PostScript

LETTERS

Hypernatraemic dehydration in breast-fed neonates

A retrospective study was carried out using patient files from January 2004 to December 2005 at the Ministry of Health Göztepe Teaching Hospital Neonatology Unit, Istanbul, Turkey. Enrolment criteria included breast-fed term neonates admitted with clinical manifestations of hypernatraemic dehydration (serum sodium level ≥ 150 mmol/l).

In all, 29 cases of hypernatraemic dehydration were identified. Tables 1 and 2 show the demographic data, clinical variables and laboratory values.

Sixteen infants were treated with a fluid bolus of 0.9% saline once or twice with oral feed and 13 infants were given free water for 48–72 h in addition to saline boluses. Peritoneal dialysis was used for two infants with severe acute renal failure. No deaths were reported.

In this report, there is no explanation for hypernatraemia other than inadequate milk intake. Mothers are often primigravidae (59% in our study) with low milk production owing to insufficient lactation caused by poor milk removal from the breast by sleepy and

quiet babies. In our study, 65% of the infants were admitted because of poor suckling.

Interestingly, 45% of the infants were born during winter and only 10% were born during summer, which suggests that lactation failure rather than environmental heat induces hypernatraemia. Besides, 59% of the babies had no fever, showing that fever may have a moderate effect on the aetiology of hypernatraemia.

The proportion of infants with jaundice was 24%, which is considerably lower than the rate of 81% reported by Moritz et al.¹ Our patients might have been admitted with hyperbilirubinaemia earlier, before hypernatraemia ensued, because of racial reasons.

In hypernatraemic dehydration, there may be changes in brain-cell osmolality and cerebral oedema resulting in lethargy and seizure. Rapid correction of plasma osmolality increases oedema and brain damage.² Three infants had seizures in the present study. The infant with a sodium level of 188 mmol/l had a seizure during the rehydration phase, which took 72 h. The other two infants undergoing dialysis treatment had seizures on admission to the emergency department. Their neurological examination at 1 year of age, electrocardiograms and cranial tomographies were normal.

Our hospital gained the title “Baby Friendly” towards the end of 2005, owing to

staff training in breastfeeding support would include location problems. As a result of this training, hypernatraemic dehydration should decrease in our unit. Another way to prevent hypernatraemic dehydration is to weigh the breast-fed infants between 72 and 96 h after birth, as recommended by the American Academy of Pediatrics.³

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Use of buccal midazolam in children

We read with much interest the recent letter on buccal midazolam by Hindley and Jameson.¹

The main reason that some recommend a buccal midazolam test dose is concern about the risk of an adverse reaction, particularly respiratory depression or apnoea, especially should it occur outside hospital. However, there is evidence that buccal midazolam is more effective than rectal diazepam and is not associated with an increased incidence of respiratory depression.²

We recently undertook a survey of epilepsy nurse specialists. Thirty questionnaires were sent to paediatric neurology and epilepsy nurse specialists looking after children with epilepsies from all regions of the UK. Twenty four responses were received (80% response rate). All preferred the buccal to the rectal route and reported an increased use and acceptance of buccal midazolam in recent years. Only 4 of the 24 (16%) responders reported that their patients were routinely given a test dose in hospital for safety reasons, to demonstrate the procedure, to reassure parents or because of local guidance. Conversely, 20 of 24 (84%) of the responders' patients were not routinely admitted to hospital for a test dose. However, 14 of 20 (70%) whose patients were not admitted for a test dose, recommended that the parent or carers call the paramedics if their child was having a prolonged seizure and wait for the paramedics to be present before giving the first-ever dose of buccal midazolam. In all, 20

Table 1 Demographic data, clinical variables and laboratory values in breast-fed infants

Clinical variables	n	Mean
Male	17	
Female	12	
Caesarean section	9	
Vaginally delivered	20	
Born at home	10	
Outborn	19	
Mother's age (years)	18–36*	25
Primigravida	17	
Summer admission	3	
Hyperbilirubinaemia	7	
Fever	12	
Winter admission	13	
Age at presentation (days)	2–12*	5.6
Lethargy and poor feeding	19	
Sepsis	None	
Seizure	3	

*Range

Table 2 Demographic data, clinical variables and laboratory values in breast-fed neonates

Clinical variables	Range	Mean
Birth weight (g)	2100–4400	3350
Weight on admission (g)	1800–3900	2825
Weight loss (%)	6–29	15.5
Sodium (mmol/l)	150–188	160.7
Potassium (mmol/l)	4–7	4.9
Blood urea nitrogen (mg/dl)	18–135	57.9
Creatinin (mg/dl)	0.6–6.6	1.5
Uric acid (mg/dl)	3.1–28	7.2
Glucose (mg/dl)	11–124	70.9

of 24 (83%) of the responders' patients used Epistatus, a proprietary oral solution (10 mg/ml), 2 of 24 (8.3%) used midazolam intravenous solution (5 mg/ml) and 2 of 24 (8.3%) used either preparation. There was no consistent practice with respect to the lower age limit for its use, the recommended dose (0.2–0.5 mg/kg to 2.5 mg for a 6–12-month old, 5 mg for a 1–5-year old, 7.5 mg for a 5–10-year old and 10 mg for a 10–16-year old) or the frequency of repeated doses.

We agree with the authors that the use of routine buccal midazolam test doses in hospital is not supported by current evidence. However, more evidence on the risks and safety of use of both rectal diazepam and buccal midazolam outside hospital is urgently needed.

Therefore, at present, it seems sensible to recommend an individualised approach, after discussion with the epilepsy nurse specialist, the family and carers. Furthermore, there are good reasons to recommend basic life-support training for all citizens, particularly the parents and carers of children with epilepsy, and especially those whose children are prescribed rectal diazepam or buccal midazolam for use outside hospital.

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Tongue-tie and infant feeding

Although for centuries doctors recognised tongue-tie as a congenital anomaly that might interfere with infant sucking, since 1950 paediatricians had emphatically denied that the condition exists or causes a problem.¹ Judging from Mr Mervin Griffiths' recent letter,² it appears that this scepticism persists.

I became convinced in 1960 that tongue-tie sufficiently severe to impair sucking was a real, although uncommon, entity. My experience during the next 28 years suggested that its prevalence was at least 1 in 1000 newborn infants. Diagnosis was made on the basis of the presence of a tight fraenum that prevented the tongue from projecting beyond the lower gum margin, combined with a weak and intermittent suck; when diagnosis had been delayed for any reason, there was a history of slow and difficult feeding and a poor weight gain. Careful division of the tongue-tie in the early neonatal period proved to be straightforward and without complication. Justification for this small operation came from the mothers who testified at once to the improved sucking capacity of their

infant and to the shorter feeding times. In cases where weight gain had been poor, there was usually a dramatic and rapid increase in weight. In one case, I measured the strength and pattern of the suck with a pressure transducer before and after snipping the fraenum. There was a dramatic improvement.¹

The time has come, I think, for those who deny the existence of symptomatic tongue-tie to undertake an objective clinical trial. Among the parameters that might be measured before and after division of tongue-tie are

- a. the strength and pattern of the suck;
- b. the time taken to complete a feed or to ingest a given volume of milk;
- c. the rate of weight gain.

It is splendid news that NICE³ has recently recognised that tongue-tie may interfere with infant feeding and has supported the need for frenotomy when required.

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Prejudice against test weighing, like tongue-tie division, should be re-assessed

I read with interest the dialogue stemming from Hall and Renfrew's article about tongue-tie, and the interest in designing trials to empirically measure the effects of tongue-tie on breastfeeding. I have photographed many tongue-tied babies. It is my impression that the degree of lift and ability to sustain lift of the tongue provides information about tongue mobility.¹ Measuring lift is described in the literature² and may prove a useful diagnostic indicator. Ultrasound studies provide another fruitful area of research, and preliminary studies by Ramsay *et al*³ show changes in the breastfeeding behaviour of tongue-tied infants before and after frenotomy.

Secondly, I disagree with the prejudice that test weighing creates undue parental stress and must therefore be ruled out as a way to measure deficits and improvement in feeding behaviour. Having used smart scales for many years in my private practice, I find them to be quite effective in terms of helping identify infants who are transferring milk poorly and growing at below reference standards. Although the place to begin assisting any breastfeeding problem is with correcting the latch and helping poor feeders obtain more access to the breast, test weights provide a very useful tool.⁴ They often confirm parental impressions that their baby is struggling to get enough to eat. This confirmation has not, in my experience,

stressed the mothers. They are already stressed owing to painful, unhappy feeds. Information helps everyone know where they stand, affirms the mother's concerns and provides a starting place to find a solution.

Test weights of the same infant before and after frenotomy provide one way to measure improved ability to breastfeed.⁵ Good counselling skills help support the parents when weighing is undertaken. Other researchers have found test weighing to be useful and non-stressful.

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NICE guidelines and the epilepsies: how should practice change?

I read with interest the article by Dunkley and Cross.¹ I looked at service delivery for a secondary care epilepsy service using data from a prospective study of all patients attending an average-sized district general hospital (DGH) over a period of 1 year.²

One particular area of interest is the concept of the paediatrician with an interest in epilepsy, the role which Dunkley and Cross say is the key to the proposals. In this study, all five consultants saw children with epilepsy, and for 211 children (60 newly diagnosed cases) with epilepsy a total of 544 outpatient appointments were given during the year studied, of which 30% were in an epilepsy clinic, 55% in a general clinic and 25% in a child development clinic. If we were to plan a dedicated epilepsy service for all patients delivered by one consultant offering 30-min appointments for new patients and 15-min appointments for return patients allowing for a 10% non-attendance rate, study leave and annual leave, it would require one and a half clinics per week to provide a service. If we were to include all the children with non-epileptic episode disorders, up to three clinics per week may be required.² Most paediatricians in a DGH have three clinics per week in their job plan. The paediatrician with a special interest could therefore become a full-time seizure specialist. Our department now has a consultant of