section and costly (financial and emotional) care of this infant could have been avoided.

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*Mr Taylor and Mr Walkinshaw comment:*

We are aware of the case report literature on fetal brain death, almost all of which is secondary to known severe hypoxia.[1] In our case we aimed to illustrate cranial nerve defects, and our dynamic ultrasound findings evolved over several weeks. There was no evidence from the history or later postmortem examination to suggest an anoxic insult. The confirmation of fetal varicella IgM antibodies was delayed for several weeks. Local laboratory testing was negative, but referral to the Manchester PHSL Reference Laboratory gave the reported result. The cardiotocograph (our fig 1) does not fulfil all of the criteria for fetal brain death. The baseline was appropriate for the gestation and close scrutiny of the illustration reveals shallow decelerations.[1] We accept, however, that this should have been elaborated on in our discussion.

Lessons are being learned from anatomical prenatal diagnosis and false positive diagnoses illustrate its lack of precision. We would urge caution in the new field of functional diagnosis rather than to take a didactic approach.

We are not aware of any reported literature on how often a potential false positive diagnosis of fetal brain death is made. Even a biophysical profile score of 2 before delivery is not always associated with a poor neonatal outcome.[2] We felt after discussion with the parents that the baby should be given the benefit of these doubts.

The paediatric management was aggressive and reflected the reluctance of the paediatricians to accept the reliability of a functional diagnosis. The obstetric authors (WGT, SAW) agree with Sheila Gahagan and Claudine Amiel-Tison that some of the paediatric investigations and management was unnecessary.

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## Should we look after babies less than 800 g?

EDITOR,—Dr Roberton writes that 'babies between 500 g and 800 g ... clearly have an excellent chance of neurologically intact survival' (p 329) and yet provides us with scant data to support this.[1]

The results quoted (based on the survival rate of all babies born in the state of Victoria, Australia and weighing under 1000 g) show no survivors under 600 g and a neurologically intact survival rate of 8% for babies weighing 600–699 g.[2 3] This is a very commendable achievement but surely not 'clearly an excellent chance'.

Dr Roberton tells us that 'the cost of looking after these babies is not prohibitive, about £10 000–£15 000 per survivor'. This figure comes from the estimated cost per survivor of £13 720 in a trial of 19 babies in Belfast Royal Maternity Hospital in 1985–7. The mean birth weight was 1287 g and the cost per day in special care £77.[4] I suggest that the cost of caring for a 500–800 g baby in 1993 is considerably more than this. (Dr Roberton says earlier in the article that '8–10 year old data apply to a standard of neonatal care that is obsolete' (p 327)). He describes the above cost as 'phenomenally cheap compared with ... hormone treatment for the menopausal middle aged' (p 328). The March 1993 edition of the *British National Formulary* gives the cost of one year's treatment with ethinyloestradiol as £11.31.

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## When will my baby go home?

EDITOR,—Powell *et al* report that parents of preterm babies frequently ask: 'when will my baby go home?'.[1] This question is being asked in the Netherlands as well. In the study cited by Powell *et al* as reporting mortality and morbidity only,[2] we asked exactly that question. The results have been published in a Dutch language medical journal.[3] Of the nationwide cohort of very preterm (gestational age less than 32 completed weeks) and (or) very low birthweight (less than 1500 g) infants the 992 surviving infants were discharged home after a mean hospital stay of 68 days (range 6–380 days).

For all infants, the difference was calculated between the expected date of delivery and the date of discharge. The figure shows the number of infants discharged per category of days before and after term. The distribution shows that, as in the study of Powell *et al*, many of these infants were discharged around 36 to 37 weeks' postconceptional age (term minus 25 to minus 15 days). A total of 60% of all infants went home at or before 42 weeks' postconceptional age. However, 10% of these very preterm infants were still in the hospital at five weeks after the expected date of birth. We concluded that for this category of preterm babies, the answer to the parents' question should be: half of these babies can go home at or before the expected date of delivery, 70% are home two weeks after that, and 90% are home at five weeks after term.

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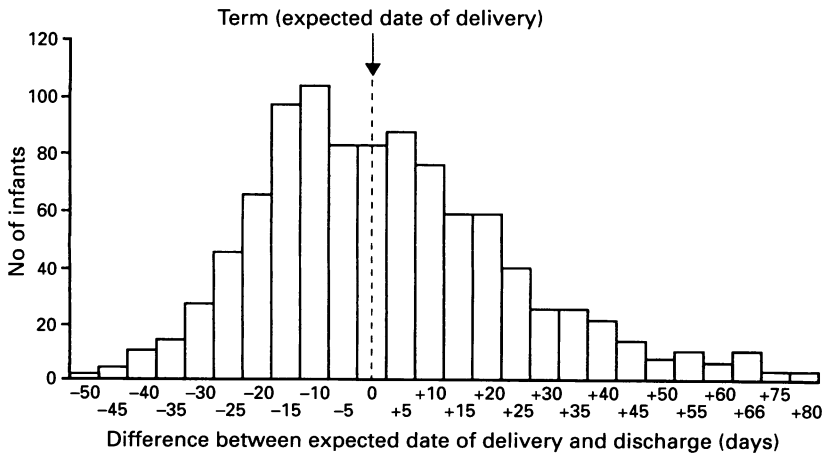
## Bilateral congenital diaphragmatic hernia – delayed presentation of the contralateral defect

EDITOR,—Approximately 3% of congenital diaphragmatic defects are bilateral.[1] Historically this condition has been almost uniformly fatal, with the first UK survivor reported in 1990.[2] We have successfully treated two patients in whom diagnosis of the contralateral hernia was delayed.




*Number of infants discharged at various ages in relation to term date (expected date of delivery) (n=992). (Figure adapted from* Het Nederlands Tijdschrift voor Geneeskunde *(1988; 132: 1687–8[3]) and reproduced with permission.)*

### Case reports

A boy of 33 weeks' gestation presented at birth with a left Bochdalek hernia, which was repaired within 24 hours. Postoperatively he required prolonged ventilatory support. A chest radiograph on day 33 showed right basal shadowing initially attributed to collapse/consolidation, but screening revealed a right diaphragmatic hernia. At operation a Bochdalek defect containing three quarters of the liver was repaired. Ventilation was continued for 14 days, and chronic lung disease evolved requiring oxygen treatment until 5 months of age. However, follow up to 4 years showed normal growth and development.

A boy of 31 weeks' gestation presented at birth with a right Bochdalek hernia. Primary repair was performed at 24 hours of age, with four days' postoperative ventilation. Progress was thereafter satisfactory with no respiratory symptoms until two weeks later when tachypnoea developed and a chest radiograph showed apparent cardiomegaly, though cardiac examination was normal. A lateral film showed the presence of a left diaphragmatic hernia, which was repaired aged 6 weeks. He required supplemental oxygen until one year but aged 6 years growth and development were normal with no respiratory symptoms.

### Discussion

In both cases early postoperative radiographs showed no evidence of a contralateral hernia and this diagnosis was not initially considered with the appearance of shadowing compatible with other pathology. Radiographic appearances in diaphragmatic hernia can occasionally be misleading, and may rarely be normal, with delayed herniation occurring secondary to falling intrathoracic pressure.[3] It seems that contralateral defects may also become apparent subsequent to changing intrathoracic and intra-abdominal pressure after reduction of viscera from the chest. We recommend palpation of the contralateral diaphragm at primary surgery, and in cases of unusual postoperative radiological appearances early examination of the contralateral diaphragm by ultrasound or screening to exclude bilateral defects.

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1 David TJ, Illingworth CA. Diaphragmatic hernia in the south-west of England. *J Med Genet* 1976; **13:** 253–62.
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### Ranitidine in infants

EDITOR,—We were interested to note the findings of Fontana *et al.*[1] In the study paired samples of serum are used to measure ranitidine concentrations in term newborn infants, and with the use of an interpretative model, pharmacokinetic indices are derived. The authors emphasise the difficulty in interpreting this, as the rationale behind the use of ranitidine is the production of adequate suppression of gastric acid secretion, and the maintenance of an increased pH in the stomach. There are therefore a considerable number of assumptions which need to be made before measurements of serum concentrations can be extrapolated to the point of calculating bolus dose or infusion rates in the management of neonatal problems related to intragastric acidity.

Using continuous intragastric pH monitoring we have been able to measure the main end point of ranitidine therapy.[2] Ranitidine was given at three infusion rates, based on our own theoretical calculation: 0·125 mg/kg per hour, 0·0625 mg/kg per hour, and 0·031 mg/kg per hour. Intragastric pH was satisfactorily raised to pH greater than 4 in all patients with an infusion of 0·0625 mg/kg per hour, and no significant benefit was conferred by using the higher dose. A smaller dose did not produce sufficient acid suppression. Interestingly the theoretical calculation by Fontana and his coauthors suggest infusion rates between 0·03 and 0·06 mg/kg per hour in the term infant. Ranitidine is largely secreted unchanged in the urine, a mode of elimination which we anticipate will be more efficient in the term infant. If in the preterm infant a rate of 0·0625 mg/kg per hour is required, we recommend that this dose should be the minimum dose used in the term infant.

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### Use of sedatives and muscle relaxants in newborn babies receiving mechanical ventilation

EDITOR,—It is well known that pancuronium may have a prolonged action in premature infants,[1] but it is not widely appreciated that some patients who receive a neuromuscular blocking drug continuously for more than two days may remain profoundly weak long after the drug is discontinued.[2] This has so far been reported for pancuronium and vecuronium, but not for atracurium. The complication has occurred in all age groups, but I have found only three reports about neonates,[3–5] and these infants had been paralysed for very long periods (two to five weeks).

Persistent blockade of the neuromuscular junction with paralysis for as long as a week has occurred particularly in patients with renal failure, and may be caused by accumulation of active 3-hydroxy metabolites of pancuronium and vecuronium. In this respect, atracurium is an attractive alternative, as it is degraded non-enzymatically in plasma to compounds not active at the neuromuscular junction. Other patients have developed a severe, generalised myopathy persisting for several weeks. This has most often occurred in asthmatic patients, and is probably caused by an adverse interaction between muscle relaxants and corticosteroids. Both these mechanisms are of concern in ventilated neonates, who have an impaired renal function, and are now often treated with steroids early in the course of lung disease. Moreover, prolonged muscle weakness and failure of weaning in tiny babies may easily be misinterpreted as caused by immaturity or cerebral depression.

Neonatologists should be aware of these potential complications and are urged to report such cases if seen. For the time being, it may be wise to avoid continuous relaxation for several days, and let the baby intermittently come to an unparalysed state. I believe this policy is already used in many neonatal intensive care units.

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