Correspondence

Effects of sleep state and feeding on cranial blood flow of the human neonate

Sir,

We were interested in Rahilly's recent speculation that metabolic changes after feeding may be important factors which could explain the fall in cerebral blood flow he reported one hour after a breast feed in term neonates.¹ Rahilly went on to imply that the metabolic fluctuations induced by bolus feeding regimens in preterm infants could predispose such infants to intraventricular haemorrhage. The concept that phasic changes in metabolism induced by feeding could induce a variety of cyclical physiological events in neonates is an important and a relatively unresearched subject which may indeed have implications for the nutritional management of sick low birthweight infants. We should like to comment on how far Rahilly's suggestions agree with some of our own data² on the metabolic and endocrine effects of feeding term and preterm infants in the first days after birth.

An argument which links postprandial changes in blood flow to metabolic events presupposes that there are indeed changes in metabolism after a feed. With respect to several circulating metabolites and alimentary hormones, many of which change after a feed in adults, we found that in breast-fed term infants on the 6th day and in preterm infants at means of either 2.5 or 6 days of age, there was no postprandial alteration in ketones (acetoacetate and β -hydroxybutyrate), alanine, pyruvate, lactate, glucagon, enteroglucagon, vasoactive intestinal peptide, gastrin, secretin, gastric inhibitory polypeptide, or pancreatic polypeptide. In contrast older neonates (>3 weeks, and older than Rahilly's patients), showed pronounced postprandial changes in many of these factors. We do accept however, that the measurement of circulating metabolites may not necessarily reflect the metabolic effects of feeding, which can be assessed fully only by turnover studies.

Nevertheless, phasic metabolic and endocrine changes after a feed in the first week postnatally are not *totally* absent. Insulin and glucose rose postprandially in all the groups we studied and, perhaps of most interest, the neurotensin response to feeding at this age is pronounced. Neurotensin is one of several gut hormones to have been isolated in both gut and brain.³⁻⁴ In the brain neurotensin is believed to have a neurotransmitter role, and pharmacological studies in animals suggest that in the circulation this hormone may affect vascular tone. Could the neurotensin response to feeding partly explain Rahilly's findings? Clearly the whole subject needs further study.

References

- ¹ Rahilly P M. Effects of sleep state and feeding on cranial blood flow of the human neonate. *Arch Dis Child* 1980; **55**: 265-70.
- ² Lucas A, Bloom S R, Aynsley-Green A. Development of gut hormone responses to feeding in neonates. *Arch Dis Child* 1980; **55**: 678-82.
- ³ Brown M, Rivier J, Kobayashi R, Vale W. Neurotensin-like and bombesin-like peptides: CNS distribution and actions. In: Bloom S R, ed. *Gut hormones*. Edinburgh: Churchill Livingstone, 1978; 550-8.
- ⁴ Buchan A M, Polak J M, Sullivan S, Bloom S R, Brown M, Pearse A G E. Neurotensin in the gut. In: Bloom S R, ed. *Gut hormones*. Edinburgh: Churchill Livingstone, 1978: 544-9.

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Upper airway abnormalities and their functional implications in respiratory disease, especially cot death in infancy

Sir,

The discussion on the interesting findings reported by Wailoo and Emery¹ could have been developed further and therefore might have been less speculative. In particular the relationship between tracheal enlargement and function might have been more conclusive if more information had been provided.

A comparison between cot deaths with and without established respiratory disease would have been helpful. It would have clarified any causative role of respiratory disease in tracheal enlargement and the possibility that an enlarged or distensible trachea might predispose to cot death, perhaps by compromising the respiratory response to airways challenge. Wailoo and Emery¹ referred to previous 'unexplained' findings of vocal cord necrosis in infants dying from cot deaths. As their paper sought a better understanding of abnormal function, surely these two findings could have been compared in the same children. It has been suggested that vocal cord necrosis may be caused by excessive laryngeal adduction, as occurs with the expiratory grunt in respiratory distress.²

We now know that expiratory laryngeal adduction often leading to transient glottic closure and thus to