To the Editor:

Break, referred to as cluster 3, was reported in P. aeruginosa linked to endoscopes contaminated in 1991, a report documenting patient infection, even if the cleaning and disinfecting the endoscope to prevent patient infection. Also not discussed in this report (and several others) is whether the AFER’s filtered rinse water and the hospital’s water supply, water faucets, and sink drains were sampled microbiologically.

Obtaining cultures of these sites, as well as of the AFER’s internal components and water filters, has been recommended during an investigation to identify the source of, and risk factors for, patient infection. Indeed, these sampling data are crucial to the conclusion reported by MMWR that human error—that is, inappropriate connection of the AFER to the bronchoscope by hospital staff—was the cause of the patient injuries described in cluster 3. If the filtered rinse water had been sampled and found to be contaminated with P. aeruginosa (the outbreak microorganism in cluster 3), then the MMWR’s conclusion, which suggests patient-to-patient transmission, would likely be incomplete, and the reported patient injuries might have occurred even if hospital staff had properly connected the bronchoscopes to the AFER.

Although effective and routinely employed by healthcare facilities to reprocess bronchoscopes and gastrointestinal endoscopes, AFERs have their limitations. For example, unlike other processes that use heat, gas, or a plasma to disinfect or sterilize reusable instruments, current AFERs immerse the endoscope in a liquid chemical sterilant, requiring that the endoscope be rinsed with a large volume of water to remove potentially toxic chemical residues. This step is arguably the Achilles’ heel of AFERs (and other liquid-chemical sterilant-based processes), as the success of these devices is therefore vulnerable to, and depends significantly on, the quality of the rinse water, which is difficult to monitor and control. Rinse water that contains microorganisms can recontaminate the endoscope and result in patient infection, even if the cleaning and chemical immersion steps were effective.

More recently, an outbreak linked to endoscopes contaminated with P. aeruginosa was reported in MMWR. Investigators of this outbreak, referred to as cluster 3, concluded that inadequately trained healthcare staff improperly connect bronchoscopes to an AFER, resulting in multiple patient infections and one fatality (for unclear reasons, the fatality was not reported in this MMWR).

Although several noteworthy recommendations were provided in the MMWR, absent was a needed discussion of the importance of drying the endoscope to prevent patient infection. Also not discussed in this MMWR was whether the AFER’s filtered rinse water and the hospital’s water supply, water faucets, and sink drains were sampled microbiologically. In conclusion, to prevent patient infection caused by inadequate dried endoscopes, I encourage federal regulatory agencies and professional endoscopy and infection control organizations to reemphasize the importance of thoroughly drying and properly storing the endoscope. Also encouraged, to prevent contamination and patient infection, are discussions aimed at clarifying and detailing the definitions and microbiological dif-
ferences between the different types of water used to rinse endoscopes. Lacking in the medical literature is a clear description of the parameters of sterile water, how it is produced, and how it compares to, and differs microbiologically from, filtered water claimed to be bacteria-free or sterile. Finally, although the CDC does not recommend routine microbiological sampling of endoscopes or the water used to rinse them, I recommend revisiting the conditions under which such a practice might be indicated to reduce the risk of patient infection.

REFERENCES

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Triclosan and Antibiotic Resistance in S. aureus

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The debate on the connection between chemical germicides and antibiotic resistance continues. Suller and Russell, from the Welsh School of Pharmacy, Cardiff University, recently reported on their studies with triclosan. Triclosan (2,4,4’-trichloro-2’-hydroxydiphenyl ether) is an antimicrobial agent used in hygiene products, plastics, and kitchenware. Handwashing products containing triclosan are used by healthcare workers in wards with patients infected with methicillin-resistant S. aureus. S. aureus strains with low-level resistance to triclosan have emerged. It has been claimed that strains with decreased susceptibility to biocides may also be less susceptible to antibiotics.

They tested the susceptibility of S. aureus clinical isolates to triclosan and several antibiotics. Triclosan minimum inhibitory concentrations (MICs) ranged between 0.025 and 1 mg/L. Some, but not all, strains were resistant to several antibiotics and showed low-level triclosan resistance. S. aureus mutants with enhanced resistance to triclosan (<1 mg/L) were isolated. In several cases this resistance was stably inherited in the absence of triclosan. These mutants were not more resistant than the parent strain to several antibiotics. Changes in triclosan MICs associated with the acquisition of a plasmid encoding mupirocin resistance were not observed, suggesting that the triclosan and mupirocin co-resistance seen in a previous study was not the result of a single resistance gene or separate genes on the same plasmid.

The continuous exposure of a triclosan-sensitive S. aureus strain to sub-MIC concentrations of triclosan for 1 month did not result in decreased susceptibility to triclosan or to several antibiotics tested. Triclosan-induced potassium leakage and bactericidal effects on a triclosan-sensitive strain, a resistant strain, and a strain selected for increased resistance were compared with those of non-growing organisms, exponentially growing organisms, and organisms in the stationary phase. No significant differences between the strains were observed under these conditions despite their different MICs.

The authors point out that biocides have multiple target sites, and so MICs often do not correlate with bactericidal activities. The ability of S. aureus to develop resistance to triclosan and the current view that triclosan may have a specific target in Escherichia coli, namely enoyl reductase, underline the need for more research on the mechanisms of action and resistance.