (NNIS) System. Am J Infect Control 1996;24 (5):380–8.
13. Cruse PJ, Foord R. The epidemiology of wound infection. A 10-year prospective study of 62,939 wounds. Surg Clin N Am 1980;60:27–40.
14. Appelgren P, Bjornhagen V, Bragderyd K, CE J, Ransjo U. A prospective study of infections in burn patients. Burns 2002;28:39–46.
15. Taylor GD, Kibsey P, Kirkland T, Burroughs E, Tredget E. Predominance of staphylococcal organisms in infections occurring in a burn intensive care unit. Burns 1992;18:332–5.
16. Wurtz R, Karajovic M, Dacumos E, Jovanocic B, Hanumadass M. Nosocomial infections in a burn intensive care unit. Burns 1995;21:181–4.
17. Pottinger JM, Herwaldt LA, Peri TM. Basics of surveillancean overview. Infect Control Hospital Epidemiol 1997;18:513–27.
18. Ehrenkranz NJ, Richter EI, Phillips PM, Shultz JM. An apparent excess of operative site infections: analyses to evaluate false-positive diagnoses. Infection Control Hospital Epidemiol 1995;16:712–6.
19. Sands K, Vineyard G, Platt R. Surgical site infections occurring after hospital discharge. J Infect Dis 1996;173:963–70.
20. Mozingo DW, McManus AT, Kim SH, Pruitt BA. Incidence of bacteremia after burn wound manipulation in the early postburn period. J Trauma 1997;42:1006–10; discussion 1010–1.
21. O'Grady NP, Alexander M, Dellinger EP, et al. Guidelines for the prevention of intravascular catheter-related infections. MMWR 2002;51:1–36
22. Tablan OC, Anderson LJ, Besser R, Bridges C, Hajjeh R. Guidelines for Preventing Health Care-Associated Pneumonia, 2003 Recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee. MMWR 2004;53:1–40.
23. Jernigan JA, Titus MG, Groschel DH, Getchell-White S, Farr BM. Effectiveness of contact isolation during a hospital outbreak of methicillin-resistant Staphylococcus aureus. [Erratum appears in Am J Epidemiol 1996;143:1079]. Am J Epidemiol 1996;143:496–504.
24. Muto CA, Jernigan JA, Ostrowsky BE, et al. SHEA guideline for preventing nosocomial transmission of multidrug-resistant strains of *Staphylococcus aureus* and enterococcus [See comment]. Infect Control Hospital Epidemiol 2003;24:362–86.
25. Pittet D, Hugonnet S, Harbarth S, et al. Effectiveness of a hospital-wide programme to improve compliance with hand hygiene. Infection Control Programme. [See comment; erratum appears in Lancet 2000;356:2196.] Lancet 2000;356:1307–12.
26. Boyce JM, Pittet D. Guideline for Hand Hygiene in Health-Care Settings. Recommendations of the Healthcare Infection Control Practices- Advisory Committee and the HICPAC/SHEA/APIC/IDSA-Hand Hygiene Task Force. MMWR 2002;51:1–56.
27. Sheridan RL, Weber JM, Pasternack MS, Tompkins RG. Antibiotic prophylaxis for group A streptococcal burn wound infection is not necessary. J Trauma 2001;51:352–5.
28. Desai MH, Rutan RL, Heggers JP, Herndon DN. Candida infection with and without nystatin prophylaxis. An 11-year experience with patients with burn injury. Arch Surg 1992;127:159–62.
29. Vasquez JE, Walker ES, Franzus BW, Overbay BK, Reagan DR, Sarubbi FA. The epidemiology of mupirocin resistance among methicillin-resistant *Staphylococcus aureus* at a Veterans' Affairs hospital. Infect Control Hospital Epidemiol 2000;21:459–64.
30. Hall CS, Ost DE. Effectiveness of programs to decrease antimicrobial resistance in the intensive care unit. Semin Respir Infect 2003;18:112–21.
31. Paradisi F, Corti G, Sbaragli S, Benedetti M. Effect of antibiotic pretreatment on resistance. Semin Respir Infect 2002;17:240–5.
32. Anonymous. Intensive Care Antimicrobial Resistance Epidemiology (ICARE) Surveillance Report, data summary from January 1996 through December 1997: A report from the National Nosocomial Infections Surveillance (NNIS) System. Am J Infect Control 1999;27:279–84.