

# Handwashing and cohorting in prevention of hospital acquired infections with respiratory syncytial virus

David Isaacs, Harriet Dickson, Chris O'Callaghan, Richard Sheaves, Andrew Winter, E Richard Moxon

## Abstract

**Hospital acquired infections with respiratory syncytial virus are a major problem. The virus is spread predominantly by infected nasal secretions and we investigated whether we could reduce its incidence by cohorting babies on each ward into designated areas and encouraging staff and parents to wash their hands.**

We examined the incidence of hospital acquired infection due to respiratory syncytial virus in all children <2 years old and in those with congenital heart disease. In 1986-7, before any intervention, 18 (4.2%) of 425 hospitalised children <2 years old developed hospital acquired infection due to respiratory syncytial virus. In 1987-8, after intervention, five (0.6%) of 840 children developed hospital acquired infection but there were fewer ward admissions with community acquired infections due to the virus. In 1988-9, when there were more community acquired infections than 1986-7, six (1.1%) of 552 children developed hospital acquired infection. In 1986-7, eight (34.8%) of 23 children <2 years old with congenital heart disease developed hospital acquired infection due to respiratory syncytial virus; all eight were among 11 children with congenital heart disease hospitalised for more than 14 days. In 1987-8, one (3.3%) of 30 children with congenital heart disease developed hospital acquired infection due to respiratory syncytial virus and in 1988-9 there was one (2.1%) case out of 47 children with congenital heart disease.

**Handwashing and cohorting significantly reduce the incidence of nosocomial respiratory syncytial virus infection.**

Respiratory syncytial virus is a major cause of morbidity and mortality in children world wide.<sup>1</sup> Children's services in hospital present a particular problem as not only are large numbers of infants admitted from the community each year with bronchiolitis and other infections caused by respiratory syncytial virus but there is a subpopulation of hospitalised children who are at relatively high risk from infection. Children at increased risk from respiratory syncytial virus infection include those with congenital heart disease, particularly if they have pulmonary hypertension,<sup>2</sup> babies who are born preterm and have chronic lung disease such as bronchopulmonary dysplasia,<sup>2</sup> immunocompromised children,<sup>3</sup> and those with cystic fibrosis.<sup>4</sup>

Respiratory syncytial virus spreads readily within hospitals to infect children and staff.<sup>5</sup>

Gloves and gowns<sup>6</sup> and even goggles<sup>7</sup> have been found by some authors to be of value in decreasing the incidence of hospital acquired (nosocomial) infections but others have been unconvinced by the efficacy of gowns and masks.<sup>8</sup> It is now widely accepted in neonatal units that handwashing is an important way of preventing nosocomial infections, but this has been less firmly emphasised on general paediatric wards, at least in the United Kingdom, and its efficacy in preventing respiratory syncytial virus infection has not been evaluated. In an effort to decrease the incidence of nosocomial respiratory syncytial virus infections we introduced cohorting of babies with suspected or proved respiratory syncytial virus infection and mounted an educational programme to emphasise the importance of handwashing to staff and relatives.

## Patients and methods

In order to assess the importance of nosocomial respiratory syncytial virus infection we retrospectively collected data on all children with respiratory syncytial virus infection on any of the paediatric medical wards in the winter of 1986-7. Infection by the virus was diagnosed by indirect immunofluorescence on nasopharyngeal secretions using a bovine antiserum against respiratory syncytial virus (Central Public Health Laboratory, Colindale) and a fluorescein conjugated antiserum (Wellcome) and/or by culture of secretions on MRC-5 fibroblasts and HeLa cells.

In order to define a population at risk of developing respiratory syncytial virus infection we determined the number of children under 2 years of age hospitalised on the two paediatric medical wards (one 17 and one 19 bed) and the (six bed) paediatric intensive care unit and the number of days they spent in hospital. Each medical ward contained six single rooms. To examine a group at high risk for severe respiratory syncytial virus infection the records were reviewed of all children under 2 years of age with congenital heart disease admitted to the paediatric medical wards over the period of the 1986-7 outbreak, from the first reported case until the last case had been discharged or was no longer infectious. In our hospital cardiac patients are admitted onto one of the general paediatric medical wards. Cases were classified as having either community acquired infection if they were admitted with a respiratory illness and had not been in hospital in the 10 days before developing symptoms or hospital acquired infection if they had previously been in

Infectious Diseases Unit,  
Department of  
Paediatrics,  
John Radcliffe Hospital,  
Oxford  
David Isaacs  
Harriet Dickson  
Chris O'Callaghan  
Richard Sheaves  
Andrew Winter  
E Richard Moxon

Correspondence and requests  
for reprints to:  
Dr David Isaacs,  
Department of Immunology  
and Infectious Diseases,  
The Children's Hospital,  
Pymont Bridge Road,  
Camperdown,  
NSW 2050,  
Australia.

Accepted 16 August 1990

this hospital in the preceding 10 days. All children with hospital acquired infection had in fact previously been in this hospital within five days of developing symptoms; that is the average incubation period of infection with the virus.<sup>6</sup> Follow up after discharge to identify late onset hospital acquired infection was not routinely performed. Children who acquired their respiratory syncytial virus infection at another hospital were considered to have community acquired infections for the purpose of this study. Children, not previously hospitalised, admitted with a non-respiratory condition but developing a respiratory illness due to respiratory syncytial virus within five days of admission were deemed to have community acquired infection, although no such cases actually occurred in the study period. Children were defined as having recurrences of infection with the virus if their symptoms recurred more than 30 days after their initial infection and respiratory syncytial virus could again be isolated.

The severity of the infection, both hospital and community acquired, was assessed using a clinical grading.<sup>9</sup> Grade 1: children tolerated oral feeds, grade 2: required nasogastric tube feeds, grade 3: required nasogastric tube feeds and supplemental oxygen, grade 4: required intravenous fluids and supplemental oxygen, and grade 5: required artificial ventilation.

For the next two winters (1987-8 and 1988-9) the same data were prospectively collected. In addition some interventions were made to try to reduce the incidence of hospital acquired respiratory syncytial virus infection. Because the medical wards alternate in taking emergency admissions it was not practicable to have a ward

dedicated to children with the infection. A six bedded area in each of the two paediatric medical wards including two single rooms was, therefore, cordoned off by screens and designated the 'respiratory syncytial virus area'. Children admitted with suspected respiratory syncytial virus infection were nursed in this area until the result of an indirect immunofluorescent stain of nasopharyngeal secretions was available. If this proved negative they were moved into the rest of the ward. Children on the ward developing respiratory symptoms were not transferred to this area unless they proved to be positive for the virus by indirect immunofluorescence. Where possible nurses who developed a respiratory illness were cohorted to care for babies in the respiratory syncytial virus area, but no formal attempt was made to diagnose the virus in staff. It was not possible to cohort babies on the paediatric intensive care unit. It has been hospital policy for nursing but not medical staff to wear gowns when handling infants under 1 year old, and change these between each infant, and this policy was not changed. Alcohol based hand rubs (Amphisept 80, Goldschmidt Ltd) were placed on each sink. Staff were instructed on the importance of handwashing and this was reinforced on ward rounds. A leaflet was prepared for parents (figure) detailing the way respiratory syncytial virus infection is spread, asking them to wash their hands and to keep older siblings with colds away from the play areas. A leaflet was given to the parents of every child admitted with the infection.

Statistical analysis was by Student's *t* test to compare means and by  $\chi^2$  or Fisher's exact test to compare incidence and severity of infection.

◆ Your baby is in hospital with a condition called bronchiolitis.

◆ This is caused by a virus called respiratory syncytial virus (RSV).

◆ The virus is usually caught from a brother or sister or parent who has a cold or chestiness.

◆ The disease is very infectious and is passed on by infected nasal secretions carried on hands or toys but not usually by coughing. The secretions are rubbed into the nose or eyes to cause infections.

◆ The best way of preventing spread of RSV infection is, therefore, by washing your hands after handling your baby. If you have a cold yourself try to wash your hands before handling other children.

◆ Many children on the ward have conditions such as heart disease which can be made much worse by RSV infection. To prevent these children being infected please wash your hands. If you have an older child with a cold do not let them play in the play areas on the ward until they are better.  
Thank you

Information leaflet given to parents.

#### Stop Bronchiolitis:-



#### Results

In 1986-7 (9 December to 26 March), before any intervention, there were 122 isolates of respiratory syncytial virus from study children. Altogether 101 isolates were from community acquired infections, 18 children had hospital acquired infections, and three with hospital acquired infection had recurrences. In 1987-8 (12 September to 11 April) there were 55 admissions with community and five with hospital acquired infection. In 1988-9 (18 October to 16 February) there were 115 admissions with community and six with hospital acquired infection (table 1).

During the first study period (1986-7) there were 425 children admitted to or present on the paediatric wards, excluding children admitted with respiratory syncytial virus infection, the latter not being considered at risk of acquiring the virus. The children stayed in hospital for a

Table 1 Occurrence of community acquired infections and hospital acquired infections of respiratory syncytial virus

Year	Total admissions during epidemic	No (%) with community acquired infection	Remainder (at risk of hospital acquired infection)	No (%) with hospital acquired infection
1986-7	526	101 (19)	425	18 (4.2)*
1987-8	895	55 (6)	840	5 (0.6)*
1988-9	667	115 (17)	552	6 (1.1)*

\*1986-7 v 1987-8,  $\chi^2=19.0$ ,  $p<0.001$ ; 1986-7 v 1988-9,  $\chi^2=8.7$ ,  $p<0.01$ ; 1987-8 v 1988-9,  $\chi^2=0.5$ ,  $p=0.5$ .

Table 2 Duration of hospital stay for children with community acquired infection due to respiratory syncytial virus

Year	Total days in hospital	Mean (SD) stay in days*	Median stay in days
1986-7	414	5.24 (4.27)	4
1987-8	259	4.71 (4.29)	3
1988-9	487	4.24 (4.25)	3

\*No significant difference between years.

total of 1731 days within the study period, a mean (SD) stay of 4.05 (3.16) days with a median of 2 days. In the second study period (1987-8), which was a much longer outbreak, there were 840 such admissions for 3749 days, a mean (SD) stay of 4.46 (3.92) days and a median of 2 days. In the third study period (1988-9) there were 552 admissions for 2082 days, mean (SD) was 3.77 (3.69) days and median 2 days. Thus in the first study period 18 of 425 (4.2%) children exposed developed respiratory syncytial virus infection compared with five of 840 (0.6%) in the second period and six of 552 (1.1%) in the third period ( $\chi^2=24.3$ , 2df,  $p<0.001$ ). The difference between the first and second periods ( $\chi^2=19.0$ , 1df,  $p<0.001$ ) and between the first and third periods ( $\chi^2=8.7$ , 1df,  $p<0.01$ ) was significant, but not between the second and third periods ( $\chi^2=0.5$ , 1df,  $p=0.5$ ).

Children infected in the community with respiratory syncytial virus were hospitalised for a similar mean duration in each study period (table 2). The total duration of hospitalisation of children with community acquired infection was similar in 1986-7 and 1988-9, but was considerably less in 1987-8.

There was an increasing number of children under 2 years old admitted with congenital heart disease over the three year study period, concomitant with the appointment of a second cardiologist. In 1986-7 none of 12 children <2 years with congenital heart disease (excluding babies with community acquired infection due to respiratory syncytial virus) admitted for less than 14 days, but eight of 11 (72.7%) admitted for more than 14 days were infected with the virus in hospital (table 3). In 1987-8 one of 30 (3.3%) children with congenital heart disease became infected in hospital compared with eight of 23 (34.8%) in 1986-7 ( $p<0.008$ , Fisher's exact test). In 1988-9 one of 47 (2.1%) became infected in hospital ( $p<0.0008$  compared with 1986-7).

Three children with congenital heart disease were admitted with community acquired infection due to respiratory syncytial virus in 1986-

Table 3 Number of children <2 years old with congenital heart disease admitted to paediatric wards during respiratory syncytial virus season (number who developed a hospital acquired infection with the virus)

Year	Duration of hospital stay (days)			Total
	1-7	8-14	>14	
1986-7	7 (0)	5 (0)	11 (8)	23 (8)*
1987-8	19 (0)	5 (0)	6 (1)	30 (1)*
1988-9	26 (0)	9 (1)	12 (0)	47 (1)*

Children with congenital heart disease admitted with respiratory syncytial virus infection have been excluded.

\* $\chi^2=24.3$ , 2df,  $p<0.001$ .

Table 4 Severity of community acquired infections with respiratory syncytial virus

Year	Grade of severity					Total
	1	2	3	4	5	
1986-7	49	20	8	21	3	101
1987-8	30	10	8	2	5	55
1988-9	62	26	20	6	1	115

$\chi^2=16.4$ , 6df,  $p<0.02$ .

7, none in 1987-8, and seven in 1988-9. Three of the children with respiratory syncytial virus infection in 1986-7 had pulmonary hypertension, none in 1987-8, and four in 1988-9. There were no deaths attributable to infection with the virus. The mean (SD) duration of hospital stay of children under 2 years with congenital heart disease (excluding babies with community acquired infection) was not significantly different in the three study years: 33.6 (33.1) days in 1986-7, 33.7 (16.1) days in 1987-8, and 31.0 (18.2) days in 1988-9. The median stay was 21, 28, and 23 days respectively. Thus children with congenital heart disease were equally likely to be exposed to respiratory syncytial virus infection, other factors being equal, in each of the three years.

The median duration of hospital stay of children with congenital heart disease before they developed respiratory syncytial virus infection was 14 days (range 8-114).

In 1986-7, four babies with chronic lung disease due to bronchopulmonary dysplasia developed respiratory syncytial virus infection (three infections acquired in hospital) and of two children with cystic fibrosis who developed the virus one infection was hospital acquired. In 1987-8 one baby with bronchopulmonary dysplasia became infected with the virus in hospital. In 1988-9 two babies with bronchopulmonary dysplasia were infected (one hospital acquired) and one child with cystic fibrosis had a community acquired infection.

The severity of infection acquired in the community in each of the study years is shown in table 4. A  $\chi^2$  analysis (combining grades 4 and 5 because of small numbers in these cells) showed that the severity of infection was not the same in each of the three years. There was no significant difference in severity between 1986-7 and 1987-8 ( $\chi^2=4.0$ , 3df,  $p>0.2$ ), but infection with the virus was significantly more severe in 1986-7 than 1988-9 ( $\chi^2=15.8$ , 3df,  $p<0.01$ ). In 1988-9 three babies with congenital heart disease and respiratory syncytial virus infection were treated with nebulised ribavirin, but ribavirin was not used for children in previous years nor for any other children in 1988-9.

## Discussion

Nosocomial infections with respiratory syncytial virus are a major problem within hospitals,<sup>5</sup> particularly as children with serious underlying disorders who are at risk of severe infection may become infected. In one study the mortality for infants with congenital heart disease and respiratory syncytial virus was 37%, and 73% if they had pulmonary hypertension,<sup>2</sup> although subse-



quent studies including the current one would suggest the mortality has fallen substantially. The risk of nosocomial infection with the virus has previously been shown to rise with increasing duration of hospital stay.<sup>2</sup>

Prevention of hospital acquired respiratory syncytial virus infection depends on understanding how the infection is spread. Babies shed large quantities of virus for prolonged periods.<sup>10</sup> Adults can readily be reinfected, by rubbing infected secretions into their nose or eyes,<sup>11</sup> and act as a reservoir for the infection, which is probably important in infecting babies. Babies are most likely to be infected by contaminated nasal secretions, which have come from another infected baby or infected member of staff and are carried on the hands of staff.<sup>12</sup> Other possible mechanisms of spread include fomites, as respiratory syncytial virus can remain viable on environmental surfaces for several hours.<sup>13</sup> Respiratory droplet spread, however, is probably an unusual route of transmission.<sup>12</sup> This evidence suggests that efficient handwashing might be an effective way of preventing infections both in staff and babies. It has been recommended that gowns be worn when soiling of clothes with respiratory secretions is likely,<sup>14</sup> but we found an unacceptably high level of nosocomial respiratory syncytial virus infection despite following this suggestion. We do not have sufficient isolation rooms to isolate all infected babies, as has also been suggested.<sup>14</sup> Various interventions have been evaluated. Leclair and colleagues found that glove and gown isolation precautions were highly effective in preventing nosocomial respiratory syncytial virus infections,<sup>6</sup> but others have found gowns and masks to be ineffective.<sup>8</sup> Eye-nose goggles prevent staff infections over a short period but are unacceptable to staff.<sup>7</sup> There are obvious advantages to using handwashing and cohorting as the main methods for preventing infections. Many studies have shown that handwashing practises, even in intensive care units, are suboptimal and we have previously observed handwashing to be extremely poorly performed on paediatric wards. We did not formally examine compliance with handwashing, but feel the reduction in nosocomial respiratory syncytial virus infection is strong evidence that compliance was good.

In order to show convincingly that our interventions were effective in reducing nosocomial infection with the virus we need to show that there was comparable exposure in the three study winters. In 1987–8 the epidemic was much smaller and more drawn out than in the other two years, and children on the wards were less exposed to the virus. Thus the fall in the incidence of hospital acquired infection in 1987–8 alone cannot be attributed to our interventions. In 1988–9 there were more children admitted with community acquired infection than in 1986–7 over a similar period of time, however, and the total number of infections in the two winters was virtually the same. We did not evaluate duration of viral shedding in children with respiratory syncytial virus infection, but they were hospitalised for a comparable

length of time each year. Leclair and colleagues estimated that children would shed virus for seven days, and calculated exposure on this basis.<sup>6</sup> On the other hand children often shed virus for much longer,<sup>9</sup> and neither they nor we formally examined this. As children with hospital acquired infection often remain in hospital because of their underlying illness they will expose other children, leading to a cascade effect if infections are not prevented. Another consideration is that the severity of the annual epidemics may vary from year to year, possibly as a result of different subgroups of respiratory syncytial virus being prevalent,<sup>15</sup> and more severe infections might be more contagious. We did not look at subgroups of the virus but we did examine severity by a clinical grading and found that respiratory syncytial virus infection was less severe in 1988–9 than in the two previous years. Despite this the number of admissions with community acquired infection due to the virus in 1988–9, and reports to the Communicable Disease Surveillance Centre for the three years, would suggest the respiratory syncytial virus epidemic was at least as large in 1988–9 as the two previous years and the virus no less contagious. We did not formally examine nursing levels, which might have influenced spread of infection. However these certainly did not improve between 1986–7 and 1988–9.

Our observations are not based on an ideal study design, as the data on the incidence of nosocomial respiratory syncytial virus infections before intervention were not collected prospectively. Our rate of nosocomial infection with the virus was unacceptably high, however, and we felt ethically constrained to intervene and then attempt to evaluate our intervention. Cases of respiratory syncytial virus infection were identified by positive viral cultures and the techniques used for identification did not change over the study period. Children were classified as having hospital or community acquired infection on strict criteria not amenable to bias. Nasopharyngeal aspirates are routinely taken from all children <2 years admitted with respiratory illness and from those hospitalised children who develop respiratory symptoms. Bias might have been introduced by failure to identify children with hospital acquired infection due to the virus after intervention, but we feel that increased awareness of respiratory syncytial virus infection generated by the study is more likely to have resulted in more rather than fewer cases of hospital acquired infection being identified.

We showed a reduction of at least 66% in the number of hospital acquired infections due to respiratory syncytial virus in the two winters after emphasising the importance of handwashing and simple cohorting of babies. Furthermore, despite comparable lengths of stay each year, we showed that the proportion of babies with congenital heart disease hospitalised for more than 14 days who acquired the infection fell from 73% to less than 4%. We acknowledge that we cannot prove that the decrease in incidence of infection with the virus was due to our interventions, but feel we have provided convincing circumstantial evidence to this effect.

One minor problem with cohorting was that babies could not remain in the accident and emergency department until a diagnosis of respiratory syncytial virus infection was virologically confirmed. Hence they were cohorted on the basis of a clinical diagnosis of bronchiolitis. In 1988–9 one baby with congenital heart disease and clinical bronchiolitis was cohorted in the respiratory syncytial virus area and subsequently shown to be negative for the virus. Despite being moved within 24 hours to the main ward and discharged home shortly thereafter the baby was readmitted within five days with respiratory syncytial virus positive bronchiolitis. This problem might be overcome if a more rapid antigen detection test for respiratory syncytial virus infection was available, and one hour tests are currently being evaluated.

Handwashing is simple, cheap, and highly effective in preventing nosocomial respiratory syncytial virus infections. We should make every effort to reinforce this message to staff and parents on the general paediatric wards as well as in the neonatal unit.

DI was funded by the Wellcome Trust. We should like to thank parents and staff for washing their hands and Doctors Archer, Bower, Dunger, Gardiner, Moncrieff, and Ostman-Smith for allowing us to study their patients. We also thank the virology department for performing antigen detection and virus isolation; the department of medical illustration for preparing the leaflet; Christine Hatton and Virginia Butler of the information centre, Manor House, for ward statistics; and Gail Davies and Betty Nicholson for secretarial assistance.

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### Infant care at the Appeal Court

A very premature baby needed ventilator treatment for a month and two further periods of ventilation when he collapsed off the ventilator. At 4 months postnatal age he was breathing independently but was cyanosed when crying and prone to periods of apnoea. Ultrasound scans had shown 'very severe brain damage'. The child was a ward of court and a judge had agreed with the doctors' view that ventilation should be withheld if the child collapsed again. The Official Solicitor appealed on the child's behalf and the case went to the Court of Appeal on 19 October. (*The Daily Telegraph*, Law reports, 25 October 1990).

The Master of the Rolls, Lord Donaldson, with Lords Justice Balcombe and Taylor, upheld the judge's decision. Lord Donaldson said that, while there was a very strong presumption in favour of a course of action that would prolong life, the quality of life and the pain and suffering that the child would experience if life was prolonged also had to be taken into account. In the end there would be cases where the answer would be that it was not in the child's interests to subject it to treatment which would cause increased suffering and produce no commensurate benefit.

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