

Environmental Study of a Methicillin-Resistant *Staphylococcus aureus* Epidemic in a Burn Unit

WILLIAM A. RUTALA,* E. BLYTHE SETZER KATZ,† ROBERT J. SHERERTZ,‡ AND FELIX A. SARUBBI, JR.

Department of Hospital Epidemiology, North Carolina Memorial Hospital, and Division of Infectious Diseases, Department of Medicine, University of North Carolina School of Medicine, Chapel Hill, North Carolina 27514

Received 7 April 1983/Accepted 10 June 1983

During an outbreak of infections caused by methicillin-resistant (MR) *Staphylococcus aureus* in our burn unit, we conducted an extensive 10-week study to define the environmental epidemiology of the organism. The inanimate environment in patient rooms and adjacent areas was examined by using volumetric air samplers and Rodac plates. Airborne and surface level contamination with MR *S. aureus* was quantitated, and overall, MR *S. aureus* comprised 16, 31, and 40% of all bacterial growth from air, elevated surfaces, and floor surfaces, respectively. Mean air, elevated surface, and floor surface MR *S. aureus* contamination in rooms of MR *S. aureus*-infected burn patients were 1.9 MR *S. aureus* per ft³ (ca. 0.028 m³), 20 MR *S. aureus* per Rodac plate and 48 MR *S. aureus* per Rodac plate, respectively. Peak patient room environmental contamination levels were 6.9 MR *S. aureus* per ft³ of air, 70 MR *S. aureus* per Rodac plate per elevated surface and 138 MR *S. aureus* per Rodac plate per floor surface. Environmental contamination levels in the adjacent work areas were considerably lower than in infected patient rooms. There was ample opportunity for contamination of personnel through the inanimate environment in this unit.

A considerable body of literature concerning infections caused by methicillin-resistant (MR) *Staphylococcus aureus* has appeared in recent years (7). A number of reports have addressed the problem of nosocomial MR *S. aureus* infections and mechanisms for cross infection with these organisms have been proposed (12). Although it is likely that hospital personnel, primarily by means of hand carriage of MR *S. aureus*, represent the main vehicle for spread of the organism from patient to patient, the role of the inanimate environment surrounding the colonized or infected patient has not been carefully studied and may be more important in disease transmission than is currently recognized. Certain investigators (9, 11) rarely found MR *S. aureus* in environmental sites during non-burn unit outbreaks of MR *S. aureus* infections, whereas others (2, 4, 6, 12) have reported the limited to extensive recovery of MR *S. aureus* from environmental sites during burn unit outbreaks.

In view of the contrasting results reported in existing environmental studies and the fact that levels of environmental contamination with MR *S. aureus* have not been quantitated, we undertook the present study during an outbreak of MR *S. aureus* infections in our burn unit. Results of volumetric air sampling and Rodac plate cultures showed that MR *S. aureus* was ubiquitous throughout the unit. Environmental MR *S. aureus* counts were highest in the rooms of patients with MR *S. aureus* burn wound infections.

MATERIALS AND METHODS

The study was conducted during a 10-week period in the burn unit at the North Carolina Memorial Hospital, which is the 640-bed teaching hospital for the University of North Carolina School of Medicine. Infection surveillance in the burn unit was carried out by an infection control practitioner and one of the authors (R.J.S.) on a nearly daily basis and the designation of nosocomial infections were according to criteria established by the Centers for Disease Control (3). Burn wound infections were defined by the recovery of $\geq 10^5$ organisms per g of tissue from the burn wound, the presence of purulent drainage from the wound (5), or both. Patients were considered to be colonized with MR *S. aureus* when the organism was recovered on culture but there were no signs or symptoms of infection. Each patient underwent a swab culture of the burn wound at the time of admission to the unit.

† Present address: Health Effects Research Program, Northrop Services, Inc., Research Triangle Park, NC 27709.

‡ Present address: Division of Infectious Diseases, Department of Medicine, J. Hillis Miller Health Center, University of Florida, Gainesville, FL 32610.

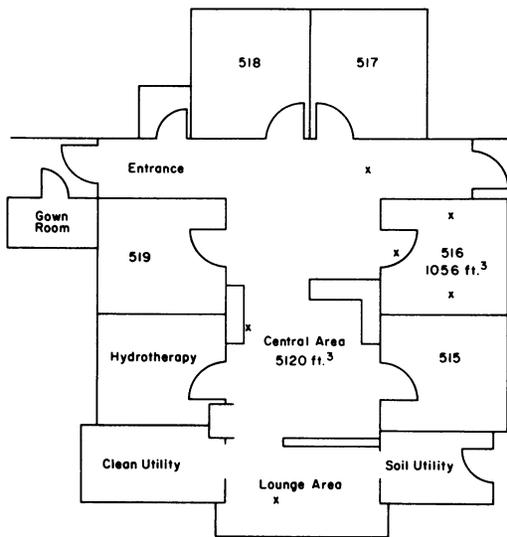


FIG. 1. Schematic drawing of the burn unit showing patient rooms and contiguous areas. The Xs indicate air sampling sites.

Additional cultures of the burn wound were generally taken on a weekly basis and cultures of other sites were done when clinically indicated. All clinical specimens were processed in the hospital clinical microbiology laboratory, and staphylococcal isolates were identified to the species level by using standard techniques. Antibiotic susceptibility testing was carried out at 35°C by the Kirby-Bauer disk method (1).

Patients admitted to the unit were placed in one of the five single-bed rooms (Fig. 1), and hydrotherapy treatment was carried out in the designated hydrotherapy room. All patients were treated with topical silver sulfadiazine. Each of the rooms opened directly into a central nursing station area and all doors were maintained in a closed position except for traffic needs. The volume of an individual patient room was 1,056 ft³ (12 ft by 11 ft by 8 ft) (ca. 29.88 m³ [ca. 3.66 m by 3.35 m by 2.44 m]). Four rooms (516, 517, 518, and 519) were studied. The patient profiled in Fig. 2 occupied rooms 516, 517, and 518 during the study period and the three patients profiled in Fig. 3 occupied rooms 518 and 519. The average temperature in these rooms (516, 517, 518, and 519) during sampling was 27.2, 26.1, 26.7, and 27.2°C, respectively, whereas the average relative humidities (measured with a Bacharach sling psychrometer; Bacharach Instrument Co., Pittsburgh, Pa.) were 38, 48, 40, and 38%, respectively. Air pressure relationships between the patient rooms and the adjacent nursing station area were frequently evaluated during the study. Rooms 515, 516, and 519 were generally at a positive air pressure with regard to the adjacent area, whereas rooms 517 and 518 were generally at a negative air pressure. Each of the rooms in the unit underwent approximately 6 air exchanges per h.

Infection control practices in the burn unit included the wearing of a clean, disposable gown on entering the unit and the washing of hands with chlorhexidine gluconate when entering and exiting the unit at sinks located outside of rooms 516 and 519. Strict hand

washing practices were observed before and after entering a patient room or the hydrotherapy room, and a second clean gown and a cap, mask, and disposable gloves were worn when entering the room of a patient colonized or infected with MR *S. aureus*.

Housekeeping services cleaned the unit on a daily basis (including wet mopping floors), using a phenolic disinfectant. Unoccupied rooms were extensively disinfected with a phenolic agent.

Environmental studies. The burn unit environment was sampled on a twice weekly basis (Monday afternoon and Thursday morning) during patient care activities. Air samples were collected with an Anderson two-stage cascade impactor set to sample 1 ft³/min (ca. 0.028 m³/min). Air sample plates were prepared with sheep blood agar, stored at 4°C, and incubated for sterility before use. After sampling, each plate was incubated for 48 h at 35°C and counted. Three air samples in two patient rooms and three in the central and lounge area (indicated by X on Fig. 1) were taken on each of the sample dates. Each 20-min air sample (20 ft³ [ca. 0.57 m³]) was taken 3 ft [ca. 0.91 m] above the floor and all patient room samples were collected with the door closed. The three patient room air samples represented approximately 6% of the total air volume in the room and the samples in the central area represented about 1% of the total air volume in that area.

Rodac plates filled with 17.5 ml of sterile Dey-Engley neutralizing agar (Difco Laboratories, Detroit, Mich.) and containing 10% additional agar were used to culture the floors and elevated surfaces in the burn unit. These plates have a planar contact surface area of approximately 4 in² (ca. 25.8 cm²) and the Dey-Engley agar neutralizes all commonly employed disinfectants. All plates were stored at 4°C and were incubated at 35°C for 48 h before use to assure sterility. All surface sample sites in the contiguous work area (central, utility rooms, hydrotherapy room, and lounge) and the floor sites in the patient rooms were randomly selected by using a grid map. The number of surface samples per room was based on the size of the room so that the average MR *S. aureus* counts per room would reasonably estimate the true level of MR *S. aureus* surface contamination. A total of 25 surface samples (one Rodac plate per 7 ft² [ca. 0.6503 m²] area) were taken from each of two patient rooms on each sampling date and included the floor (12 samples), sink (2 samples), supply shelf (2 samples), bedside table (2 samples), over-the-bed table (2 samples), supply cabinet (2 samples), linen hamper (1 sample), windowsill (1 sample), and ventilator (1 sample, if present). Sixty-six surface samples (one Rodac plate per 14 ft² [ca. 1.3 m²] area) were taken from the contiguous work areas (central room, 45 samples; hydrotherapy, 10 samples; clean utility, 6 samples; soiled utility, 5 samples) twice weekly and included 46 floor samples, and 20 elevated surface samples (counter tops, desk tops, and shelves). After sampling, the Rodac plates were incubated for 48 h at 35°C. Total colony counts were made after 24 h and a pigmented colony count (MR *S. aureus*) was made at 48 h.

Data displayed in the figures and table represent the average number of air and elevated and floor surface counts recorded in the designated area during individual (figures) or grouped (table) sample periods. Environmental data in figures are drawn as continuous

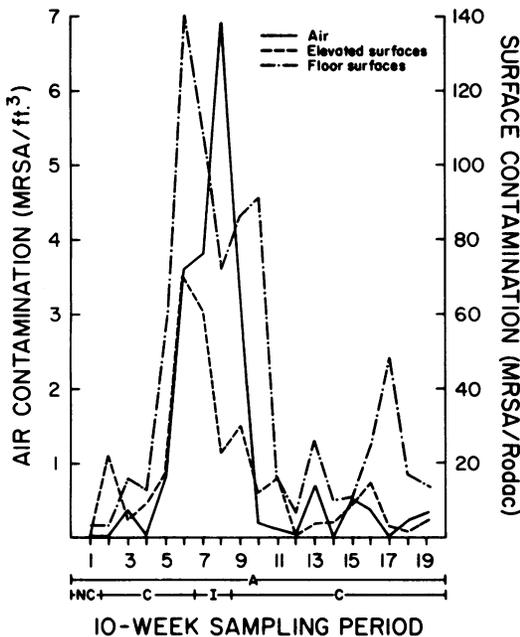


FIG. 2. Air and surface contamination with MR *S. aureus* (MRSA) in the room of patient A. NC, C, and I refer to noncolonized, colonized and infected states, respectively, of the burn wound with MR *S. aureus*. The numbers 1 through 19 refer to sampling days during the 10-week period.

curves for ease of comparison although they actually represent discrete sampling points and each point is a mean of several samples. For example, a particular point referring to the floor surface contamination with MR *S. aureus* on a single sample date in a patient room represents the mean number of MR *S. aureus* on the 12 Rodac plates.

Microbiology. All environmental cultures were processed in the hospital epidemiology laboratory. On each sampling period throughout the study, selected colonies with typical morphology and gold pigmentation on the sheep blood agar and Dey-Engley neutralizing agar plates were Gram stained and subjected to tube coagulase testing as well as to susceptibility testing by the Kirby-Bauer disk diffusion method (1). A total of 649 of 666 colonies (97.5%) thought to be *S. aureus* on the basis of colonial morphology and pigmentation were shown to be *S. aureus* when Gram stained and coagulase tested. Of the 649 staphylococcal colonies, 618 (95.2%) proved to be MR *S. aureus* on susceptibility testing. Since visual inspection of the plates and assessing colony morphology and pigmentation allowed for the correct identification of MR *S. aureus* colonies 93% of the time, this method was used to identify the remaining 50,574 colonies, which were not subjected to the previously described tests.

Phage typing of all clinical isolates and of 155 randomly selected environmental isolates was performed at the Centers for Disease Control, Atlanta, Ga.

Statistical analyses were carried out by using the Wilcoxon signed rank test.

RESULTS

Occupied patient rooms. Rooms occupied by patients A, B, C, and D, who received care in the burn unit during the study, underwent environmental sampling.

Patient A, a 27-year-old male with 60% second-degree burns, was neither colonized nor infected with MR *S. aureus* when the study began but developed burn site colonization by the second sampling period and remained colonized/infected with MR *S. aureus* for the duration of the study (Fig. 2). He had a non-staphylococcal lower respiratory tract infection when first evaluated, and this process waxed and waned throughout the 10-week period. MR *S. aureus* was recovered from sputum from the 5th through the 11th sampling periods and from blood from the 15th through the 19th periods. Peak environmental contamination in the room of patient A occurred between the 5th and 11th sampling periods and included air, elevated surface, and floor contamination levels of 6.9 MR

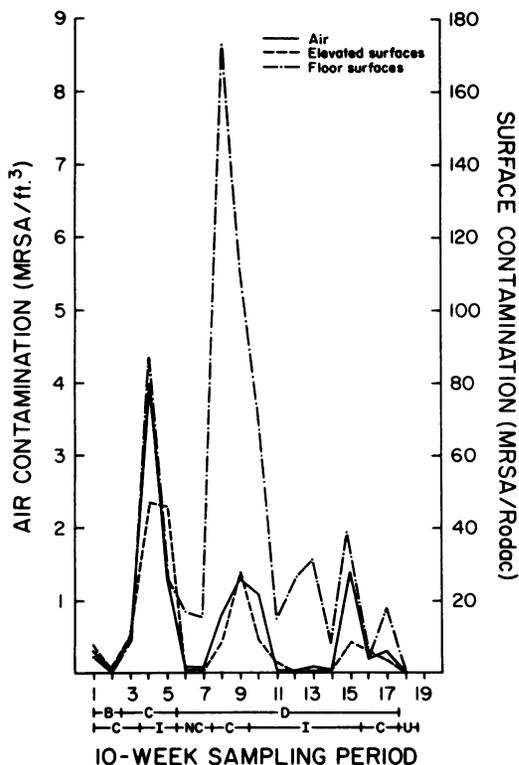


FIG. 3. Air and surface contamination with MR *S. aureus* (MRSA) in the rooms of patients B, C, and D. NC, C, and I refer to the noncolonized, colonized, and infected states, respectively, of the burn wounds with MR *S. aureus*. U refers to sampling in an unoccupied room. The numbers 1 through 19 refer to sampling days during the 10-week period.

S. aureus per ft³, 70 MR *S. aureus* per Rodac plate, and 138 MR *S. aureus* per Rodac plate, respectively.

Environmental culture results in three additional patient rooms are shown in Fig. 3. Patient B, a 52-year-old male with 42% third-degree burns, was receiving treatment for a previously documented MR *S. aureus* bloodstream infection and had MR *S. aureus* burn wound colonization during the two sampling periods depicted. Low levels of environmental contamination with MR *S. aureus* were found during these samplings. Patient C, a 59-year-old male with 30% second-degree burns, had an MR *S. aureus* burn wound infection 1 week before his first room sampling and was subsequently felt to have MR *S. aureus* burn wound colonization and then infection during environmental sampling in his room. The peak level of MR *S. aureus* contamination in his room appeared to coincide with the time of his burn wound infection. Patient D, an 80-year-old male with 34% second- and third-degree burns, was not colonized or infected with MR *S. aureus* when his room was first sampled. However, MR *S. aureus* burn wound colonization was noted within 1 week of his admission to the unit, and this was followed by burn wound infection. He also experienced an MR *S. aureus* respiratory tract infection and bloodstream infection during the period of burn wound infection. Mean environmental MR *S. aureus* counts for floor, elevated surface, and air sites on sample period 9 just before his documented MR *S. aureus* infections showed 110 MR *S. aureus* per Rodac plate, 28 MR *S. aureus* per Rodac plate, and 1.3 MR *S. aureus* per ft³, respectively.

Unoccupied patient rooms. Environmental samples were taken from three unoccupied, cleaned burn unit patient rooms. One room was sampled twice and the remaining two were sampled once. Each of these rooms had previously housed patients with MR *S. aureus* infections and were ready for use by newly admitted patients. Average MR *S. aureus* counts from air, elevated surfaces, and floor surfaces for the four samplings were 0.004 MR *S. aureus* per ft³, 0.7 MR *S. aureus* per Rodac plate, and 1.0 MR *S. aureus* per Rodac plate, respectively.

Environmental contamination with MR *S. aureus* was significantly greater in the rooms of patients with colonized or infected burn wounds when compared to unoccupied burn unit rooms ($P < 0.05$), and the elevated surface contamination with MRSA was significantly greater in the rooms of patients with colonized burn wounds when compared to the noncolonized, noninfected room ($P < 0.05$) (Table 1).

Contiguous support areas. Environmental contamination with MR *S. aureus* in the central area

TABLE 1. Environmental contamination with *S. aureus* in burn patient rooms related to colonization/infection status of the burn wound

Room	Air (MR <i>S. aureus</i> per ft ³)	Surface (MR <i>S. aureus</i> per Rodac plate)	
		Elevated	Floor
Unoccupied	0.004	0.7	1.0
Noncolonized, noninfected patient	0.04	1.0	12
Colonized, noninfected patient	0.6 ^a	12.6 ^{a,b}	39 ^a
Infected patient	1.9 ^a	20 ^a	48 ^a

^a Significant ($P < 0.05$) compared to unoccupied burn patient room.

^b Significant ($P < 0.05$) compared to unoccupied or to noncolonized, noninfected patient room.

was generally lower than that found in patient rooms. Peak air, elevated surface, and floor surface contamination levels with MR *S. aureus* were 0.7 MR *S. aureus* per ft³, 2.7 MR *S. aureus* per Rodac plate, and 84 MR *S. aureus* per Rodac plate, respectively. These levels correlated with the peak contamination times in the room of patient A, which was under positive air pressure, and to some extent with the contamination in the room of patient D. Overall surface contamination in the hydrotherapy room was substantially greater than the surface contamination found in other contiguous area sites. For the entire study period, the mean elevated surface and floor contamination with MR *S. aureus* in the hydrotherapy room was 21 and 69 MR *S. aureus* per Rodac plate, respectively. This is in contrast to the overall mean elevated surface and floor counts in the central area, clean utility room, and soiled utility rooms of 0.9 and 20, 1.0 and 13, and 0.7 and 9 MR *S. aureus* per Rodac plate, respectively.

Phage types. The phage types of 155 randomly selected environmental MR *S. aureus* isolates included nonreactive, 75, D11/1136, and 80/83A/95/D11/1136. The predominant MR *S. aureus* phage type found in the rooms of patients A and D as well as in the contiguous areas in the unit was type 75. This phage type as well as nontypable MR *S. aureus* were recovered from the blood or wound cultures from patients A and D. MR *S. aureus* isolates from patients B and C were phage type D11/1136, and this phage type was recovered from the rooms of these patients.

DISCUSSION

During the past five years, a number of outbreaks of MR *S. aureus* infections have been reported in U.S. hospitals. Several of these outbreaks have occurred in burn units, and

detailed epidemiological studies have focused on personnel, patients, and the inanimate environment in an attempt to elucidate mechanisms for disease transmission (2, 4, 6). Although the acquisition of MR *S. aureus* in the nares of hospital personnel caring for colonized or infected patients appears to occur infrequently (12), nasal carriage of the organism by personnel has been implicated in certain MR *S. aureus* infection outbreaks (9, 13). It seems more likely, however, that personnel transmit the organism from patient to patient by means of the transient carriage of MR *S. aureus* on their hands (4, 11, 12). The patient colonized or infected with MR *S. aureus* represents perhaps the most important reservoir for the organism, which may subsequently be dispersed to other patients via the hands of hospital personnel or through the inanimate environment. In contrast to the findings of Mortimer et al. (10) who demonstrated the airborne and hand transmission of *S. aureus* in a nursery setting, more recent studies of the inanimate environment with regard to its role in the epidemiology of nosocomial MR *S. aureus* infections have been limited in their scope. Previous investigators have not attempted to quantify the environmental MR *S. aureus* content surrounding colonized or infected patients, nor has a detailed, systematic survey of a unit experiencing an outbreak of MR *S. aureus* infections been carried out over an extended period of time. Published reports of MR *S. aureus* environmental contamination in burn units provide contrasting results, which in part may be due to certain limitations in study design. For example, Everett and co-workers (6) and Boyce et al. (2), infrequently found environmental contamination with MR *S. aureus* in their burn unit settings, whereas Crossley and associates (4) and Thompson and co-workers (12) recovered MR *S. aureus* from a variety of environmental sites in other burn units.

Although the latter two studies showed that MR *S. aureus* could regularly be recovered from the burn unit environment, the lack of any quantitation of the level of contamination and the undocumented consistency of recovery of the organism over a period of time made it difficult to estimate the role played by the inanimate environment in the transmission of MR *S. aureus* to personnel or patients. We undertook the present study to more precisely define the levels of MR *S. aureus* environmental contamination over a period of time in our burn unit when the MR *S. aureus* colonization/infection rate was 66.7%. Environmental samples recovered from four occupied, single-patient rooms revealed MR *S. aureus* on every sampling period. Significant differences in quantitative MR *S. aureus* counts in air and on elevated surfaces

and floor surfaces were found in the rooms of burn wound colonized or infected patients when compared to cleaned, unoccupied patient rooms. Elevated surface contamination with MR *S. aureus* in colonized patient rooms was significantly greater than in noncolonized, noninfected patient rooms (Table 1). The highest counts were noted in the rooms of infected patients where the overall mean air contamination was nearly 2 MR *S. aureus* per ft³ and the mean elevated surface count was 20 MR *S. aureus* per Rodac plate. The highest level of environmental contamination was found in the room of patient A (Fig. 2), who had the largest percent body burn (60%) of the four patients. This is consistent with the findings of Hambræus (8), who correlated the *S. aureus* dispersal from burned patients with the size of the burn wound.

Figures 2 and 3 also demonstrate that the levels of MR *S. aureus* environmental contamination can vary over a period of time and that samplings carried out at isolated points (for example, sampling periods 13 and 14) would have suggested that environmental contamination with MR *S. aureus* was minimal and perhaps not important to the overall epidemiology of these infections in the unit. Although variations of MR *S. aureus* environmental contamination in patient rooms appeared to be associated with the status of MR *S. aureus* colonization/infection in the patient, it was not possible to completely exclude effects of patient movement, nursing care activities, and housekeeping activities. However, we specifically noted these activities during each sampling time and were unable to clearly correlate them with periods of high-level MR *S. aureus* environmental contamination. It is also unlikely that our findings are due to a carrier among the burn unit personnel since the same personnel worked during the various sampling periods, and anterior nares cultures taken from 29 burn unit workers after the completion of this study showed that none were nasal carriers of MR *S. aureus*.

Environmental contamination with MR *S. aureus* in cleaned, unoccupied patient rooms and the contiguous non-patient room areas in the unit was, overall, substantially less than that noted in occupied patient rooms. The terminal cleaning and disinfection practices in the unoccupied patient rooms were very effective in reducing but not eliminating MR *S. aureus* environmental contamination. The hydrotherapy room accounted for a significant proportion of the contiguous work area surface contamination with MR *S. aureus*. In fact, the elevated surfaces in the central work area and in the clean and soiled utility rooms showed extremely low-level MR *S. aureus* contamination with mean counts of 0.9, 1.0, and 0.7 MR *S. aureus* per

Rodac plate, respectively, compared to the mean of 21 MR *S. aureus* per Rodac plate in the hydrotherapy room. The higher level of MR *S. aureus* surface contamination in the hydrotherapy room likely reflects contamination from colonized or infected patients treated in this room. Although MR *S. aureus* floor surface contamination in the central area may have largely been due to the tracking of organisms out of patient rooms and the hydrotherapy room on the soles of shoes, it appears that infection control measures (strict hand washing and use of gloves) were effective in limiting the elevated surface contamination in the central area and utility rooms. Although certain patients with MR *S. aureus* burn wound colonization and infection received care in positive air pressure patient rooms, MR *S. aureus* air contamination in the central area was generally less than 0.5 MR *S. aureus* per ft³ of air (mean air counts were 0.2 MR *S. aureus* per ft³). This may have been due to the practice of limiting patient access to this area, maintaining patient room doors closed as much as possible, and to the level of dilution ventilation in the rooms.

Finally, after tabulating all colonies on the more than 2,700 cultures of air and various surfaces, MR *S. aureus* was found to account for 16, 31, and 40% of all bacterial growth from air, elevated surface, and floor surface cultures, respectively. Interestingly, 51% of the total 2,551 MR *S. aureus* collected by the air sampler were of a size (<6 μ m), which would have allowed for their deposition into the respiratory tract.

Although we were unable to demonstrate a clear relationship between environmental contamination and clinical infections, we did show that MR *S. aureus* can be ubiquitous in the inanimate environment of a burn unit caring for patients with MR *S. aureus* colonization or infection. Environmental contamination may reach considerable levels, and this could result in contamination or colonization of patient equipment, personnel, or other patients in the vicinity. Patient rooms containing individuals who are colonized or infected with MR *S. aureus* should preferably be at an even or negative air pressure with respect to the adjacent area. Hands of personnel could easily become colonized, even without direct patient contact, and the need for frequent hand washing is underscored.

ACKNOWLEDGMENTS

We thank Greg Samsa for assistance with the statistical analysis, Mary Boutilier for technical assistance, and the burn unit staff for their support and cooperation throughout the study.

This study was supported in part by Public Health Service training grant A107151-05 from the National Institute of Allergy and Infectious Diseases.

LITERATURE CITED

1. Bauer, A. W., W. M. Kirby, J. C. Sherris, and M. Turck. 1966. Antibiotic susceptibility testing by a standardized single disk method. *Am. J. Clin. Pathol.* 196:493-496.
2. Boyce, J. M., M. Landry, T. R. Deetz, and H. L. DuPont. 1981. Epidemiologic studies of an outbreak of nosocomial methicillin-resistant *Staphylococcus aureus* infections. *Infect. Control* 2:110-116.
3. Centers for Disease Control. 1980. Algorithms for diagnosing infections. *Am. J. Epidemiol.* 111:635-643.
4. Crossley, K., B. Landesman, and D. Zasko. 1979. An outbreak of infections caused by strains of *Staphylococcus aureus* resistant to methicillin and aminoglycosides. II. Epidemiologic studies. *J. Infect. Dis.* 139:280-287.
5. Crossley, K., D. Loesch, B. Landesman, K. Mead, M. Chern, and R. Strate. 1979. An outbreak of infections caused by strains of *Staphylococcus aureus* resistant to methicillin and aminoglycosides. I. Clinical studies. *J. Infect. Dis.* 139:273-279.
6. Everett, E. D., A. E. Rahm, T. R. McNitt, D. L. Stevens, and H. E. Peterson. 1978. Epidemiologic investigation of methicillin-resistant *Staphylococcus aureus* in a burn unit. *Mil. Med.* 143:165-167.
7. Haley, R. W., A. W. Hightower, R. F. Khabbaz, C. Thornsberry, W. J. Martone, J. R. Allen, and J. M. Hughes. 1982. The emergence of methicillin-resistant *Staphylococcus aureus* infections in United States hospitals. *Ann. Intern. Med.* 97:297-308.
8. Hambraeus, A. 1973. Studies on transmission of *Staphylococcus aureus* in an isolation ward for burned patients. *J. Hyg.* 71:171-183.
9. Klimek, J. J., F. J. Marsik, R. C. Bartlett, B. Weir, P. Shea, and R. Quintilliani. 1976. Clinical, epidemiological and bacteriologic observations of an outbreak of methicillin-resistant *Staphylococcus aureus* at a large community hospital. *Am. J. Med.* 61:340-345.
10. Mortimer, E. A., E. Wolinsky, A. J. Gonzaga, and C. H. Rammelkamp. 1969. Role of airborne transmission in staphylococcal infections. *Br. Med. J.* 1:319-322.
11. Peacock, J. E., F. J. Marsik, and R. P. Wenzel. 1980. Methicillin-resistant *Staphylococcus aureus*: introduction and spread within a hospital. *Ann. Intern. Med.* 93:526-532.
12. Thompson, R. L., I. Cabezedo, and R. P. Wenzel. 1982. Epidemiology of nosocomial infections caused by methicillin-resistant *Staphylococcus aureus*. *Ann. Intern. Med.* 97:309-317.
13. Winn, R. E., T. T. Ward, A. I. Hartstein, T. P. McAnally, D. L. Sewell, C. J. Falvey, J. L. Bailey, and L. K. Krause. 1980. Epidemiological, bacteriological, and clinical observations on an interhospital outbreak of nafcillin-resistant *Staphylococcus aureus*, p. 1096-1097. In J. D. Nelson, and C. Grassi (ed.), *Current chemotherapy and infectious disease*, vol. 2. American Society for Microbiology, Washington, D.C.