A boy of 33 weeks’ gestation presented at birth with a right 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 byonic 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 ascites 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.


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 stress 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. This is an important factor as an increased pH is associated with gastrointestinal tract complications. In this respect, atropine 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, in all aged groups, 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 randomly contract an unparalysed state. I believe this policy is already used in many neonatal intensive care units.

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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 continue to breathe for two days may remain profoundly weak 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 aged 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).

Prolonged 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. In this respect, atracurium is a more 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, in all aged groups, weakness and failure of weaning in tiny babies may easily be misinterpreted as caused by immaturity or cerebral depression.

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