LETTERS TO THE EDITOR

Audit of screening for congenital hypothyroidism

EDITOR,—Professor Pharoah and Dr Madden raised the issue of administrative practices causing inefficiency in screening for congenital hypothyroidism.¹ We share their concerns particularly over the delay in the reporting of normal results and in identifying infants who have not been tested at all.

Under the present system the identification of infants who have never been tested is by comparing a list of birth notifications with a list of all the results, normal and abnormal. Infants whose names appear on the list of birth notifications but not on the results list are those who have failed to have a test. This sounds simple but in practice it can take over two months to compile the two lists (figure).

In order to short circuit the system to identify infants who have never been tested, our proposal is that a copy of the Guthrie form should be given to the mother on testing. At the statutory new birth visit at 10 days the health visitor would check that every mother has a form and arrange immediate screening for any infant who had been missed.

At present there is also an unacceptable delay in compiling a list of normal test results. There

are two main difficulties. One is that the mother's name which is used to identify the infant on the birth notification form differs from the baby's name used on the Guthrie test form. Another is that because the mother and child either move house or go to stay with relatives the Guthrie test specimen is sent to a laboratory in another health authority.

We suggest the Guthrie form should be modified to include the mother's name and the neonatal laboratory to which the report will be sent. These proposals would reduce costs, avoid confusion, encourage parental involvement, and avoid delays in diagnosis.

Before we consider adding other metabolic screens to the Guthrie test it is imperative to develop a streamlined and fail-safe system to handle the results.

M E TORRANCE
H BANTOCK
M FREEMAN

Bloomsbury and Islington Health Authority,
Insurance House,
Insurance Street,
London WC1X 0JB

1 Pharoah POD, Madden MP. Audit of screening for congenital hypothyroidism. *Arch Dis Child* 1992; 67: 1073–6.

Sleep related upper airway obstruction and hypoxaemia in sickle cell disease

EDITOR,—I read with interest the report of Samuels and his colleagues on respiratory sleep disturbances in sickle cell disease.¹ In the discussion the authors suggest that changes in

the anatomy of the upper airway may have an important role, besides the underlying mechanism of sickling of red cells which is enhanced by hypoxia.

I therefore think that it is worthwhile to draw attention to the peculiar type of nasal obstruction recently described as 'priapism of the turbinates' observed in two young patients with sickle cell disease.² The respiratory involvement of such nasal obstruction is likely to be equivalent to that caused by nasal packing, which is known to induce a significant decrease in oxygen saturation during sleep.^{3,4}

It would be of great interest to know if any evidence of nasal turbinate enlargement was found in the 53 patients with sickle cell disease studied by Samuels and his colleagues, as they all underwent ear, nose, and throat examination. It may contribute to the recognition of this newly described condition, which up to now has probably been overlooked.

NICOLA D'ALOYA
Lung Function Unit,
Ospedale 'A. Murri' di Jesi,
Via F. del Cossa 32,
I-44100 Ferrara, Italy

- 1 Samuels MP, Stebbens VA, Davies SC, Picton-Jones E, Southall DP. Sleep related upper airway obstruction and hypoxaemia in sickle cell disease. *Arch Dis Child* 1992; 67: 925–9.
- 2 Ajulo SO. Priapism of the turbinates: a cause of nasal obstruction in sickle cell anaemia. *J Laryngol Otol* 1991; 105: 851–2.
- 3 Hady MRA, Kodeira KZ, Nasel AH. The effect of nasal packing on arterial blood gases and acid base values and its clinical importance. *J Laryngol Otol* 1983; 97: 599–601.
- 4 Buckley TG, Hickey SA, Fitzgerald O'Connor AF. Does post-operative nasal packing cause nocturnal oxygen desaturation? *J Laryngol Otol* 1991; 105: 109–11.

Drs Samuels, Southall, and Davies comment:

Our ear, nose, and throat colleagues made no comment with regard to the finding of nasal turbinate enlargement in any of the 53 patients we studied. Because priapism and sickling are acute events and resolve in the stable state, they are unlikely to be the cause of chronic sleep related upper airway obstruction or hypoxaemia. We thank Dr D'Aloia for drawing our attention to this unusual syndrome.

Teachers and epilepsy

EDITOR,—Encouraging as the knowledge and awareness of epilepsy was among the teachers sampled in the study of Bannon *et al*,¹ it would be naive to expect a similar response nationwide. For instance, within the Mersey region recently, two children have been denied commencing their primary school education because of even the possibility of epileptic seizures.

The point regarding the need for improved communication was well made. This could be facilitated by the provision of nurse specialists in epilepsy, such as currently exist for other chronic childhood disorders including diabetes, asthma, and cancer. The nurse specialist could establish a much needed liaison between the hospital and community child health services, and could also fulfil a major educational role within schools.

RICHARD E APPLETON
Mersey Region Paediatric Epilepsy Clinic
and Department of Neurology,
Alder Hey Children's Hospital,
Eaton Road,
Liverpool L12 2AP

- 1 Bannon MJ, Wildig C, Jones PW. Teachers' perceptions of epilepsy. *Arch Dis Child* 1992; 67: 1467–71.

Day

Present system for processing Guthrie tests and how it may be improved. Earliest detection of those not tested is at 62–76 days. ***Untested infants could be identified by health visitor at 10 days.

**Steps made unnecessary by proposals. CHCS=child health computerised system. Figures from Bloomsbury and Islington Health Authority, 1992.