

Research from the South

Pneumonia associated with infection with pneumocystis, respiratory syncytial virus, chlamydia, mycoplasma, and cytomegalovirus in children in Papua New Guinea

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Abstract

Paired serum samples were collected from 94 children with pneumonia admitted to Goroka Hospital, Papua New Guinea. All but three of the children were aged 1-24 months. Only nine children were malnourished, with weight for age less than 70% of the Harvard median (three had weight for age less than 60% of the Harvard median). *Pneumocystis carinii* antigen was detected in the serum of 23 children. Twenty two children had serological evidence of recent infection with respiratory syncytial virus. Five children were probably infected with *Chlamydia trachomatis* at the time of the study, and there was less convincing serological evidence of current infection in a further 11 children. Five children showed a fourfold rise in antibody to *Mycoplasma pneumoniae*. Although only one child showed a fourfold rise in antibody to cytomegalovirus, 86 children had this antibody. No child showed a fourfold rise in antibody to *Ureaplasma urealyticum* or *Legionella pneumophila*.

P. carinii, respiratory syncytial virus, *C. trachomatis*, *M. pneumoniae*, and cytomegalovirus may be important causes of pneumonia in children in developing countries.

Introduction

Pneumonia and diarrhoea are the commonest causes of death among children. Of the 15 million children aged under 5 who die every

year, 96% die in developing countries¹ and about 30% of these die from an acute respiratory infection.² Thus a child dies from an acute respiratory infection every seven seconds. Too little is known about the aetiology of these infections³; knowledge is urgently required so that improved treatment regimens and effective vaccines can be developed.

At Goroka Hospital, in the highlands of Papua New Guinea, pneumonia accounted for 45% of the 12 371 paediatric admissions and 43% of the 666 paediatric deaths in the five years 1979-83. In a prospective study of children admitted to the hospital with pneumonia⁴ *Haemophilus influenzae*, *Streptococcus pneumoniae*, or other bacteria were isolated from lung aspirates or blood cultures from 51 (61%) of 83 children, and viruses were isolated from 18 (29%) of 62 children. Neither bacteria nor viruses were isolated from 26 (31%) of the 83 children, but no attempt was made to find evidence of infection with *Chlamydia trachomatis*, *Pneumocystis carinii*, *Ureaplasma urealyticum*, *Mycoplasma pneumoniae*, or *Legionella pneumophila*.

Stagno *et al* suggested that cytomegalovirus, *C. trachomatis*, *P. carinii*, and *U. urealyticum* are important causes of pneumonia in American infants in Birmingham, Alabama.⁵ We performed a prospective study at Goroka Hospital in which we looked for serological evidence of infection with *C. trachomatis*, cytomegalovirus, *L. pneumophila*, *M. pneumoniae*, *P. carinii*, respiratory syncytial virus, and *U. urealyticum* in children with pneumonia.

Methods

We studied all children with pneumonia who were over 2 weeks old and were admitted to the paediatric ward of Goroka Hospital between mid-October 1981 and mid-January 1982. All the children had radiological evidence of consolidation. The study was approved by the Papua New Guinea Medical Research Advisory Committee. Blood was collected from each child on the day of admission and seven to 10 days later. Within two hours after collection the blood was centrifuged and the serum separated and stored at -20°C. Clinical details were not available to the participating laboratories at the time they performed their assays.

P. carinii antigen was detected in serum by counterimmunoelectrophoresis.^{6,7} Serum samples were treated by electrophoresis against antiserum prepared by inoculating rabbits with *P. carinii* grown in cell culture.⁸ IgG antibody to *P. carinii* was measured by a microenzyme linked immunosorbent assay.⁹ A 100 µl volume of acid solubilised *P. carinii* antigen suspended in carbonate buffer (pH 9.6) was bound to flat bottomed flexible microtitre plates (Dynatech Laboratories) by overnight incubation at 4°C. The plates were then rinsed with phosphate buffered saline containing Tween 20 at a dilution of 1/19 and allowed to dry. Each well was treated with 200 µl of blocking solution containing 10% bovine serum albumin in phosphate buffered saline containing Tween (Kirkegaard and Perry Laboratories), and the plates were tapped to remove excess liquid. Diluted specimens of serum were dropped into the wells and incubated for one hour at room temperature, and the plates were then rinsed with phosphate buffered saline containing Tween. Goat antihuman IgG specific to heavy and light chains

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and tagged with peroxidase (Kirkegaard and Perry Laboratories) was added to each well and allowed to incubate for one hour at room temperature. After being rinsed with phosphate buffered saline containing Tween 50 μ l of substrate consisting of ABST (2,2'-azino-di-(3-ethyl-benzthiazoline sulfonate (6))) in cacodylic acid buffer combined with equal parts of hydrogen peroxide was added to each plate. The plates were then incubated in the dark for 30 minutes at room temperature and were read at room temperature on an Abbott EIA microenzyme linked immunosorbent assay plate reader.

Antibody to *C trachomatis* was detected by a microimmunofluorescence test¹⁰ using elementary body antigens of each of the standard serotypes of the trachoma biovar, except Ba, and the L2 serotype of the lymphogranuloma biovar. Serum samples were screened at 1/10 dilution against pools of antigens using sheep antihuman immunoglobulins conjugated with fluorescein isothiocyanate (Wellcome, Australia). Serum samples with antibody to *C trachomatis* were then titrated in fourfold dilution steps against the appropriate single serotype antigens with fluorescein isothiocyanate conjugated goat antihuman Fc specific IgM, IgG, and IgA (Nordic Immunological Laboratories, Netherlands).¹¹

IgG and IgM antibodies to respiratory syncytial virus were detected using enzyme immunoassay.¹² A complement fixation test was used to detect antibodies against *M pneumoniae* and cytomegalovirus.¹³ A fourfold rise in titre was regarded as evidence of primary infection. IgM, IgG, and IgA antibodies to *U urealyticum* were measured by an enzyme immunoassay.¹⁴ An indirect immunofluorescence test was used to detect antibody to *L pneumophila*.¹⁵

Analysis of the data was performed with Statpro (Wadsworth Electronic Publishing, Boston, USA). The Mann-Whitney U test was used to test whether two groups had come from the same population; two proportions were compared by the χ^2 test, or by Fisher's exact test when any cell had a value of less than five. Weight for age was expressed as a percentage of the Harvard median.¹⁶

Results

Altogether 179 children were admitted to the study. Nine children died, 55 absconded from hospital before the second blood sample was taken, and 21 were discharged and failed to return to be bled a second time, leaving 94 children from whom paired serum samples were available. Age, weight, severity of illness, and duration of illness before admission were similar in the 94 children who remained in the study and the 85 children lost to follow up. The mean age of the 94 children was 10.6 months (median 8 months, range 1-72 months); 70 children were 1-12 months old, 21 were 13-24 months old, and only three were over 24 months. Sixty nine children were 80% or more of the Harvard median weight for their age, 15 were 70-79%, six were 60-69%, and three were less than 60%.

Serological evidence of infection with pneumocystis was found in 23 of the 94 children (25%), respiratory syncytial virus in 22 (23%), mycoplasma in five of 74 (7%), chlamydia in five of 91 (6%), cytomegalovirus in one of 74 (1%), ureaplasma in none of 94, and legionella in none of 94. No evidence of infection with any of these organisms was detected in 62 children (66%). Mixed infections occurred in 11 children: respiratory syncytial virus was found with either *P carinii* (six cases) or *C trachomatis* (three), and *M pneumoniae* was found with either *P carinii* (one case) or *C trachomatis* (one). Paired serum samples were obtained from three children who died: evidence of infection with *P carinii* was detected in one, *P carinii* and respiratory syncytial virus in one, and respiratory syncytial virus and *C trachomatis* in one.

TABLE I—Titres of *P carinii* antibody in children with and without *P carinii* antigen. (Figures are numbers of children)

	Titre					
	32	64	128	256	512	1024
Positive for antigen			1	7	12	3
Negative for antigen	1	2	9	5	4	2

P carinii antigen was detected by counterimmunoelectrophoresis in 23 of the 94 children. The geometric mean *P carinii* IgG antibody titre in these children was 427, while it was 201 in 23 children in whom *P carinii* antigen was not detected ($p < 0.01$, *t* test, 44 df) (table I). When compared with the 71 other children, children with *P carinii* antigenaemia had a faster pulse rate ($p < 0.05$) and respiratory rate ($p < 0.05$) and were more likely to be cyanosed ($p < 0.05$) or not feeding ($p < 0.1$).

Twenty two of the 94 children had evidence of recent infection with respiratory syncytial virus (IgM detected, or rising IgG titre); 12 had

evidence of infection in the past (no IgM, stationary IgG titre); 20 had antibody that was probably maternal (falling IgG titre); eight had low immunoassay absorbance values that were borderline negative; and 32 had no antibody in either sample of serum. The 22 children with evidence of infection with respiratory syncytial virus spent longer in hospital than the 72 other children ($p < 0.02$).

Chlamydia antibodies were detected in 38 of 91 children (no serum was available from one child, and the sera from two children destroyed the antigen spots). Five children probably had infection with *C trachomatis* at the time of the study: two had IgM and seroconverted in IgG, and three showed fourfold or greater rises in both IgM and IgG (the highest IgM antibody titre in each child was 40, 160, 160, 2560, and 640, and the highest IgG antibody titre was 2560, 40, 2560, 2560, and 160 respectively). Eleven children had evidence of possible current infection with *C trachomatis*, with low titres of IgM and little or no IgG antibody. Table II shows *C trachomatis* serotypes detected. When compared with the 89 other children the five children with probable infection with *C trachomatis* were younger (median ages 10.6 and 2.8 months respectively, $p < 0.02$) and were more likely to have severe intercostal retraction ($p < 0.1$).

TABLE II—Serotypes of *C trachomatis* antibodies detected in 91 children

	No of children	Serotypes
Probable recent infection	5	5 B/DE
Possible recent infection	11	1 A, 1 AB, 2 AC/J, 2 BD/E, 2 F/G, 1 H, 2 X-r*
No evidence of recent infection	22	1 A, 1 AB, 3 AC/J, 1 B, 3 BD/E, 1 C/J, 1 D/E, 2 F/G, 3 H, 1 J, 4 X-r*, 1 L ₂
No <i>C trachomatis</i> antibodies	53	

*X-r=Cross reactive.

Twenty children had anticomplementary activity in one of their paired serum samples. Five of the remaining 74 children showed a fourfold change in antibody to *M pneumoniae* and one a fourfold change in antibody to cytomegalovirus. Table III shows the highest titres of antibody to cytomegalovirus and *M pneumoniae* in each child. When compared with the 89 other children the five children with probable infection with *M pneumoniae* had a higher temperature ($p < 0.05$) and a lower white cell count ($p < 0.1$).

TABLE III—Highest titre of antibodies to cytomegalovirus and *M pneumoniae* found in the 94 children. (Figures are numbers of children)

	Titre					
	0	16	32	64	128	256
Cytomegalovirus	8	11	26	25	21	3
Mycoplasma	47	28	12	5	1	

Only two children had antibodies to *L pneumophila*; neither had evidence of recent infection. Six children had antibodies to *U urealyticum*: four had IgG and two IgA antibodies. Two of the children with specific IgG antibodies were 2 months old, and the antibody may have been maternal IgG. No child had IgM antibody against *U urealyticum*.

Discussion

This serological study may have underestimated the true prevalence of infection with all the organisms sought, with the exception of *P carinii*, as diagnosis depended on the child's ability to mount an antibody response. This particularly applies to the 38 children who were aged under 6 months or were malnourished (weight for age less than 70% of the Harvard median). Although all 179 children admitted to the hospital with pneumonia were entered into the study, a second blood sample was not obtained in 85 cases. Fortunately, it is unlikely that this was an important source of bias as these children appeared to be similar to the 94 children from whom paired serum samples were obtained.

Twenty three of the 94 children tested had *P carinii* antigen detected in their serum by counterimmunoelectrophoresis. These

children were more ill than those who did not have *P carinii* antigenaemia: more of them were cyanosed, and they were breathing faster and had faster pulse rates. Two of the three children in the study who died had *P carinii* antigenaemia. The children with *P carinii* antigenaemia had a substantially higher titre of antibodies to *P carinii* than those without. Stagno *et al* reported that 19 of 104 North American infants with pneumonia had *P carinii* antigen detected by counterimmunoelectrophoresis and suggested that pneumonia due to *P carinii* may be quite common, even in infants with no underlying immune defects.⁵ In our study the nutritional state of the 23 children with *P carinii* antigenaemia (median weight for age 85%) was no worse than that of the 71 other children (median weight for age 88%).

As we did not do lung biopsies we cannot say how many of the children in our study actually had pneumonia due to *P carinii*. The *P carinii* antigen that we detected might merely have been released from lung tissue that had been damaged by other pathogens, although the clinical picture in these children was typical of that of pneumonia due to *P carinii*, with tachypnoea, tachycardia, and cyanosis without severe changes in chest radiographs. None of 184 normal American children and only six of 208 normal American adults had *P carinii* antigen detected by counter-immunoelectrophoresis, but 10% of 83 patients with pulmonary infection, non-pulmonary infection, or non-malignant lung disease had antigenaemia; these may have been false positive results, or they may have represented colonisation or subacute infection with *P carinii*.

Twenty two children had evidence of recent infection with respiratory syncytial virus, and 12 other children had evidence of past infection. Although the children with recent infection were clinically no more ill than the others, they stayed in hospital longer, and two of the three children who died had infection with respiratory syncytial virus. Respiratory syncytial virus was often associated with consolidation in the chest radiograph, whereas bronchiolitis, with a wheeze and hyperinflation, was noted in only one of the 22 children. The proportion of children in the hospital with IgG specific to respiratory syncytial virus was substantially lower than that in a group of children in Melbourne, Australia, tested with the same enzyme immunoassay.¹² Only 15 (71%) of the 21 children in our hospital aged under 3 months had specific IgG compared with 41 (91%) of 45 Melbourne children ($p < 0.05$); and 39 (53%) of 73 of our children aged over 3 months had specific IgG compared with 50 (94%) of 53 Melbourne children ($p < 0.001$).

Beem and Saxon first reported that *C trachomatis* was a cause of pneumonia in infants,¹⁷ and this finding has been confirmed by several other studies.¹⁸ Five children had strong serological evidence of recent infection with *C trachomatis* at the time of our study; all were aged 1-6 months, and all had antibodies to the B/DE group. Eleven other children had equivocal evidence of current infection with *C trachomatis* (table II). Thirty eight of the 91 children tested had antibodies to *C trachomatis*, suggesting that infection with this organism is common among children in Papua New Guinea. Serotypes A-C cause trachoma; serotypes D-K cause genital infections, conjunctivitis, and pneumonia in infants; and serotypes L1-L3 cause lymphogranuloma venereum. The serotypes were determined in 37 children in the present study: 17 had antibody to the genital strains that cause pneumonia in infants (table II).

In developed countries pneumonia due to *M pneumoniae* is rare in children under 2.¹⁹ The five children in our study who showed a fourfold rise in antibodies to *M pneumoniae*, however, were aged between 6 months and 2 years. These children had a significantly higher temperature than the 89 other children in the study; high fever is common in pneumonia due to *M pneumoniae*.¹⁹ Forty six of the 93 children tested had antibody to *M pneumoniae* (table III), which suggests that infection with this organism is common among young children in Papua New Guinea.

Eighty five of the 93 children tested had antibody to cytomegalovirus, and 48 had a titre of 1/64 or more. Although only one child had a fourfold rise in titre, this does not exclude cytomegalovirus as a common cause of pneumonia in these children as the paired serum samples were taken only seven to 10 days apart and it takes many weeks for cytomegalovirus antibody to rise.²⁰ We did not attempt to culture cytomegalovirus in this study, and we did not study

controls. It has been suggested that cytomegalovirus is an important cause of pneumonia in infants in the United States, often in association with other agents.^{5, 21, 22} The high antibody titres found in our study suggest that cytomegalovirus may be a direct cause of pneumonia in children in Papua New Guinea; it may also predispose children to pneumonia caused by other agents as infection with cytomegalovirus is known to cause immunosuppression.²³

No child had IgM antibody against *U urealyticum*, which suggests that none had active infection at the time of the study. As culture of nasopharyngeal secretions has suggested that *U urealyticum* may cause pneumonia in infants in the United States⁵ it is unfortunate that we were unable to culture nasopharyngeal aspirates from our children. *L pneumophila* is a rare cause of pneumonia in North American children, and most children who seroconvert are asymptomatic.^{24, 25} Only two of the 94 children in our study had antibody to *L pneumophila*, and the titre did not change in either child.

H influenzae, *Str pneumoniae*, *Staphylococcus aureus*, and tuberculosis are important causes of severe pneumonia in children in developing countries.^{4, 26} This study suggests that *P carinii*, respiratory syncytial virus, *C trachomatis*, *M pneumoniae*, and perhaps cytomegalovirus also cause pneumonia in children in developing countries. In Papua New Guinea children with mild pneumonia are treated with procaine or benzylpenicillin and those with severe pneumonia are treated with chloramphenicol.²⁷ Chloramphenicol is active against most strains of *H influenzae*, *Str pneumoniae*, and *Staph aureus*, and although clinical experience is limited, *M pneumoniae* is often sensitive to chloramphenicol in vitro.²⁸

Although most children with severe pneumonia respond to treatment with chloramphenicol, a few have persistent cough and tachypnoea. In the past these children were often assumed to have viral pneumonia or tuberculosis, and antituberculous treatment was started; we suggest that, though some of them will indeed have viral pneumonia or tuberculosis, others may have infection with *P carinii* or *C trachomatis*. Co-trimoxazole should perhaps be tried before antituberculous treatment is given in children who do not respond to chloramphenicol because, unlike chloramphenicol, co-trimoxazole is active against *P carinii* and *C trachomatis*. High doses of co-trimoxazole are needed to treat pneumonia due to *P carinii*—that is, at least 5 mg trimethoprim/kg and 25 mg sulphamethoxazole/kg every six hours. Our findings lend support to the World Health Organisation's plans to promote the use of co-trimoxazole for pneumonia in children in developing countries.²⁹

This study suggests that, in addition to *H influenzae* and *Str pneumoniae*,⁴ *P carinii*, respiratory syncytial virus, *C trachomatis*, *M pneumoniae*, and perhaps cytomegalovirus are important causes of pneumonia in children in developing countries. Prospective studies should be performed in several developing countries using culture techniques, as well as serological examination, to detect these organisms in children with pneumonia and in controls matched for age.

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MATERIA NON MEDICA

Anatomy of horror

Staring at me as I sit at my desk is a bony skull. Despite the passage of nine years in an anatomy department, and a special respect and affection for head and neck anatomy, I can still recall the time when I associated such objects primarily with horror films. I mean, of course, not the modern "nasties," but the classics of the past and present: films which rely more for their effect on the viewer's imagination than on offensiveness. The resurgence of the British gothic cinema in the 1950s and 1960s, largely due to Hammer, was something of a mini phenomenon and the first two Hammer *Dracula* films are very good indeed, especially the resurrection scene in *Dracula*, *Prince of Darkness* where the victim's blood imparts life to the ashes of the Count. Religious symbolism in these films is never far from the surface and often quite explicit; so, too, is eroticism, especially in the vampire films.

Anatomists have been likened to vampires, particularly in *viva voce* examinations; I cannot deny that such were my thoughts on one occasion in 1969. Horror films and anatomy meet most obviously with the Edinburgh resurrectionists, and their activities call to mind the Frankenstein story. This evolved partly, I suppose, from the eighteenth and nineteenth century predilection in art and architecture for the cult of the sublime, as witnessed by the many follies and specially built ruins which are scattered about country estates.

There are also many memorable moments in the later films. *Frankenstein Created Woman* has a wench with a naevus on the right side of her face and so, we assume, a right sided brain lesion and a left sided motor loss. Not so: Hammer knows nothing of decussations, and so the poor girl has an ipsilateral motor loss. A remark to savour comes from *I Was A Teenage Frankenstein*: "I know you have a civil tongue in your head: I sewed it there myself." And how on earth do Peter Cushing *et al* manage in brain transplants with all those cranial nerves? When you've seen mortuary technicians (or, for that matter, anatomy lecturers) hack out a brain from the cranial cavity, you can appreciate some of the problems solved by Dr Frankenstein.

Not only are there plenty of references in horror films to organs of the anatomical kind, but also to those of the musical kind. Vincent Price has been seated at his organ in at least four films, and he is by no means the only maniac organist on film. That is another story, though, and there is scope for another thesis or, at very least, another *Materia non Medica*.—STANLEY MONKHOUSE, anatomist, Nottingham.

In like a lion and out like a lamb

In 1926 when the coal strike was at its height I had a sudden telephone call from one of my former professors. This was to see if I could go at once to England to take over an RSO job in St Helens, Lancashire. The matter was urgent because the Resident Surgical Officer in post must return home at once—his brother had died, and the practice and the dispensary attached to it must be filled without delay.

The professor knew that I was unemployed. I had just passed the first part of the FRCS and had been unsuccessful in getting any house job in England although I had applied for over 20 such posts—I was part of the postwar 1 bulge. It is good for the boys today to see that even in those days we also had our problems.

I agreed to go at once. I packed my bag with a few clothes and a lot of

books, hoping to work for the final Fellowship. I got the overnight boat to Liverpool, but what I did not know was that at midnight a general strike had been declared. At 7 am when I disembarked at the Liverpool docks there was no transport of any sort, so here I was, a small man with a big bag, large and heavy, faced with a journey on foot of 20 miles. The position seemed hopeless until I discovered a small greengrocer's cart loaded to the top with bags of potatoes, carrots, sprouts, etc with a pony between the shafts and a rather unshaven gentleman in charge, but what did matter was that there was a small plate on one of the shafts which said "John Gribbon, Green Grocer, St Helens." I approached him and with great deference suggested that for £2 perhaps I might sit on one of the bags of potatoes as far as the hospital at St Helens. After a small monetary adjustment had taken place and when £3 had changed hands, I mounted the cart with my luggage and made my state entry into my job on a bag of spuds. I was told later that it was the first time that an RSO had arrived in such style.

As the miners' strike was the daily topic, and A J Cooke was the Scargill of that time, I decided that I should go to one of his meetings. It took place on the local Rugby League football pitch. About 1000 people were there. I went in an old suit not to be conspicuous—this was not difficult as it was my only one. All went well and I mingled with the miners unnoticed until something happened that I had not foreseen. In those days miners did not stand, they hunkered down on their heels. They often did this in the mines working at the coal face and so as the crowd got ready for the speeches they all hunkered down, reducing the overall height to about half. I naturally did so with the others, but I did not realise that after 15 minutes of this I could not stand it any longer, and so the inevitable took place: I had to stand up and became the only visible figure in this vast audience—the very thing I had wanted to avoid. I had great difficulty escaping with nothing worse en route than being cursed both in English, which I did understand, and in Welsh, which I did not.

Looking back on it, I suppose although it was worrying to a young man at that time it was very much more peaceful than what we have seen recently.—IAN FRASER, Belfast.

Is it true that women who shave their legs are likely to have an increase in the number and strength of hairs on legs and that those who remove hairs from legs with heated wax will not have to continue the process because each application diminishes the hair growth?

Shaving the legs is a quick, cheap, and effective method of depilation, although some women object to the initial stubbly appearance of the regrowing hair. Nevertheless, shaving of the legs has no effect on the rate of hair growth or on the diameter of the hair shaft.¹ Waxing of the legs plucks out the hair from the root and therefore its effect may last for up to six weeks before repeat treatment is required. As the hair has to be moderately long before waxing may be performed application of a bleaching agent in between treatments may be helpful for dark haired women.² Waxing does not destroy the follicle and therefore will not result in permanent depilation.—E C BENTON, senior registrar, Edinburgh.

1 Lynfield YL, MacWilliams P. Shaving and hair growth. *J Invest Dermatol* 1970;55:170-2.

2 Rentoul JR, Aitken AA. The cosmetic treatment of hirsutism. *Practitioner* 1980;224:1171-5.