Outbreak of Severe Pseudomonas aeruginosa Infections Caused by a Contaminated Drain in a Whirlpool Bathtub

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During a 14-month period, 7 patients with hematological malignancies acquired serious infections caused by a single strain of multiply resistant Pseudomonas aeruginosa. A case-control study, culture surveys, and pulsed-field gel electrophoresis implicated a whirlpool bathtub on the unit as the reservoir. All case patients and 32% of control patients used this bathtub (P = .003). The epidemic strain was found only in cultures of samples taken from the bathtub. The drain of the whirlpool bathtub, which was contaminated with the epidemic strain, closed ~2.5 cm below the drain’s strainer. Water from the faucet, which was not contaminated, became contaminated with P. aeruginosa from the drain when the tub was filled. The design of the drain allowed the epidemic strain to be transmitted to immunocompromised patients who used the whirlpool bathtub. Such tubs are used in many hospitals, and they may be an unrecognized source of nosocomial infections. This potential source of infection could be eliminated by using whirlpool bathtubs with drains that seal at the top.

Pseudomonas aeruginosa causes infections in patients whose immune systems or general host defenses are impaired. Patients who are at particular risk of infection with P. aeruginosa are those with neutropenia, cystic fibrosis, or thermal injury; those who have undergone organ transplantation; or those hospitalized in intensive care units, or those treated with mechanical ventilation [1–4].

P. aeruginosa, which has few nutritional requirements, thrives in moist environments, such as drains, and creates biofilms that allow it to grow in macrocolonies within plumbing systems [1, 2, 4, 5]. In addition, P. aeruginosa commonly contaminates plants and vegetables [6–8]. Consequently, the inanimate environment in hospitals may be an important reservoir for these organisms.

Investigators still debate about whether plumbing systems in hospitals transmit P. aeruginosa to susceptible patients [2, 3, 9–13]. However, contaminated hydrotherapy tanks in burn units and pools in physical therapy departments were reservoirs for previous outbreaks of P. aeruginosa infections, such as folliculitis and burn wound infections [14–18]. Similarly in the general community, whirlpool spas have been the source of P. aeruginosa strains causing outbreaks of folliculitis [19–21]. Although they happen rarely, keratitis, pneumonia, urinary tract infections, and bacteremia have been diagnosed in healthy people who used whirlpool spas in motels or in their homes [22–25].

Commercial and residential whirlpools recirculate water that is treated with chlorine, bromide, or other disinfectants. However, P. aeruginosa can persist in biofilms within the hoses, pipes, and filters despite the use of a disinfectant, and P. aeruginosa can proliferate rapidly if disinfectant levels decrease below recommended concentrations [19, 21, 26].

Whirlpool bathtubs, which are drained after every use, are now common in homes, hotels, and hospitals [27, 28]. These tubs have not been associated with outbreaks of folliculitis or serious P. aeruginosa infections. Whirlpool bathtubs are used in several wards, including the hematology and oncology units, at the University of Iowa Hospitals and Clinics (UIHC), Iowa City, Iowa. These tubs were used for many years and had not been shown to be a source of nosocomial infections until a cluster of 3 P. aeruginosa infections was identified during a 30-day period. We describe the epidemic, the epidemic investigation, the results of the investigation, and the interventions used to terminate transmission.

Methods

Hospital and surveillance. UIHC is a 900-bed tertiary health care center that serves patients throughout Iowa and its bordering states. Approximately 30,000 patients are admitted each year. The prospective, hospital-wide surveillance system for nosocomial in-
Infections, begun in 1976, was 81% sensitive and 97% specific when previously validated [29].

Epidemic investigation. After affected patients B, E, and F (table 1) were identified, staff in the Program of Hospital Epidemiology (PHE) reviewed their medical records to gather information on possible risk factors for infection. On several occasions, PHE staff members talked with personnel who worked in the affected unit to determine whether a reservoir could be identified. In addition, PHE staff members searched the infection control database to determine whether other patients had nosocomial infections caused by multiply resistant *P. aeruginosa* during the 2 years preceding the first cluster-related infection. PHE staff members also searched the Clinical Microbiology Laboratory’s database to identify multiply resistant *P. aeruginosa* isolates obtained from patients during the same time.

Culture surveys. After patients B, E, and F (table 1) were identified, staff members from PHE conducted 4 different culture surveys. Staff members obtained samples for cultures from possible environmental reservoirs in the affected unit: water from patients’ rooms, povidone iodine swabs and solutions, hand lotions, bath oil, shampoo, soap dispensers, ice machines, water faucets, drains in sinks and showers, several sites in the unit’s whirlpool bathtub, and solutions used in the whirlpool bathtub. Samples for surveillance cultures were obtained from the perirectal area of 4 patients hospitalized in the unit after the cluster of affected patients was identified.

Samples from the ice machine, water faucets, and drains and from the perirectal area of patients were all plated on blood agar and MacConkey agar plates (Remel, Lenexa, KS). Water samples were filtered, and the filters were placed on blood agar plates. Samples of povidone iodine, hand lotions, bath oil, shampoo, soap, and solutions were inoculated into tubes containing 5 mL of TLSO (Tween 80, lecithin, sodium oleate, proteose peptone, tryptone, and distilled water), incubated at 35°C overnight, plated onto blood agar plates, and incubated for 72 h at 35°C. Samples from hands were obtained by use of the broth-bag method, as described previously [30]. After inoculation, the agar plates were incubated at 35°C for up to 72 h. Oxidase-positive colonies growing on the agar medium were identified to species level by use of the Vitek Systems (BioMerieux, St. Louis, MO).

Antimicrobial susceptibility testing. Isolates were tested for susceptibility to a panel of 10 antimicrobial agents: piperacillin, piperacillin and tazobactam, ticarcillin, gentamicin, tobramycin, amikacin, ciprofloxacin, cefotaxime, ceftazidime, and imipenem. Testing was performed by use of a broth microdilution method according to the National Committee for Clinical Laboratory Standards [31].

Pulsed-field gel electrophoresis (PFGE). Chromosomal DNA

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex/Age, y</th>
<th>Underlying disease</th>
<th>Days of admissionsa</th>
<th>Days of positive culture resultsa</th>
<th>Site</th>
<th>Diarrhea</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>M/40</td>
<td>AML, new</td>
<td>−14−8</td>
<td>0</td>
<td>Blood</td>
<td>Yes; acquired a perianal fissure</td>
<td>Died with ongoing sepsisb</td>
</tr>
<tr>
<td>B</td>
<td>M/54</td>
<td>AML, new</td>
<td>121−164</td>
<td>146</td>
<td>Blood</td>
<td>Tracheal aspirate</td>
<td>Died</td>
</tr>
<tr>
<td>C</td>
<td>M/22</td>
<td>ALL, relapse</td>
<td>198−246</td>
<td>276</td>
<td>Blood</td>
<td>Tracheal aspirate</td>
<td>Died</td>
</tr>
<tr>
<td>D</td>
<td>M/51</td>
<td>AML, new</td>
<td>248−276</td>
<td>Blood</td>
<td>No</td>
<td>Died</td>
<td>Lived</td>
</tr>
<tr>
<td>E</td>
<td>M/57</td>
<td>AML, new</td>
<td>327−373</td>
<td>Blood</td>
<td>Stool</td>
<td>Yes, severe; developed a perianal ulcer</td>
<td>Lived</td>
</tr>
<tr>
<td>F</td>
<td>M/54</td>
<td>RA with subsequent AML</td>
<td>399−404</td>
<td>Hickman site</td>
<td>Urine</td>
<td>Yes; incontinent; slipped on stool in bathroom</td>
<td>Died</td>
</tr>
<tr>
<td>G</td>
<td>M/59</td>
<td>AML, new</td>
<td>397−413</td>
<td>Blood</td>
<td>Hickman site</td>
<td>Admitted with severe diarrhea; later developed anal ulcer</td>
<td>Died</td>
</tr>
</tbody>
</table>

NOTE. ALL, acute myelogenous leukemia; AML, acute lymphocytic leukemia; BAL, bronchial alveolar lavage; M, male; RA, refractory anemia.

a Day 0 is defined as the day patient A’s first positive culture result was obtained.

b Patient also had prolonged *S. epidermidis* bacteremia but died with negative blood culture results.

c Patient was in the bone marrow transplant unit at the onset of infection but was previously in the hematology unit.

d Patient’s initial *P. aeruginosa* bacteremia was caused by a susceptible strain.

Table 1. Characteristics of patients with *Pseudomonas aeruginosa* infections.
was extracted from the stored isolates, the DNA was digested with SpeI, and PFGE was performed as described elsewhere [32]. The pulse time was ramped from 10–90 s and gels were run for 24 h. The gels were stained with ethidium bromide and photographed under ultraviolet light. Isolates were considered to be the same strain if all bands matched, to be subtypes of the same strain if 1–3 bands differed, and to be different strains if >3 bands differed [33].

Case-control study. A “case patient” was defined as any patient who had the epidemic P. aeruginosa strain isolated from a clinical specimen. Each case patient was matched to 4 control patients. “Control patients” were people who: (1) were hospitalized in the affected unit at the same time but whose clinical specimens did not yield the epidemic strain on culture; (2) were hospitalized on the affected unit for at least as long as the time from admission to infection for the matched case patient (i.e., exposure time); (3) had an underlying disease (i.e., primary diagnosis) that was similar to that of the affected patient; and (4) were within 10 years of age of the case patient. If a perfect match could not be identified, the selection criteria were prioritized in the order listed above.

Data on possible risk factors were abstracted from the patients’ medical records and entered into EpiInfo (Centers for Disease Control and Prevention, Atlanta, GA). Statistical analyses were performed by StatXact and LogXact (Cytel Software, Cambridge, MA) [34, 35]. Because the number of affected patients was small, statistical tests based on exact methods were used [36–38]. Frequencies and percentages were calculated to evaluate the distributions of potential risk factors for patients and control patients. Stratified analysis, based on the noncentral hypergeometric distribution (i.e., stratifying on case-control groupings to adjust for matching in the design), was used to determine the probability of detecting differences between variables for the case patients and control patients that were at least as large as those observed, if no association existed. Conditional logistic regression analysis was performed by use of exact methods to estimate the risk, adjusted for potential confounders, of developing infection with the epidemic strain found in the whirlpool bathtub. Because the sample size was small, the analysis was adjusted for 1–2 potential confounders at a time. The median unbiased estimator was calculated with its associated P value rather than the maximum-likelihood estimate of the OR, because all patients used the whirlpool bath, making the OR undefined.

Results

Description of the epidemic. Clinicians in a hematology unit notified staff members in the PHE that 3 hospitalized patients (patients B, E, and F; table 1) were infected during a 30-day period with multiply resistant P. aeruginosa isolates. The isolates were resistant to gentamicin (MIC, 32 μg/mL), tobramycin (MIC, 16 μg/mL), ticarcillin (MIC, 128 μg/mL), and piperacillin (MIC, 256 μg/mL); were intermittently susceptible or resistant to cefotaxime and ceftriaxone (MIC, 32–64 μg/mL); and were susceptible to amikacin, ceftazidime, imipenem, and ciprofloxacin. Staff members in the PHE immediately implemented isolation precautions and began an epidemiologic and microbiologic investigation.

Despite these precautions, 43 days after patient B was found to have bacteremia, cultures of blood samples obtained from a fourth patient (patient G; table 1) yielded P. aeruginosa that had an antibiogram similar to that described above. Infection control personnel searched the databases maintained by PHE and the Clinical Microbiology Laboratory and identified 3 other patients (patients A, C, and D; table 1) who had been infected with multiply resistant P. aeruginosa isolates in the preceding year. PFGE results indicated that all 7 patients (8 separate infections) were infected with the same strain of P. aeruginosa (figure 1) that was different from strains isolated from 18 other patients (6 patients from the affected unit, 5 patients from the medical intensive care unit, and 7 patients who had multiply resistant isolates and who were hospitalized in other units).

The 7 affected patients were all men who had hematological malignancies (table 1). Six patients had bloodstream infections; of these 6, 1 (patient B) had 2 episodes of pneumonia and bloodstream infection caused by the same strain. One patient had a urinary tract infection and an infection of his Hickman catheter exit site. The primary clinical signs of infection were high fever (mean temperature, 39.2°C; range, 38.3–39.7°C), tachycardia (mean heart rate, 123 beats/min; range, 93–140 beats/min), and tachypnea (mean respiratory rate, 31 breaths/min; range, 16–60 breaths/min). The mean blood pressure at the onset of infection was 110/57 mm Hg (range, 69/30 to 140/70). Three of the patients had chills.

Culture surveys. None of the patient-care solutions or medications were contaminated with P. aeruginosa. Surveillance culture results of samples obtained from the perirectal areas of 4 patients in the affected unit were negative, as was the result for a sample obtained from a nursing student’s hands after she cared for 1 of the affected patients. Results of cultures of samples of the quaternary ammonium compound used by the unit staff to clean the whirlpool bathtub after each use were also negative. Numerous cultures of samples from the environment (i.e., sinks and drains) yielded P. aeruginosa, but only the drain and a ledge in the whirlpool bathtub yielded organisms that had the same antibiogram as did the outbreak strain. These multiply resistant isolates were the A2 subtype of the epidemic strain (figure 1).

PHE staff members evaluated the design of the drain and found that it closed ~2.54 cm below the surface of the tub (figure 2). Thus, an area of the drain that was contiguous with the bathtub was filled with water when the tub was used. Cultures of water samples obtained directly from the faucet did not yield P. aeruginosa, but water aspirated gently from the accessible area of the drain, and water added to the tub yielded a susceptible strain of P. aeruginosa that was identified in previous cultures of the drain.

PHE staff members obtained samples for culture from the drains of all whirlpool bathtubs in nursing units. These tubs were made by the same manufacturer and had the same basic
design as the tub implicated in the outbreak. Cultures from 5 of 8 drains were positive for *P. aeruginosa*. Three drains were colonized with 4 different *P. aeruginosa* strains each, and 2 drains were colonized with 3 different strains each. These strains were susceptible to most antimicrobial agents tested, and they were determined by use of PFGE to be different than the epidemic strain. Laboratory staff members used PFGE to compare the *P. aeruginosa* isolates from those drains with isolates from 41 patients who acquired nosocomial infections while hospitalized in those units during the 6 months after cultures were obtained from the drains. The PFGE patterns of isolates from 1 patient’s wound and from the tub in the patient’s unit (i.e., the vascular surgery unit) were identical. PHE staff did not assess whether any of the 41 patients used the whirlpool bathtubs.

**Case-control study.** All of the case patients and control patients were matched for the admission period, exposure time, and primary diagnosis. Eleven control patients were either >10 years older or younger than their matched case patients. The overall success of the matching was 90%.

All 7 case patients were men, compared with only 12 (43%) of the control patients (*P*=.007) (table 2). Case patients and control patients were matched for age and underlying disease. However, case patients were more likely than control patients to be <60 years old, and 86% of case patients had acute myelogenous leukemia, compared with 61% of control patients. Case patients were more likely to have a WBC count <500 cells/mm$^3$ and a serum albumin level <25 g/L (table 2). Disease category (i.e., newly diagnosed vs. relapsed) was not associated with infection. Four patients (57%) died; their deaths were re-
The association between use of the whirlpool bath and infection remained strong when adjusted for the patients’ age and sex (OR, 7.47; P = .042) and for the patients’ WBC count (OR, 6.70, P = .063). Some of the patients used the tub as treatment for perianal fissures or ulcers associated with severe diarrhea. All of the case patients and 79% of the control patients had diarrhea (table 3). Case patients (6 [86%]) were more likely than control patients (10 [37%]; P = .023) to have had samples obtained for stool culture. However, a high proportion both of case patients (5 [71%]) and control patients (18 [67%]; P = .62) had test results negative for *Clostridium difficile*. Three case patients (43%) and 3 control patients (11%) had perianal or rectal fissures or ulcers (P = .089). The association of whirlpool use with infection remained strong when adjusted for the presence of fissures or ulcers (OR, 8.8; P = .025).

**Intervention.** The whirlpool bathtub was withdrawn from service, removed from the unit, and replaced with a completely new system. Before the outbreak, staff in the affected unit cleaned the whirlpool bathtub, as recommended by the manufacturer of the tub, with a quaternary ammonium compound sold by the manufacturer for this purpose. Because the routine cleaning protocol did not prevent the outbreak, PHE staff members worked with personnel from the Housekeeping Department to develop a weekly cleaning protocol for the drains of the remaining whirlpool bathtubs.

In addition, PHE staff members recommended the following interventions: Febrile patients with neutropenia who were previously hospitalized in the unit or who were in the unit when they developed fever should be treated empirically with cefazidime and amikacin. Once culture and susceptibility results were available, the antimicrobial agents should be changed appropriately. Patients who were not hospitalized in the affected unit or who were in the unit when they were available, the antimicrobial agents should be changed appropriately. Patients who were not hospitalized in the affected unit during 1994 and who were admitted with neutropenic fever must be placed in contact precautions.

### Table 2. Demographic and clinical characteristics of case patients and control patients.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Case patients (n = 7)</th>
<th>Control patients (n = 28)</th>
<th>1-Sided P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>7 (100)</td>
<td>12 (43)</td>
<td>.007</td>
</tr>
<tr>
<td>Age, y</td>
<td>48.7 ± 3.3 years</td>
<td>51.3 ± 4.4 years</td>
<td>ND</td>
</tr>
<tr>
<td>Disease</td>
<td>Acute myelogenous leukemia</td>
<td>6 (86)</td>
<td>ND</td>
</tr>
<tr>
<td>Other malignant disease</td>
<td>5 (71)</td>
<td>14 (11)</td>
<td>.011</td>
</tr>
<tr>
<td>Newly diagnosed disease</td>
<td>7 (100)</td>
<td>22 (79)</td>
<td>.205</td>
</tr>
<tr>
<td>Mucositis</td>
<td>2 of 6 (33)</td>
<td>12 (43)</td>
<td>.544</td>
</tr>
<tr>
<td>WBC count, cells/mm³</td>
<td>7.1 (14)</td>
<td>3685 (28)</td>
<td>ND</td>
</tr>
<tr>
<td>Serum albumin, g/L</td>
<td>26.9 ± 10 g/L</td>
<td>30.5 ± 14 g/L</td>
<td>.044</td>
</tr>
<tr>
<td>Mean</td>
<td>4 (57)</td>
<td>0 (0)</td>
<td>.010</td>
</tr>
</tbody>
</table>

**NOTE.** Data are no. (%) unless otherwise indicated. ND, not done.

### Table 3. Drugs and other treatments to which case patients and control patients were exposed.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Case patients (n = 7)</th>
<th>Control patients (n = 28)</th>
<th>1-Sided P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Piperacillin</td>
<td>6 (86)</td>
<td>16 (57)</td>
<td>.164</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>7 (100)</td>
<td>20 (71)</td>
<td>.148</td>
</tr>
<tr>
<td>H₂-blockers</td>
<td>5 (71)</td>
<td>17 (61)</td>
<td>.472</td>
</tr>
<tr>
<td>Diphenoxylate hydrochloride with atropine sulfate</td>
<td>2 (28.6)</td>
<td>9 (32.1)</td>
<td>.616</td>
</tr>
<tr>
<td>Systemic steroids</td>
<td>5 (71)</td>
<td>13 (46)</td>
<td>.226</td>
</tr>
<tr>
<td>Cytarabine</td>
<td>6 (86)</td>
<td>16 (57)</td>
<td>.154</td>
</tr>
<tr>
<td>Packed red blood cells</td>
<td>7 (100)</td>
<td>26 (92.9)</td>
<td>.640</td>
</tr>
<tr>
<td>Central venous nutrition</td>
<td>4 (36.4)</td>
<td>7 (63.6)</td>
<td>.088</td>
</tr>
<tr>
<td>Hickman catheter</td>
<td>6 (85.7)</td>
<td>23 (85.2)</td>
<td>.744</td>
</tr>
<tr>
<td>Foley catheter</td>
<td>2 (28.6)</td>
<td>2 (7.14)</td>
<td>.181</td>
</tr>
<tr>
<td>Whirlpool bath tub</td>
<td>7 (100)</td>
<td>9 (32)</td>
<td>.003</td>
</tr>
</tbody>
</table>

**NOTE.** Data are no. (%) unless otherwise indicated.
tions, and patients who were previously colonized or infected with the outbreak strain and who were readmitted must be placed in contact precautions until samples of the previous sites of colonization or infection were obtained and culture results tested negative.

One year after the whirlpool tub was removed and replaced, PHE staff members identified an additional patient who had a Hickman site infection caused by a multiply resistant \textit{P. aeruginosa} isolate. The PFGE pattern of this isolate indicated that it was a subtype (A3) of the outbreak strain. This patient was hospitalized twice in the affected unit just before the whirlpool tub was removed, but no information was found in his medical record to document that he used this device during either admission. In addition, this patient’s hospital admissions did not coincide with the admission dates of the 7 patients previously infected with the epidemic strain. Furthermore, infection control surveillance has not identified clusters of \textit{P. aeruginosa} infections in units with whirlpool bathtubs since the outbreak was terminated (i.e., >5 years).

**Discussion**

Contaminated whirlpool spas in the community have caused numerous outbreaks of folliculitis and a few sporadic systemic \textit{P. aeruginosa} infections [23–25]. However, we could not find any published reports of serious infections caused by \textit{P. aeruginosa} from contaminated whirlpool bathtubs. Hollyoak et al. [39] reported that a resident of a long-term care facility who used a whirlpool bathtub, which was colonized with \textit{P. aeruginosa}, acquired a wound infection caused by this organism. These authors did not determine whether the strain causing the infection also colonized the tub. A subsequent survey demonstrated that 17 tubs in 16 nursing homes were colonized with \textit{P. aeruginosa} [40]. One year later, the investigators obtained 100-mL samples of water from 6 of these tubs, all of which were still contaminated with \textit{P. aeruginosa} [27].

The outbreak we report had several unusual features. First, cases occurred over 14 months, which seems peculiar given the severity of the infections. However, the first 3 infections occurred ~5 months apart and appeared to be sporadic. Thus, the staff did not suspect nosocomial transmission of a common strain until a cluster of 3 cases occurred during a 30-day period. Second, none of the patients acquired folliculitis, which is the most common infection associated with whirlpool spas. Some investigators have postulated that water must be contaminated with large amounts of \textit{P. aeruginosa} before people who use a contaminated spa will acquire folliculitis [17]. To reach such high numbers, the organism must grow in the water over time. However, whirlpool bathtubs, unlike whirlpool spas, are refilled and drained with every use. Therefore, the conditions necessary to achieve a very high bacterial load would not be present in a whirlpool bathtub.

Third, the reservoir for the epidemic strain was unique. Other investigators have identified the gastrointestinal tract as an important host reservoir for \textit{P. aeruginosa} [1, 3, 41], but none have proposed whirlpool bathtubs as a source of the organism. We did not determine how the drain became contaminated, but we suspect that a patient whose gastrointestinal tract was colonized with the epidemic strain may have been the original source of the organism.

We believe that the design of the drain in the whirlpool tub was an important factor in the persistence and spread of the epidemic \textit{P. aeruginosa} strain. The drain plate closed ~2.54 cm below the surface of the bathtub and a strainer (i.e., the metal grill that covers the drain), which appeared to be permanent, covered this area (figure 2). The area of the drain that was contiguous with the tub stayed moist, providing a good environment in which \textit{P. aeruginosa} could form macrocolonies under a protective slime layer. When water was added to the tub, \textit{P. aeruginosa} colonies in the drain could have been dislodged and thus contaminated the water in which the patient bathed. The staff members of the affected unit cleaned the tub after each use with a quaternary ammonium compound produced by the tub’s manufacturer for this purpose. However, because the strainer was in place, staff could not scrub the area of the drain that was contiguous with the tub. Therefore, the epidemic \textit{P. aeruginosa} strain survived and multiplied undisturbed in the drain.

The patient’s perirectal and perineal areas could have become contaminated with \textit{P. aeruginosa} when immersed in the contaminated water, even if only a few organisms were present. In addition, the whirlpool jets sprayed water vigorously, and areas of the patient’s body that normally would not have been immersed (e.g., exit sites for implanted catheters) could have become contaminated. Subsequently, the organisms could have multiplied when the patient took antimicrobial agents to which the epidemic strain was resistant. If the patient developed neutropenia, he or she could have acquired a serious systemic infection.

Some of the patients who used the whirlpool bathtub during the 14-month period during which the epidemic strain was transmitted did not become infected with the epidemic strain. The patients who were infected with the epidemic strain after they used the whirlpool bathtub were younger, but sicker (i.e., lower WBCs and albumin levels), than the control patients who were also exposed to the tub and thus may have been at higher risk of infection after being exposed to the organism. In addition, men were at a significantly higher risk of becoming infected with the epidemic strain than were women; however, we cannot explain this observation.

To our knowledge, this is the first reported outbreak in which a whirlpool bathtub was the reservoir for an outbreak of severe \textit{P. aeruginosa} infections. The whirlpool bathtubs used in our institution are used in many other institutions, and even regular bathtubs often have a drain that closes below the level of the tub. People who are immunosuppressed, who have implanted...
catheters, or who have open wounds could be exposed to \textit{P. aeruginosa} while bathing in such tubs. Therefore, drains that close beneath the level of the bathtub may be an unrecognized source of both hospital- and community-acquired \textit{P. aeruginosa} infections. This potential source could be eliminated simply by using bathtubs with drains that seal from the top.

**Acknowledgments**

We thank the nurses and medical staff of the affected unit, who cooperated fully with this investigation; Cheryl Carter, Marlene Schmid, Brenda Barr, and Jean Pottinger, who helped with the investigation; and Connie K. Quee and Khen Mac, who assisted with the molecular typing.

**References**


