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.

Diler Coskun, MD
Department of Infectious Diseases and Clinical Microbiology
Kadir Has University
Jale Aytaç, MD
Seda Deveci
Florence Nightingale Hospital
Emine Sönmez, MD
Department of Infectious Diseases and Clinical Microbiology
Kadir Has University
Istanbul, Turkey

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-

tion, which is defined to achieve at least a 6 $\log_{10}$ reduction of mycobacteria, would have been expected to destroy the entire titer of *T. whipplei* used in this study (ie, 10^5 inclusion-forming units/mL), instead of reportedly achieving only a 3 $\log_{10}$ reduction. Contrary to the results of this study by La Scola et al., other studies have consistently demonstrated that high-level disinfection, achieved using several different products including 2% (alkaline) glutaraldehyde, destroys all pathogenic microorganisms including vegetative bacteria, mycobacteria, and some types of spore-forming bacteria, such as *Clostridium difficile*.[2]

Several factors may have contributed to the unique results of the study by La Scola et al. For example, the temperature of the 2% glutaraldehyde solution (and the two peracetic acid products) to which *T. whipplei* was exposed was not recorded or discussed. According to the label of virtually every 2% glutaraldehyde solution sold in the United States, it is necessary to elevate (and monitor) the immersion temperature to 25°C or higher to achieve high-level disinfection.[2] Indeed, small increases in the temperature of a high-level disinfectant can significantly increase its biocidal properties. It is unclear whether the temperature of the 2% glutaraldehyde solution, which the study reported was produced by thawing and diluting a frozen concentrate just prior to testing, was several degrees below 25°C during testing, reducing its effectiveness and preventing it from achieving high-level disinfection. Studies that do not report the temperature of a high-level disinfectant used to destroy bacteria or reprocess instruments can provide data and conclusions of limited, if any, significance.

Moreover, the 2% glutaraldehyde solution used for this study was produced on site and was not a prepackaged product manufactured in accordance with appropriate regulatory guidelines, such as those typically required by the U.S. Food and Drug Administration of manufacturers of high-level disinfectants and liquid chemical sterilants. As a result, the effectiveness, consistency, and chemical composition of this 2% glutaraldehyde solution, each of which is a factor crucial to the reproducibility, reliability, and integrity of the study's data, may be questioned. Also, although it was demonstrated to destroy *P. aeruginosa*, this 2% glutaraldehyde solution, like the study's two peracetic acid products, was not shown to destroy mycobacteria, which generally are the appropriate and necessary microorganisms to use as positive controls whenever testing the biocidal effectiveness of high-level disinfectants. Failure to have used mycobacteria as a positive control limits the validity and significance of the study's data and conclusions.

Although perhaps due to a protective effect provided by an amorphic glycoprotein material that La Scola et al. reportedly observed surrounding its cells,[1] it is unlikely that *T. whipplei* is more resistant to high-level disinfection than other vegetative bacteria and mycobacteria (to which *T. whipplei* is phylogenetically related) and may require sterilization for its destruction. Some other factor, such as the temperature and/or chemical composition of the three high-level disinfectants used during testing, is a more likely explanation for this study's reported unusual resistance of *T. whipplei* to high-level disinfection.

Noteworthy, this study reported that both of the patients diagnosed as having Whipple's disease 3 years earlier had undergone intestinal biopsy during gastroscopy. This study, however, solely focused on the potential for inadequate high-level disinfection of the GI endoscope and did not consider or discuss whether instead inadequate reprocessing of the biopsy forceps used during gastroscopy could have played a significant role in the study's two reported cases of transmission of Whipple's disease. Recent studies have identified failure to adhere to reprocessing guidelines for reusable biopsy forceps, which require cleaning and sterilization, as the cause of transmission of infectious agents, including hepatitis C, during GI endoscopy.[3]

The finding by La Scola et al. that *T. whipplei* may survive high-level disinfection and be transmitted via GI endoscopes should not cause alarm or raise undue concern. Failure to record the temperature of each high-level disinfectant during testing, as well as selection of a vegetative bacterium for a positive control instead of mycobacteria, limit this study's scope and significance. Most important, application of this study to the clinical setting is somewhat tenuous. Specifically, the results of this study do not reflect the significant reduction in the risk of disease transmission and nosocomial infection achieved by cleaning, a mechanical process that is the standard of care, required before disinfection (and sterilization) of endoscopes and their accessories and, as acknowledged by La Scola et al., reported to achieve a 5 $\log_{10}$ reduction of microorganisms. If this study had either mechanically cleaned surfaces or devices contaminated with *T. whipplei* prior to exposure to each of the three high-level disinfectants, or acknowledged, corrected for, and incorporated into its methodology and results the expected log reduction reportedly achieved during cleaning, then for each of these three high-level disinfectants the entire titer of *T. whipplei* would have been destroyed and high-level disinfection achieved. In short, cleaning followed by high-level disinfection of a GI endoscope contaminated with *T. whipplei* (or any other infectious agent) would be expected to prevent disease transmission.

Therefore, although it is plausible that GI endoscopy and intestinal biopsy may be risk factors for Whipple's disease, more research is necessary to evaluate this study's conclusion and place its results in better perspective. Publication of additional corroborating data is essential before specific conclusions can be drawn and guidelines provided that recommend that patients who have previously undergone intestinal biopsy during esophagogastroduodenoscopy be examined and assessed for Whipple's disease.

REFERENCES

1. La Scola B, Rolain J-M, Maurin M, Raoult D. Can Whipple's disease be transmitted by gastroscopes? *Infect Control Hosp Epidemiol* 2003;24:191-194.
2. Muscarella LF. Soaking endoscopes in 2% glutaraldehyde for 20 minutes: a risk management perspective. *Am J Infect Control* 1998;26:153-155.
3. Bronowicki JP, Venard V, Botte C, et al. Patient-to-patient transmission of hepatitis C virus during colonoscopy. *N Engl J Med* 1997;337:237-240.

Lawrence F. Muscarella, PhD
Custom Ultrasonics, Inc.
Ivyland, PA

The authors reply.