

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.

D P BARKER
S HUSSAIN
J D FRANK
H R NOBLETT
P J FLEMING
Neonatal Unit,
St Michael's Hospital,
Southwell Street,
Bristol BS2 8EG

- 1 David TJ, Illingworth CA. Diaphragmatic hernia in the south-west of England. *J Med Genet* 1976; **13**: 253-62.
- 2 Bruce J, Doig CM. Survival following repair of bilateral diaphragmatic hernia. *J R Coll Surg Edin* 1990; **35**: 389.

3 Gleeson F, Spitz L. Pitfalls in the diagnosis of congenital diaphragmatic hernia. *Arch Dis Child* 1991; **66**: 670-1.

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.

SIMON J NEWELL
ERIC J KELLY
St James's University Hospital,
Beckett Street,
Leeds LS9 7TF

- 1 Fontana M, Massironi E, Rossi A, *et al.* Ranitidine pharmacokinetics in newborn infants. *Arch Dis Child* 1993; **68**: 602-3.
- 2 Kelly EJ, Chatfield SL, Brownlee KG, *et al.* The effect of intravenous ranitidine on the intragastric pH of preterm infants receiving dexamethasone. *Arch Dis Child* 1993; **69**: 37-9.

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.

LARS J BJÖRKLUND
Neonatal Intensive Care Unit,
Department of Paediatrics,
Lund University Hospital,
S-221 85 Lund, Sweden

- 1 Levene MI, Quinn MW. Use of sedatives and muscle relaxants in newborn babies receiving mechanical ventilation. *Arch Dis Child* 1992; **67**: 870-3.
- 2 Hansen-Flaschen J, Cowen J, Raps EC. Neuromuscular blockade in the intensive care unit. More than we bargained for. *Am Rev Respir Dis* 1993; **147**: 234-6.
- 3 Torres CT, Maniscalco WM, Agostinelli T. Muscle weakness and atrophy following prolonged paralysis with pancuronium bromide in neonates. *Ann Neurol* 1985; **18**: 403.
- 4 Rutledge ML, Hawkins EP, Langston C. Skeletal muscle growth failure induced in premature infants by prolonged pancuronium treatment. *J Pediatr* 1986; **109**: 883-6.
- 5 Rossiter A, Souney PF, McGowan S, Carvajal P. Pancuronium-induced prolonged neuromuscular blockade. *Crit Care Med* 1991; **19**: 1583-7.