

## Women in paediatrics

Sir,

It took an Act of Parliament in 1876 to enable women to graduate from British medical schools and at the present rate another Act will be required now, 100 years later, to give them realistic opportunities to complete their postgraduate training.

Your editorial<sup>1</sup> gives hope that attitudes may be changing although, in general, this is not the impression gained from my paediatric colleagues. I do not believe that there will be any appreciable change until women are on national committees in sufficient numbers to influence decisions, for I do not think enough men are seriously motivated to change the *status quo* (after all it works to their advantage).

I hope this will be one of many letters requesting that the British Paediatric Association appoint a committee, on which at least half the members are married women, to examine their particular needs in paediatrics so that suitable adaptations to career grades and instructions to appointment committees will give those struggling in the system a fairer deal.

### Reference

- <sup>1</sup> Anonymous. Women in paediatrics. *Arch Dis Child* 1981; **56**: 409.

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## Perinatal mortality

Sir,

Other readers who were surprised by Dr Robertson's letter<sup>1</sup> should re-read what Pamela Davies actually wrote in her review of the Short Committee Report.<sup>2</sup>

I failed to find any suggestion that 'money would be better spent in primary care and prevention of prematurity'. The actual comment was 'prevention of preterm labour would of course lead to a reduction in neonatal mortality . . . but preterm labour and low birthweight are to a large extent part of the basic problem—poverty', a statement with which all but the most blinkered would concur. Nor could I find any implication that 'as money is in short supply . . . we should not complain bitterly about lack of perinatal provision', described as 'an amazing council of inertia'. On the contrary Dr Davies made constructive suggestions about deployment of existing medical staff to ensure that 24-hour supervision of mothers and newborns was provided; she acknowledged that deaths could be prevented and gave as examples the decline in spastic/ataxic diplegia in low birthweight infants, the two achievements of improved perinatal facilities about which there is little dispute.

In view of the extravagant claims made before the Short Committee, and widely publicised in the media, that 20–50 % of childhood handicaps could be prevented by an increase in highly sophisticated neonatal facilities

(a claim based on very little evidence), I suspect that the real cause of Dr Robertson's anger was Dr Davies's statement that 'the bulk of severe disabling handicap in childhood is not caused by perinatal factors', and her reservations about the proportion of time in special care units spent on 'keeping ever smaller and more immature infants alive (for a future of uncertain quality sometimes)'.

A study of a total population of Dundee-resident children born 1974/1975, in whom the incidence of handicap is very similar to that reported in other surveys, confirms that genetic or early embryonic factors are implicated in most children with severe handicaps and that social factors are predominant in children with mild mental retardation, and particularly in those of normal intellectual potential who are failing in normal schools. Although only a few severe handicaps could be attributed in any way to adverse perinatal factors, there was among children with less severe disabilities a highly significant excess of complications of pregnancy and delivery, associated with later evidence of neurological dysfunction in many cases, which could be explained only in part by socioeconomic factors. It is possible that minimal damage resulting from such complications renders the child more vulnerable to adverse factors in the postnatal environment. Dr Davies's comments about antenatal care and supporting health services for mothers and infants are most pertinent at this point.

Too seldom do neonatologists emerge from their highly specialised cocoons into the real world of childhood disability and handicap, but when they do, attention should be paid to the plea that paediatricians responsible for newborn care should 'urge with all the force at their disposal that available resources should be used where they can do most for the health of all children'.

### References

- <sup>1</sup> Robertson N R C. Letter: Perinatal mortality. *Arch Dis Child* 1981; **56**: 484–5.  
<sup>2</sup> Davies P A. Perinatal mortality. *Arch Dis Child* 1980; **55**: 833–7.

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## Recurrent croup and allergy

Sir,

I congratulate Zach *et al.*<sup>1</sup> on their report of a highly significant association between recurrent croup and allergy/airways hyperreactivity. In a group of children admitted for adenoidectomy<sup>2</sup> we found that out of 202 without recurrent croup 44 of them had had atopic disease, whereas out of 21 with recurrent croup 10 had had atopic disease ( $P < 0.01$ ).

Perhaps an old debate between ear, nose, and throat and paediatric departments may soon be settled. Recurrent croup should be included among unspecific symptoms of an atopic constitution together with—for example, recurrent wheezy bronchitis and follicular hyperkeratosis. High total IgE concentrations are often found to be

present with such symptoms, including croup (N-I M Kjellman, 1981, unpublished data), before any specific symptoms of atopic allergy.<sup>3 4</sup>

Preventive measures should be instituted if a high risk of atopic allergy is present.<sup>5</sup> I suggest that levels of IgE should be determined and a family history be taken in any infant or child with recurrent croup so as to choose the children who would benefit the most from such preventive measures—for example, children coming from families that do not smoke.<sup>6</sup>

#### References

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- 2 Kjellman N-I M, Synnerstad B, Hansson L O. Atopic allergy and immunoglobulins in children with adenoids and recurrent otitis media. *Acta Paediatr Scand* 1976; **65**: 593–600.
- 3 Foucard T. A follow-up study of children with asthmatoïd bronchitis. II. Serum IgE and eosinophil counts in relation to clinical course. *Acta Paediatr Scand* 1974; **63**: 129–39.
- 4 Kjellman N-I M, Johansson S G O. IgE and atopic allergy in newborns and infants with a family history of atopic disease. *Acta Paediatr Scand* 1976; **65**: 601–7.
- 5 Anonymous. Towards prevention of allergy. *Lancet* 1977; **i**: 339.
- 6 Kjellman N-I M. Letter: Effect of parental smoking on IgE levels in children. *Lancet* 1981; **i**: 993–4.

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## Transient tachypnoea of the newborn

Sir,

The interesting paper by Halliday *et al.*<sup>1</sup> illustrated the problems of nomenclature in neonatal cardiorespiratory disorders. The authors described a subgroup of babies in whom we would diagnose persistent fetal circulation, even without resort to echocardiography. They chose to label the disorder 'transient tachypnoea of the newborn (TTN)', but observed that it differed in many ways from 'classical TTN', and postulated that there were two types of TTN, so lo and behold a disease variant was born!

It is clear that there is a group of babies who suffer from respiratory distress soon after birth but in whom there seems to be no major identifiable cause—such as hyaline membrane disease, congenital pneumonia, meconium aspiration syndrome, or structural malformation of the cardiorespiratory system—and it is difficult to put a diagnostic label on such babies. In terms of aetiology some have a delay in the clearance of lung fluid, some are suffering from post-asphyxial pulmonary oedema, some have persistent fetal circulation with pulmonary hypertension and right-to-left shunt; others have transient and patchy atelectasis possibly caused by minor aspiration and airways obstruction. Often a combination of causes operate. We suggest that the term 'maladaptation syndrome' describes these babies,

qualified by a statement about the probable major aetiological factor. This is of more than academic interest because delayed clearance of lung fluid is benign and self-limiting, pulmonary oedema may respond to frusemide and continuous positive airways pressure, persistent fetal circulation may be controlled by tolazoline, and patchy atelectasis is helped by chest physiotherapy.

We are concerned that someone will label the so-called more severe form of TTN of Halliday *et al.* as 'type II TTN'; bearing in mind that TTN is sometimes referred to as 'type II respiratory distress syndrome',<sup>2 3</sup> we shall then all be talking at cross-purposes.

#### References

- 1 Halliday H L, McClure G, Reid M McC. Transient tachypnoea of the newborn: two distinct clinical entities? *Arch Dis Child* 1981; **56**: 322–5.
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- 3 Sundell H, Garrott J, Blankenship W J, Shepard F M, Stahlman M T. Studies on infants with type II respiratory distress syndrome. *J Pediatr* 1971; **78**: 754–64.

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Dr Halliday and co-workers comment:

Our aim in writing the paper was not to suggest changes in nomenclature of neonatal respiratory disorders but rather to stress to non-specialist physicians who care for the newborn that some babies with fairly clear chest radiographs do have severe illness.

The diagnosis of transient tachypnoea of the newborn (TTN) can only be made with certainty retrospectively. That is, an infant with tachypnoea, mild distress, and a characteristic chest radiograph<sup>1</sup> who recovers in 3 or 4 days.<sup>2</sup> We attempted to make the diagnosis prospectively on the basis of clinical presentation and x-ray films in our 25 babies. Six of our infants had profound hypoxaemia and 3 needed mechanical ventilation though their initial clinical and radiological findings were similar to those of babies with Avery's classical TTN.<sup>2</sup> These 6 babies probably had pulmonary hypertension (raised RPEP/RVET ratios) and right-to-left shunting, but initial clinical findings would not have distinguished them from the other 19 babies.

We are concerned that some nurseries who only rarely care for ill newborn infants may decide on the basis of fairly clear chest x-ray films (absence of signs of respiratory distress syndrome) not to transfer a potentially hypoxaemic infant. Blood gas analysis may not be routinely practised in such nurseries, and it is only when the baby later collapses that transfer to the regional centre is arranged. Perhaps, worse still, some of these babies may be transferred as cases of cyanotic congenital heart disease and even undergo cardiac catheterisation<sup>3</sup> without resort to echocardiographic assessment.

Although echocardiography suggests disordered left