

Respiratory Syncytial Virus: Its Transmission in the Hospital Environment

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Respiratory syncytial virus (RSV) over the past two decades has been recognized as the most important cause of lower respiratory tract disease in infants and young children. Recently, it has also been identified as a major nosocomial hazard on pediatric wards. The potential for RSV to spread on such wards is underlined by several singular characteristics of RSV. It arrives in yearly epidemics and is highly contagious in all age groups. Immunity is of short duration, allowing repeated infections to occur. Thus, during an epidemic 20-40 percent of infants admitted for other conditions may acquire nosocomial RSV infection, as well as 50 percent of the ward personnel. The usual infection control procedures for respiratory illnesses have had limited success in controlling the spread of RSV. This may be due in part to the modes of transmission of RSV. Inoculation occurs mainly through the eye and nose, rather than the mouth. This may be via large-particle aerosols or droplets, requiring close contact. The virus, however, does not seem capable of traversing distances by small-particle aerosols. Nevertheless, it is able to remain infectious on various environmental surfaces, suggesting fomites as a source of spread. Indeed, inoculation after touching such contaminated surfaces can occur, and may be a major second means of spread, in hospitals as well as in families.

Twenty-five years ago a virus was isolated from a chimpanzee suffering from a cold [1]. It was subsequently entitled "CCA," chimpanzee coryza agent, emphasizing its animal source and its upper respiratory tract signs. Over the ensuing two decades, however, this virus, respiratory syncytial virus (RSV), has been recognized as the major respiratory pathogen of infants and young children.

Initially its contagiousness was not entirely appreciated nor its recalcitrant behavior known. Subsequently its ubiquitous and epidemic profile has been painted with its yearly peaks of bronchiolitis and pneumonia in young children [2,3,4]. Essentially all children acquire RSV infection during their first few years of life. Specific neutralizing antibody is present in all adults, and thus passively in all newborns [5,6,7]. Studies in Washington, D.C., have indicated that approximately half of the infants in that urban setting will acquire RSV infection during the first epidemic to which they are exposed [7-9]. Furthermore, about 40 percent of these infants infected in their first year of life will develop lower respiratory tract disease, and 1 percent of those will require hospitalization [7-10].

RSV not only spreads easily among the young, but also produces re-infections in older age groups. Indeed, older siblings appear most commonly to introduce the virus into the family setting [11,12]. In families with an infant and at least one older child who were studied prospectively during an RSV outbreak, the secondary attack

rate was highest in infants. Once the virus was introduced into a household, however, it was highly contagious to family members of all ages (Table 1) [11].

Certain characteristics of RSV infection suggest why it spreads with such facility. First, RSV is the only virus that regularly produces outbreaks of infection in urban areas each year [4,7,9]. Second, all of us are potentially susceptible to RSV re-infection despite our possession of measurable levels of specific serum antibody. Immunity to RSV remains a conundrum; it is of short duration and repeated infections occur throughout life [1,11-14]. RSV infection manifest as pneumonia and bronchiolitis is mostly confined to infancy and early childhood. However, a child may have more than one bout of bronchiolitis or pneumonia within this time period [13]. In subsequent years RSV infection may be manifest as a "cold," otitis media, febrile upper respiratory tract infection, or tracheobronchitis [14,15]. Hence, susceptible populations are readily available for the spread of RSV.

These same characteristics suggest that RSV would spread with ease on hospital wards during a community outbreak of RSV infection. Indeed, its potential as a nosocomial hazard has recently been recognized both in England and in this country [16,17]. On pediatric wards during winter and early spring an appreciable proportion of admissions may be infants with RSV lower respiratory tract disease. These infants shed the virus in particularly high titer and for prolonged periods [18]. The potential for nosocomial spread of RSV is further enhanced by the crowding on pediatric wards that may occur during such a community outbreak.

The risk of an infant acquiring RSV infection as a result of nosocomial contact appears to be related to the duration of the infant's hospitalization, to the design of the ward, and to the age of the infants it houses [16,17,19]. Studies on our pediatric floors in Rochester over the past few years have indicated that 20-45 percent of babies admitted with other conditions will acquire RSV nosocomially [17,20-22]. The morbidity and occasional mortality associated with these nosocomial infections underscore the importance of delineating how RSV is transmitted. Lower respiratory tract involvement occurs in 30 to 50 percent of these infants with nosocomial RSV infection; in the rest it may be manifest as an upper respiratory tract infection

TABLE 1
Attack Rate of Respiratory Syncytial Virus (RSV) in Families According to Age

Age (Yr)	Attack Rate*					
	Crude Rate		Rate in RSV-Positive Families		Secondary Rate	
	No.*	%	No.*	%	No.*	%
< 1	10/34	29.4	10/16	62.5	5/11	45.4
1-<2	2/7	28.6	2/5	40.0	0/3	0.0
2-<5	9/34	26.4	9/19	47.0	2/12	16.6
5-<17	9/48	18.7	9/24	38.0	4/19	21.0
17- 45	<u>9/55</u>	16.8	<u>9/21</u>	43.0	<u>6/18</u>	33.3
Totals	39/178	21.9	39/85	45.9	17/63	27.0

*Crude attack rate according to age is shown for all family members studied and for members of RSV-positive families. The secondary attack rate is also shown for members of RSV-positive families, excluding all primary and co-primary cases.

*No. of persons infected with RSV

total no. of persons exposed

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with fever or otitis, or occasionally even as apnea [17,20-22]. In certain groups of compromised infants, especially those with congenital heart disease or those who are immunosuppressed, RSV infection may be particularly severe and accompanied by a high rate of mortality [23,24].

The children on a ward are not the only ones to acquire a nosocomial infection with RSV. Thirty to 50 percent of the personnel working on these infant wards are also apt to acquire RSV infection [17,20-22]. Illness in the staff may further aggravate the problems of a busy infant ward during a community outbreak of RSV infection. About 80 percent of infections in these personnel are symptomatic and commonly require absence from work. Furthermore, staff members appear to be important in the spread of the virus within the hospital.

We have used a variety of infection control procedures in an attempt to limit the nosocomial spread of RSV [21,22]. Infected infants have been isolated or divided into cohorts to whom staff are solely assigned. In addition, hand washing has been emphasized and gowns worn whenever direct patient contact occurs. The number of patient contacts and visitors to the ward have also been limited. Such techniques have resulted in a diminished number of nosocomial infections in infants, but appear to have no effect on the rate of nosocomial infections in exposed personnel [21].

How, then, was this virus being transmitted if such control procedures did little to prevent infections in our ward staff? Theoretically three modes of viral transmission are possible; (1) by small particle aerosols, which could travel appreciable distances (greater than six feet) (2) by large-particle aerosols or droplets, which can travel only short distances and, therefore, whose spread would require close contact, or (3) by fomites and inoculation from contaminated hands. The continued high rate of infection in personnel suggested that this last mode might be important. However, transmission of RSV from contaminated surfaces to hands would require its survival in the environment for periods sufficient for such spread. Yet RSV is well recognized as a labile virus.

Subsequently we examined the survival of RSV in fresh secretions on various surfaces surrounding an infant's bed, and found that on some surfaces RSV was capable of relatively long life (Fig. 1) [25]. Its infectivity varied with the type of surface, the environmental humidity, and the temperature. On non-porous surfaces,

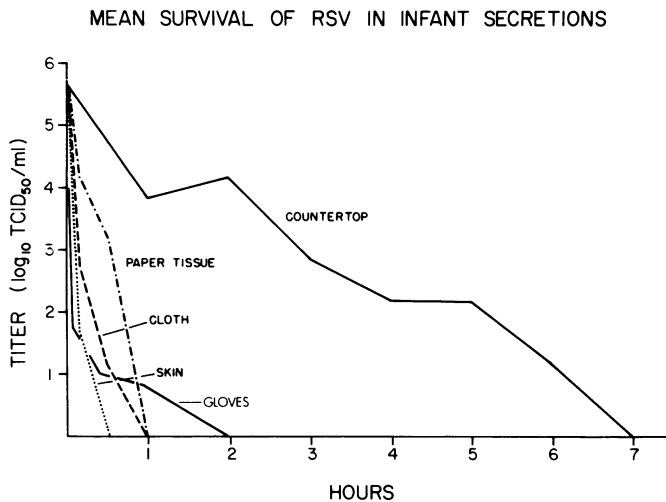


FIG. 1. Mean titer and duration of recovery of respiratory syncytial virus (RSV) from skin (hands) and four environmental surfaces that were contaminated with RSV in nasal secretions freshly obtained from infants hospitalized with lower respiratory tract disease caused by RSV.

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such as counter tops, plastic or glass, RSV can survive for six to 12 hours or sometimes even longer. Furthermore, RSV may be transferred from these surfaces to hands with subsequent recovery of infectious virus from the skin. Similar patterns of transmission have been found with rhinoviruses by Hendley and colleagues [26]. Therefore, it appeared possible that RSV might be spread by touching contaminated surfaces and then inoculating one's self. Whether such a mode actually occurred on hospital wards or within families remained to be determined.

In a further study, adult volunteers were exposed in one of three ways to infected babies admitted with RSV lower respiratory tract infection [27]. The first group, called "cuddlers," were exposed by caring for an infant in the usual manner, such as feeding, changing, and playing with the baby. These caretakers wore gowns on direct contact, but no masks. The second group, called "touchers," were exposed by touching various objects and surfaces about the infant's bed and then touching their eyes or nose at a time when the infected infant was out of the room. The third group, called "sitters," were exposed by sitting at a distance of greater than six feet from an infected infant. The sitters were gowned, gloved, and could read but were not allowed to touch anything. Hence, cuddlers could be exposed by any of the three possible modes of transmission—large-particle aerosols, self-inoculation after touching fomites, and small-particle aerosols. Touchers, on the other hand, would be exposed only by self-inoculation from touching contaminated surfaces. Sitters would be infected only by small-particle aerosols. Cuddlers and touchers became infected, but none of the sitters. This suggests that RSV may be spread by close contact with direct inoculation of large-particle aerosols or by self-inoculation after touching contaminated surfaces. However, spread by small-particle aerosols does not seem to be a major mode of transmission.

RSV appears able to infect through the nose or eye, but not by mouth [28]. In these studies of volunteers challenged with RSV by nose, eye, and mouth in various doses, the nose and eye appeared to be equally sensitive routes of inoculation. Hence, the inadvertent rubbing of the eye or nose by contaminated hands may lead to RSV infection. Infection control procedures, therefore, should take into consideration that masks cover only one of the potential portals of entry for RSV.

In summary, RSV is only about 25 years old—in terms of recognized birthdays. Yet during this period it has been demonstrated to be an ubiquitous and highly contagious virus of pathogenic import. Transmission of RSV appears to require close contact. Inoculation occurs primarily through the eye or nose directly by large-particle aerosols or indirectly after touching contaminated surfaces. This labile virus has the ability to survive on various environmental surfaces long enough for spread by fomites to be feasible. The infectivity of the virus and the potential for transmission are variable, depending on the type of surface and the environmental conditions.

A susceptible population for the spread of RSV is usually readily available, because immunity to this virus is of short duration. Problems which remain arcane include, first, the host or other factors associated with resistance to RSV infection, and, second, the reason for its striking winter-spring epidemic activity, followed by its mystical disappearance and period of hibernation.

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