

## LETTERS TO THE EDITOR

### The extended role of the new school doctor

SIR,—With the movement of preschool preventive medicine towards primary care, the focus in community child health services is changing towards school age children. We believe that the school health service is an underutilised yet potentially powerful child health resource and put forward the following views for discussion.

### Children's health in schools

The emphasis in school health services should be moved away from primary care medicine for all schoolchildren towards the development of the science of (re)habilitation for those with *special needs* (including support for the psychosocially vulnerable). The new school doctor should not only be well versed in the medical details of conditions causing chronic health impairment in childhood, but also be familiar with the theoretical and practical concepts underlying handicap and developmental disability (for example, the sociology of poverty and stigma, methods of measuring handicap, and the physiological basis of physiotherapy).

A further necessary part of this individual 'clinical' role of the new school doctor is the *early identification* of children with special needs (and the generation of data to audit this early identification process).

### Healthy schools for children

#### Healthy environments

The school doctor has a 'public health' responsibility for children at school. Why for instance do schools not keep a register of accidental injuries occurring among their children? Not a book in the secretary's office for legal purposes, but an educational exercise to discover the patterns and causes of injury, whether they occur in or out of school. Schools may be an ideal medium for the conduct of a campaign for better road crossings and safer play areas. These campaigns would involve a number of disciplines both in and out of the school and could be based on hard local data collected by the school.

#### Health(y) education

A great deal of effort is already invested in schools in more or less explicit health education but what do we know about the results? Our new school doctor should be working with the school nursing service on a continuously updated survey on smoking, diet, alcohol/drug abuse, and accident related behaviours for the school population. These are legitimate measures of local 'positive' child health and may provide vital feedback into curriculum development.

These initiatives in extending the habilitation and health promotion roles of the school health service will require new skills from our school doctors. Not only will they need to

extend their knowledge of basic sciences (epidemiology, statistics, sociology as well as developmental paediatrics) but also they will require insight into organisational structures, together with communication and assertiveness training to help fulfil their true position as public health advocates for school children.\*

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\*This paper is based on a discussion document used in the design of a new MSc in Community Child Health now established in Newcastle.

### Iron supplementation in the preterm or low birthweight infant

SIR,—In September of 1988 we carried out a similar survey to that of Barclay *et al.*,<sup>1</sup> by telephone, contacting 26 British neonatal units with more than four intensive care cots. It appears that the two surveys have similar results: the statistical modes for the dose of iron supplement, the time of initiation, and duration of treatment are identical. However, we reached a different conclusion. In view of the recommended supplement given by the European Society for Paediatric Gastroenterology and Nutrition or the American Academy of Pediatrics,<sup>2</sup> the great majority of neonatal units oversupplement their patients. Although there is no reported difficulty associated with these larger oral supplements, if taken in conjunction with transfused iron, it is conceivable that the raised iron load will increase susceptibility to infection, and potentiate other pathologies via free radical mechanisms in a small or premature neonate.<sup>3</sup> Therefore on the basis of *primum, non nocere* we concluded that iron supplements should be reduced to the level of current recommendations, and the consequences carefully monitored.

In our survey we further identified another complication associated with iron supplements: in two units iron and phosphate supplements were given to patients during the same drug round. This combination produces an insoluble complex, rendering both supplements ineffective.

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- 1 Barclay SM, Lloyd DJ, Duffy P, Aggett PJ. Iron supplements for preterm or low birthweight infants. *Arch Dis Child* 1989;64:1621-2.
- 2 Wharton BA (chair). *Nutrition and feeding of preterm infants*. Oxford: Blackwells, 1987.
- 3 Herschko C, Peto TA, Weatherall DJ. Iron and infection. *Br Med J* 1988;296:660-4.

### Arteriovenous malformations in the vein of Galen

SIR,—We enjoyed reading the annotation on arteriovenous malformations of the vein of

Galen by Dr Nicholson *et al.*<sup>1</sup> In the annotation reference was made to our case report of a newborn with a pattern I lesion who had her severe congestive heart failure treated by embolisation of the malformation using Gianturco coils.<sup>2</sup> Nicholson *et al.* suggested this procedure is a useful interim measure allowing elective surgery to be carried out when the child is older and fitter.

In our case, however, we reported a successful outcome to the age of 21 months with normal growth and development.<sup>2</sup> Indeed, our little girl is now 4.3 years old and appears perfectly normal, without any additional procedures having been necessary.

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- 1 Nicholson AA, Hourihan MD, Hayward C. Arteriovenous malformations involving the vein of Galen. *Arch Dis Child* 1989;64:1653-5.
- 2 McCord FB, Shields MB, McNeil A, Halliday HL, McCure G, Reid MMc. Cerebral arteriovenous malformation in the neonate: treatment by embolisation. *Arch Dis Child* 1987;62:1273-5.

### Increases in plasma concentrations of a prostaglandin metabolite in acute airway obstruction

SIR,—Skoner *et al.* elegantly demonstrated raised plasma concentrations of a prostaglandin metabolite during and after recovery from acute airway obstruction in infants.<sup>1</sup> They suggested that prostaglandin F<sub>2α</sub> is involved in airway obstruction and therefore encourage trials of specific anti-inflammatory agents for the treatment of airway obstruction.

There are, however, some disturbing data in the article that should preclude assuming any causal relationship between prostaglandins and airway obstruction in these studied infants. The authors have not actually found any difference in prostaglandin concentrations between infants studied before treatment and those studied shortly after initial treatment. They have not demonstrated such a difference even in the few infants whose prostaglandin concentrations were measured before and shortly after airway obstruction (their fig). It is also not clear from the data whether group II (infants after treatment) had their airway obstruction resolved at the time of blood collection.

In order for an appropriate correlation between prostaglandin plasma concentrations and airway obstruction, more objective parameters (lung function tests) need to be studied rather than the clinical assessment used in this study. Furthermore, airway obstruction such as described was probably associated with a marked degree of stress. Hypoxaemia may have been present as well. The possibility also exists that prostaglandins are released into the circulation in response to bronchoconstriction. Tachypnoea, bronchoconstriction, hypoxaemia, and stress are all variables that should be excluded as a possible cause for prostaglandin secretion. Thus it is still to be determined whether prostaglandins play a mechanistic role in airway obstruction or whether their increases are merely a morphological marker for some other aspects of the acute clinical situation.

Treatment directed against byproducts of

airway obstruction and not against its causes will not be beneficial.

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- 1 Skoner DP, Fireman P, Davis HW, Wall R, Caliguini LA. Increases in plasma concentrations of a prostaglandin metabolite in acute airway obstruction. *Arch Dis Child* 1989;64:1112-7.

*Drs Skoner and Fireman comment:*

In response to Dr Amirav: (1) Persistence of post treatment rises of the measured prostaglandin  $F_{2\alpha}$  metabolite were not unexpected, as this metabolite is stable in plasma and may be detectable for hours after the release of prostaglandin  $F_{2\alpha}$ , which has a relatively short half life. (2) All of the infants in group II (infants after treatment) had residual signs of airway obstruction. (3) Pulmonary function testing would certainly be superior to clinical parameters in assessing the degree of airway obstruction, but this methodology was unavailable and may be unreliable in infants. (4) Inflammatory mediators are released either as a primary event causative of a disease process or as a secondary event, which may not be related to pathophysiology. In either case, the relationship of the mediator to the pathophysiological process is very complex, as inflammation is likely an orchestrated response, with release of multiple mediators simultaneously and synergism or antagonism between various mediators in provoking pathophysiology. Additionally, the desired human pathophysiological response may be inaccessible to direct measurement. As such, absolute proof of a causal relationship between a mediator and a pathophysiological process is frequently lacking and rests on a body of indirect rather than direct evidence.

Three criteria for implicating a mediator in disease pathophysiology are generally accepted: (1) recovery at or near the site of active disease; (2) reproduction by exposure to the mediator; and (3) attenuation or prevention by pretreatment with a mediator antagonist. The results of our study indicate that a metabolite of prostaglandin  $F_{2\alpha}$  was detectable in plasma during acute airway obstruction and satisfy criteria (1). Other investigators have documented that inhaled prostaglandin  $F_{2\alpha}$  can cause bronchoconstriction (criteria 2).<sup>1</sup> This evidence, although suggestive, is insufficient to prove a causal relationship. In situations such as this, clinical trials with specific antagonists (criteria 3) can provide confirmation that a given mediator participates in a pathophysiological process. For this reason, we believe that therapeutic trials using specific anti-inflammatory agents for the treatment of airway obstruction are warranted.

- 1 Mathe AA, Hedqvist P, Holmgren A, Svanborg. Bronchial hyperreactivity to prostaglandin  $F_{2\alpha}$  and histamine in patients with asthma. *Br Med J* 1973;i:193-6.

**Diagnostic accuracy of pH monitoring in gastro-oesophageal reflux**

SIR,—Da Dalt and colleagues believe their data show that a pH study has 100% sensitivity and 94% specificity for the diagnosis of

gastro-oesophageal reflux.<sup>1</sup> In particular, in their discussion they make comment of the fact that they did not find any false negatives and conclude that a negative result must be thought of as being sufficient to rule out gastro-oesophageal reflux.

In order to comment about the sensitivity or specificity, a comparison must be made with a gold standard. In the study by Da Dalt and colleagues, the indication for 24 hour oesophageal pH study was a clinical suspicion of gastro-oesophageal reflux. If the pH study was negative, the children were followed up clinically for a period ranging from eight months to two years. On the basis of the observations made over this period of time, the authors decided none of these children had gastro-oesophageal reflux. I would be very sceptical that clinical follow up was sufficient to use as a gold standard and would be reluctant to accept that clinical suspicion would be used as an indication for a test on the one hand, and then subsequently as the confirmation that the test was accurate.

By contrast, the gold standard used for confirmation that abnormal tests were indeed abnormal was the combination of a barium swallow or oesophagoscopy, or both. Although no indication is made of what constituted a positive barium swallow examination or an oesophagoscopy, we can be more confident with the specificity of the test. It would be useful to know whether the oesophagoscopy included a biopsy.

Finally, I question the conclusion that pH monitoring should be used as the first line of investigation in the diagnosis of gastro-oesophageal reflux. A case may be able to be made for this suggestion in children admitted to hospital, but the extrapolation that the diagnosis of gastro-oesophageal reflux in a child who is an outpatient should involve his or her admission to hospital, is premature.

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- 1 Da Dalt L, Mazzoleni S, Montini G, Donzelli F, Zachello F. Diagnostic accuracy of pH monitoring in gastro-oesophageal reflux. *Arch Dis Child* 1989;64:1421-6.

*Drs Da Dalt, Mazzoleni, Montini, Donzelli, and Zachello comment:*

We are grateful to Dr Henry for the comments on our paper and for the opportunity to clarify some aspects of our study.

We agree with the necessity of a gold standard but we would like to point out that neither barium swallow nor oesophagoscopy have got the characteristics to be a gold standard in the diagnosis of gastro-oesophageal reflux.<sup>1</sup>

For that reason and for ethical considerations we decided not to perform these examinations on children with normal pH study and we thought that a clinical follow up would be sufficient. In fact in most of these children symptoms disappeared spontaneously because they were due, we think, to a physiological gastro-oesophageal reflux, which is not detected as abnormal by pH study and for which no treatment is necessary. Only in a few children, in whom symptoms were persistent, did we reach a different final diagnosis such as coeliac disease, anorexia, etc.

We also would like to point out that in

patients with an abnormal pH study we performed a barium swallow and/or oesophagoscopy especially in order to rule out structural anomalies; however, a radiological gastro-oesophageal reflux was diagnosed when at least two episodes of spontaneous reflux were present in five minutes (according to Meyers *et al*<sup>2</sup>). A biopsy was performed in all our oesophagoscopies and signs of oesophagitis were always present.

Finally we agree with Dr Henry that not all children who vomit need a pH study or other examinations, but we think that when a major clinical problem is present and an important gastro-oesophageal reflux is suspected, admission is to be considered. An alternative could be to perform an ambulatory 24 hour pH study; this has recently been attempted even in children.<sup>3</sup>

- 1 Sondheimer JM. Gastroesophageal reflux: update on pathogenesis and diagnosis. *Pediatr Clin North Am* 1988;35:103-16.  
2 Meyers WF, Roberts CC, Johnson DG, Herbst JJ. Value of tests for evaluation of gastroesophageal reflux. *J Pediatr Surg* 1985;20:515-20.  
3 Sondheimer JM, Haase GM. Simultaneous pH recording from multiple esophageal sites in children with and without gastroesophageal reflux. *J Pediatr Gastroenterol Nutr* 1988;7:46-51.

**A catabolic state in dexamethasone treatment of bronchopulmonary dysplasia**

SIR,—Dexamethasone treatment is being increasingly used in babies with bronchopulmonary dysplasia because of reports of its benefit.<sup>1,2</sup> We have reviewed the effect of treatment in the first nine infants we have treated. All were infants with bronchopulmonary dysplasia who had been ventilated from birth for respiratory distress syndrome. Gestation ranged from 25 to 30 weeks (mean 27.5) and birth weight ranged from 740 to 1510 g (mean 1050). Four babies had required surgical ligation of a patent ductus arteriosus. Dexamethasone was commenced between days 32 and 67, and the starting dosage varied between 0.46 and 2.13 mg/kg/day (mean 1.07). The infants' ventilatory requirement had been either static or deteriorating over the week before treatment. We found the treatment effective in the short term managing successfully to extubate all the infants within a week and seven of nine by the third day of treatment.

There has been concern over the incidence of side effects including hypertension, septicaemia, necrotising enterocolitis, hyperglycaemia, and the possibility of longer term adrenal suppression. In our patients infection, hypertension, and hyperglycaemia did not cause any real problem. Three of our nine patients, however, required prolonged courses in excess of five weeks to maintain the effect and appeared to exchange dexamethasone for ventilator dependence; this is of particular concern with regard to possible adrenal suppression.

We would highlight the fact that a pronounced catabolic state developed on commencing treatment, to our knowledge this has not been commented on in previous reports. Mean weight gain fell to -57 g/week from +190 g/week; only one infant gained any weight in the first week of treatment and all had previously been gaining weight. Blood urea concentrations averaged over the first week on treatment were 5.43 times those of