reported. The meta-analysis of Saint et al.[6] is an early publication that incorporated clinical trials only to 1993, which were also incorporated into the later meta-analyses.[3-5,7] The meta-analysis by Johnson et al.[7] concluded that there is only "fair quality evidence"[7(p116)] that antimicrobial catheters can prevent bacteriuria in hospitalized patients during short-term catheterization and that there is no evidence for prevention of symptomatic infection. Johnson et al.[7] concluded that the poor quality of published studies and the lack of valid economic analysis mean that further studies are required to clearly define the role of these catheters. The articles by Newton et al.[8] and Karchmer et al.[9] to which Ciavarella and Ritter[1] referred were considered in the systematic review of Johnson et al.[7] As noted in the compendium, several more-recent publications not included in these meta-analyses[10,11] raise further questions about the effectiveness of antimicrobial catheters.

Thus, the recommendation in the compendium to "not routinely use silver-coated or other antibacterial catheters"[2(pS46)] is appropriate, given the evidence. This topic, however, remains controversial, and this is acknowledged by the inclusion of "use of antimicrobial-coated catheters for selected patients at high risk for infection"[2(pS46)] as an unresolved issue in the compendium.

The ultimate solution for catheter-acquired urinary infection seems to require the development of catheter materials that are biofilm resistant. Device manufacturers certainly have an important role to play in achieving this goal. The introduction of potentially beneficial devices, however, must be accompanied by clinical trials that are methodologically rigorous, evaluate important clinical outcomes, and support the use of the devices.

ACKNOWLEDGMENTS

Potential conflicts of interest. Both authors report no conflicts of interest relevant to this article.

Evelyn Lo, MD; Lindsay E. Nicolle, MD

From the St. Boniface General Hospital (E.L.) and the Health Sciences Center (L.E.N.), Winnipeg, Manitoba, Canada.

Address reprint requests to Lindsay E. Nicolle, MD, Health Sciences Center, 820 Sherbrook St., Room GG443, Winnipeg, Manitoba R3A 1R9, Canada (lnicolle@hsc.mb.ca).

Infect Control Hosp Epidemiol 2009; 30:405-406

 0899-823X/2009/3004-0022$15.00. DOI: 10.1086/597381

REFERENCES

1. Ciavarella DJ, Ritter J. Strategies to prevent catheter-associated urinary tract infection. *Infect Control Hosp Epidemiol* 2009; 30:404-405 (in this issue).
2. Yokoe DS, Mermel LA, Anderson DJ, et al. Compendium of strategies to prevent healthcare-associated infections in acute care hospitals. *Infect Control Hosp Epidemiol* 2008; 29:S12-S21.
3. Niel-Weise BS, Arsend SM, van den Broek PJ. Is there evidence for recommending silver-coated urinary catheters in guidelines? *J Hosp Infect* 2002; 52:81-87.
4. Brosnahan J, Jull A, Tracy C. Types of urethral catheters for management of short-term voiding problems in hospitalized adults. *Cochrane Database Syst Rev* 2004; 1:CD004013.
5. Schumm K, Lam TBL. Types of urethral catheters for management of short-term voiding problems in hospitalized adults. *Cochrane Database Syst Rev* 2008; 2:CD004013.
6. Saint S, Elmore JG, Sullivan SD, et al. The efficacy of silver alloy-coated urinary catheters in preventing urinary tract infection: a meta-analysis. *Am J Med* 1998; 105:236-241.
7. Johnson JR, Kuskowki MA, Wilt TJ. Systematic review: antimicrobial urinary catheters to prevent catheter-associated urinary tract infection in hospitalized patients. *Ann Intern Med* 2006; 144:116-127.
8. Newton T, Still JM, Law E. A comparison of the effect of early insertion of standard latex and silver-impregnated latex Foley catheters on urinary tract infections in burn patients. *Infect Control Hosp Epidemiol* 2002; 23: 217-218.
9. Karchmer TB, Gianotte ET, Muto CA, Strain BA, Farr BA. A randomized crossover study of silver-coated urinary catheters in hospitalized patients. *Arch Intern Med* 2000; 160:3294-3298.
10. Crnich CJ, Drinka PJ. Does the composition of urinary catheters influence clinical outcomes and the results of research studies? *Infect Control Hosp Epidemiol* 2007; 28:102-103.
11. Srinavasan A, Karchmer T, Richards A, Song X, Perl T. A prospective trial of a novel, silicone-based, silver-coated Foley catheter for the prevention of nosocomial urinary tract infection. *Infect Control Hosp Epidemiol* 2006; 27:38-43.

Importance of Postoperative Factors in the Study of the Epidemiology of Surgical Site Infection Due to Methicillin-Resistant *Staphylococcus aureus*

We read the recent article by Anderson et al.[1] with interest and commend their effort to shed light on the timely topic of surgical site infection (SSI) due to methicillin-resistant *Staphylococcus aureus* (MRSA). However, we wish to comment on some of the limitations and conclusions of their study.

With regard to surgical site isolates, the definition of MRSA and the method for identifying MRSA were not stated. Since this was a multicenter study, it would have been desirable to have used a uniform definition and method for identifying MRSA across the entire network of participating hospitals. In addition, the frequency with which polymicrobial results were detected (ie, MRSA and other organisms growing concurrently from the same specimen) and how they were handled in the data analysis (if at all) were not presented.

It was interesting that the postulate by Anderson et al.[1] that preoperative patient debility is a risk factor for MRSA colonization—and therefore infection—was not consistently supported by their own data. Specifically, they failed to find a significant association between MRSA SSI and admission from outside facilities that are likely to house debilitated patients (eg, a nursing home or a rehabilitation facility).[1] Is the failure to confirm such an association due to a type 2 error,

or does debility impact SSI rate indirectly through factors not examined by the study (eg, stay at a long-term care facility or hospital within the previous year or postoperative factors)?

Even though the authors cited 2 references[2,3] in support of their argument linking functional status with MRSA colonization, they failed to mention that the population of both of these studies was limited to nursing home patients whose risk of exposure to MRSA is expected to be higher than that of the general population. Whether debility is an independent predictor of MRSA colonization outside of nursing homes deserves further study.

Data with regard to postoperative variables were largely ignored. For example, no data were presented with regard to the number of days (both inpatient-days and days after discharge) between surgery and the diagnosis of SSI for patients with SSI due to MRSA, compared with that for patients with SSI not due to MRSA. Similarly, Anderson et al.[1] did not discuss the setting (outpatient, in a hospital, or in a long-term care facility) where the diagnosis of SSI was made, how frequently wound drains were used, the duration of antibiotic therapy after surgery, or the postdischarge destination of patients (eg, long-term care facility vs others). Consideration of these and other postoperative factors is important before invoking only a preoperative mechanism of association between debility and MRSA SSI. Although intraoperative wound contamination is considered to be a common cause of SSI,[4] inoculation of the surgical site either directly or through hematogenous routes during the postoperative period may also occur.[4-6]

The sole study[7] cited by the authors to support their view that MRSA colonization in surgical patients increases SSI risk due to the same organism involved primarily patients with non–surgical site infections (eg, pneumonia, urinary tract infection, bacteremia, and vascular access–related infection). Of interest, 2 other relevant studies[8,9] have failed to find an association between preoperative MRSA colonization and SSI due to the same organism. Thus, preoperative colonization with MRSA may play a lesser role in causing SSI than in causing infections that do not involve the surgical site.

Even though the study by Anderson et al.[1] was not a randomized controlled study, it would have been useful for the authors to present data on the potential impact of vancomycin prophylaxis on the risk of MRSA SSI, as some of their patients almost certainly received vancomycin prophylaxis. Was this issue studied but never reported, or was it not studied at all?

Anderson et al.[1] suggest screening of patients "with decreased functional status" for MRSA colonization as well as decolonization of carriers or a change in their perioperative antibiotic regimen to include an agent with activity against MRSA.[1(p838)] We believe such recommendations are premature for the following reasons: (1) it has not been clearly demonstrated that preoperative MRSA carriage is a predictor of MRSA SSI,[8,9] (2) lack of demonstration of several preoperative factors (eg, American Society of Anesthesiologists risk category, diabetes, dialysis, or older age) that may be associated with functional impairment as independent predictors of MRSA SSI when studied simultaneously with selected postoperative factors,[10] (3) the benefit of preoperative vancomycin prophylaxis with respect to MRSA SSI has not been clearly demonstrated,[10,11] and (4) the benefit of mupirocin nasal decolonization therapy in preventing *S. aureus* SSI has not been clearly demonstrated, despite several randomized controlled studies[12,13] (in fact, the benefit of mupirocin nasal decolonization therapy in preventing *S. aureus* SSI has not been demonstrated even in nonrandomized studies, in the case of general surgery procedures[12]).

In conclusion, the weight of the evidence to date suggests that when investigating the epidemiology of MRSA SSI, the potential impact of postoperative variables should not be ignored. Indeed, if corroborated as independent predictors of MRSA SSI, postoperative factors such as how frequently wound drains are used[14] or the duration of antibiotic prophylaxis[10,14] may be more amenable to intervention than preoperative variables such as Medicaid insurance coverage or debility.[1]

ACKNOWLEDGMENTS

Potential conflicts of interest. Both authors report no conflicts of interest relevant to this article.

Farrin A. Manian, MD, MPH; Jad A. Khoury, MD

From the Division of Infectious Diseases, St. John's Mercy Medical Center, St. Louis, Missouri (both authors).

Address reprint requests to Farrin A. Manian, MD, MPH, Division of Infectious Diseases, St. John's Mercy Medical Center, 621 S. New Ballas, 7018B, St. Louis, Missouri 63141 (manianfa@aol.com).

Infect Control Hosp Epidemiol 2009; 30:406-408

 0899-823X/2009/3004-0023$15.00. DOI: 10.1086/596046

REFERENCES

1. Anderson DJ, Chen LF, Schmader KE, et al. Poor functional status as a risk factor for surgical site infections due to methicillin-resistant *Staphylococcus aureus*. *Infect Control Hosp Epidemiol* 2008; 29:832-839.
2. 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.
3. Mody L, Maheshwari S, Galecki A, Kauffman CA, Bradley SF. Indwelling device use and antibiotic resistance in nursing homes: identifying a high risk group. *J Am Geriatr Soc* 2007; 55:1921-1926.
4. Kernodle D, Kaiser AB. Surgical and trauma-related infections. In: Mandell GL, Bennett JE, Dolin R, eds. *Principles and Practice of Infectious Diseases*. New York: Churchill Livingstone; 2000:3177-3191.
5. Lowry PW, Blankenship RJ, Gridley W, Troup NJ, Tompkins LS. A cluster of *Legionella* sternal-wound infections due to postoperative topical exposure to contaminated tap water. *N Engl J Med* 1991; 324:109-113.
6. Ehrenkranz NJ, Pfaff SJ. Mediastinitis complicating cardiac operations: evidence of postoperative causation. *Rev Infect Dis* 1991; 13:803-814.
7. Huang SS, Platt R. Risk of methicillin-resistant *Staphylococcus aureus* infection after previous infection or colonization. *Clin Infect Dis* 2003; 36: 281-285.
8. San Juan R, Chaves F, Lopez Gude MJ, et al. *Staphylococcus aureus* poststernotomy mediastinitis: description of two distinct acquisition pathways with different potential preventive approaches. *J Thorac Cardiovasc Surg* 2007; 134:670-676.
9. Harbath S, Huttner B, Gervaz P, et al. Risk factors for methicillin-resistant

Staphylococcus aureus surgical site infection. *Infect Control Hosp Epidemiol* 2008; 29:890-893.
10. Manian FA, Meyer PL, Setzer J, Senkel D. Surgical site infections associated with methicillin-resistant *Staphylococcus aureus:* do postoperative factors play a role? *Clin Infect Dis* 2003; 36:864-868.
11. Finkelstein R, Rabino G, Mashiah T, et al. Vancomycin versus cefazolin prophylaxis for cardiac surgery in the setting of a high prevalence of methicillin-resistant staphylococcal infections. *J Thor Cardiovasc Surg* 2002; 123:326-332.
12. Kallen AJ, Wilson CT, Larson RJ. Perioperative intranasal mupirocin for the prevention of surgical-site infections: systematic review of the literature and meta-analysis. *Infect Control Hosp Epidemiol* 2005; 26:916-922.
13. van Rijen MML, Bonten M, Wenzel RP, Kluytmans JW. Intranasal mupirocin for reduction of *Staphylococcus aureus* infections in surgical patients with nasal carriage: a systematic review. *J Antimicrob Chemother* 2008; 61:254-261.
14. Ross H. Postoperative wound infection with methicillin-resistant staphylococci in general surgical patients. *Aust N Z J Surg* 1984; 55:13-17.

Pseudoinfection Due to Mislabeling

To the Editor—Pseudoinfections and pseudooutbreaks are mainly caused by transfer of organisms between patient specimens (cross-contamination) and by contamination of patient specimens with environmental organisms.[1,2] Other causes are clinical misdiagnosis and surveillance artifacts.[2-4] The following example shows that further causes must be considered.

Salmonella enterica serovar Hadar was isolated on the same day from a stool specimen of patient A and from an intestinal biopsy specimen of patient B. The patients were hospitalized in the same hospital but in different wards. An investigation was prompted, revealing that the patients had gone to the endoscopy suite concurrently on the day of specimen collection. An ileocoloscopy had been performed on patient A, including collection of mucosal biopsy specimens, whereas patient B had undergone gastroscopy without biopsy. No specimens at all had been collected from patient B that day to be sent to the microbiology laboratory. A stool specimen had been collected for microbiological examination from patient A, before patient A went to the endoscopy suite that day. Patient B did not show clinical signs of salmonellosis, and a pseudoinfection was suspected. However, the pseudoinfection obviously could not have been caused by specimen contamination or cross-contamination. Observations of the work flow within the endoscopy suite led us to conclude that specimen mislabeling was the most likely cause of the pseudoinfection. The charts of the 2 patients had been deposited on the same desk. When the biopsy specimen was taken to the desk to be marked with a patient label, a label of patient B was erroneously used for the biopsy specimen of patient A.

As in other cases published, this case of a pseudoinfection was noticed because of the unusual pathogen involved. Coincidentally, no biopsy specimen had been obtained from patient B on the day of specimen collection. If this had not been the case, the pseudoinfection would not have been noticed at all, or cross-contamination would have been regarded as the most likely cause of pseudoinfection, leading to a costly analysis of endoscope processing as well as of each step in specimen collection and processing.[1,3] Taking into account frequent errors in daily routine work, we hypothesize that pseudoinfection due to mislabeling of specimens is not an infrequent event.

ACKNOWLEDGMENTS

Potential conflicts of interest. Both authors report no conflicts of interest relevant to this article.

Roland Schulze-Röbbecke, PD, MD; Claudia Schmitz

From the Institute of Medical Microbiology and Hospital Hygiene, Heinrich-Heine-University of Düsseldorf, Düsseldorf, Germany (both authors).

Address reprint requests to Roland Schulze-Röbbecke, PD, MD, Institut für Medizinische Mikrobiologie und Krankenhaushygiene, Universitätsstr. 1, 40225 Düsseldorf, Germany (schulzrr@uni-duesseldorf.de).

Infect Control Hosp Epidemiol 2009; 30:408-408

 0899-823X/2009/3004-0024$15.00. DOI: 10.1086/596041

REFERENCES

1. Cunha BA. Pseudoinfections and pseudo-outbreaks. In: Mayhall GC, ed. *Hospital Epidemiology and Infection Control.* 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2004:123-133.
2. Manangan LP, Jarvis WR. Healthcare-associated pseudo-outbreaks. *Semin Infect Control* 2001; 1:102-110.
3. Weinstein RA, Stamm WE. Pseudoepidemics in hospital. *Lancet* 1977; 2: 862-864.
4. Wendt C, Herwaldt LA. Epidemics: identification and management. In: Wenzel RP, ed. *Prevention and Control of Nosocomial Infections.* 3rd ed. Baltimore: Williams & Wilkins; 1997:175-213.