tious Diseases, 26th Ed. Elk Grove Village, IL: American Academy of Pediatrics, **2003**:296.

Reprints or correspondence: Dr. Laura Hammitt, Arctic Investigations Program, Centers for Disease Control and Prevention, 4055 Tudor Centre Dr., Anchorage, AK 99508 (lhammitt@cdc.gov).

Clinical Infectious Diseases 2006; 43:386–7
 1058-4838/2006/4303-0024

Control of Nosocomial Methicillin-Resistant *Staphylococcus aureus* Infection

To the Editor—We write to express agreement with the statement of Klevens et al. [1] that, "regardless of which [methicillin-resistant *Staphylococcus aureus* (MRSA)] strains are present in hospitals, action is necessary to control further spread" (p. 391). We believe, however, that their next sentence, "Aggressive programs in several European countries have documented the success of identifying and treating colonized patients quickly," (p. 391) misled readers by implying that health care facilities in those countries (and in Western Australia, which has had similar success with a similar approach [2]) quickly treat—but do not isolate—colonized patients, and that treating colonized patients is the key secret to those countries' success in controlling MRSA infection. On the contrary, Dutch eradication therapy is often postponed until conditions are optimal (frequently after discharge), whereas isolation is used for all patients with known or suspected MRSA colonization [3]. A recent Dutch study illustrated the importance of isolation, reporting that MRSA was transmitted to 38 individuals when 3 MRSA-colonized patients were admitted to an intensive care unit (ICU) unsuspected, uncultured, and unisolated, compared with transmission to only 1 individual when 3 other patients were suspected, cultures were performed, and the patients were isolated at admission to the same ICU [4]. Successes at the University of Virginia (Charlottesville) over 26 years, as well as at other American hospitals, confirm the importance of identifying and isolating all colonized patients [5–8], including many situations where eradication therapy was not used [5, 8–10]. Active detection and isolation have worked well for other contagious pathogens, such as smallpox virus [11], severe acute respiratory syndrome (SARS) coronavirus [11], *Mycobacterium tuberculosis* [12], and other antibiotic-resistant pathogens, such as vancomycin-resistant *Enterococcus* species, for which eradication therapy was not possible [13–16].

The Centers for Disease Control and Prevention (CDC) has never explicitly recommended routine use of active surveillance cultures for control of MRSA and vancomycin-resistant *Enterococcus* species; so only few US health care facilities have used them routinely to identify and isolate all colonized patients [17]. Standard precautions, as recommended by the CDC, have failed to stem the growing tide of MRSA infections in many settings [18], including in the CDC's National Nosocomial Infections Surveillance (NNIS) system hospitals [1], where standard precautions have been required since 1996 (including mandatory annual infection-control retraining of all health care workers). In 1983, the CDC began recommending that contact isolation of patients with known or suspected colonization or infection be used to control spread of epidemiologically important antibiotic-resistant pathogens like MRSA. Data were already available at that time that suggested that success would require active surveillance cultures to identify and isolate all MRSA-colonized patients [5].

Eradication therapy to eliminate MRSA colonization can help control the spread of infection, because an individual who is no longer a carrier is no longer a reservoir for spread [3, 19, 20]; however, the very high MRSA prevalence in many US health care facilities would make it difficult to use eradication therapy for primary control of infection (i.e., for logistical reasons, and because of the potentiation of mupirocin resistance). Because active detection and isolation of all colonized patients results in major reductions in rates of infection, even in hospitals with very high levels of endemicity and without the use of eradication therapy [5, 9, 10], this practice should remain as the mainstay [21]. After MRSA prevalence falls, eradication therapy could then be added judiciously, making its use safe and convenient.

Acknowledgments

Potential conflicts of interest. C.A.M. has been a member of the speakers' bureau of ViroPharma. W.R.J. has been a consultant for BD (Becton, Dickinson and Company), Kimberly-Clark, 3M, and J&J (Advanced Sterilization Products, a division of ETHICON). All other authors: no conflicts.

Carlene A. Muto,[1] Margreet C. Vos,[4] William R. Jarvis,[2] and Barry M. Farr[3]

[1]University of Pittsburgh, Pittsburgh, Pennsylvania; [2]Jarvis and Jason Associates, Hilton Head Island, South Carolina; [3]University of Virginia, Charlottesville, Virginia; and [4]Erasmus University Medical Center, Rotterdam, The Netherlands

References

1. Klevens RM, Edwards JR, Tenover FC, et al. Changes in the epidemiology of methicillin-resistant *Staphylococcus aureus* in intensive care units in US hospitals, 1992–2003. Clin Infect Dis **2006**; 42:389–91.
2. Dailey L, Coombs GW, O'Brien FG, et al. Methicillin-resistant *Staphylococcus aureus*, Western Australia. Emerg Infect Dis **2005**; 11: 1584–90.
3. Verhoef J, Beaujean D, Blok H, et al. A Dutch approach to methicillin-resistant *Staphylococcus aureus*. Eur J Clin Microbiol Infect Dis **1999**; 18:461–6.
4. Vriens MR, Fluit AC, Troelstra A, Verhoef J, Van Der Werken C. Are MRSA more contagious than MSSA in a surgical intensive care unit. Infect Control Hosp Epidemiol **2002**; 23: 491–4.
5. Thompson RL, Cabezudo I, Wenzel RP. Epidemiology of nosocomial infections caused by methicillin-resistant *Staphylococcus aureus*. Ann Intern Med **1982**; 97:309–17.
6. Jernigan JA, Clemence MA, Stott GA, et al. Control of methicillin resistant *Staphylococcus aureus* at a university hospital: one decade later. Infect Control Hosp Epidemiol **1995**; 16: 686–96.
7. Jernigan JA, Titus MG, Groschel DHM, Getchell-White SI, Farr BM. Effectiveness of contact isolation during a hospital outbreak of methicillin-resistant *Staphylococcus aureus*. Am J Epidemiol **1996**; 143:496–504.
8. Calfee DP, Farr BM. Infection control and cost control in the era of managed care. Infect Control Hosp Epidemiol **2002**; 23:407–10.
9. Blank MK, Haas L, Donahoe M, Kramer P,

Muto CA. Sustained effect in reducing methicillin-resistant *Staphylococcus aureus* (MRSA) hospital acquired infections (HAIs) using active MRSA surveillance cultures (MSC)—three-year follow-up [abstract 22]. In: Conference of the 15th Annual Society for Healthcare Epidemiology of America (Los Angeles, CA). **2005**:64.
10. Huang SS, Yokoe DS, Rego VH, et al. Impact of ICU surveillance for MRSA on bacteremia [abstract 1074]. In: 43rd Annual Meeting of The Infectious Diseases Society of America (San Francisco, CA). **2005**:237.
11. Sommer A. SARS: paradigm for an emerging pathogen in a global community. In: the opening plenary session of the Fourteenth Annual Meeting of The Society for Healthcare Epidemiology of America (Philadelphia, PA). **2004**.
12. Beck-Sague CM, Dooley SW, Hutton MD, et al. Outbreak of multidrug-resistant tuberculosis among persons with HIV infection in an urban hospital: transmission to staff and patients and control measures. JAMA **1992**; 268: 1280–6.
13. Byers KE, Anglim AM, Anneski CJ, et al. A hospital epidemic of vancomycin-resistant *Enterococcus:* risk factors and control. Infect Control Hosp Epidemiol **2001**; 22:140–7.
14. Christiansen KJ, Tibbett PA, Beresford W, et al. Eradication of a large outbreak of a single strain of vanB vancomycin-resistant *Enterococcus faecium* at a major Australian teaching hospital. Infect Control Hosp Epidemiol **2004**; 25:384–90.
15) Mascini EM, Troelstra A, Beitsma M, et al. Genotyping and preemptive isolation to control an outbreak of vancomycin-resistant *Enterococcus faecium.* Clin Infect Dis **2006**; 42: 739–46.
16. Ostrowsky BE, Trick WE, Sohn AH, et al. Control of vancomycin-resistant enterococcus in health care facilities in a region. N Engl J Med **2001**; 344:1427–33.
17. Sunenshine RH, Liedtke LA, Fridkin SK, Strausbaugh LJ, Infectious Diseases Society of America Emerging Infections Network. Management of inpatients colonized or infected with antimicrobial-resistant bacteria in hospitals in the United States. Infect Control Hosp Epidemiol **2005**; 26:138–43.
18. Marshall C, Harrington G, Wolfe R, Fairley CK, Wesselingh S, Spelman D. Acquisition of methicillin-resistant *Staphylococcus aureus* in a large intensive care unit. Infect Control Hosp Epidemiol **2003**; 24:322–6.
19. Karchmer TB, Jernigan LJ, Durbin LJ, Simonton BM, Farr BM. Eradication of MRSA colonization with different regimens [abstract 65]. In: the Ninth Annual Meeting of the Society for Healthcare Epidemiology of America, (San Francisco, CA). **1999**:42.
20. Boyce JM, Cookson B, Christiansen K, et al. Methicillin-resistant *Staphylococcus aureus.* Lancet Infect Dis **2005**; 5:653–63.
21. Muto CA, Jernigan JA, Ostrowksy BE, et al. SHEA guideline for preventing nosocomial transmission of multidrug-resistant strains of *Staphylococcus aureus* and *Enterococcus.* Infect Control Hosp Epidemiol **2003**; 24:362–86.

Reprints or correspondence: Dr. Carlene A. Muto, Infection Control and Hospital Epidemiology, University of Pittsburgh Medical Center, Presbyterian Campus, 3471 Fifth Ave., 1215 Kaufmann Bldg., Pittsburgh, PA 15213 (mutoca@msx.upmc.edu).

Clinical Infectious Diseases 2006; 43:387–8

Rash as a Prognostic Factor in West Nile Virus Disease

To the Editor—As West Nile virus (WNV) becomes widespread throughout the United States, larger cohort data allow investigators to more precisely identify determinants of clinical outcomes of disease. We commend Bode et al. [1] for uncovering additional factors associated with adverse events in patients hospitalized with WNV infection during an outbreak in 4 Colorado counties in 2003. In this population with high morbidity, multivariate analysis of the presence of rash as a prognostic factor for severe disease and mortality would have been of interest.

Two recent studies of large-scale outbreaks of WNV in the United States have highlighted this finding. In 2002, the Illinois Department of Public Health reported 884 cases of WNV infection and 66 deaths due to infection [2]. Rash was a common finding among all patients for whom information was available (301 [46%] of 654 patients), as well as among patients with neuroinvasive disease (151 [39%] of 390 patients). Among patients with reported rash, age-adjusted risks were significantly decreased for encephalitis (relative risk, 0.67; 95% CI, 0.53–0.84), encephalitis plus death (relative risk, 0.44; 95% CI, 0.21–0.92), and death (relative risk, 0.39; 95% CI, 0.19–0.81). In 2003, the Colorado Epartment of Public Health and Environment reported 2947 cases of WNV infection and 63 deaths due to infection throughout the entire state [3]. A total of 1564 patients (60%) had signs of rash among evaluable cases. Age-adjusted risks for meningitis (OR, 0.7; 95% CI, 0.6–0.9), encephalitis (OR, 0.5; 95% CI, 0.3–0.6), and death (OR, 0.3; 95% CI, 0.1–0.8) were also similarly decreased in patients with reported rash [4].

A characteristic rash typically appears transiently in a diffuse maculopapular pattern at the height of febrile symptoms [4–6]. Few studies have examined the histopathological characteristics of rash lesions in WNV infection. In 1 case series, skin biopsy revealed superficial perivascular lymphocytic infiltrates seen commonly in viral exanthems [6]. Whether the development of rash in WNV infection reflects a functional immunoprotective response to circulating viral antigens requires further investigation and validation. Future surveillance activities should include prospective studies assessing features of rash that might account for its apparent favorable effect against severe disease and mortality in WNV disease.

Acknowledgments

Potential conflicts of interest. G.D.H. and M.S.D.: no conflicts.

Gregory D. Huhn[1] and Mark S. Dworkin[2]

[1]Division of Infectious Diseases, ACCESS Community Health Network, and [2]Division of Infectious Diseases, Illinois Department of Public Health, Chicago, Illinois

References

1. Bode AV, Sejvar JJ, Pape WJ, Campbell GL, Marfin AA. West Nile virus disease: a descriptive study of 228 patients hospitalized in a 4-county region of Colorado in 2003. Clin Infect Dis **2006**; 42:1234–40.
2. Huhn GD, Austin C, Langkop C, et al. The emergence of West Nile virus during a large outbreak in Illinois in 2002. Am J Trop Med Hyg **2005**; 72:768–76.
3. Colorado Department of Public Health and Environment. West Nile virus: human West Nile virus infections: Colorado, 2003. Available at: http://www.cdphe.state.co.us/dc/zoonosis/wnv/HUMAN_WNV_03.HTML. Accessed 5 April 2006.
4. Ferguson D. Rash observations about West Nile virus—Colorado, 2003. 6th National Conference on West Nile Virus in the United States (San Jose, CA). **2005**. Available at: http://www.cdc.gov/ncidod/dvbid/westnile/conf/february_2005.htm. Accessed 7 June 2006.
5. Ferguson DD, Gershman K, LeBailly A, Peterson LR. Characteristics of the rash associated with West Nile virus fever. Clin Infect Dis **2005**; 41:1204–7.
6. Anderson RC, Horn KB, Hoang MP, Gottlieb E, Bennin B. Punctate exanthem of West Nile