

## ENVIRONMENTAL CONTAMINATION DUE TO METHICILLIN-RESISTANT *STAPHYLOCOCCUS AUREUS*: POSSIBLE INFECTION CONTROL IMPLICATIONS

John M. Boyce, MD; Gail Potter-Bynoe, BS; Claire Chenevert, BS; Thomas King, MD

### ABSTRACT

**OBJECTIVE:** To study the possible role of contaminated environmental surfaces as a reservoir of methicillin-resistant *Staphylococcus aureus* (MRSA) in hospitals.

**DESIGN:** A prospective culture survey of inanimate objects in the rooms of patients with MRSA.

**SETTING:** A 200-bed university-affiliated teaching hospital.

**PATIENTS:** Thirty-eight consecutive patients colonized or infected with MRSA. Patients represented endemic MRSA cases.

**RESULTS:** Ninety-six (27%) of 350 surfaces sampled in the rooms of affected patients were contaminated with MRSA. When patients had MRSA in a wound or urine, 36% of surfaces were contaminated. In contrast, when MRSA was isolated from other body sites, only 6% of surfaces were contaminated (odds ratio, 8.8; 95% confidence interval, 3.7-25.5;  $P < .0001$ ). Environmental contamination occurred in the rooms of 73% of infected patients and 69% of colonized

patients. Frequently contaminated objects included the floor, bed linens, the patient's gown, overbed tables, and blood pressure cuffs. Sixty-five percent of nurses who had performed morning patient-care activities on patients with MRSA in a wound or urine contaminated their nursing uniforms or gowns with MRSA. Forty-two percent of personnel who had no direct contact with such patients, but had touched contaminated surfaces, contaminated their gloves with MRSA.

**CONCLUSIONS:** We concluded that inanimate surfaces near affected patients commonly become contaminated with MRSA and that the frequency of contamination is affected by the body site at which patients are colonized or infected. That personnel may contaminate their gloves (or possibly their hands) by touching such surfaces suggests that contaminated environmental surfaces may serve as a reservoir of MRSA in hospitals (*Infect Control Hosp Epidemiol* 1997;18:622-627).

The prevalence of methicillin-resistant *Staphylococcus aureus* (MRSA) has slowly, but progressively, increased in hospitals in the United States since the early 1980s.<sup>1,2</sup> MRSA has become highly endemic in some hospitals, even though the affected institutions have adopted infection control policies such as "contact isolation"<sup>3</sup> or "body substance isolation,"<sup>4,5</sup> which were designed to limit transmission of nosocomial pathogens. Of the many possible factors that may have accounted for the inability of some hospitals to control MRSA, perhaps the most likely is poor compliance with recommended infection control policies by healthcare work-

ers.<sup>6,7</sup> However, it is also possible that barrier precautions that have been used widely in recent years do not take into account all possible reservoirs from which MRSA can spread.

To assess the possible role of contaminated surfaces as a reservoir of MRSA in the hospital setting, we prospectively obtained cultures of environmental surfaces in the rooms of patients with endemic MRSA. We also examined the relationship between the level of environmental contamination and the frequency with which personnel contaminated their clothing and gloves when caring for patients with MRSA.

*From the Miriam Hospital and Brown University, Providence, Rhode Island.*

*Address reprint requests to John M. Boyce, MD, Division of Infectious Diseases, Miriam Hospital, 164 Summit Ave, Providence, RI 02906.*

*95-OA-204. Boyce JM, Potter-Bynoe G, Chenevert C, King T. Environmental contamination due to methicillin-resistant Staphylococcus aureus: possible infection control implications. Infect Control Hosp Epidemiol 1997;18:622-627.*

## METHODS

Thirty-eight consecutive patients colonized or infected with MRSA were enrolled prospectively in the study. A majority of patients were identified by cultures obtained for clinical purposes, and the remainder were identified by surveillance cultures. The following data were recorded on each patient: age, gender, body sites that were positive for MRSA; whether the patients were colonized or infected with MRSA; and the number of days in the room before environmental cultures were obtained. Patients with tracheostomies and MRSA in the sputum were classified as having the organism in a wound, rather than being classified as having MRSA in sputum.

### Environmental Cultures

From 7 to 11 environmental cultures were obtained in each patient's room. A sterile, moistened, rayon-tipped Culturette swab (Baxter Healthcare, Deerfield, IL) was moved several times over the surface of the item being sampled. Cultures were obtained routinely from the following surfaces: patient gowns, bed linens, side rails of beds, blood pressure cuffs, overbed tables, floor adjacent to patients' beds, room door handles, and bathroom door handles. Other surfaces sampled included intravenous pump or ventilator buttons, chairs next to patients' beds, commodes, walkers, overbed monitors, and a few additional items.

On 20 occasions, cultures of gowns or uniforms of nurses were obtained after routine morning care activities had been performed on patients with MRSA in wounds or urine. New gloves (and new gowns, if worn) were donned before beginning patient care. Moistened swabs were moved several times over the front surface of the gown or uniform above and below the waist. On 12 occasions, cultures of the nurses' gloves also were obtained. In addition, cultures of the gloves of 12 nurses were obtained following nursing activities that did not require direct patient contact, but involved contact with environmental surfaces near the patient.

To determine if contamination of surfaces was widespread in the hospital, cultures of environmental surfaces were obtained in rooms of four patients who were not colonized or infected with MRSA.

Swabs were used to inoculate blood agar plates, which were incubated for 24 hours at 35°C (direct plating). Immediately after inoculating blood agar plates, swabs were used to inoculate thioglycolate broth, which was incubated for 24 hours at 35°C. The broth cultures then were subcultured onto blood agar plates, which were incubated for 24 hours

at 35°C (broth enrichment cultures). *S aureus* was identified using standard methods.

### Antimicrobial Susceptibility Tests

Disk diffusion susceptibility tests were performed using recommended methods.<sup>8</sup> Isolates resistant to oxacillin were inoculated onto oxacillin-salt screening plates containing 6 µg/mL of oxacillin and 4% NaCl, and isolates that yielded growth after incubation for 24 hours at 35°C were considered to be MRSA.<sup>9</sup>

### Molecular Typing of Strains

To confirm that isolates of MRSA recovered from environmental surfaces represented the same strains as those recovered from patients in the respective rooms, 9 clinical patient isolates and 16 associated environmental isolates, 3 epidemiologically unrelated MRSA isolates from hospitalized patients, 9 MRSA isolates from other geographic areas of the United States, and 11 *S aureus* control isolates from epidemiologically unrelated inpatients and personnel were analyzed by arbitrary-primed polymerase chain reaction (PCR) methods. Colonies recovered from 24-hour blood agar plates were picked with a sterile wooden stick, and DNA was prepared using InstaGene Purification Matrix (BioRad, Hercules, CA) according to the manufacturer's directions. Samples either were analyzed immediately or stored at -20°C until needed. Typically, 1 to 10 µL of sample (approximately 0.05 to 0.5 ng DNA) was added to a 25 to 50 µL PCR reaction containing oligonucleotide primers at 1 µM, GeneAmp 1X PCR buffer II (Perkin-Elmer, Norwalk, CT), 2.0 mM MgCl<sub>2</sub>, 200 µM GeneAmp deoxynucleotide triphosphates (Perkin-Elmer), and 1.5 units of AmpliTaq DNA polymerase (Perkin-Elmer). Random primers included HLWL74 primer (ACGTATCTGC)<sup>10</sup> and a 9-mer primer (TGGTCCTGC; MH Nicolas-Chanoine, Ambroise-Pare Hospital, Boulogne, France, unpublished data) used in combination. Samples were overlaid with mineral oil and subjected to 35 to 40 cycles of amplification (94°C for 1 minute, 20°-25°C for 1 minute, 72°C for 2 minutes), with a final extension of 10 minutes at 72°C using a Perkin-Elmer 480 thermal cycler. PCR products were analyzed in 1.0% to 2.0% agarose gels in tris borate EDTA buffer, stained with ethidium bromide, and photographed under ultraviolet light. In some experiments, <sup>32</sup>P α dCTP (Amersham Life Sciences, Arlington, IL) was included in PCR reactions (0.1 µCi per tube), and gels were dried and autoradiographed. PCR control reactions included

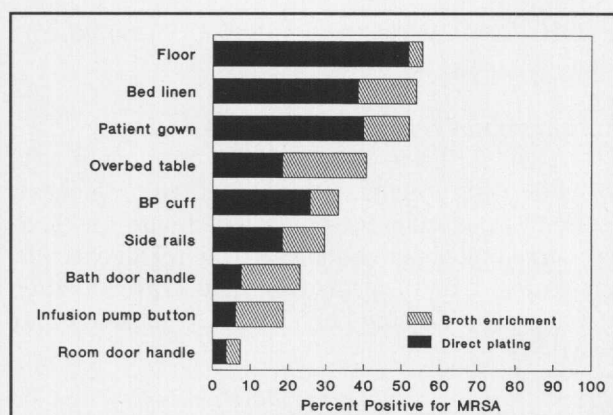


FIGURE 1. Percentage of environmental cultures positive for MRSA, by direct plating and by broth enrichment, by item cultured.

controls without added DNA to monitor for contamination. Molecular weight standards were used to assess band size.

### Statistical Analysis

Dichotomous variables were analyzed by using chi-squared tests or Fisher's Exact Test. Continuous variables were analyzed by using the Mann-Whitney U Test.

## RESULTS

### Cultures of Environmental Surfaces

Of the 38 patients (20 women) included in the study, 22 had infections caused by MRSA, and 16 were colonized. Body sites from which MRSA was recovered included wounds (25), sputum (19), nares (6), urine (5), blood (3), and other (8).

Sixty-five (19%) of 350 environmental surfaces yielded MRSA in direct-plate cultures. Thirty-one cultures yielded fewer than five colonies, 8 cultures yielded from five to nine colonies, and the remaining 26 cultures yielded 10 or more colonies of MRSA. An additional 31 cultures were positive only by the broth enrichment method. Therefore, a total of 96 (27%) of 350 environmental surface cultures yielded MRSA. For the remaining analyses, the yield is expressed as the sum of both direct plating and broth enrichment cultures.

The frequency of environmental contamination by MRSA depended, to a large degree, on the body site(s) at which the respective patients were colonized or infected. Twenty-three (85%) of 27 patients who had MRSA in a wound or urine had contaminated surfaces in their rooms, whereas only 4 (36%) of 11 patients who had MRSA in sputum, blood, or conjunctivae (but not wounds or urine) had contaminated surfaces in their rooms (odds ratio [OR], 10.1; 95% confidence interval [CI<sub>95</sub>], 1.6-69;  $P=0.005$ ). Ninety

(36%) of 250 cultures obtained in the rooms of the patients who had the organism in wounds or urine yielded MRSA, whereas only 6% of 100 cultures obtained in the rooms of the patients who had the organism in sputum, blood, or conjunctivae yielded MRSA (OR, 8.8; CI<sub>95</sub>, 3.7-25.5;  $P<0.0001$ ).

The median number of days that patients had been in their rooms before environmental cultures were performed was 11 (range, 2-86) for patients with MRSA in wounds or urine and 8 (range, 3-44) for patients who had MRSA at other body sites ( $P=0.4$ ). When we examined the relation between the number of days the patients had been in their rooms and the proportion of environmental surfaces that yielded MRSA, we found that the yield was 30% (33 of 110 cultures) for patients who had been in the room for 1 to 5 days, 16% (13/83) for those in the room for 6 to 10 days, and 31% (49/157) for those present in their room for more than 10 days.

Environmental contamination occurred in the rooms of 73% of 22 patients infected with MRSA and in the rooms of 69% of 16 patients colonized with MRSA. However, the proportion of surfaces contaminated with MRSA was greater in the rooms of infected patients. Thirty-two percent of surfaces in the rooms of infected patients were contaminated, compared to 20% of surfaces in the rooms of colonized patients (OR, 1.9; CI<sub>95</sub>, 1.1-3.2;  $P=0.01$ ).

The frequency with which various environmental surfaces yielded MRSA in the rooms of patients with the organism in wounds or urine is shown in Figure 1. The surfaces most commonly contaminated with the organism included the floor next to the patient's bed, bed linens, patient gowns, overbed tables, and blood pressure cuffs. Items that were contaminated less than 30% of the time included bedside rails, infusion pump buttons, and door handles. MRSA also was recovered from two bedside commodes, a stethoscope, and a window sill.

In the four instances where environmental cultures were positive in the rooms of patients who did not have MRSA in a wound or urine (but were positive at other body sites), only one or two colonies of MRSA were recovered, usually from a single item. On the four occasions when cultures of environmental surfaces were obtained in rooms where no patient was colonized or infected with MRSA, none of the cultures yielded MRSA.

### Cultures of Gowns and Gloves

Cultures of the gowns or uniforms of 20 personnel who had performed routine morning nursing-care activities on patients having MRSA in a wound or urine revealed that 13 (65%) of the 20 had contami-

nated their gowns or uniforms with MRSA. Cultures were positive for MRSA by direct plating in eight individuals and positive only by broth enrichment in five additional individuals. Cultures of the gloves of 12 of these personnel revealed that 7 (58%) of the nurses had contaminated their gloves, also. All were positive by direct plating.

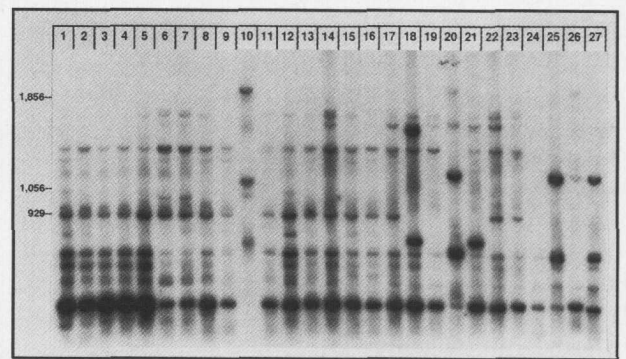
When cultures were performed on the gloves of 12 nurses who had performed activities that required no direct patient contact but involved touching side rails, linens, infusion pump buttons, or other potentially contaminated objects in the rooms of patients with MRSA in a wound or urine, we found that the gloves of five (42%) of the nurses were contaminated with MRSA. In four of the seven instances when glove cultures were negative, cultures of the environmental surfaces in the patient's room revealed little or no MRSA.

### Typing of Isolates

Analysis of the antimicrobial susceptibility patterns of MRSA isolates revealed that, in all instances, environmental isolates had the same susceptibility patterns as clinical isolates recovered from the patient in the room. Arbitrary-primed PCR typing using two random primers revealed that patient isolates and the corresponding environmental isolates had identical or very similar banding patterns (Figure 2, lanes 1-4, 6-7, 8-9, 11-13, 15-16). MRSA control isolates from the study hospital (lanes 10 and 14) and epidemiologically unrelated *S aureus* isolates from inpatients and personnel revealed different banding patterns (lanes 17-27). MRSA isolates from other geographic areas all had banding patterns different than those produced by isolates recovered from study patients (data not shown).

### DISCUSSION

Colonized or infected patients represent the major reservoir of *S aureus* in hospitals.<sup>11</sup> Personnel with persistent nasal carriage of the organism also can serve as a reservoir from which *S aureus* may be transmitted.<sup>11-16</sup> Although contaminated environmental surfaces have been considered by some authorities to be a possible reservoir of *S aureus* in hospitals, their importance is controversial. In the 1950s, extensive contamination of environmental surfaces by *S aureus* was documented in the rooms of some patients with staphylococcal infections.<sup>17,18</sup> *S aureus* were shown to be viable and pathogenic for animals and humans after being allowed to dry on environmental surfaces for several days to more than a week.<sup>18-22</sup> Based in part on these earlier studies, contaminated surfaces were felt to represent a possible



**FIGURE 2.** Autoradiograph of 2% agarose gel electrophoresis of polymerase chain reaction products produced with the combination of arbitrary primers TGGTCCTGC and ACGTATCTGC. Lanes 1-4, patient A and environmental surface, and nursing uniform and glove isolates; lane 5, patient B isolate; lanes 6-7, patient C and environmental isolates; lanes 8-9, patient D and environmental isolates; lane 10, epidemiologically unrelated MRSA control isolate; lanes 11-13, patient E and environmental and nursing uniform isolates; lane 14, MRSA control isolate; lanes 15-16, patient F and environmental isolates; lanes 17-27 epidemiologically unrelated *S aureus* isolates from inpatients and personnel.

reservoir of the organism. As a result, during the 1970s and early 1980s, many hospitals required that all personnel wear gloves, gown, and mask when entering the rooms of patients with major *S aureus* infections.<sup>23</sup>

Since the emergence of MRSA, a majority of outbreak investigations that included environmental cultures found the organism in the inanimate environment. In burn units, from a few percent to 64% of surface cultures have yielded MRSA,<sup>11,24-31</sup> whereas fewer than 5% (range 1%-18%) of environmental surfaces were reported to be contaminated in most studies performed on other wards.<sup>27,32-45</sup> One recent study conducted during an outbreak in an intensive-care unit found that environmental surfaces frequently were contaminated.<sup>46</sup>

Unlike most earlier investigations, which involved outbreaks, the present study assessed the frequency of environmental contamination associated with endemic cases of MRSA. Also, in contrast to other studies, we examined the relationship between the frequency of environmental contamination and the body sites at which patients were colonized or infected. One of the unique findings of our study was that the frequency of environmental contamination by MRSA was sixfold greater when patients had the organism in a wound or urine (36%) than when MRSA was not present at these body sites (6%). Both phenotypic and genotypic typing of isolates confirmed that isolates recovered from environmental surfaces and from affected patients were the same strain or closely related strains.

Another unique aspect of the present study was

that we demonstrated that the level of environmental contamination in the rooms of patients with MRSA in a wound or urine frequently was sufficient to cause contamination of the gloves of nurses who had touched contaminated inanimate surfaces, but had no direct contact with affected patients. Presumably, their hands would have become contaminated if they had not been wearing gloves. These findings may have important infection control implications. Barrier precaution systems adopted in the late 1980s did not require the use of gloves for touching dry, intact skin of affected patients, the patient's gown, or surfaces near the patient.<sup>3,5</sup> Although these precaution systems recommended that personnel wash their hands upon leaving the patient's room, personnel frequently do not wash their hands when leaving the rooms of patients who are in isolation.<sup>6,7</sup> We suspect that personnel who are caring for some MRSA patients may contaminate their hands by touching either the patient or objects in the immediate environment and then fail to wash their hands because they do not appear visibly soiled with blood or body secretions. Based on the present study, we favor the wearing of gloves by all personnel entering the rooms of patients with MRSA. Gloves have been shown to reduce contamination of healthcare worker hands<sup>47</sup> and should minimize contamination of the hands of personnel caring for patients with MRSA. Further studies are needed to establish if the number of viable organisms that contaminate the hands of personnel who have contact only with environmental surfaces is sufficient to result in efficient transmission of MRSA to patients.

We also found that, when the patient's immediate environment was heavily contaminated with MRSA, personnel frequently contaminated the front of their uniforms or gowns during activities that did not result in obvious soiling of their clothing. Our findings corroborate earlier studies that found that personnel may contaminate their clothing while caring for patients.<sup>48-50</sup> A few studies have suggested that organisms may be transferred from one patient to another by contaminated nurses' uniforms.<sup>49,51,52</sup> However, neither these earlier studies nor the present study provide direct evidence that *S aureus* is transmitted from one patient to another via the clothing of personnel. Further investigations are needed to establish whether *S aureus* is transmitted effectively on the clothing of personnel.

Recently, the Hospital Infection Control Practices Advisory Committee (HICPAC) published new guidelines for isolation precautions in hospitals.<sup>53</sup> The HICPAC guidelines recommend that gloves be worn routinely by all personnel entering the rooms of patients with MRSA. Gowns are recom-

mended if the healthcare worker's clothing will have substantial contact with the patient or environmental surfaces in the patient's room. The guidelines also recommend that personnel remove their gloves before leaving the room and wash their hands with an antimicrobial agent. We believe that the findings of the present study provide additional rationale for the use of such precautions. However, further studies are necessary to establish the levels of environmental contamination that are epidemiologically important and to elucidate the mechanisms by which organisms are transmitted from the inanimate objects to susceptible patients.

## REFERENCES

1. Boyce JM. Increasing prevalence of methicillin-resistant *Staphylococcus aureus* in the United States. *Infect Control Hosp Epidemiol* 1990;11:639-642.
2. Panlilio AL, Culver DH, Gaynes RP, et al. Methicillin-resistant *Staphylococcus aureus* in US hospitals, 1975-1991. *Infect Control Hosp Epidemiol* 1992;13:582-586.
3. Garner JS, Simmons BP. Centers for Disease Control guideline for isolation precautions in hospitals. *Infect Control* 1983;4:245-325.
4. Lynch P, Jackson MM, Cummings MJ, Stamm WE. Rethinking the role of isolation practices in the prevention of nosocomial infections. *Ann Intern Med* 1987;107:243-246.
5. Jackson MM, Lynch P. An attempt to make an issue less murky: a comparison of four systems for infection precautions. *Infect Control Hosp Epidemiol* 1991;12:448-449.
6. Larson E. Compliance with isolation technique. *Am J Infect Control* 1983;11:221-225.
7. Pettinger A, Nettleman MD. Epidemiology of isolation precautions. *Infect Control Hosp Epidemiol* 1991;12:303-307.
8. National Committee for Clinical Laboratory Standards. *Performance Standards for Antimicrobial Disk Susceptibility Tests*. 5th ed. Approved Standard. NCCLS Document M2-A5. Villanova, PA: NCCLS; 1993.
9. National Committee for Clinical Laboratory Standards. *Methods for Dilution Antimicrobial Susceptibility Tests for Bacteria That Grow Aerobically*. 3rd ed. Approved Standard M7-A3. Villanova, PA: NCCLS; 1993.
10. Mazurier S-I, Wernars K. Typing of *Listeria* strains by random amplification of polymorphic DNA. *Res Microbiol* 1992;143:499-505.
11. Thompson RL, Cabezudo I, Wenzel RP. Epidemiology of nosocomial infections caused by methicillin-resistant *Staphylococcus aureus*. *Ann Intern Med* 1982;97:309-317.
12. Reagan DR, Doebbeling BN, Pfaller MA, et al. Elimination of coincident *Staphylococcus aureus* nasal and hand carriage with intranasal application of mupirocin calcium ointment. *Ann Intern Med* 1991;114:101-106.
13. Coovadia YM, Bhana RH, Johnson AP, Haffeejee I, Marples RR. A laboratory-confirmed outbreak of rifampicin-methicillin resistant *Staphylococcus aureus* (RMRSA) in a newborn nursery. *J Hosp Infect* 1989;14:303-312.
14. Gaynes R, Marosok R, Mowry-Hanley J, et al. Mediastinitis following coronary artery bypass surgery: a 3-year review. *J Infect Dis* 1991;163:117-121.
15. Boyce JM, Opal SM, Potter-Bynoe G, Medeiros AA. Spread of methicillin-resistant *Staphylococcus aureus* in a hospital after exposure to a health care worker with chronic sinusitis. *Clin Infect Dis* 1993;17:496-504.
16. Sherertz RJ, Reagan DR, Hampton KD, et al. A cloud adult: the *Staphylococcus aureus*-virus interaction revisited. *Ann Intern Med* 1996;124:539-547.

17. Walter CW, Kundsins RB, Shilkret MA, Day MM. The spread of staphylococci to the environment. *Antibiotics Annual* 1958;952-957.
18. Colbeck JC. Environmental aspects of staphylococcal infections acquired in hospitals. *Am J Public Health* 1960;50:468-473.
19. Beard-Pegler MA, Stubbs E, Vickery AM. Observations on the resistance to drying of staphylococcal strains. *J Med Microbiol* 1988;26:251-255.
20. Farrington M, Brenwald N, Haines D, Walpole E. Resistance to desiccation and skin fatty acids in outbreak strains of methicillin-resistant *Staphylococcus aureus*. *J Med Microbiol* 1992;36:56-60.
21. Hinton NA, Maltman JR, Orr JH. The effect of desiccation on the ability of *Staphylococcus pyogenes* to produce disease in mice. *American Journal of Hygiene* 1960;72:343-350.
22. McDade JJ, Hall LB. Survival of *Staphylococcus aureus* in the environment, I: exposure on surfaces. *American Journal of Hygiene* 1963;78:330-337.
23. Centers for Disease Control. *Isolation Techniques for Use in Hospitals*. Washington, DC: US Government Printing Office; 1975.
24. Boyce JM, White RL, Causey WA, Lockwood WR. Burn units as a source of methicillin-resistant *Staphylococcus aureus* infections. *JAMA* 1983;249:2803-2807.
25. Arnow PM, Allyn PA, Nichols EM, Hill DL, Pezzlo M, Bartlett RH. Control of methicillin-resistant *Staphylococcus aureus* in a burn unit: role of nurse staffing. *J Trauma* 1982;22:954-959.
26. Saroglou G, Cromer M, Bisno AL. Methicillin-resistant *Staphylococcus aureus*: interstate spread of nosocomial infections with emergence of gentamicin-methicillin resistant strains. *Infect Control* 1980;1:81-89.
27. Crossley K, Landesman B, Zaska D. An outbreak of infections caused by strains of *Staphylococcus aureus* resistant to methicillin and aminoglycosides, II: epidemiologic studies. *J Infect Dis* 1979;139:280-287.
28. Everett ED, McNitt TR, Rahm AE Jr, Stevens DL, Peterson HE. Epidemiologic investigation of methicillin-resistant *Staphylococcus aureus* in a burn unit. *Military Medicine* 1978;143:165-167.
29. Espersen F, Nielsen PB, Lund K, Sylvest B, Jensen K. Hospital-acquired infections in a burn unit caused by an imported strain of *Staphylococcus aureus* with unusual multi-resistance. *J Hyg (Lond)* 1982;88:535-541.
30. Rutala WA, Katz EBS, Sherertz RJ, Sarubbi FA Jr. Environmental study of a methicillin-resistant *Staphylococcus aureus* epidemic in a burn unit. *J Clin Microbiol* 1983;18:683-688.
31. Snyder LL, Wiebelhaus P, Boon SE, Morin RA, Goering R. Methicillin-resistant *Staphylococcus aureus* eradication in a burn center. *J Burn Care Rehabil* 1993;14:164-168.
32. Walsh TJ, Vlahov D, Hansen SL, et al. Prospective microbiologic surveillance in control of nosocomial methicillin-resistant *Staphylococcus aureus*. *Infect Control* 1987;8:7-14.
33. Duckworth GJ, Lothian JL, Williams JD. Methicillin-resistant *Staphylococcus aureus*: report of an outbreak in a London teaching hospital. *J Hosp Infect* 1988;11:1-15.
34. Bitar CM, Mayhall CG, Lamb VA, Bradshaw TJ, Spadora AC, Dalton HP. Outbreak due to methicillin- and rifampin-resistant *Staphylococcus aureus*: epidemiology and eradication of the resistant strain from the hospital. *Infect Control* 1987;8:15-23.
35. Bartzokas CA, Paton JH, Gibson MF, Graham F, McLoughlin GA, Croton RS. Control and eradication of methicillin-resistant *Staphylococcus aureus* on a surgical unit. *N Engl J Med* 1984;311:1422-1425.
36. Ward TT, Winn RE, Hartstein AI, Sewell DL. Observations relating to an inter-hospital outbreak of methicillin-resistant *Staphylococcus aureus*: role of antimicrobial therapy in infection control. *Infect Control* 1981;2:453-459.
37. Shanson DC, Kensit JC, Duke R. Outbreak of hospital infection with a strain of *Staphylococcus aureus* resistant to gentamicin and methicillin. *Lancet* 1976;2:1347-1348.
38. O'Toole RD, Drew WL, Dahlgren BJ, Beaty HN. An outbreak of methicillin-resistant *Staphylococcus aureus* infection. Observations in hospital and nursing home. *JAMA* 1970;213:257-262.
39. Craven DE, Reed C, Kollisch N, et al. A large outbreak of infections caused by a strain of *Staphylococcus aureus* resistant to oxacillin and aminoglycosides. *Am J Med* 1981;71:53-58.
40. Lejeune B, Buzit-Losquin F, Simitzis-Le Flohic AM, Le Bras MP, Alix D. Outbreak of gentamicin-methicillin-resistant *Staphylococcus aureus* infection in an intensive care unit for children. *J Hosp Infect* 1986;7:21-25.
41. Cookson B, Peters B, Webster M, Phillips I, Rahman M, Noble W. Staff carriage of epidemic methicillin-resistant *Staphylococcus aureus*. *J Clin Microbiol* 1989;27:1471-1476.
42. Bradley SF, Terpenning MS, Ramsey MA, et al. Methicillin-resistant *Staphylococcus aureus*: colonization and infection in a long-term care facility. *Ann Intern Med* 1991;115:417-422.
43. Strausbaugh LJ, Jacobson C, Sewell DL, Potter S, Ward TT. Antimicrobial therapy for methicillin-resistant *Staphylococcus aureus* colonization in residents and staff of a Veterans' Affairs nursing home care unit. *Infect Control Hosp Epidemiol* 1992;13:151-159.
44. Farrington M, Ling J, Ling T, French GL. Outbreaks of infection with methicillin-resistant *Staphylococcus aureus* on neonatal and burn units of a new hospital. *Epidemiol Infect* 1990;105:215-228.
45. Haddad Q, Sobayo EI, Basit OBA, Rotimi VO. Outbreak of methicillin-resistant *Staphylococcus aureus* in a neonatal intensive care unit. *J Hosp Infect* 1993;23:211-221.
46. Maki DG, McCormick RD, Zilz MA, Stolz SM, Alvarado CJ. An MRSA outbreak in an SICU during Universal Precautions: new epidemiology for nosocomial MRSA: downside for universal precautions. In: Program and Abstracts of the 30th Interscience Conference on Antimicrobial Agents and Chemotherapy; October 1990; Atlanta, GA. Abstract 473.
47. Olson DR, Lynch P, Coyle MB, Cummings J, Bokete T, Stamm WE. Examination gloves as barriers to hand contamination in clinical practice. *JAMA* 1993;270:350-353.
48. Babb JR, Davies JG, Ayliffe GAJ. Contamination of protective clothing and nurses' uniforms in an isolation ward. *J Hosp Infect* 1983;4:149-157.
49. Lidwell OM, Towers AG, Ballard J, Gladstone B. Transfer of micro-organisms between nurses and patients in a clean air environment. *J Appl Bacteriol* 1974;37:649-656.
50. Nystrom B. The contamination of gowns in an intensive care unit. *J Hosp Infect* 1981;2:167-170.
51. Hambraeus A. Transfer of *Staphylococcus aureus* via nurses' uniforms. *J Hyg (Lond)* 1973;71:799-814.
52. Hambraeus A, Ransjo U. Attempts to control clothes-borne infection in a burn unit, I: experimental investigations of some clothes for barrier nursing. *J Hyg (Lond)* 1977;79:193-202.
53. Centers for Disease Control and Prevention. Guideline for isolation precautions in hospitals. *Infect Control Hosp Epidemiol* 1996;17:53-80.