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Correspondence

The State of the World's Children

Sir,

We were interested to read in David Morley's recent annotation 'The State of the World's Children' that in the Gambia there is a 10% mortality in the subsequent nine months after infection with measles, and this compares with only 1% in controls.[1]

Our own interest in the possible predisposition to subsequent death from measles infection was aroused by two deaths within 24 hours from meningococcaemia, which occurred in this hospital earlier this year. Both children were girls aged 2 years, and one had had measles eight weeks before her death and the other six weeks before.

Meningococcal infection is an important cause of morbidity and mortality in childhood, causing 2% of all deaths between 1 and 4 years in the United Kingdom. It has been suggested that host factors—for example, familial complement deficiency[2]—may be operative in some cases of fulminating disease, but in most cases no such factors are recognised and immunosuppression is a well known associate with measles. Niwa *et al* have recently shown that suppressor T lymphocytes from patients with measles can significantly depress oxygen radical generation by normal polymorphs.[3] This effect was shown to last for two months and was more pronounced in patients with secondary bacterial complications. There has been speculation that other viral infections may predispose to infection with *Neisseria meningitidis* as Young *et al* reported a simultaneous outbreak of influenza and meningococcal infection in Mississippi where there was a significant association between serologic evidence of influenza and systemic infection with or nasopharyngeal carriage of *Neisseria meningitidis*.[4]

The figures from the Gambia would seem to support further the essential inclusion of measles immunisation in any population immunisation programme, and we would be interested in any other instances of fatal meningococcal infection with preceding measles, because although both measles and meningococcal infection are both common in children, particularly in the first years of life, demonstration of a possible association between the two would provide further support for an effort to promote measles immunisation in the United Kingdom.

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References

[1] Morley D. The state of the world's children. *Arch Dis Child* 1985;**60**:693–4.
[2] Nicholson A, Lepow IH. Host defence against Neisseria meningitidis requires a complement-dependent bactericidal activity. *Science* 1979;**205**:298–9.
[3] Niwa Y, Sakane T, Somiya K, *et al*. Decreased oxygen radical generation by neutrophils from patients with measles, presumably owing to activation of suppressor T lymphocytes. *J Clin Microbiol* 1985;**21**:318–22.
[4] Young LS, La Force FM, Head JJ, *et al*. A simultaneous outbreak of meningococcal and influenzal infections. *N Engl J Med* 1972;**287**:5–9.

Small bowel biopsy

Sir,

I read with interest the Personal practice concerning small bowel biopsy of Dr Collins and colleagues.[1] I would concur that semi-rigid catheters are superior to the very soft tubing supplied with some capsules. Indeed we have found that metal braided angiocardiographic catheters are superior to KIFA catheter tubing, which does not satisfactorily transmit torque and has a tendency to coil in the stomach. We have also found it helpful to inject some air into the stomach through the capsule and lie the child on the right side so that the capsule falls into the antral region.

In view of the extrapyramidal signs sometimes associated with metoclopramide, perhaps this should not be used routinely?

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Drs Rolles and Collins comment:

We agree with Dr Shaw that the rigidity of the capsule tubing is of major importance and shall try the catheters he suggests with interest. As mentioned in our article, we always lie the child on the right side and have also found the injection of air to be useful if there is delay in the capsule passing through the pylorus.

With a combined experience of around 1500 biopsies we have never had difficulties with the use of metoclopramide as described. On those occasions when it was omitted or when intubation was unduly delayed after giving the metoclopramide the biopsy procedure was prolonged.

Reference

[1] Collins AL, Brookfield DSK, Hyde I, Rolles CJ. Small bowel biopsy. *Arch Dis Child* 1985;**60**:1082–5.

Early congenital syphilis still occurs

Sir,

We read with interest the article on the presentation of early congenital syphilis by Ewing *et al*.[1] Of the seven

infants described, none seems to have had pneumonia or pneumonitis.

We have recently had occasion to treat a newborn whose main presenting complaint was respiratory failure and pneumonia.

Case history

A girl weighing 2290 g was born to a 21 year old single primigravida mother in a general practitioner maternity unit outside Belfast. The mother had not booked for delivery and had received no antenatal care. The baby was believed to be 31 weeks' gestation and was transferred to a nearby district general hospital for treatment of respiratory distress. On arrival there blood gas analysis showed pH 6·88, arterial carbon dioxide tension 11·7 kPa (89 mmHg), arterial partial pressure of oxygen 5·5 kPa (42 mmHg), bicarbonate 8·6 mmol/l, and base excess −18·7. The baby was immediately intubated and transferred to Royal Maternity Hospital, Belfast, for mechanical ventilation.

At 4 hours of age the baby was examined at the Regional Neonatal Intensive Care Unit and found to have pronounced hepatosplenomegaly with liver 4 cm and spleen 5 cm below the costal margins. In addition, the hands and feet showed reddish-purple blotchy areas with white patches. Estimated getational age on clinical appearance was 34 weeks. Chest radiograph showed extensive patchy opacity throughout both lungs, most pronounced in the upper zones. The dorsal spine was 79 mm in length, corresponding with a gestational age of 35 weeks. Other investigations showed haemoglobin 123 g/l, white cell count $23{\cdot}3\times10^9$/l, and the platelet count 82×10^9/l.

A diagnosis of congenital pneumonia was made, and it was believed that the likely cause was either syphilis or group B β haemolytic streptococcus. The baby was treated with a high dose intravenous penicillin and made a very satisfactory recovery. All bacterial cultures subsequently proved negative. Serology results were Venereal Disease Research Laboratory test negative, *Ireponema pallidum* haemagglutination test positive in titre >1280, and fluorescent treponemal antibody (absorbed) test positive. At follow up one year after birth the baby was normal.

We believe that it is important to add congenital pneumonia or pneumonitis to the list of presenting features of early congenital syphilis given by Ewing *et al.*[1] We agree entirely that the presence of hepatosplenomegaly and rash, especially if confined to the palms and soles, is very suggestive of a diagnosis of congenital syphilis.

Reference

[1] Ewing CI, Roberts C, Davidson DC, Arya OP. Early congenital syphilis still occurs. *Arch Dis Child* 1985;**60**:1128–34.

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Cot deaths

Sir,

I wish to bring to the attention of your readers the very positive role that our local coroner plays in helping mothers whose babies have died of a 'cot death'.

Most mothers feel an enormous sense of guilt or failure, or both, when their baby dies in this manner, and it is often very difficult to remove such feelings.

Our local coroner writes a letter to the parents in every case of an uncomplicated 'cot death', and, apart from explaining very briefly the nature of a 'cot death' and enclosing literature about the Local Cot Death Support Group, he also takes care to include the words 'the pathologist found your baby to be in good physical condition and a credit to you'.

All the mothers who have received such a letter say that they find that particular sentence particularly reassuring, and indeed many keep the letter for many years and refer back to it from time to time. It is, they say, 'official' recognition of the fact that they were not to blame, and it is a great help in coming to terms with the death.

Might I suggest that paediatricians contact their local coroner to see if such a letter could be sent to all parents whose babies have died from an uncomplicated 'cot death'?

S E Barnes
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