Professor Milner comments:

May I reply to Dr Wright's points in turn:

(1) We entirely agree and in fact stated in the text that the introduction of apnoea monitors was based on the hypothesis that 'babies dying from sudden infant death have cessation of breathing probably for longer than 30 seconds before death or irreversible brain damage has occurred'.

(2) There are published reports of babies dying while attached to monitors.^{5 6}

(3) We would agree that the Graseby MR 10 is the most commonly used monitor but it is certainly not the only device used in the United Kingdom.

(4) Since we do not know how babies are victims of sudden infant death syndrome we do not know whether obstructive apnoea is an important factor, but studies have shown that obstructive apnoea certainly occurs in babies suffering near miss sudden infant death, who are often provided with apnoea monitors for use at home.⁷

(5) We would certainly agree that false positive alarms can sometimes reassure parents that the device is working, although in our experience frequent alarms often cause the parents to stop using monitors after relatively short periods.

References

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- ³ Callan K, Wright BM. *Proceedings of the 3rd International conference on ambulatory monitoring*. London: Academic Press, 1980:329-34.
- ⁴ Stewart-Brown S. Personal view. *Br Med J* 1982;**284**:1628.
- ⁵ Kelly DH, Shannon DC, O'Connor K. Care of infants with near-miss sudden infant death syndrome. *Pediatrics* 1978;**61**:511-4.
- ⁶ Lewak N. Sudden infant death syndrome in a hospitalized infant on an apnoea monitor. *Pediatrics* 1975;**56**:296-8.
- ⁷ Guillemainault C, Ariagno R, Korobkin R, *et al*. Mixed and obstructive sleep apnea and near miss for sudden infant death syndrome. 2. Comparison of near miss and normal control infants by age. *Pediatrics* 1979;**64**:882-91.

Costs and outcomes in a regional neonatal intensive care unit

Sir,

News *et al*¹ report the costs of intensive neonatal care in Birmingham in 1980 and 1981. In succeeding to calculate the costs of this service to the National Health Service they are to be congratulated. No similar data are available. There is, however, the danger that in the absence of other cost evaluations many health authorities may use this information as a benchmark to price their own services now that 'Sainsbury' style management is imminent.

Nursing staff salaries, the major component of their costs, have risen considerably in excess of the average compounded inflation rates. Furthermore, the nursing

staff levels at Birmingham Maternity Hospital not commented upon directly by the authors were, in 1981 to 1983, to quote a West Midlands Health Authority report 'grossly unsatisfactory in the ratio of trained to untrained staff and unsatisfactory at night'.

That they cared so well for their neonates while being so short staffed is to their credit but for those of us being called upon to project the present costs for intensive neonatal services a more realistic estimate would be to double the quoted figures.

Reference

- ¹ News B, Drummond MF, Durbin GM, Culley P. Costs and outcomes in a regional neonatal intensive care unit. *Arch Dis Child* 1984;**59**:1064-7.

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Clinical significance of gastro-oesophageal reflux

Sir,

Am I to assume that Professor Carré examines radiologically all regurgitating babies he sees?¹

Reference

- ¹ Carré IJ. Clinical significance of gastro-oesophageal reflux. *Arch Dis Child* 1984;**59**:911-2.

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Professor Carré comments:

Dr Connor is mistaken in his assumption. There was indeed a time when I would have had the great majority of regurgitating infants examined radiologically, but resulting from experience gained at that time only selected cases have been studied in this manner during the past 10 to 15 years. These comprise infants with persistent and particularly troublesome regurgitation, especially if associated with occasional vomiting or other clinical disturbance such as respiratory illness, and all younger regurgitating siblings of children with a partial thoracic stomach.

Coagulation defect in congenital tyrosinaemia

Sir,

We agree with Professor Özsoylu¹ that low fibrinogen

values make interpretation of coagulation tests unreliable, but our diagnosis² of dysfibrinogenaemia was based on the grossly prolonged reptilase time compared with the thrombin time. If simple hypofibrinogenaemia were responsible, we would have expected both times to have been similarly prolonged. Furthermore, the coagulation factor assays are independent of the patient's fibrinogen value.

As regards the diagnosis of congenital or hereditary tyrosinaemia, the record needs to be put right. It is correct to say that tyrosinaemia (with abnormal excretion of phenolic acids) and hypermethioninaemia alone, do not indicate the diagnosis. In all three infants reported, not only was there evidence of acute liver necrosis but also of severe proximal renal tubular disease characterised by proteinuria, gross generalised aminoaciduria, phosphaturia, hypophosphataemia, and early rickets accompanied by considerably raised serum alkaline phosphatase, mainly of bone origin. The galactose-1-phosphate uridyl transferase deficiency galactosaemia was excluded in all, and none of the infants received fructose in the diet or in medication.

These observations strongly favour the diagnosis of congenital or hereditary tyrosinaemia of acute variety; unfortunately necropsy examination in the first two patients was not available. As for galactokinase deficiency galactosaemia, liver disease does not occur in this disorder.

References

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- ² Evans DIK, Sardharwalla IB. Coagulation defect of congenital tyrosinaemia. *Arch Dis Child* 1984;**59**:1088-90.

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Lest we forget

Sir,

Your editorial¹ was a splendid reminder to us to be primarily people-orientated. Your question, 'How often do we in our postgraduate and in-service training discuss the problems of children in hospital, their psychological needs, and the needs and the problems of unrestricted access for parents?' is timely.

Now we are in the third decade of 'deinstitutionalisation' of the severely mentally handicapped, do we need to ask a similar question? 'How often do we in our postgraduate and in-service training discuss the problems of severely subnormal children living at home, the physical and psychological needs, and all the problems of parents continuing to care for over grown toddlers who are extraordinarily slow to learn'?

How often do we reflect on the needs of the mothers of the severely handicapped? How often do we consider her fourfold role as: (a) Mother of the handicapped child; (b) Mother of the other child/children; (c) Wife; (d) Needing time/space to be 'herself'.

If we are to support the severely subnormal in the community then we must be acutely aware of the needs and stresses of those who provide day and night cover, too often, without a break.

Reference

- ¹ Anonymous. Lest we forget. [Editorial]. *Arch Dis Child* 1985;**60**:93.

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Who should get growth hormone?

Sir,

The four papers on the assessment of potential candidates for growth hormone treatment¹⁻⁴ contain some contradictions that may lead to misunderstandings among readers. These contradictions originate mainly from different definitions of growth hormone deficiency.

The two papers on exercise tests^{2,3} emphasise the latter half of the classic definition, that is the combination of a growth velocity below the 25th centile for age or bone age and a growth hormone response to adequate insulin hypoglycaemia under 15 mU/l. The commentary⁴ emphasises the first half of this definition. Low growth velocity is considered as an absolute prerequisite for the diagnosis, but no indication is given as to which growth velocity is abnormally low. If the classic 25th centile were used, 25% of the children would have to be tested.

There is increasing evidence that the classic definition has lost its usefulness. Studies on 24 hour growth hormone profiles in short children have shown that the results of provocation tests are poor parameters of growth hormone secretory status and poor predictors of growth response to long term treatment.

In my opinion, growth hormone deficiency should be defined as the condition in which either the total 24 hours' production of growth hormone, or the frequency or amplitude of peaks, or both, is below normal. This does not imply that the classic criteria (growth velocity and biochemical tests) would lose their role in the diagnostic process. In case of low growth velocity and low responses to provocative stimuli, growth hormone deficiency can be diagnosed without 24 hour profiles. Such profiles, however, could be made in children with short stature or low growth velocity, or both, and growth hormone responses greater than 15 mU/l in order to confirm or exclude growth hormone deficiency. In case of normal growth hormone production a therapeutic trial might still be indicated, as children with abnormal growth hormone molecules or partial receptor defects seem to respond favourably to treatment.

In conclusion, in one issue of the *Archives* growth hormone deficiency is defined in the classic way^{2,3} as well as in a more updated way.^{1,4} Suggestions that growth