Airborne Nosocomial Infection: A Contemporary Perspective

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ABSTRACT

The history of airborne nosocomial infections is reviewed, and current beliefs about such infections are placed into their historical context. Possible sources, both animate and inanimate, of airborne nosocomial infections in the hospital environment are identified. Viruses, bacteria, and fungi that have been important causes of airborne nosocomial infections in the past are discussed, and examples of key studies that have confirmed an airborne route of transmission are presented. Where relevant, measures that have been used to control airborne transmission of nosocomial pathogens are discussed. Although outbreaks of airborne nosocomial infection have been uncommon, airborne transmission appears to account for about 10% of all endemic nosocomial infections.

This review has four objectives: first, to place current beliefs about airborne nosocomial infections into their historical context; second, to review the possible sources of airborne infection in the healthcare setting; third, to review the microorganisms that have been transmitted by the airborne route in hospitals; and finally, to estimate the relative importance of airborne transmission of infection in the overall problem of nosocomial infection.

HISTORICAL CONTEXT

Any discussion of routes of transmission of infectious diseases must acknowledge the cyclic nature of beliefs about this topic in history. In 400 B.C., Hippocrates believed that air, water, and places influenced the health of populations. In the 2nd century A.D., Galen noted that when many sicken and die at once, one should consider the air that we breathe. His observations were underscored by the occurrence of dreaded epidemics such as the Black Death in Europe during the 14th century. Two hundred years later, Fracastorius noted that infection could be transmitted by direct contact, by indirect contact, or from a distance, that is, through the air.

For the next several hundred years, virtually all infectious diseases were thought to be transmitted through the air, and so the “miasmic” theory of infection gained credence, leading to names like malaria. After the microbial nature of infectious diseases was recognized in the mid-19th century, the role of contact in infection transmission was clearly identified and gained acceptance rapidly. By 1910, Charles Chapin could write in his treatise On the Sources and Modes of Infection: “Without denying the possibility of [airborne] infection, it may be fairly affirmed that there is no evidence that it is an appreciable factor in the maintenance of most of our common contagious diseases. We are warranted, then, in discarding it as a working hypothesis and devoting our chief attention to the prevention of contact infection.” He did waver a bit in the case of tuberculosis (TB), however, and considered that disease more likely than any other to be airborne.

Chapin’s views persisted for the next 35 years. In
1935, however, William Firth Wells, an engineer at Harvard, began to challenge this dogma and argued that certain diseases, such as measles, were spread through the air by droplet nuclei. Ultraviolet (UV) lights were introduced into a few schools to test this hypothesis and, initially at least, met with success. As recently as 1946, however, a committee of the American Public Health Association, in its final report, wrote: "Conclusive evidence is not available at present that the airborne mode of transmission of infection is predominant for any particular disease." Among the committee members was Dr. Alexander Langmuir, who later was converted.

The next 25 years, of course, sharply changed beliefs about airborne transmission of infectious disease and put epidemiological theory on a more scientific basis. Langmuir, in a thoughtful review published in 1980, identified four areas of study that led to a more substantive understanding of the role of airborne infection. These were, first, an understanding of the creation and behavior of aerosols of microorganisms; second, an understanding of the physiology and function of the respiratory tract, particularly the respiratory host defense mechanisms; third, the study of experimental airborne infections in animals and humans; and fourth, increased understanding of the epidemiology of both naturally occurring and accidentally acquired infection.

Knowledge and understanding of the role of airborne infection in the healthcare setting has paralleled understanding of the role of airborne infection more generally. In fact, it probably is fair to state that studies of nosocomial infection transmission often have been pivotal in understanding the broad role of airborne infection. The classic studies of Richard Riley in the Baltimore Veterans Administration Hospital, for example, were of landmark significance, finally convincing even the skeptics that TB was, for the most part, an airborne infection. These studies were elegant in the simplicity of their design (Figure 1). Room air was exhausted from pilot ward rooms, in which were patients with pulmonary TB; exhaust air then was circulated to test chambers in which were housed guinea pigs, highly susceptible to infection by any tubercle bacilli that might be in their inspired air. The effect of UV light could be assessed simultaneously in a parallel series of exposure chambers.

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![Figure 1. Schematic drawing of ward, ducts, and exposure chambers. Reprinted with permission.](image-url)
TABLE 1
POSSIBLE SOURCES OF AIRBORNE NOSOCOMIAL INFECTION*

Inside the hospital:
- Infected or colonized patients, staff, and visitors
- Infective dusts and aerosols
- Ventilation or air-conditioning systems

Outside the hospital:
- Soil
- Water (eg, cooling towers)
- Decaying organic materials
- Dust from construction or renovation

* Adapted from Schaal.7

SOURCES OF AIRBORNE INFECTION IN HOSPITALS

Possible sources of airborne nosocomial infection are summarized in Table 1. Within the hospital, the most important and most obvious sources are human beings: patients, personnel, or visitors. To be an efficient source of airborne infection, a person needs to be a disseminator, or spreader, of some pathogenic organism. Such a disseminator may be a person with symptomatic disease, as has been described in nosocomial outbreaks of TB and smallpox; alternatively, a disseminator may be wholly asymptomatic, a kind of microbial “Pigpen,” to recall the Peanuts character. Such asymptomatic carriers have been well described as sources of airborne nosocomial staphylococcal infections. Sites from which airborne dissemination has occurred include the nares, pharynx, anus, skin, and skin scales. Other possible sources of airborne infection within the hospital include dusts or aerosols from the floor or furniture, from potted plants or flower vases, sinks, showers, nebulizers, humidifiers, or aspirating devices. Contaminated ventilation or air-conditioning systems have been implicated in some nosocomial airborne outbreaks, via infective aerosols, dust, or even colonized filters.7

Outside the hospital, there are a number of possible inanimate sources as well. These must include soils, acting as a natural habitat of certain pathogens, or soil that has been contaminated by feces. Water supplies may be contaminated by potential pathogens and the contaminants then may be amplified in certain settings such as cooling towers or in holding areas within the hospital. Legionnaire’s disease has been spread both from contaminated cooling tower water and by the generation of infective aerosols from water supplies within the hospital. Infective dusts may be generated from building construction or renovation activities within the hospital or located in immediately adjacent areas.

TABLE 2
VIRUSES IMPLICATED IN AIRBORNE NOSOCOMIAL INFECTIONS

- Rhinoviruses
- Influenza and parainfluenza viruses
- Respiratory syncytial virus
- Adenoviruses
- Varicella zoster virus
- Measles
- Rubella
- Smallpox
- Certain enteroviruses

In the last 15 years, we have come to appreciate that airborne nosocomial pathogens derived from the inanimate environment generally have been less virulent than those derived from animate sources and tend to occur primarily in areas in which very highly susceptible hosts are located, eg, oncology units, organ transplantation units, and the like. Furthermore, the number of pathogens that can spread via the airborne route from dusts, soils, or construction areas appears to be limited to those bacteria and fungi that can survive in a dry environment for extended periods of time.

ETIOLOGIC AGENTS IN AIRBORNE NOSOCOMIAL INFECTION

A substantial number of viruses, bacteria, and fungi are capable of spreading via the airborne route in hospitals. Possibility of airborne transmission and documentation of airborne transmission are quite different, however, and the problem is complicated by the fact that many if not most of the pathogens to be discussed are capable of spreading by more than one route. Many common respiratory viral infections, for example, may be spread by large droplets, actually a form of indirect contact, and by airborne droplet nuclei. This discussion will focus on pathogens for which there is good evidence of at least some airborne transmission.

Viruses believed to be spread at least in part by the airborne route in hospitals are shown in Table 2. The common respiratory viruses, including rhinoviruses, influenza and parainfluenza viruses, respiratory syncytial virus, and adenoviruses are included in this category. The evidence in support of airborne rather than droplet spread of many of these viruses often is incomplete; however, there is good epidemiological evidence for airborne transmission of respiratory syncytial virus and adenoviruses in pediatric wards.8,10 The strongest epidemiological evidence of airborne transmission of influenza comes not from the hospital setting, but rather from a well-documented outbreak that occurred on a commercial aircraft.11
There also is some epidemiological evidence in support of such transmission in hospital wards. Among the common viral exanthems, the evidence in support of airborne transmission is quite strong with respect to *Varicella zoster* virus and measles. One of the best recent examples of the airborne spread of *Varicella* in a hospital was published in 1980 by investigators at Children's Hospital in Boston. Figure 2 shows the strikingly high attack rates for nosocomial airborne *Varicella* among susceptible children in other rooms on the ward. Rubella also may be spread by the airborne route, but the evidence is not as compelling.

Since the eradication of smallpox, any consideration of nosocomial airborne transmission of this disease is only of academic interest. That this has occurred, however, is established beyond any doubt. In 1970, a major outbreak of smallpox occurred in a small hospital in Meschede, West Germany; a single index patient infected 17 other persons, including patients and personnel. Two additional cases occurred in a second generation, a total of 19 cases, with three deaths. As illustrated in Figure 3, the investigators showed with a smoke generator that aerosols from the index patient's room spread not only out of the window, but also into the corridor, up a stairwell, and into patient rooms on floors above. Ironically, the very last case of smallpox in the world was due to airborne transmission, a tragic laboratory accident that resulted not only in the death of the victim, a 40-year-old medical photographer in the medical school at the University of Birmingham, England, but also in the suicide of the director of the laboratory.

There are theoretical concerns about possible spread of viral hemorrhagic fevers such as Lassa fever or Ebola virus disease transmission via the airborne route in the hospital setting, but evidence in support of this possibility is fragmentary. The recent outbreak of *Hantavirus*-associated adult respiratory distress syndrome in many parts of the United States also raises such concerns, which thus far seem to have been entirely theoretical.

Some evidence suggests that certain enteric viruses may be transmitted through the air. Particularly intriguing was an outbreak of what apparently was Norwalk virus-like gastroenteritis that occurred in a 600-bed general hospital in Toronto, Ontario, Canada, in November 1985. The outbreak occurred over a 3-week period and involved 635 hospital personnel, more than one quarter of the staff. No common food or water source was found, and the investigators concluded that spread of the organism within the hospital probably was by the airborne route.

Although a theoretical possibility, there is no evidence to support transmission of bloodborne viral pathogens such as hepatitis B virus or human immunodeficiency virus (HIV) through generation of aero- sols in blood banks, patient care areas, operating rooms, clinical laboratories, or autopsy rooms.

Moving up from viruses, there is one rickettsial agent that should be mentioned, that being *Coxiella burnetti*, the etiologic agent of Q fever. This organism has never been transmitted in the hospital setting, to my knowledge, but it has caused airborne infection in medical school research laboratories that used partu- rient sheep to study perinatal physiology. In a 1980 outbreak at the University of Colorado Health Sciences Center, most of the 137 cases occurred in staff members working in laboratories or offices along the routes used in transporting sheep to their destination.

Bacteria that have been implicated in airborne transmission in healthcare facilities are shown in Table 3. Evidence in support of airborne transmission of bacteria generally is easier to obtain than in the case of viruses simply because it is technically easier to recover bacteria using air sampling techniques. Yet the mere demonstration of viable bacterial pathogens in the air does not establish that airborne transmission has occurred.

Bacteria that may be transmitted airborne directly from infected persons or healthy carriers include group A streptococci, *Staphylococcus aureus*, the Menin-
gococcus, Corynebacterium diphtheriae, Bordetella pertussis, and, of course, Mycobacterium tuberculosis. Bacteria that may be airborne from dust particles or from aerosols generated within the hospital include S. aureus, tubercle bacilli, other mycobacteria, Nocardia species, pseudomonads, enteric bacteria, and legionellae. Contaminated or colonized ventilation or air-conditioning systems have resulted in airborne spread of legionellae, pseudomonads, clostridia, Nocardia, and probably Chlamydia psittaci.

Among the bacteria spread directly from infected persons, patients or personnel, or from asymptomatic carriers, S. aureus and tubercle bacilli are the most important by far. Airborne staphylococcal infections in hospitals have been particularly important in nurseries and operating theaters. Key experiments documenting airborne spread of staphylococci in nurseries were carried out by Mortimer et al in the early 1960s. These studies are another example of elegance in simplicity. One such nursery study is illus-

**TABLE 3**

**BACTERIA THAT CAUSE AIRBORNE NOSOCOMIAL INFECTION***

<table>
<thead>
<tr>
<th>From patients, staff, and visitors:</th>
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<tbody>
<tr>
<td>Group A Streptococcus</td>
</tr>
<tr>
<td>Staphylococcus aureus</td>
</tr>
<tr>
<td>Neisseria meningitidis</td>
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<tr>
<td>Bordetella pertussis</td>
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<tr>
<td>Mycobacterium tuberculosis</td>
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<tr>
<td>From infective aerosols:</td>
</tr>
<tr>
<td>Pseudomonads</td>
</tr>
<tr>
<td>Acinetobacter</td>
</tr>
<tr>
<td>Legionellae</td>
</tr>
<tr>
<td>Other nonfermenters</td>
</tr>
<tr>
<td>From ventilation/air-conditioning systems:</td>
</tr>
<tr>
<td>Legionellae</td>
</tr>
<tr>
<td>Clostridia</td>
</tr>
<tr>
<td>Nocardia</td>
</tr>
</tbody>
</table>

* Adapted from Schaal.7

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**FIGURE 3.** Floor plan and rear elevation of Meschede Hospital showing locations of all smallpox cases. Reprinted with permission.16
treated in Figure 4. Carrier infants were placed in the “X” bassinets; the same nurses that cared for the “X” infants also cared for the “T” infants, and spread of the index staphylococci to the “T” infants probably was primarily a result of contact spread. That this was so could be demonstrated by a sharp reduction in colonization of the “T” infants when rigorous handwashing techniques were used. The “AB” infants, however, were cared for by other nurses who were restricted to the area in the nursery bounded by the solid line shown in Figure 4. Thus, these nurses had no contact with the “X” or “T” infants, and the spread of index staphylococci to the “AB” infants was primarily airborne. In recent decades, however, staphylococcal cross-infection in nurseries appears to have become much less prominent, although staphylococcal colonization almost surely continues to occur.

In contrast, staphylococcal postoperative wound infections remain a significant problem, particularly in procedures involving the insertion of prosthetic devices, including joints and valves. There continues to be a great deal of controversy, however, as to the relative contribution to the problem made by airborne transmission of staphylococci, as compared with transmission by direct or indirect contact. For example, when total hip arthroplasty first was introduced, postoperative infections, mostly due to staphylococci, were unacceptably frequent. Using ultraclean vertical laminar airflow plus exhaust-ventilated clothing in the operating room, Charnley et al. were able to show in the late 1960s a striking reduction in postoperative sepsis rates from 9% to 1%. Critics pointed out that there were no concurrent controls in those studies and that several other changes were introduced during the study period. Surgeons improved their skills as they gained more experience, operative techniques were changed, and operation duration decreased.

Furthermore, in some other centers, notably at the Mayo Clinic, where ultraclean air was not used for total hip arthroplasties, infection rates were comparatively low.

The role of airborne bacteria in operating rooms as major determinants of postoperative wound infection rates in other kinds of surgical procedures remains controversial as well. Some surgeons in the United States, notably Deryl Hart at Duke University, were so convinced of the significant role of airborne transmission that they installed UV lights in their operating rooms during the 1940s and 1950s. Published data suggested that the use of UV lights in those operating rooms was associated with a very low infection rate, approximately 0.5% in “refined clean wounds,” a category of surgical wounds in which one would expect an infection rate of 1% or less.

Continuing controversy about the value, if any, of UV irradiation in operating rooms prompted the National Research Council to sponsor a multihospital controlled trial of the role of UV light in preventing postoperative wound infection. The results published in 1964 suggested that there was indeed a reduction of the rate of postoperative infections in “refined clean wounds,” from 3.8% to 2.9%, but this category of wounds represented only 19% of all infections studied; thus, this modest beneficial effect was lost in the overall experience of the study and was offset by an apparent detrimental effect of UV light in nonclean wounds. However, UV light was effective in reducing the counts of airborne bacteria in the operating rooms.

Controversy about the relationship of quantitative bacterial counts in the operating room and the risk of subsequent development of sepsis continues. Lidwell et al. in Great Britain found a good correlation between the level of air contamination and subsequent sepsis rates in joint replacement procedures. Fitzgerald et al. at the Mayo Clinic were not able to relate the level of airborne bacteria to the risk of wound sepsis; however, they did note that older operating rooms with lower rates of air exchange seemed to have higher postoperative infection rates than newer rooms, with higher rates of air exchange.

Today, by far the most serious threat in airborne nosocomial infection is that posed by M.
tuberculosis. The nature of the threat is clear enough and is highlighted by a number of recent investigations of hospital outbreaks of multidrug-resistant TB. All of them have been associated with highly immunosuppressed acquired immunodeficiency syndrome (AIDS) patients acting as index cases, and spread occurred within the hospital to other AIDS patients, patients highly immunosuppressed for some other reason, and to hospital staff. In one instance, a healthcare worker with HIV infection and TB was the index case in a major outbreak in a city hospital. Even before the AIDS epidemic, there already was abundant evidence that TB was transmitted via the air in hospitals. TB is in many ways the prototype airborne infection because there is evidence that tubercle bacilli are transmitted more effectively by the airborne route than by any other. Droplet nuclei, owing to their very small size, may be inhaled directly into terminal alveoli without even encountering the pulmonary host defense mechanisms that protect us so well against larger particles.

Group A streptococcal airborne transmission in hospitals fortunately is infrequent, but has occurred. The source almost invariably has been a physician or nurse, and spread has been from the nares, pharynx, vagina, or anus. Meningococcal nosocomial infection has been rare, but probably has occurred as well.

In general, enteric gram-negative bacteria are spread only rarely, if at all, via the air because they are quite susceptible to drying. However, other nonenteric gram-negative organisms, including Pseudomonas and Acinetobacter, have been transmitted through the air. Allen and Green reported an outbreak of multidrug-resistant Acinetobacter anitratutus infections in patients in neurosurgical wards and the intensive care unit of a general hospital. Most of the infections involved the respiratory tracts of ventilated patients, but the respiratory equipment could not be implicated as the source of the outbreak. The investigators believed that airborne transmission played a major role in perpetuation of this outbreak, but the proportion of infection caused by airborne spread could not be determined. It is worth noting, however, that this particular organism has been found to be unique among gram-negative bacilli in its relative resistance to drying.

In the past two decades, Legionella pneumophila and related species have emerged as significant nosocomial pathogens that may be spread via air. Probably the lack of evidence of person-to-person spread facilitated acceptance of Legionnaire's disease as an airborne infection. Spread through infectious aerosols has been demonstrated amply and this, of course, is not truly airborne infection. However, several other epidemics have implicated ventilation systems; these represent true airborne transmission. Perhaps the most vivid outbreak occurred in Memphis in the summer of 1978; 44 cases of L pneumophila pneumonia occurred in patients in a particular wing of a hospital. The investigation revealed (Figure 5) that the cooling tower for an auxiliary air-conditioning system was contaminated with the organism; normal aerosol drift occurred and was drawn into the air intake of the hospital's ventilation system. This outbreak emphasized again that careful consideration must be given to locating air intakes for ventilation systems. This appears to be a lesson that must be relearned at periodic intervals.

Other bacteria implicated in spread through ventilation systems have included clostridia, Nocardia, and perhaps atypical mycobacteria. There have been several recent reports of possible airborne transmission of Nocardia, usually involving high-risk patients in special care units, such as transplant recipients.

Among the fungi (Table 4), only Aspergillus and to a lesser extent Zyomyces have been implicated as major airborne hazards in the hospital setting. Most of these outbreaks have been associated with hospital construction or renovation. Airborne Aspergillus infections have proven to be a particular hazard in special

**TABLE 4**

Fungi that cause airborne nosocomial infection

<table>
<thead>
<tr>
<th>Fungi</th>
<th>Cause</th>
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<tbody>
<tr>
<td>Aspergillus</td>
<td>Zyomyces (Mucor and others)</td>
</tr>
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care units in which severely granulocytopenic patients are housed. Bone marrow transplant patients are at particular risk, but the increased risk can be controlled by HEPA filtration and laminar airflow.\textsuperscript{45,46}

There is abundant evidence that \textit{Pneumocystis carinii} may be transmitted via air in animal experiments. There is circumstantial evidence that it has been transmitted in nursery settings. There is no direct evidence, however, that \textit{P carinii} is a significant nosocomial pathogen, or that airborne transmission occurs in healthcare settings.\textsuperscript{47}

**THE RELATIVE ROLE OF AIRBORNE SPREAD IN NOSOCOMIAL INFECTION**

Finally, it is instructive to examine the relative contribution of airborne transmission to the overall problem of hospital infection. At the 1970 International Conference onNosocomial Infection, held at the Centers for Disease Control (CDC), Brachman\textsuperscript{48} reviewed the topic and concluded that although airborne spread certainly accounted for some nosocomial infections, the exact size of the piece was unknown. He estimated, based largely on data available from the then-infant National Nosocomial Infections Surveillance Study, that airborne spread accounted for 10% to 20% of all endemic nosocomial infections.

In a 1980 review of airborne contagion, sponsored by the New York Academy of Sciences, Kundsin\textsuperscript{49} concluded, based largely on studies carried out at the Peter Bent Brigham hospital during the previous 20 years, that airborne spread in the operating theater accounted for 20% to 24% of all postoperative wound infections. Others doubted that the proportion was that high and were skeptical of the importance of absolute levels of bacteria in operating room air, although instances of staphylococcal transmission from a surgeon to patients in the operating room had been documented thoroughly. The Cooperative Ultraviolet Light Study,\textsuperscript{27} although it did not show a dramatic effect of UV light in reducing rates of postoperative wound infection, did not evaluate directly possible routes of transmission of bacteria causing postoperative wound infection.

In a thoughtful review, Ayliffe\textsuperscript{50} cited an unpublished study carried out in Birmingham, England, in which the postoperative wound infection rate in an unventilated operating suite during the year preceding installation of a ventilation system was 8.8%, in the year following installation of a plenum ventilation system with 20 air changes per hour, the infection rate was 12.6%! Furthermore, there was a 50% reduction in airborne bacterial counts after the ventilation system was installed in this admittedly uncontrolled study. He cited evidence that most wound infections are acquired in the operating room from the patient’s own microbial flora, the balance being acquired mainly from staff present in the operating room during surgery. Since air is an important source of infection involving insertion of prostheses of various kinds, the use of ultraclean air and exhaust-ventilated clothing frequently is recommended. The value of this technology in other kinds of surgical procedures, however, is doubtful.

It appears likely today that Brachman was not far off in his 1970 estimate,\textsuperscript{48} and a more recent estimate of the relative incidence of airborne infections is about 10% of the whole of endemic nosocomial infection.\textsuperscript{7}

Epidemic nosocomial infections must be considered, as well. The CDC studies carried out during the early 1970s suggested that outbreaks of nosocomial infection in seven hospitals participating in an intensive surveillance study represented only about 2% of all patients with nosocomial infection.\textsuperscript{51} Wenzel et al.\textsuperscript{52} estimated that outbreaks accounted for 3.7% of nosocomial infections in a large university tertiary care referral center. Among nosocomial outbreaks investigated by the CDC from 1986 to 1990, more than 67% were related to products, procedures, or devices.\textsuperscript{53} Thus, airborne outbreaks of nosocomial infection have not been prominent, at least on a simple statistical basis.

Although reassuring, there have been some disquieting trends in the last decade. Particularly worrisome has been the resurgence of airborne nosocomial transmission of TB, a problem made all the more urgent by the multidrug-resistant nature of recent outbreaks. Outbreaks of airborne legionellosis in hospitals continue to occur, as does airborne transmission of \textit{Aspergillus} causing both endemic disease in certain special care units and construction-related outbreaks. These concerns relate primarily to outbreaks, unanticipated and unpredictable in occurrence. The only predictable thing about epidemic nosocomial infections is that they will continue to occur.

Should we be doing surveillance cultures for airborne nosocomial pathogens? Our surgical colleagues would love us if we did! In 1970, at the behest of the American Hospital Association Advisory Committee on Infections in Hospitals, I published recommendations against routine environmental sampling programs, including air sampling, even with simple techniques such as settle plates.\textsuperscript{54} At that time, patient-based surveillance programs literally were just getting started in a few hospitals, and environmental sampling programs were, in fact, what most hospitals were doing to “prevent” nosocomial infection. It mattered not that no one knew how to interpret the data generated by such programs.

Did we overstate the case against environmental
sampling? Not at all, in view of the circumstances that existed at that time. Should this policy be re-examined? I believe the answer is yes; in some very limited circumstances, it may be appropriate to re-consider directed environmental sampling in ultrahigh-risk units such as bone marrow transplant units or other settings in which patients temporarily have no functioning host defense mechanisms. In such settings, any stray opportunistic organism that comes along may in fact cause a fatal infection. At the very least, the issue certainly merits some thoughtful consideration.

REFERENCES

independent risk factor for invasive aspergillosis and zygomycosis in patients with hematologic malignancy. Infect Control 1987;8:71-75.

Whirlpool Bath Source of Outbreak of Legionnaires’ Disease Aboard Cruise Ship

by Gina Pugliese, RN, MS
Medical News Editor

As of August 10, 1994, a total of 14 passengers aboard the cruise ship Horizon had Legionnaires’ disease (LD) confirmed by either sputum culture, detection of antigens of Legionella pneumophila serogroup 1 (LP1) in urine by radioimmunoassay, or fourfold rise in antibody titers between acute- and convalescent-phase serum specimens. Under investigation are 28 other passengers with pneumonia that occurred within 2 weeks after sailing aboard the cruise ship. Cases have occurred from separate week-long cruises between April 30 and July 9, 1994.

A case-control study revealed a strong association between exposure to whirlpool baths and illness. Cultures taken from a sand filter, used for recirculation of whirlpool water, yielded an isolate of LP1; this isolate and the clinical isolate had matching monoclonal antibody subtyping patterns. Various interventions were completed, including hyperchlorination of the ship’s potable water supply, removal of the whirlpool filters, and discontinuation of the whirlpool baths.

Additional recommendations to reduce the risk of transmission from whirlpool baths aboard cruise ships will be the subject of a special meeting scheduled to be held this fall. Information about the meeting is available from CDC's National Center for Environmental Health: (404) 488-7093.