

## Neonatal tuberculosis

Sir,

We were interested to read the short report by Bate, Sinclair, and Robinson as it reminded us of a similar case, previously unreported, seen by us some years ago.<sup>1</sup>

A baby girl was delivered by forceps at 36 weeks' gestation to a 19 year old married primigravida, who was a Spanish hotel waitress. After a normal pregnancy she was an emergency admission with what seemed initially to be a pyogenic meningitis but was subsequently shown to be tuberculous meningitis, although culture for acid fast bacilli yielded negative results. Her uncle had almost certainly died from pulmonary tuberculosis.

The baby was separated from her mother at birth in good condition but became jaundiced, with a maximum serum bilirubin concentration of 190  $\mu\text{mol/l}$  on the fourth day. She failed to thrive, developing a series of staphylococcal infections followed on the 22nd day by a fever of 38°C (rectal), pulse 140–172/min, irregular respirations averaging 48/min, fine crepitations in the right lower zone, and moderate hepatosplenomegaly. A chest x ray film (Figure) showed extensive coarse miliary mottling due to miliary tuberculosis. Scanty acid fast bacilli were identified on the Ziehl-Nielsen film from gastric washings, and subsequently *Mycobacterium tuberculosis* was grown on culture. Treatment was started with rifampicin 10 mg/kg/day, isoniazid 20 mg/day, and prednisone 2 mg/kg/day, and she began to gain weight slowly and was well on discharge aged 5 months, soon afterwards emigrating with her mother to Caracas, Venezuela, where she was thriving at 13 months.

Our case shows features of the 'aspiration' type of congenital tuberculosis, as opposed to the 'haematogenous' transplacental type,<sup>2</sup> with infected amniotic fluid entering the fetal lungs, probably intrapartum,<sup>3</sup> and is unusual in that both mother and baby survived.

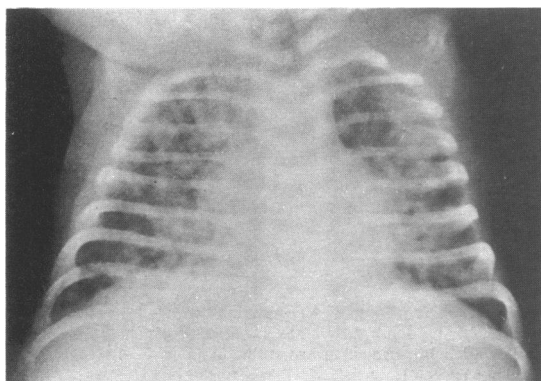


Figure Chest x ray of the case aged 1 month, showing extensive bilateral miliary mottling due to miliary tuberculosis.

## References

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- <sup>2</sup> Voyce MA, Hurst AT. Congenital tuberculosis. *Arch Dis Child* 1966;**41**:299–300.
- <sup>3</sup> Corner BD, Brown NJ. Congenital tuberculosis: report of a case with necropsy findings in mother and child. *Thorax* 1955;**10**: 99–103.

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## Height measurements at the onset of acute lymphoblastic leukaemia

Sir,

I read the article by Bessho with interest.<sup>1</sup> The author could not find significant height differences between children with acute lymphocytic leukaemia and healthy controls matched for age and sex. This indeed is remarkable, for most other investigators have claimed the opposite, leukaemic children being taller than controls. Yet I am not convinced for the following reasons.

It is known that severe illnesses result in a decrease in growth velocity. This has been investigated also in the case of acute lymphoblastic leukaemia by Berglund *et al.*<sup>2</sup> The authors observed a decrease in growth before the start of treatment and suggested that the disease might cause the growth failure. Most interestingly, they not only compared height with a reference group at the clinical onset of the disease but also one year before diagnosis. Thus I am sceptical indeed whether single measurements of body height at the instant of the diagnosis of acute lymphoblastic leukaemia in fact represent valid information on growth. Could pre-existing tall stature have vanished by the time of the diagnosis?

Though the role of growth hormone as cited by Bessho is indeed questionable, there is overwhelming evidence for the involvement of polypeptide growth factors not only in the regulation of normal growth but also in growth factor initiated pathways in the aetiology of cancer.<sup>3</sup>

With respect to this, child growth before the onset of acute lymphoblastic leukaemia is an interesting variable. Bessho's patient group is very large and obviously well matched with its controls. I would like to know more about the patients' growth, not just mean height at one point of their development.

## References

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<sup>2</sup> Berglund G, Karlberg J, Marky I, Mellander L. A longitudinal study of growth in children with acute lymphoblastic leukaemia. *Acta Paediatr Scand* 1986;74:530-3.

<sup>3</sup> Goustin AS, Lcof EB, Shipley GD, Moses HL. Growth factors and cancer. *Cancer Res* 1986;46:1015-29.

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Dr Bessho comments:

I do not deny the importance of information about the height and its velocity before the onset of acute lymphoblastic leukaemia and the possibility that height at diagnosis may be a function of growth enhancing effect of possible growth factor(s) and growth suppressing effect of the disease (acute lymphoblastic leukaemia). It is worth while to confirm Dr Berglund's finding as a phenomenon with a larger scale study using an appropriate control.

I do not think, however, that this kind of study could clarify the possible role of growth factor(s) on leukaemogenesis. No one can solve single equations with two or more variables. Obviously, other kinds of studies are required for this purpose. Therefore, I carefully avoided making the statement that growth hormone had no role concerning leukaemogenesis.

colic duplication, suspected by antenatal echography at 33 weeks' gestation, was confirmed by operation on day 7. On day 17 she had a second abdominal operation for a hypertrophic pyloric stenosis. Further history has been uneventful.

This association could be explained by a simple embryological mechanism as the development of the thyroid gland (formation and migration between 16th and 50th day of fetal life) and the gut (formation and rotation of the umbilical loop between 21st and 42nd day) both occur early in the fetal development.

The question arises as to whether the results reported by Bamforth *et al* could represent the consequences of local genetic or environmental factors, or both. As suggested by the authors, this point deserves further investigation.

#### References

<sup>1</sup> Bamforth JS, Hughes I, Lazarus J, John R. Congenital anomalies associated with hypothyroidism. *Arch Dis Child* 1986;61:608-9.

<sup>2</sup> Goujard J, Safar A, Rolland A, Job JC. Epidémiologie des hypothyroïdies congénitales malformatives. *Arch Fr Pédiatr* 1981;38:875-9.

<sup>3</sup> Hulse JA, Grant DB, Clayton BE, Lilly P, Jackson D, Sprachlon A. Population screening for congenital hypothyroidism. *Br Med J* 1980;ii:675-8.

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## Congenital anomalies associated with hypothyroidism

Sir,

The report by Bamforth and colleagues of a high incidence of various extrathyroidal abnormalities associated with congenital hypothyroidism detected by neonatal screening<sup>1</sup> is of interest as it could throw new light on the aetiology of these congenital malformations. Such an association had been suggested in infants with congenital hypothyroidism detected on the basis of clinical signs before the era of screening<sup>2</sup> but has not been reported in large series of hypothyroid infants detected by screening.<sup>3</sup>

In a series of infants similar to the one of Bamforth *et al* we could not confirm their findings: since the introduction of neonatal thyroid screening in Belgium in 1974, our own centre has screened 125 257 infants and has detected 41 infants with persistent primary hypothyroidism—that is, an incidence of 1/3055. Only one of them had associated anomalies: this girl was born after a normal pregnancy of 38 weeks' gestation, with biological signs of severe congenital hypothyroidism (serum thyroid stimulating hormone at diagnosis 100 mU/ml and thyroxine 69 nmol/l). Thyroid scintigraphy showed a lingual thyroid gland. A

## Endotracheal suction techniques in the neonate

Sir,

Endotracheal suction of the ventilated neonate is often performed during neonatal intensive care. It has many side effects, including hypoxia, increased blood pressure and cerebral blood flow velocity,<sup>1</sup> and atelectasis.<sup>2</sup> In an attempt to reduce side effects many neonatal units have developed special procedures to achieve efficient suction in the shortest possible time. In one such technique the endotracheal tube suction catheter is attached to a mucus extractor and suction applied by the nurse rather than by the traditional wall mounted vacuum source with a gauge.

We investigated the pressures generated by this technique. The patient end of a mucus extractor was attached directly to a water manometer. Four doctors and six trained experienced nurses were then asked to apply