was reduced in the presence of organic load (Figure 1). On patient wards, application of Sterilox HG resulted in significant reductions in total aerobic and facultative bacterial counts (mean CFU, 39 vs 0.73; $P = .0003$) and in positive *C. difficile* cultures (8/66 [12%] vs 0/66; $P = .006$). Spraying of Sterilox HG on sets of equipment was simple and required only approximately 15 seconds per application. Application of Sterilox HG did not result in production of noticeable noxious fumes but was described as producing an odor similar to that of swimming pool water. There were no reported complaints from nursing staff or patients.

Our results demonstrate that spraying equipment with an electrochemically activated saline solution is a simple and effective means to reduce contamination with *C. difficile* and other healthcare-associated pathogens. The potential advantages of this method for equipment disinfection include efficiency, ability to maintain sufficient disinfectant contact time when surfaces were thoroughly sprayed, thorough application of disinfectant on objects with irregular surfaces that might be difficult to reach with a cloth, relatively low risk for skin or respiratory irritation, and ability to perform disinfection in patient care areas. Potential disadvantages of this method include lack of mechanical removal of pathogens and organic material, dependence on the operator to apply sufficient disinfectant to thoroughly wet the surfaces, and infeasibility of leaving sprayed surfaces to air dry for 15–30 minutes if equipment is needed for immediate reuse.

Our study has some limitations. A small number of strains were tested, and only 1 disinfectant was tested as a comparator to Sterilox HG. We did not perform a complete assessment of materials compatibility and did not determine the effect of different surfaces on effectiveness of Sterilox. Finally, only a 10-minute contact time was used in the complete set of laboratory studies.

ACKNOWLEDGMENTS

We thank Svetlana Panicheva, PhD, and Mark Sampson, PhD (PuriCore), for providing the Sterilox HG used in this study and for helpful discussions.

Financial support. This work was supported by the Department of Veterans Affairs.

Potential conflicts of interest. All authors report no conflicts of interest relevant to this article. All authors submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest, and the conflicts that the editors consider relevant to this article are disclosed here.

Dennis Fertelli;[1] Jennifer L. Cadnum, BS;[2] Michelle M. Nerandzic, BS;[2] Brett Sitzlar, BS;[3] Sirisha Kundrapu, MD;[4] Curtis J. Donskey, MD[3,4]

Affiliations: 1. Infection Control Department, Cleveland Veterans Affairs Medical Center, Cleveland, Ohio; 2. Research Service, Cleveland Veterans Affairs Medical Center, Cleveland, Ohio; 3. Geriatric Research Education and Clinical Center, Cleveland, Ohio; 4. Case Western Reserve University School of Medicine, Cleveland, Ohio.

Address correspondence to Curtis J. Donskey, MD, Geriatric Research Education and Clinical Center, 10701 East Boulevard, Cleveland, OH 44106 (curtisd123@yahoo.com).

Infect Control Hosp Epidemiol 2013;34(5):543-544

 0899-823X/2013/3405-0021$15.00. DOI: 10.1086/670226

REFERENCES

1. Manian FA, Meyer L, Jenne J. *Clostridium difficile* contamination of blood pressure cuffs: a call for a closer look at gloving practices in the era of universal precautions. *Infect Control Hosp Epidemiol* 1996;17:180–182.
2. Walker N, Gupta R, Cheesbrough J. Blood pressure cuffs: friend or foe? *J Hosp Infect* 2006;63:167–169.
3. Porwancher R, Sheth A, Remphrey S, Taylor E, Hinkle C, Zervos M. Epidemiological study of hospital acquired infection with vancomycin-resistant *Enterococcus faecium*: possible transmission by an ear-probe thermometer. *Infect Control Hosp Epidemiol* 1997;18:771–774.
4. Livornese LL, Dias S, Samuel C, et al. Hospital-acquired infection with vancomycin-resistant *Enterococcus faecium* transmitted by electronic thermometers. *Ann Intern Med* 1992;117:112–116.
5. Brooks SE, Veal RO, Kramer M, et al. Reduction in the incidence of *Clostridium difficile*–associated diarrhea in an acute care hospital and a skilled nursing facility following replacement of electronic thermometers with single-use disposables. *Infect Control Hosp Epidemiol* 1992;12:98–103.
6. Jernigan JA, Siegman-Igra Y, Guerrant RC, Farr BM. A randomized crossover study of disposable thermometers for prevention of *Clostridium difficile* and other nosocomial infections. *Infect Control Hosp Epidemiol* 1998;19:494–499.
7. Shetty N, Srinivasan S, Holton J, Ridgway GL. Evaluation of microbicidal activity of a new disinfectant: Sterilox 2500 against *Clostridium difficile* spores, *Helicobacter pylori*, vancomycin resistant *Enterococcus* species, *Candida albicans* and several *Mycobacterium* species. *J Hosp Infect* 1999;41:101–105.
8. Selkon JB, Babb JR, Morris R. Evaluation of the antimicrobial activity of a new super-oxidized water, Sterilox, for the disinfection of endoscopes. *J Hosp Infect* 1999;41:59–70.
9. Niezgoda JA, Sordi PJ, Hermans MH. Evaluation of Vashe Wound Therapy in the clinical management of patients with chronic wounds. *Adv Skin Wound Care* 2010;23:352–357.
10. Nerandzic MM, Donskey CJ. Effective and reduced-cost modified selective medium for isolation of *Clostridium difficile*. *J Clin Microbiol* 2009;47:397–400.

Peripheral Venous Catheter and Bloodstream Infection Caused by *Pseudomonas aeruginosa* after a Contaminated Preoperative Shower

To the Editor—Pseudomonas aeruginosa, a ubiquitous gram-negative bacillus frequently involved in healthcare-associated infections, is usually found in water-related environmental reservoirs—such as pipes, taps, or showers—where it develops in a naturally resistant and adherent biofilm.[1] Several *P.*

aeruginosa outbreaks have been linked to an environmental source, especially in intensive care, surgery, and hematology units. However, the route of contamination (from environment to patients or vice versa) is difficult to ascertain, and reports where the route of transmission from environment to patients is proven remain scarce. We report here a case of bloodstream infection due to direct contamination of a peripheral venous catheter (PVC) by a *P. aeruginosa* strain found in the water used for the preoperative shower of an immunocompetent patient.

A 57-year-old man was admitted to the cardiology unit on the eve of a programmed percutaneous catheter ablation of atrial fibrillation. The patient's medical history was thyroid dysfunction and hypertension, both controlled by treatment, and minor mental retardation. He had never been hospitalized before, had no chronic wounds, and bore no indwelling medical device. Upon admission, a first preoperative antiseptic shower with povidone-iodine-based soap was followed by insertion of a PVC on the forearm (after skin antisepsis with 2% alcoholic chlorhexidine) and shearing of the inguinal area. The next morning, shortly before the invasive procedure, a second shower with povidone-iodine scrub was performed, with the catheter allegedly protected by an occlusive dressing (Tegaderm). The radiofrequency procedure was successfully performed via transient femoral artery catheterization.

The next morning, the patient had a fever of 37.8°C, reaching 38.7°C in the evening, with chills and inflammation of the PVC insertion. The PVC was removed and cultured, as well as 3 blood cultures, one of which yielded *P. aeruginosa*, as did the endoluminal catheter tip. The *P. aeruginosa* strain was susceptible to all antibiotics tested except cefotaxime. Clinical evolution was favorable under anti-infective treatment (ceftazidime + amikacin), and the patient returned home on day 11 with no relapse. The case fulfilled the definition of healthcare-associated infections and was thus reported to the infection control team for further investigation.

Retrospective survey of the microbiology database for the cardiology unit found only 1 unrelated case of community-acquired *P. aeruginosa* infection in 2009. Considering that the patient had no risk factor for prior *P. aeruginosa* colonization, an environmental source was suspected. Microbiological investigation was performed in the month following the event and consisted of multiple samplings of water and swabs from faucets, sink drains, and showerheads. Swabs and water samples were submitted to qualitative and quantitative cultures, respectively. Positive cultures were identified by classical biochemical tests, including growth on cetrimide media and pigmentation on King A/King B media for *P. aeruginosa*. Results of environmental sampling are summarized in Table 1. *P. aeruginosa* was abundantly found in cold and hot water of the shower (shower 1) used by the patient and in cold water of the other shower (shower 2) of the ward. The bacterium was also present on showerheads and sink drains. All strains were genotyped by *Spe*I macrorestriction followed by pulsed-field gel electrophoresis.[2] The clinical strains from blood and catheter cultures were identical and shared the same pulsotype as strains from water in showers 1 and 2 as well as the sink of shower 2 (Table 1). All the unit's faucets and showerheads were replaced. Maintenance procedures (monthly scaling and disinfection of terminal valves) were enforced, and subsequent microbiological controls remained negative. No further case of *P. aeruginosa* infection was reported in the unit.

TABLE 1. Microbiological Investigation: Pulsed-Field Gel Electrophoresis (PFGE) Typing of *Pseudomonas aeruginosa* Strains from Patient and Environment

Origin and samples	Culture results	PFGE type
Patient		
Catheter	*P. aeruginosa*	1
Blood culture	*P. aeruginosa*	1
Shower room 1		
Showerhead	*P. aeruginosa*	1
Siphon	Negative culture	
Cold water	800 CFUs/100 mL *P. aeruginosa*	1
Hot water	2,000 CFUs/100 mL *P. aeruginosa*	1
Shower room 2		
Showerhead	*P. aeruginosa*, coagulase-negative staphylococci	2
Siphon	*P. aeruginosa*, coagulase-negative staphylococci	2
Cold water	400 CFUs/100 mL *P. aeruginosa*, 800 CFUs/100 mL *Bacillus* sp.	1
Hot water	1,200 CFUs/100 mL *Bacillus* sp.	
Patient room		
Shower tap	*Enterococcus* sp., *Bacillus* sp., coagulase-negative staphylococci	
Sink drain	*P. aeruginosa*, coagulase-negative staphylococci, *Enterobacter cloacae*	1
Showerhead	*P. aeruginosa*, coagulase-negative staphylococci	3
Cold water	$>10^3$ CFUs/100 mL *Bacillus* sp.	
Hot water	$>10^3$ CFUs/100 mL *Bacillus* sp.	

NOTE. CFU, colony-forming unit.

We report the first documented case of a catheter-related bloodstream infection unambiguously related to a preoperative shower with *P. aeruginosa*–contaminated water. Hospital environment provides many potential reservoirs for *P. aeruginosa*, whose ability to form biofilms increases the risk of contamination of healthcare-related water devices,[1] such as faucets and drains,[3] hydrotherapy bathtubs,[4] bronchoscopes,[2] and endoscope reprocessors.[5] Consistent with French recommendations, in our institution, microbiological control surveys are periodically carried out in high-risk areas (operating theatres, hematology department) or critical devices (endoscope reprocessors). The medical cardiology ward was not under surveillance; thus, we could not determine how long the water and/or pipes had been contaminated before the case occurred. However, identity of patient and environmental strains, together with the short delay between expo-

sure and infection, leave little doubt that contamination originated from shower water.

As many as 2% of community drinking water samples have been found to contain *P. aeruginosa*;[6] thus, chances of encountering the bacterium in everyday life are not nil. Moreover, community-acquired cutaneous infections or malignant otitis linked to contaminated public pools or spas[7] have been reported. Otherwise, *P. aeruginosa* infections usually occur in hospitalized[8] or predisposed patients,[9] following opportunistic colonization of respiratory and digestive tracts. The high bacterial inoculum found in our water samples could explain the infection's rapid development in this patient with no predisposing factor for *P. aeruginosa* infection. The ill-protected PVC provided a direct portal of entry for bloodstream infection. This observation led us to revise clinical procedures in the cardiology department, foregoing systematic venous catheterism of patients upon admission and enforcing waterproof protection of PVC insertion dressings when a shower is required.

Following disinfection and replacement of faucets and showerheads, no further *P. aeruginosa* contamination of water points was detected, arguing against the installation of disposable antibacterial filters in this low-risk unit. By contrast, we advocate widespread use of filters for all clinically significant water points in high-risk units whenever *P. aeruginosa* contamination is detected.

ACKNOWLEDGMENTS

Potential conflicts of interest. All authors report no conflicts of interest relevant to this article. All authors submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest, and the conflicts that the editors consider relevant to this article are disclosed here.

Sara Romano, PharmD;[1,2] Alice Bourdier, PharmD;[1,2] Sylvie Parer, MD;[1,2] Agnès Masnou;[1] Lydie Burgel, MSc;[2] F. Raczka, MD;[3] Brigitte Lamy, MD;[1,4] Estelle Jumas-Bilak, PhD, PharmD;[1,2] Anne Lotthé, MD[1,2]

Affiliations: 1. Unité Mixte de Recherche 5119, Équipe Pathogènes et Environnements, Université Montpellier 1, Montpellier, France; 2. Département d'Hygiène Hospitalière, Centre Hospitalier Régional Universitaire de Montpellier, Montpellier, France; 3. Service de Cardiologie, Centre Hospitalier Régional Universitaire de Montpellier, Montpellier, France; 4. Laboratoire de Bactériologie, Centre Hospitalier Régional Universitaire de Montpellier, Montpellier, France.

Address correspondence to Estelle Jumas-Bilak, PhD, PharmD, Unité Mixte de Recherche 5119, Equipe Pathogènes et Environnements, 15 Avenue Charles Flahault, BP 14491, 34093 Montpellier Cedex 5, France (ebilak@univ-montp1.fr).

Infect Control Hosp Epidemiol 2013;34(5):544-546
 0899-823X/2013/3405-0022$15.00. DOI: 10.1086/670221

REFERENCES

1. Kerr KG, Snelling AM. *Pseudomonas aeruginosa*: a formidable and ever-present adversary. *J Hosp Infect* 2009;73:338–344.
2. Corne P, Godreuil S, Jean-Pierre H, et al. Unusual implication of biopsy forceps in outbreaks of *Pseudomonas aeruginosa* infections and pseudo-infections related to bronchoscopy. *J Hosp Infect* 2005;61:20–26.
3. Vianelli N, Giannini MB, Quarti C, et al. Resolution of a *Pseudomonas aeruginosa* outbreak in a hematology unit with the use of disposable sterile water filters. *Haematologica* 2006;91:983–985.
4. Berrouane YF, McNutt LA, Buschelman BJ, et al. Outbreak of severe *Pseudomonas aeruginosa* infections caused by a contaminated drain in a whirlpool bathtub. *Clin Infect Dis* 2000;31:1331–1337.
5. Shimono N, Takahiro T, Tsuchimochi N, et al. An outbreak of *Pseudomonas aeruginosa* infections following thoracic surgeries occurring via the contamination of bronchoscopes and an automatic endoscope reprocessor. *J Infect Chemother* 2008;14:418–423.
6. Mena KD, Gerba CP. Risk assessment of *Pseudomonas aeruginosa* in water. *Rev Environ Contam Toxicol* 2009;201:71–115.
7. Moore JE, Heaney N, Millar BC, Crowe M, Elborn JS. Incidence of *Pseudomonas aeruginosa* in recreational and hydrotherapy pools. *Commun Dis Public Health* 2002;5:23–26.
8. Blanc DS, Francioli P, Zanetti G. Molecular epidemiology of *Pseudomonas aeruginosa* in the intensive care units: a review. *Open Microbiol J* 2007;1:8–11.
9. Brugha RE, Davies JC. *Pseudomonas aeruginosa* in cystic fibrosis: pathogenesis and new treatments. *Br J Hosp Med* 2011;72:614–619.