planned admissions to the hospital. Emergency was the first reason for admission for more than half (52.3%) of the patients receiving dialysis infected by *S. aureus*.

The median time between admission and the principal procedure was 1 day and the interquartile range (IQR) was 0 to 4 days. The median length of stay was 7 days (IQR, 4 to 14 days) in both groups. HCUP has many gaps in the data for variables concerning the hospitals; bed size categories were based on the number of hospital beds and were specific to the hospital location and teaching status. Nearly half of the healthcare payers were insured by Medicare, as was the case in 81.2% of the patients receiving dialysis reported to be infected by *S. aureus*. This is most likely because Medicare provides health insurance to individuals 65 years and older and to those who have permanent kidney failure or certain disabilities. Nearly half of the patients (postoperative patients, 48.6%; patients receiving dialysis, 54.7%) infected by *S. aureus* had routine discharge status. The discharge statuses in decreasing order were home healthcare, skilled nursing facility, and another type of facility. Among postoperative patients and those receiving dialysis who were infected by *S. aureus*, 1.1% and 9.8% of patients died, respectively. Finally, Tables 1 and 2 list the diagnoses and procedures, respectively, more frequently retrieved from patients identified with severe nosocomial infections due to *S. aureus* (n = 1,147) when we combined the selected ICD-9-CM codes for severe nosocomial infections due to *S. aureus*. All of these procedures could be the source of severe nosocomial infections, but the 1997 HCUP Nationwide Inpatient Sample did not provide the exact dates of the procedures and diagnoses. We were therefore unable to clearly determine which pathologies were responsible for the severity and the necessity of using medical devices before onset of the nosocomial infection.

This database did not permit clear description of patient profiles for those at risk of acquiring severe nosocomial infections and, ultimately, did not identify new groups of patients who could potentially benefit from a preventive vaccine against nosocomial infections due to *S. aureus*. However, for the patients receiving dialysis, the results of our analysis are in line with the data from the literature (approximately 10 complications per 100 patient-years).[7,8] The identification of patient profiles at risk of nosocomial infection to assist in the development of a preventive vaccine recommendation against *S. aureus* remains a challenge.

TABLE 2
PROCEDURES REPORTED FROM THE 1997 NATIONWIDE INPATIENT SAMPLE OF THE HEALTHCARE COST AND UTILIZATION PROJECT AND MORE FREQUENTLY RETRIEVED FROM PATIENTS WITH SEVERE NOSOCOMIAL INFECTIONS ACCORDING TO THE INTERNATIONAL CLASSIFICATION OF DISEASES, NINTH REVISION, CLINICAL MODIFICATION DURING HOSPITALIZATION (N = 1,147)

Primary and Secondary Procedure	PR1	PR–PR1	PR
Excisional debridement of wound, infection, or burn (86.22)	142 (69.6%)	62	204
Incision of skin or subcutaneous tissue with removal of foreign body or drainage (86.04, 86.05, 86.09)	69 (41.8%)	96	165
Venous catheterization, not classified elsewhere (38.93)	46 (21.8%)	165	211
Hemodialysis and venous catheterization for renal dialysis (39.95, 38.95)	42 (33.1%)	85	127
Injection of antibiotic and transfusion of packed cells (99.0, 99.21)	24 (14.9%)	137	161
Insertion of totally implantable vascular access device (86.07)	10 (18.9%)	43	53

PR1 = principal procedure in Healthcare Cost and Utilization Project (often the reason for admission to the hospital); PR–PR1 = secondary procedure; PR = principal and secondary procedures (merged together into this denomination).

REFERENCES

1. Shinefield H, Black S, Fattom A, et al. Use of a *Staphylococcus aureus* conjugate vaccine in patients receiving hemodialysis. *N Engl J Med* 2002;346:491-496.
2. Healthcare Cost and Utilization Project (HCUP). *Nationwide Inpatient Sample, Release 6*. Rockville, MD: Agency for Healthcare Research and Quality; 2003. Available at www.ahrq.gov/data/hcup/hcupnis.htm. Accessed May 3, 2004.
3. Best AE. Secondary data bases and their use in outcomes research: a review of the area resource file and the Healthcare Cost and Utilization Project. *J Med Syst* 1999;23:175-181.
4. Zhao SZ, Wong JM, Davis MB, Gersh GE, Johnson KE. The cost of inpatient endometriosis treatment: an analysis based on the Healthcare Cost and Utilization Project Nationwide Inpatient Sample. *American Journal of Managed Care* 1998;4:1127-1134.
5. Kong SX, Hatoum HT, Zhao SZ, Agrawal NM, Geis SG. Prevalence and cost of hospitalization for gastrointestinal complications related to peptic ulcers with bleeding or perforation: comparison of two national databases. *American Journal of Managed Care* 1998; 4:399-409.
6. Bentham WD, Cai L, Schulman KA. Characteristics of hospitalizations of HIV-infected patients: an analysis of data from the 1994 Healthcare Cost and Utilization Project. *J Acquir Immune Defic Syndr* 1999;22:503-508.
7. D'Agata EM, Mount DB, Thayer V, Schaffner W. Hospital-acquired infections among chronic hemodialysis patients. *Am J Kidney Dis* 2000;35:1083-1088.
8. Nielsen J, Kolmos HJ, Espersen F. *Staphylococcus aureus* bacteraemia among patients undergoing dialysis: focus on dialysis catheter-related cases. *Nephrol Dial Transplant* 1998;13:139-145.

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Evaluation of Surgical-Site Infections Following Cardiovascular Surgery

To the Editor:

Infections after cardiovascular surgery are an important cause of morbidity and mortality. During the past decade, prevention of surgical-site infections (SSIs) after cardiovascular surgery has become an important component of quality assurance

TABLE
BACTEREMIA AND MORTALITY RATES AMONG DIFFERENT ISOLATES AND TYPES OF SURGICAL-SITE INFECTIONS

Isolated Bacteria	Deep Sternal SSI			Superficial Sternal SSI			Leg Harvest-Site Infection			Total
	No.	Bacteremia	Mortality	No.	Bacteremia	Mortality	No.	Bacteremia	Mortality	
Methicillin-resistant CNS	12	3 (25%)	3 (25%)	10	-	-	-	-	-	22
MRSA	16	9 (56%)	4 (25%)	1	-	-	-	-	-	17
Methicillin-susceptible CNS	8	-	-	4	-	-	2	-	-	14
MSSA	7	1 (14%)	1 (14%)	4	-	-	-	-	-	11
Escherichia coli	5	-	-	2	-	-	-	-	-	7
Klebsiella pneumoniae	2	-	1 (50%)	1	-	-	1	-	-	4
Enterobacter cloacae	-	-	-	1	-	-	1	1 (100%)	-	2
Acinetobacter baumannii	2	1*	1 (50%)	4	-	–	1	-	-	7
Pseudomonas aeruginosa	1	1*	1 (100%)	1	-	-	-	-	-	2
None	1	-	-	2	-	-	-	-	-	3
Total	54	15 (28%)	11 (20%)	30	-	-	5	1 (20%)	-	89

SSI = surgical-site infection; CNS = coagulase-negative staphylococci; MRSA = methicillin-resistant *Staphylococcus aureus*; MSSA = methicillin-susceptible *S. aureus*.
*Bacteremia or sepsis due to MRSA that has been isolated only from blood and not from other sites of the body, including surgical site.

and hospital cost containment. Many authorities have turned to surveillance of SSIs after coronary artery bypass graft to evaluate and compare hospital performance. Most data are from university or community hospitals and few data are available from private medical centers.

Florence Nightingale Hospital is a 300-bed, private medical center in Istanbul, Turkey, affiliated with Kadir Has University. Approximately 3,000 cardiovascular and vascular operations are performed there each year. Active laboratory-based surveillance for nosocomial infections has been conducted at the hospital since 1999. We evaluated adult patients having SSIs following cardiovascular surgery, by prevalence, type, microbiology, and outcome, from January 1999 to June 2002.

Centers for Disease Control and Prevention definitions were used in the diagnosis of SSIs.[1] Infections of the skin were considered superficial and infections of the sternum, mediastinum, or both were considered deep SSIs. Surgeon diagnosis and sometimes computed tomography or magnetic resonance imaging were used to differentiate deep from superficial infection. Leg harvest-site infections were also included in the study.

Patients were recorded as having secondary bacteremia if the same pathogen was isolated from the blood and the surgical site, and no other potential source of bacteremia was present. Conventional methods were used for culture of microorganisms. Sceptor (Becton Dickinson, Cockeysville, MD) was used for speciation and antibacterial susceptibility testing.

During the study period, a total of 6,709 adults underwent cardiovascular surgery, and 89 (1.3%) had SSIs afterward. Thirty-seven (41.6%) of the patients were female and 52 (58.4%) were male. The mean age was 61.25 years (range, 42 to 80 years). Thirty-seven (41.6%) of the patients were diabetic, 4 (4.5%) were obese, and 7 (7.9%) were diabetic and obese.

Isolated bacteria, types of SSIs, and the bacteremia and mortality rates among different isolates and types of SSIs are listed in the table.

The rates of deep sternal SSI, superficial sternal SSI, and leg harvest-site infection were 0.8%, 0.45%, and 0.07%, respectively. The deep sternal SSI rate in our study was in accordance with the values that range between 0.5% and 2.3% in the literature.[2-5] However, our rates for superficial sternal SSI and harvest-site infection were lower than rates reported in the literature of 1.9% to 5.3% and 0.9% to 14.6%, respectively.[2-5] One of the reasons for that may be the strict preventive measures taken at the hospital. On the other hand, patients are discharged on the 10th day after the operation if there is no serious problem that requires extra hospitalization. Some cases of superficial SSIs and leg harvest-site infections may not result in a culture or may occur after discharge and those patients may not return to the hospital. If so, the true incidence of superficial sternal SSIs and leg harvest-site infections may be higher than that found in this study.

The distribution of bacteria causing SSIs following cardiovascular surgery was similar to that described in the literature. Coagulase-negative staphylococci and *Staphylococcus aureus* were the leading causes of SSIs following cardiovascular surgery, especially in the sternal area as in previous studies.[4,6] Methicillin resistance among staphylococci was high in our study. Methicillin-resistant staphylococci were all susceptible to vancomycin and teicoplanin, and trimethoprim–sulfamethoxazole susceptibility was greater than 90%. The rate of gram-negative infections was low.

Superficial SSIs cause morbidity, but do not seem to be directly associated with more serious adverse outcomes. The overall mortality rate following cardiovascular surgery was 1.8%. The rates of bacteremia and mortality were high in patients with deep sternal SSIs, suggesting that deep sternal SSIs increase the mortality rate.

The mortality rate of patients with bacteremia was higher than that of patients without bacteremia (50% vs 4%). Six of the 11 deaths were associated with methicillin-resistant *S. aureus* (MRSA) infections. Murphy et al. also reported that MRSA infection

had a high mortality rate in vascular surgical patients.[7]

Evaluating risk factors and preventing SSIs is important in each setting. Preoperative nasal carriage of *S. aureus* by patients was accepted as an independent risk factor for *S. aureus* SSIs and antibiotic ointment was thus proposed to decrease the risk.[8,9] Intranasal mupirocin ointment began being used at our hospital for all patients undergoing cardiovascular surgery in January 2001. SSI rates have trended higher since that time (41 of 2,650 [1.5%] vs 48 of 4,059 [1.2%]; relative risk [RR], 1.31; 95% confidence interval [CI_{95}], 0.86 to 1.98; $P = .24$) and *S. aureus* SSIs have trended lower as a proportion of all SSIs (11 of 41 [26.8%] vs 19 of 48 [39.6%]; RR, 0.68; CI_{95}, 0.37 to 1.25; $P = .29$).

SSI is an important cause of morbidity and mortality following cardiovascular surgery. Deep sternal SSIs were associated with secondary bacteremia and mortality. Coagulase-negative staphylococci and *S. aureus* were the leading causes, but MRSA seems to be associated with particularly adverse outcomes. For reliable rates of superficial sternal and harvest-site infections, postdischarge surveillance is necessary.

REFERENCES

1. Horan TC, Gaynes RP, Martone WJ, Jarvis WR, Emori TG. CDC definitions of nosocomial surgical site infections, 1992: a modification of CDC definitions of surgical wound infections. *Am J Infect Control* 1992;20:271-274.
2. Vuorisalo S, Haukipuro K, Pokela R, Syrjala H. Risk features for surgical-site infections in coronary artery bypass surgery. *Infect Control Hosp Epidemiol* 1998;19:240-247.
3. Harbarth S, Samore MH, Lichtenberg D, Carmeli Y. Prolonged antibiotic prophylaxis after cardiovascular surgery and its effect on surgical site infections and antimicrobial resistance. *Circulation* 2000;101:2916-2921.
4. L'Ecuyer PB, Murphy D, Little JR, Fraser VJ. The epidemiology of chest and leg wound infections following cardiothoracic surgery. *Clin Infect Dis* 1996;22:424-429.
5. McConkey SJ, L'Ecuyer PB, Murphy DM, Leet TL, Sundt TM, Fraser VJ. Results of a comprehensive infection control program for reducing surgical-site infections in coronary artery bypass surgery: further data from the authors. *Infect Control and Hosp Epidemiol* 1999;20:791-792.
6. Spelman DW, Russo P, Harrington G, et al. Risk factors for surgical wound infection and bacteraemia following coronary artery bypass surgery. *Australian and New Zealand Journal of Surgery* 2000;70:47-51.
7. Murphy GJ, Pararajasingam R, Nasim A, Dennis MJ, Sayers RD. Methicillin-resistant *Staphylococcus aureus* infection in vascular surgical patients. *Ann R Coll Surg Engl* 2001;83:158-163.
8. Kluytmans JA, Mouton JW, Ijzerman EP, et al. Nasal carriage of *Staphylococcus aureus* as a major risk factor for wound infections after cardiac surgery. *J Infect Dis* 1995;171:216-219.
9. Wong ES. The price of a surgical-site infection: more than just excess length of stay. *Infect Control Hosp Epidemiol* 1999;20:722-724.

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Is Gastrointestinal Endoscopy a Risk Factor for Whipple's Disease?

To the Editor:

In the March 2003 issue of *Infection Control and Hospital Epidemiology*, a study by La Scola et al. was published that investigated whether high-level disinfection may be inadequate to prevent patient-to-patient transmission of *Tropheryma whipplei* via gastrointestinal (GI) endoscopes.[1] *T. whipplei* is a poorly understood intracellular gram-positive bacterium that causes Whipple's disease, a rare and chronic disorder that usually damages the small intestines, although other organs including the heart and central nervous system may also be affected. Symptoms of this chronic disease include fever, diarrhea, weight loss, and abdominal pain. Duodenal biopsy during esophagogastroduodenoscopy is usually performed to diagnose infection. Without appropriate antibiotic treatment, Whipple's disease can be fatal. The mode of transmission of *T. whipplei* is unclear.

The rationale for this study's investigation was based primarily on the clinical examination of two patients who were each diagnosed as having Whipple's disease 3 years after gastroscopy and intestinal biopsy. Although infrequent, GI endoscopes have been reported to transmit bacteria and other infectious agents. In each case, however, at least one crucial reprocessing step was breached. Flexible endoscopes that are properly cleaned, high-level disinfected, and dried in accordance with published guidelines pose virtually no risk of disease transmission (with the exception of several defective and subsequently recalled bronchoscope models).

To evaluate whether *T. whipplei* can survive high-level disinfection and be transmitted via GI endoscopes, La Scola et al. exposed a titer of 10^5 inclusion-forming units/mL of this vegetative bacterium to three different high-level disinfectants.[1] One of the high-level disinfectants contained 2% glutaraldehyde and the other two, although different products, each contained 1.5% peracetic acid. Whereas the two peracetic acid products were preformulated and ready for use, the solution of 2% glutaraldehyde (pH, 8) was reportedly produced by thawing and diluting a frozen concentrate just prior to exposure to *T. whipplei*. Sterile distilled water was used as a negative control, and a suspension of *Pseudomonas aeruginosa* (10^5 colony-forming units/mL) was used as a positive control.

The results of the study indicated that exposure to the thawed and diluted solution of 2% (alkaline) glutaraldehyde (alkaline) for 60 minutes reduced the initial titer of viable *T. whipplei* by 3 $\log_{10}$ (or 99.9%). Similar results were recorded for both peracetic acid products. Sterile distilled water, as expected, had no biocidal effect, whereas all three of the high-level disinfectants reduced the control suspension of *P. aeruginosa* by 5 $\log_{10}$ or greater after 5 minutes of exposure. The latter result presumably demonstrated that each of the three high-level disinfectants was biocidal and destroyed vegetative bacteria (with the possible exception of *T. whipplei*).

This study by La Scola et al. is the first to report that high-level disinfection may be inadequate to prevent transmission of some vegetative bacteria including *T. whipplei* via GI endoscopes. This conclusion is unique and based on only this one study's results, however, and therefore warrants circumspect interpretation and cautious extrapolation. Because *T. whipplei* is an actinomycete (ie, bacterium) that is related to mycobacteria, high-level disinfec-