with the isolate imported by this neonate from the other institution. These two observations together highly minimize the role of a unique environmental source and lead us to consider our study as the first evidence of patient-to-patient nosocomial cross-transmission by *S maltophilia*.

We share Dr. VanCouwenberghe's opinion about the importance of defining the nosocomial transmission dynamics of this microorganism. The precise definition of either environmental sources or cross-transmission as the causes of nosocomial infections is essential in order to adopt specific strategies in the management of patients and thus efficiently contain outbreaks.

With regard to the proposal of using antibiotic susceptibility patterns to type this microorganism, in our case this approach was not possible, as all isolates were multiresistant and susceptible only to trimethoprim-sulfamethoxazole. Furthermore, in our institution, most of the *S maltophilia* isolates (70%) obtained in 1999 were susceptible to only trimethoprim-sulfamethoxazole (50%) or to aminoglycosides and quinolones (20%). This would limit seriously, in our context, the usefulness of applying susceptibility patterns for typing purposes.

We consider that, today, molecular typing is a requirement for efficient management of nosocomial infections, and it should no longer be considered as a luxury tool restricted to refined epidemiological analysis. To fill the conceptual gap between these two realities, efforts should be made to increase personnel expertise in molecular techniques in the microbiology wards and to supply them with adequate resources. The reduction in expenses derived from more efficient management of nosocomial infections would easily finance "typing units" to provide immediate assistance to the bedside clinician.

Darío García de Viedma, PhD
Mercedes Marín, PhD
Emilia Cercenado, PharmD
Roberto Alonso, PhD
Marta Rodríguez Créixems, MD, PhD
Emilio Bouza, MD, PhD
Hospital General Universitario Gregorio Marañón
Madrid, Spain

Hospital Characteristics Associated With Colonization of Water Systems by *Legionella* and Risk of Nosocomial Legionnaires' Disease: A Cohort Study of 15 Hospitals

To the Editor:

We wish to comment on the important article authored by Kool et al from the Centers for Disease Control and Prevention (CDC).[1] Kool et al found that the number of cases of hospital-acquired legionnaires' disease in San Antonio hospitals correlated better with the proportion of water sites positive for *Legionella* than with concentration of *Legionella* in water samples. That is, quantitation of *Legionella* at individual distal water sites did not correlate well with the presence of hospital-acquired legionnaires' disease. We had already documented this phenomenon,[2] as pointed out by Kool et al. This information is reflected in the Allegheny County Health Department (ACHD) guidelines mandating routine environmental cultures for *Legionella* in Pittsburgh hospitals; the proportion of sites positive, not quantitation at distal sites, is the parameter used for decision making.[3] In contrast, the CDC guidelines recommend obtaining environmental cultures after only one or two cases of hospital-acquired legionnaires' disease are discovered.[4]

Kool et al noted that it may seem counterintuitive that *Legionella* bacterial concentrations would not be an important determination of risk of transmission. The major reason why bacterial concentrations are not predictive is that *Legionella* is not concentrated in water but is harbored in the biofilm consisting of sediment and detritus at each distal water outlet.[5,6] Thus, sampling itself will affect the biofilm, and repeated samplings over time may well dislodge the biofilm present in any distal water fixture, thus decreasing the quantitative count. We have also shown that the concentration of *Legionella* in a water sample can be significantly lower (even negative) than that recovered by a swab (biofilm) sample.[5]

The CDC errs in claiming that the ACHD guidelines recommend that disinfection measures be implemented when the percentage of positive sites exceeds 30%. Actually, the ACHD guidelines recommend only that disinfection be considered at that level, but the guidelines do mandate that *Legionella* testing be made available in hospitals with a contaminated water supply. The ACHD guidelines are much less draconian than the CDC guidelines.[4] For example, the CDC guidelines recommend that the percentage of positive sites and quantitative counts be reduced to zero, which is extremely difficult to attain, especially with hyperchlorination. Since there is some debate regarding the level of site positivity that should trigger disinfection options, it would be meaningful to know whether Kool et al found a specific percentage of positive sites above which disease was more likely to be detected.

It is well recognized that the diagnosis of legionnaires' disease cannot be made on clinical grounds alone and that specialized laboratory tests are necessary, as is well shown in the article by Kool et al. The authors point out that numerous cases may have been missed, because *Legionella* testing was not routinely applied to all patients with hospital-acquired pneumonia. The authors do not mention the mortality for the legionnaires' disease cases in San Antonio.

We cannot help but point out that, had the ACHD guidelines been implemented in the 12 San Antonio hospitals, all of these hospitals would have adopted in-house *Legionella* laboratory testing many years earlier. Cases would have been uncovered earlier if urinary antigen testing and culture on selective media were applied to patients with hospital-acquired pneumonia. Initiation of earlier treatment would have minimized the mortality. Disinfection measures could be considered, in order to prevent more cases from occurring. The proactive ACHD approach versus the reactive CDC approach actually can be assessed prospectively in San Antonio.

Although *Legionella* was found in 11 hospitals, only 5 hospitals experienced hospital-acquired legionnaires' disease. We predict that the remaining 6 hospitals may well have undiagnosed hospital-acquired legionnaires' disease, if the proportion of site positivity is high. We recommend that the microbiology laboratories of these 6 hospitals perform urinary antigen

and *Legionella* culture in-house (rather than sending them to reference laboratories). Infection control practitioners merely have to ensure that all cases of hospital-acquired pneumonia undergo testing with urinary antigen and culture on selective media. If these 6 hospitals fail to diagnose any cases in 2 to 3 years, this would support the CDC reactive approach of environmental culturing only when cases are discovered.

REFERENCES

1. Kool JL, Bergmire-Sweat D, Butler JC, Brown EW, Peabody DJ, Massi DS, et al. Hospital characteristics associated with colonization of water systems by *Legionella* and risk of nosocomial legionnaires' disease: a cohort study of 15 hospitals. *Infect Control Hosp Epidemiol* 1999;20:798-805.
2. Best M, Yu VL, Stout J, Goetz A, Muder RR, Taylor F. *Legionellaceae* in the hospital water supply. Epidemiological link with disease and evaluation of a method of control of nosocomial legionnaires' disease and Pittsburgh pneumonia. *Lancet* 1983;2:307-310.
3. Allegheny County Health Department. *Approaches to Prevention and Control of* Legionella *Infection in Allegheny County Health Care Facilities.* 2nd ed. Pittsburgh, PA: Allegheny County Health Department; 1997:1-15.
4. Centers for Disease Control and Prevention. Guidelines for prevention of nosocomial pneumonia. *MMWR* 1997;46(RR-1):1-79.
5. Ta AC, Stout JE, Yu VL, Wagener MM. Comparison of culture methods for monitoring *Legionella* species in hospital potable water systems and recommendations for standardization of such methods. *J Clin Microbiol* 1995;33:2118-2123.
6. Wright JB, Ruseska I, Athar MA, Corbett S, Costerton JW. *Legionella pneumophila* grows adherent to surfaces in vitro and in situ. *Infect Control Hosp Epidemiol* 1989;10:408-415.

Victor L. Yu, MD
Janet E. Stout, PhD
Veterans' Affairs Medical Center
Pittsburgh, Pennsylvania

The authors declined to reply.

Respiratory Syncytial Virus in a General Hospital and the Need for Extensive Measures

To the Editor:

Respiratory syncytial virus (RSV) is a major cause of morbidity and mortality in children worldwide. The incubation period of RSV ranges from 2 to 8 days. It is highly contagious. The period of viral shedding is usually 3 to 8 days, but may be longer, especially in young infants in whom shedding may continue for as long as 3 to 4 weeks.

RSV in nasal secretions from infected infants can survive for up to 6 hours on surfaces and objects, and at least for half an hour on contaminated skin (hands), gowns, or paper tissues. Transmission of RSV occurs mainly by direct or close contact with persons shedding the virus. This can lead to self-contamination (hands, eyes, nose) of healthcare workers (HCWs) and cross-contamination of objects: thus, the hands of HCWs may be contaminated during direct patient care or by contact with contaminated surfaces or objects. In this context, HCWs play a major role in the nosocomial transmission of RSV infection, mostly due to hand-to-hand contact.[1] In contrast to large-droplet spread, spread by small-particle aerosol transmission is not a major route of transmission of RSV.

Reported RSV infection control interventions include use of single patient rooms or cohorting; isolation techniques (gowns, gloves, masks, eye-nose goggles); cohorting of nurses; admission screening; and visitor restrictions. The Dutch Working Party on Infection Control recommends the following infection control measures: admission to a single room (or cohorting); wearing of gowns, gloves, and masks; and, of course, hand washing with soap and disinfection of the hands with an alcoholic solution. Visitors (eg, parents) also must wear masks and wash or disinfect their hands. Goggles are not recommended.[2,3] However, in our hospital, a 950-bed general hospital, we only use patient placement in single rooms, gowns, and careful hand washing or disinfection.

To study the effectiveness of these measures, we performed a simple retrospective analysis of a 3-year period. The data file of hospital admissions, containing 3,302 children admitted from January 1995 through December 1997, was combined with the microbiology data file containing 227 children from whom a direct immunofluorescent (DIF) test or culture for RSV was performed in the same period.

A positive DIF test or culture for RSV was found for 116 children. RSV was detected in 95 of the children in a period from 4 weeks before admission at our pediatric ward until 10 days after discharge. A further selection was made of 12 children with RSV detection in a period ranging from 2 days after admission until 10 days after discharge, because these children were suspected to have had a nosocomial infection. Five of these 12 children appeared to have been admitted with respiratory symptoms fitting with RSV diagnosis and thus were unlikely to have nosocomial RSV infection. Of the remaining 7 children with a possible nosocomial RSV infection, 1 had respiratory distress directly after birth, and 1 was transferred from another hospital. Based on the incubation period and clinical symptoms, nosocomial infection in these children seems to be unlikely. One child was admitted with respiratory symptoms. This child had a negative DIF at admission, which turned positive by discharge (12 days after admission) but without new clinical symptoms. Three children developed an RSV infection between 6 and 8 days after discharge. One child was admitted with respiratory symptoms and 3 days later developed progressive respiratory distress, with a positive DIF. Thus, in 5 (4.75%) of 95 children, nosocomial RSV infection could not be ruled out. This is even lower than the rates reported by others using extensive infection control interventions.[4,5]

We conclude that placing patients with RSV in single rooms, using gowns, and washing or disinfecting the hands is sufficient to keep the rate of nosocomial RSV infections in patients in a general hospital as low as in hospitals with extensive measures.

REFERENCES

1. Thomas DJ. Respiratory syncytial virus in a special care nursery. In: Soule BM, Larson EL, eds. *Infections and Nursing Practice, Prevention and Control.* 1995:375-382.
2. Dutch Working Party on Infection Control. Prevention of infections on a neonatal ward. Guideline 30A. Leiden, The Netherlands: Dutch Working Party on Infection Control; 1998.
3. Dutch Working Party on Infection Control. Guidelines for isolation. Guideline 4A. Leiden, The Netherlands: Dutch Working Party on Infection Control; 1991.
4. Leclair JM, Freeman J, Sullivan BF, Crowley CM, Goldman DA. Prevention of nosocomial respiratory syncytial virus infections through compliance with glove and gown isolation precautions. *N Engl J Med* 1987;317:329-334.
5. Karanfil LV, Conlon M, Lykens K, Masters CF, Forman M, Griffith ME, et al. Reducing the rate of nosocomially transmitted respiratory syncytial virus. *Am J Infect Control* 1999;27:91-96.