

Correspondence

Aetiology of severe pneumonia in developing countries

Sir,

Dr Ikeogu suggests, on the basis of his lung aspirate study of 40 children hospitalised in Bulawayo, that bacterial infection is not the commonest cause of severe pneumonia in children in Zimbabwe.¹ This contrasts with published data from at least 13 lung aspirate studies from developing countries showing the importance of bacterial infections, in hospitalised children with severe (predominantly lobar) pneumonia. While geographical differences undoubtedly exist there are remarkable similarities in the results from these studies. The statement that 'negative bacterial growth from lung aspirates implies acute viral infection in most cases' should be read with caution. Bullova's study of 211 bacteraemic pneumococcal pneumonias has suggested that lung aspiration has a false negative rate of at least 22%.² Studies where viral cultures, immunofluorescence, or serology have been performed, together with lung aspiration or blood culture in children with severe pneumonia, have found evidence of viral infection only in approximately 30%. If a genuinely low bacterial isolation rate in Bulawayo is to be established convincingly, serum or urine should be tested for antibacterial activity, as history of recent antibiotic treatment can often be unreliable.²

Intervention trials from six countries have recently shown that improved case management with antibiotics may substantially reduce mortality from acute respiratory infections.³ This strategy is not necessarily one of increasing antibiotic prescription and hence escalating costs. Although viral and bacterial pneumonias cannot be differentiated reliably by clinical or radiological findings, simple clinical signs can be used to distinguish lower from upper respiratory infections thus allowing antibiotic delivery to be directed to the former group and potentially reducing antibiotic use in the community.⁴

We are concerned that the low mortality in this series suggests that these patients may not be typical of the majority of children in Zimbabwe and that the epidemic nature of respiratory infections requires that the minimum study period be one year (invasive pneumococcal disease in The Gambia appears to be noticeably seasonal). Furthermore, although Dr Ikeogu emphasises supportive care methods, it should be recognised that these are not yet proved to be effective in reducing mortality from pneumonia in developing countries. The weight of currently available evidence supports early antibiotic treatment as the primary intervention for all severe pneumonia in developing countries.

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Dr Ikeogu comments:

Drs Campbell and Forgie raise a number of issues regarding my article on acute pneumonia.¹ Space does not allow me to discuss all the points raised. They express surprise about the low mortality in the patients in the series. In our unit a lot of emphasis is placed on supportive care even in those with bacterial pneumonia. The supportive care principles are based on simple pathophysiological considerations but mechanical ventilation is used very infrequently in our unit and none of the 40 children was ventilated. The argument here is not distinguishing upper from lower respiratory infection but determining the aetiology of acute lower respiratory infection and therefore deciding when to use antibiotics. I consider the use of the suggested simple physical signs⁴ in arriving at this decision as an oversimplification, which is sometimes useful in situations where there are no doctors or paediatricians or facilities for appropriate investigations but which also, in my experience, often results in delayed referral to hospital often with disastrous consequences.

Also not treating upper respiratory infections because of absence of such simple signs as Drs Campbell and Forgie seem to suggest is dangerous in those areas with a high incidence of rheumatic fever. The difficulties in trying to simplify the management of respiratory infection in children are obvious and nothing can substitute experience backed up by appropriate investigations.

The purpose of my article was to indicate the possibility of regional differences in the patterns of pneumonia in developing countries and by so doing stimulate more investigations. Drs Campbell and Forgie agree with me that geographical differences and similarities do exist, a point reinforced by the absence of a single positive blood culture in any of the patients (this point was not mentioned in the article). The period of investigation included the peak period for respiratory infections and the population studied had no access to undocumented antibiotic exposure.

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- ⁴ Cherian T, Jacob John T, Simoes E, Steinhoff MC, John M. Evaluation of simple clinical signs for the diagnosis of acute lower respiratory infections. *Lancet* 1988;**ii**:125–8.

Seizures presenting as apnoea

Sir,

Investigations of the infant with a seizure disorder presented by Navelet *et al* do not provide adequate data to state that the

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recurrent episodes of intense cyanosis were due to seizure induced airway obstruction or a seizure induced prolonged absence of inspiratory efforts.¹ We accept that seizures may cause prolonged absence of breathing effort and hypoxaemia and have previously described one patient with this problem.² In our patient, however, the apnoea was present for more than 50 seconds before severe hypoxaemia became apparent. In situations of complete airway obstruction due to imposed apnoea the onset of sufficient hypoxaemia to produce cyanosis does not appear until after 60 seconds of absent airflow.³ In our opinion, it is more likely that the primary cause of the sudden onset and rapid progression of hypoxaemia was intrapulmonary shunting and prolonged expiratory apnoea.⁴ This may have triggered the seizure shown in the recording or may have resulted from the seizure. In order to answer this, it would be required to document the temporal association between the onset of hypoxaemia and development of seizure activity.

The onset of cyanotic episodes with meals or pain, the lack of response to antacids, metoclopramide, atropine, and phenobarbitone and the early onset and intensity of cyanosis would be typical of a sudden intrapulmonary shunt and prolonged expiratory apnoea.⁴ These latter episodes are induced in infants under 6 months typically by feeding or painful or stressful stimuli, and the resulting seizure is secondary to the hypoxaemia.

It is also possible that cerebral injury resulting from severe hypoxaemia early in life may have produced a focus for the subsequent development of epilepsy.

We suggest that all polygraphic monitoring during cyanotic episodes should include measurements of oxygenation, most reliably sensed by a pulse oximeter in a beat-to-beat mode.

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Range of neural tube defects in southern India

Sir,

The article on neural tube defects in southern India by Kulkarni *et al* mentions 'widespread prenatal screening and

termination of affected pregnancies' as contributing towards the downward trend of these defects in Western countries including the United States and Scotland.¹ However, the Report of the Chief Medical Officer for England and Wales in 1985 states that 'screening and subsequent elective abortion has not been a major cause of the decline'.² A similar decline has occurred in the Republic of Ireland, where abortion is not carried out.³

References

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Breath hydrogen excretion in infants with colic

Sir,

In some babies with troublesome crying the features of intense sympathetic activity after a prolonged screaming episode of, say, 30 minutes are very striking. These babies have a pronounced tachycardia, sweat, and become red in the face. Is it possible that the work of crying results in a shortened intestinal transit time with increased delivery of lactose to the colon reflected in high breath hydrogen excretion as Miller and colleagues have demonstrated?¹ Another observation is that babies with troublesome crying very often stop crying after hospital admission, often within 24-48 hours, with no alteration to their diet.² It would be very interesting to know if the hydrogen breath excretion dropped over this same short period. This may help to distinguish whether the abnormal results of Miller *et al* reflect a primary gut abnormality or a secondary phenomenon. A study to examine this may be easier to design than one to examine the effect of altering the lactose content of the diet.

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