we look back again at the cleanroom analogy, the international standard for cleanrooms details test methods and equipment for airflow volumes, installed filter leakage, air exchange rate (ie, recovery time) and containment, in addition to pressure differentials and airflow visualization.[3] The recent design manual for hospitals published by the American Society of Heating, Refrigerating and Air-Conditioning Engineers points out that "maintaining a negative air pressure between the AII (airborne infectious isolation room) and the corridor may not be enough to provide isolation" and "the truly significant factor in determining the amount of air volume migration from the room to the corridor is the airflow volume differential" and that it is necessary to "maintain a specific differential airflow rate" in an isolation room.[5(p134)] How can one be certain of maintaining a specific differential airflow rate if it is not periodically measured? Airflow volume differential is dependent on envelope tightness and pressure differential. Without a sufficiently tight envelope for the isolation room, pressure differentials cannot be maintained and airflow direction cannot be controlled. The isolation room guideline from Norway[6] recognizes this and explicitly calls for envelope tightness testing as part of isolation room commissioning. Unfortunately, the guideline doesn't say how to do the test nor does it give any indication as to what value is acceptable for an envelope tightness test result for an isolation room.

Because isolation room ventilation system parameters do change over time and do deviate from design values, the natural question arises as to what impact a particular deviation—for example, a pressure differential that is deemed to be too low—has on containment performance. In the international standard for cleanrooms,[4] intervals for performance testing are specified, and documentation requirements are given. If specified commissioning or maintenance test results fall outside of prescribed limits, then the cleanroom is considered to be in a state of noncompliance, and a remedial action plan is implemented to correct the out-of-compliance condition. Requalification is necessary to bring the cleanroom back into compliance.

The time is ripe for a similar standardization of performance testing for isolation rooms. A separate and distinct guideline that deals exclusively with testing and test methods is desirable. At present, considerable resources are dedicated to the design and construction stages of a project, with little thought (or budget) allocated to follow up on testing of the finished product. A consensus international standard detailing what needs to be tested and documented and how often, as well as what to do and when to do it, in the event that test results deviate from design values, will be an important step forward in minimizing the risk of exposure in hospitals and healthcare facilities. An international committee of ventilation and infection control experts needs to be established to get the ball rolling. The first step is to get governments and funding agencies interested in this development. It cannot happen, however, without the interest and support of healthcare professionals working at the forefront of infection protection.

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Colonization of a Water System by *Legionella* Organisms and Nosocomial Legionellosis: A 5-Year Report From a Large Italian Hospital

TO THE EDITOR—*Legionella* infections in the region of Piedmont, Italy, have been reported since 1980.[1,2] In a 1-year period alone (March 1984 to April 1985), 58 cases of pneumonia, 13 of which were ascribed to *Legionella pneumophila* serotype 1, were diagnosed at a major regional hospital on the basis of direct clinical observation and culture of lung specimens obtained at autopsy. Inspection of the hospital's water system, specifically the pipes delivering hot water to the wards where the patients had been hospitalized, revealed extensive contamination with *L. pneumophila* serotype 1. This raised considerable alarm and led to the implementation of corrective measures.

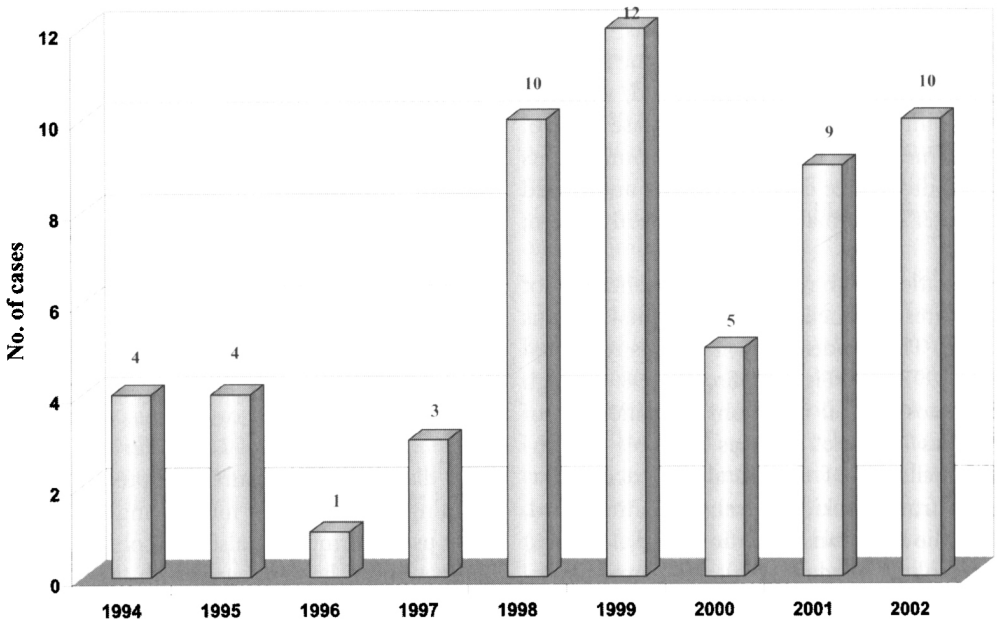

FIGURE. Annual number of cases of nosocomial *Legionella* infection at the study hospital, 1994-2002

Initially, the corrective measures comprised hyperchlorination, then, in 1984-1997, weekly superheating and flushing of the hot water supply (heating to 60°C for 24 hours at distal sites); later, in 1998–2002, other preventive control procedures were adopted (continuous chlorination at 2-3 ppm and use of bacterial filters [Filtranios PV1000; Anios Laboratories]) in addition to hyperchlorination.

Other actions undertaken in parallel included the following: (1) spout aerators were removed and faucets were periodically decontaminated by steam disinfection and descaling; (2) the hospital's cooling towers were disinfected; (3) oxygen bubble humidifiers prefilled with sterile water were installed; (4) ice makers were decontaminated, and their use for food or drinks was forbidden; and (5) air conditioning units and systems periodically cleaned and maintained.

Since 1998, environmental sampling has been performed every 2 weeks (15 and 30 days after hyperchlorination). In selecting sample-collection points, attention was directed at high-risk units (eg, the transplantation, hematology, and oncology units). Water samples (1 L after a 1-minute flow-through) and biofilm specimens (obtained with a swab) were taken from the hot water production plant (recirculation line) and from the ward faucets and shower heads. At the time of sampling, the faucet aerators had been removed. Samples were also collected from the hospital's cooling towers. Samples were analyzed according to a published International Standard Organization protocol.[3]

Cases were identified by applying international guideline definitions that include clinical and radiograph findings and the results of culture, urinary antigen tests, and serologic tests.

TABLE. Comparison of the Number of Cases of Nosocomial *Legionella* Infection and the Disinfection Methods Used at the Study Hospital, 1994-2002

Period	Water-system disinfection method used	No. of patients with legionellosis	No. (%) of distal environmental sites positive for *Legionella*[a]	No. of patients with urine antigen test performed
1994-1997	Weekly superheating (to 60°C at distal sites)	12	NA	0
1998	Monthly hyperchlorination (to 50 ppm)	10	73 (49)	75
1999	Monthly hyperchlorination (to 50 ppm)	12	57 (145)	517
2000	Monthly hyperchlorination (to 50 ppm)	5	41 (134)	502
2001	Monthly hyperchlorination (to 50 ppm), then continuous chlorination (at 2-3 ppm)[b]	9	27 (323)	547
2002	Continuous chlorination (at 2-3 ppm)	10	6 (231)	632

NOTE. NA, not available.

[a] Total number of distal sites sampled.

[b] From September 2001.

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Introduction of the urinary antigen test in the hospital in 1998 simplified the identification of legionellosis, and the study has been continued since that time. The type of infection (ie, hospital-acquired or community-acquired) was classified according to definitions in Centers for Disease Control and Prevention guidelines.[4] The strains isolated from patients and environmental sources were characterized by automated ribotyping (RiboPrinter Microbial Characterization System; DuPont Qualicon).

Despite the installation and regular implementation of costly control procedures, legionellosis in the hospital remains an unsolved problem. The incidence of infections remained relatively low until 1997 (see Figure). From 1998 through 2002, there were 46 nosocomial infections identified, 37 of which were classified as "definite" and 9 as "possible" healthcare-associated legionellosis. Of the 46 identified case patients, 7 (15%) were from the kidney transplantation ward, 10 (22%) were from the liver transplantation ward, 11 (24%) were from the hematology ward, 1 (2%) was from the nephrology ward, and the remaining 17 (37%) were from other wards (mostly internal medicine). Since 2001, a seasonal trend in the number of cases has emerged (June-September), whereas before then cases were more evenly distributed over the course of the year.

Most patients (63%) were male (age range, 30-86 years; mean, 57.3 years), among whom a common risk factor was an immunosuppressive disorder or receipt of immunosuppressant pharmacological treatment (for organ transplantation, leukopenia, tumors, severe anemia, and/or neuropathies). For the "definite" cases, the mean time interval between admission and the first signs of pneumonia was 26 days (range, 10-60 days).

From 1998 through 2002, the fatality rate was 23.9% (11 deaths among 46 cases). In only 12 of the 46 cases observed since 1998 was it possible to isolate *Legionella* strains. In these 12 cases, *L. pneumophila* serotype 1 was isolated. Ribotyping of the 12 isolates identified 3 distinct ribogroups: 20-S6 (6 strains), 20-S2 (5 strains) and 20-S4 (1 strain).

The environmental strains (isolated from samples collected from ward room sinks and showers and from the hot water delivery and return pipes) were identified as belonging to ribogroups 20-S6 and 20-S1. The strain of *L. pneumophila* serotype 1 isolated from one of the cooling towers was determined to belong to ribogroup 93-S1. In only 2 clinical cases (among 12 ribotyping tested) could we find a direct epidemiological relation between the patient and environmental contamination.

The increase in the number of reported cases of hospital-acquired legionellosis since 1998 is thought to be the result of the introduction of the urinary antigen test and to physicians' greater awareness of potential infection, as borne out by the appreciable increase in the number of physician requests for diagnostic procedures (Table).

During the first year of our analysis (1998), all cases occurred in contaminated high-risk units (the kidney transplantation, liver transplantation, hematology, and nephrology units and the ear, nose, and throat unit). The proportion of samples positive for *Legionella* species was 73%. At the time, preventive environmental measures included monthly hyperchlorination of the water supply.

After the adoption of continuous chlorination in 2002, the proportion of environmental samples positive for *Legionella* species decreased to 6%. Yet the number of cases did not decrease as expected, and new cases were also reported in wards considered to be at low risk (eg, general medicine). The constant number of cases suggests that the environmental contamination was very probably not the only factor that accounted for the risk of infection.

An additional consideration is that there was a variation in circulating strains over time, which suggests that non-*pneumophila* species of *Legionella* have acquired resistance to the disinfection measures adopted at the hospital. Similar findings from routine monitoring of other hospital facilities that use chlorine have also been observed (unpublished data). So we find it hard to agree with the rule that "if the percent of positive cultures at the distal sites is equal to or greater than 30% of the total number sampled, then disinfection of the water distribution system is appropriate,"[5] because in other hospitals in the Piedmont region where clinical surveillance has reported no cases, the percentage of positive samples is >30%. The interpretation of the significance of the environmental findings does not appear to be consistent with the patterns of circulation of the disease as reported by other investigators.[4,6]

Hospital administrators should adopt measures aimed at reducing or eliminating environmental contamination in wards where at-risk patients are hospitalized.[7] For such interventions to be effective, however, it is necessary to search for uncontrolled variables, such as neighboring cooling towers, open windows in air-conditioned wards, and occasional dismantling of shower and sink filter units by patients, which could at least partly explain some of the cases. The results of our study strongly suggest that the most effective means of controlling disease is not environmental surveillance followed by disinfection but rather active surveillance of pneumonia.

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Bacteremia Caused by *Stenotrophomonas maltophilia* in a Dialysis Patient With a Long-Term Central Venous Catheter

TO THE EDITOR—Intravascular catheters are essential in complex medical and surgical interventions, such as hemodialysis; bone-marrow and organ transplantation; cancer therapy; and abdominal, cardiothoracic, and trauma surgery.[1] *Stenotrophomonas maltophilia* has recently emerged as an important nosocomial pathogen, with at least 5 reports in the English-language literature documenting infection with this pathogen in hemodialysis patients.[2-6] We describe a hemodialysis patient who developed *S. maltophilia* bacteremia associated with use of a tunneled subclavian catheter.

A 43-year-old man with chronic pyelonephritis and recurrent nephrolithiasis first underwent hemodialysis because of end-stage renal failure in May 1995. According to the patient's history, in the seventh year after starting hemodialysis, multiple vascular accesses failed. The patient refused peritoneal dialysis, which necessitated insertion of a long-term indwelling silicone catheter (Medcomp) into the right subclavian vein on October 12, 2001. From October 19 to November 10, the patient had at least 8 episodes of bacteremia, and he presented with clinical symptoms of high fever, chills, and abdominal pain to a secondary hospital dialysis center in a city other than that where our institution is located (Dicle University Medical Hospital, Diyarbakir, Turkey). The patient had been receiving broad-spectrum antibiotic therapy, which included ceftriaxone, cefazolin, and gentamicin, for 14 days in a secondary-care hospital. However, the patient did not well respond to broad-spectrum antibiotic therapy. Therefore, he was referred to the hemodialysis center at our institution on November 11, 2001. We observed 2 additional episodes of fever and chills and observable inflammation at the catheter exit site. However, we did not find another complications of catheter-related bacteremia, such as endocarditis or abscess. Teicoplanin and cefazolin therapy was initiated after blood samples were obtained for paired blood cultures on November 18, 2001.

The patient's vital signs were as follows: blood pressure, 110/70 mm Hg; heart rate, 161 beats/minute; respiratory rate, 26 breaths/minute; and temperature (oral), 38.8°C. There was crepitation in the basal pulmonary area. A complete blood count revealed a white blood cell count of 15,600 cells/mm^3 (70% polymorphonuclear cells), a hematocrit of 38.3%, an erythrocyte sedimentation rate of 70 mm/h, and a thrombocyte count of 274,000 cells/mm^3. Chest radiographs revealed minimal bibasilar effusion. Electrocardiography revealed atrial tachycardia, T-wave abnormality, and left anterior fascicular block. Echocardiography showed a first-degree mitral valve failure and left ventricular posterior wall hypertrophy. Urinalysis was not performed because the patient was anuric. When blood cultures were indicated, 3-7 mL of venous blood was drawn from the catheter and from 2 peripheral veins after skin preparation with povidone-iodine. All blood culture bottles were incubated for up to 8 days in an automated blood culture system (Bactec 9240; Becton-Dickinson). Incubation of blood cultures for 48 hours yielded bacterial growth of *S. maltophilia.* The isolate's susceptibility to antibiotics was examined with an automated system (AutoSceptor; Becton Dickinson), which revealed resistance or intermediate susceptibility to all antibiotics except ciprofloxacin, ceftazidime, ticarcillin-clavulanate, cefoperazone, and cotrimoxazole.

Therapy with cotrimoxazole and ciprofloxacin was initiated. Despite continued therapy, the patient had 3 further episodes of fever and chills. On December 20, 2001, the long-term indwelling silicone catheter was removed according to a strict protocol under aseptic conditions. After withdrawal, the distal 5 cm part of the catheter was cut off with sterile scissors and sent in aseptic conditions to the infectious diseases laboratory of our institution, where it was cultured by means of the semiquantitative method described by Maki et al.[7] The culture again yielded *S. maltophilia.* In this instance, the diagnosis of catheter-related bacteremia was confirmed by multiple cultures positive for the pathogen (ie, at least 3 consecutive positive blood cultures) and the detection of the same pathogen in catheter culture and blood culture. Since then, the patient has had no further episodes of bacteremia.

Catheter-related bacteremia frequently occurs in outpatients undergoing hemodialysis. The incidence of bacteremia in outpatients undergoing hemodialysis with dual-lumen, tunneled, cuffed catheters has been reported to be 3.9 episodes per 1,000 catheter-days, although catheter-related bacteremia is thought to occur less frequently in patients with tunneled, cuffed catheters.[8] Organisms causing catheter-related bacteremia generally enter the bloodstream from the skin