Acute Chest Infections in Children
1–5 Years of Age

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The CARPA treatment guidelines are partly based on WHO guidelines aimed at children in developing countries.[1,2] However, they have significant differences to the WHO algorithms because of local circumstances. In particular the WHO guidelines are primarily aimed at preventing death. In Central Australia mortality rates are much lower than in developing countries and efficient evacuation is usually possible for sick children.

Most children with acute chest symptoms will have infection with bacteria, virus or sometimes both.

The most important decisions are:

1. Which children are likely to have bacterial infection and therefore need antibiotics?
2. Which children are sick enough to need evacuation?

The international literature suggests that S. pneumoniae is the most common pathogen in childhood pneumonia, but that H. influenzae is also important.[3,4] It is very likely that S. pneumoniae is the major cause of pneumonia in Central Australian children. A study of invasive pneumococcal disease in Central Australia has shown that children in this region have the highest rates of this disease in the world literature.[5]

A study of the aetiology of pneumonia in hospitalised children in Alice Springs supports a prominent role for S. pneumoniae, but also demonstrates that nearly half these children had evidence of viral infection, and co-infection may also be common.[6]

Research in developing countries has shown that in settings where X–ray is not available respiratory rate is the best clinical predictor of pneumonia.[7] A major problem with this respiratory rate ‘cut-off’ is that it lacks specificity and will lead to over-diagnosis of bacterial pneumonia, and hence increased use of antibiotics in children who do not have bacterial infection.[7,8] This problem is likely to be worse in populations where viral infection and wheeze syndromes are common. However, this approach provides the safest option for managing these children because there are no clinical signs which can exclude the presence of bacterial infection. Studies to determine the optimum cut-off respiratory rate demonstrate that increasing the cut-off increases the specificity but lowers sensitivity. In children one and up to five years a respiratory rate of 40 or more provides a

sensitivity of 71 to 87 per cent and a specificity of 72 to 85 per cent.[9,10,11,12]

Studies of lower chest wall indrawing (defined as inward movement of the lower chest wall on inspiration) have shown it to be a predictor of severe pneumonia.[7] However, the sign must depend on compliance of chest wall, degree of airway obstruction and consequent generation of intra-thoracic pressure swings. Predictably, chest indrawing can be seen in conditions such as bronchiolitis which may be due to viral rather than bacterial infection. Therefore, its value as a predictor of severity in pneumonia will vary with the incidence of severe bronchiolitis in the population being serviced.

Wheeze can occur in children with bacterial pneumonia and bacterial co-infection can occur in Respiratory Syncytial Virus and other viral-related bronchiolitis syndromes.[13,14] Therefore, it is not possible to distinguish viral bronchiolitis from pneumonia on clinical signs alone.

Fever is an obvious candidate as a predictor of infection requiring antibiotic treatment. However, it has not been shown to reliably distinguish between viral and bacterial infection. In a study of 90 infants presenting with bronchiolitis, El–Radhi[15] found that fever was more likely to be present in those with severe disease and in those with radiological evidence of pneumonia. This study identified fever, defined as a single reading >38.0°C or two consecutive readings of >37.8°C, in 28 children (31%). The children who presented with fever had a longer hospital stay and a more severe illness than other children. Weber et al.[16], in a study of bronchiolitis in The Gambia, found that the mean temperature of children with a positive blood culture was significantly higher than those with a negative blood culture. However, Cherian et al.[17] found no clinical predictors of X–ray consolidation in 114 Indian children with bronchiolitis.

Benzyl penicillin, procaine penicillin and amoxicillin all have activity against both S. pneumoniae (SP) and H. influenzae (HI). There is some evidence that, at least for SP, achieving blood levels above the mean inhibitory concentration (MIC) for significant periods of time is important in antibiotic efficacy[18], although at least one important clinical trial has failed to demonstrate this association.[19] Both benzyl penicillin and procaine penicillin given intra-muscularly will achieve serum levels greater than the MIC for pneumococci even with intermediate level resistance (0.1 to 1.0 µg/ml).[20] Benzathine penicillin and other long-acting penicillins achieve only very low serum levels and should not be used for pneumonia. They are likely to be ineffective and to facilitate resistance.[20]

The effectiveness of oral amoxicillin in the Central Australian setting is not clear. Some recent studies from Pakistan provide some basis for decision making. These studies are all in non–severe pneumonia i.e. children who do not have chest indrawing or any other signs of severity. Oral amoxicillin given three times a day in a dose of 15 mg per kg is effective[19] in childhood pneumonia although failure rates in recent studies have been in the order of 16 per cent.[21] In addition compliance with three times a day oral therapy is obviously problematic.

A recent study has shown that a three day course of three times a day amoxicillin is as effective as five days.[22] Twice daily amoxicillin is as effective as twice daily cotrimoxazole but with a failure rate of over 16%.[21] Currently studies are underway to determine if increasing the dose of Amoxicillin will decrease failure rate. The high rate of chronic lung disease in Central Australian Aboriginal children[23] raises further concerns about the impact of duration of therapy on long-term outcome. On the basis of existing evidence it would appear that oral therapy should not be considered first-line treatment option. If the health care worker does not consider parenteral therapy possible, then amoxicillin in a dose of 25–30 mg/Kg per dose two or three times daily (depending on the health care worker's estimate of compliance) is a reasonable second-line therapy option.

There are many issues to consider in the treatment of fever in young children. These issues are extensively reviewed elsewhere.[24,25]

In summary, fever may be beneficial to the host with infection. Animal studies show that moderate fever improves immune response. A series of animal studies reviewed by Shann[25] demonstrate a worse outcome in febrile animals treated with paracetamol or aspirin compared to placebo. Except in children with cardiac or respiratory failure, fever is not harmful unless greater than 41°C. Febrile convulsions are thought to occur in response to an initial rapid rise in temperature and so paracetamol is unlikely to be beneficial. The only relevant clinical trials suggest no benefit with the use of paracetamol in preventing convulsions. Finally, paracetamol has been shown to have only a modest effect in reducing distress and discomfort associated with infection.

Overall, very little toxicity has been associated with paracetamol therapy for fever in children. Thus, although anti-pyretic treatment seems relatively safe in the setting of acute respiratory infection, it is reasonable to restrict its use to children with axillary temperature over 38.0°C.[21]

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