
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 emphasize 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. Thus we are left with 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 less efficient in the 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

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 be ventilated for 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 aged groups, but I have found only three reports about neonates,3,4 and these infants had been paralysed for very long periods (two to five weeks).

Prolonged blockade of the neuromuscular junction with paralysis as long as a week has occurred particularly in patients with renal failure, and may be caused by accumulation of active 3-hydroxy metabolites or, as a more likely cause, an unparalysed state. 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, 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 become an unparalysed state. I believe this policy is already used in many neonatal intensive care units.

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S J Newell and E J Kelly

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