

PostScript

LETTERS

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Weighing alone will not prevent hypernatraemic dehydration

Having recently reviewed the case notes of babies readmitted to hospital in the first 10 days of life (over a one year period), we firmly agree with the views expressed by Laing and Wong.¹ The incidence of documented hypernatraemic dehydration secondary to the failure of lactation in Bristol is 1.7 per 1000 live births much higher than that described by Oddie *et al* in the Northern Region (2.5 per 10 000 live births).² In addition only 50% of infants readmitted with weight loss of <10% had a plasma sodium concentration measured. The true incidence of hypernatraemic dehydration secondary to lactation problems in Bristol could thus be as high 3.4 per 1000 live births. Our estimate could be an underestimate. Firstly, our study looked only at infants readmitted within 10 days (Oddie *et al* looked at infants readmitted up to 1 month of age) and secondly, due to failure to recognise of this condition.

Laing and Wong proposed weighing all infants when the Guthrie blood samples are taken, to identify those infants at risk of dehydration.¹ We believe that this is too late as in many areas this occurs on days seven or on day 10 with handover of care to the health visitor. We have already described a series of babies with hypernatraemic dehydration where all presented to hospital before day seven.³

The case has been made correctly that newborn hypernatraemia is due to unsuccessful feeding.^{1,4} While we agree that careful examination and observation of the infant while feeding and so forth may identify these babies, we would dispute that this is currently universally possible. Due to midwifery shortages, postnatal wards are short staffed and community midwives are fully stretched, so many women are discharged within a few hours of delivering. If a midwifery home visit does not coincide with a feed, the mother's assessment of feeding is assumed to be correct (as indeed it usually is). Weighing the baby will reassure most mothers that their baby is following the normal pattern of loss followed

by gain. Identification of excessive weight loss should prompt the health professional to examine the baby for evidence of illness and carefully observe breast feeding technique. These mother-baby dyads could then be given additional support and advice in the community and thus successfully establish feeding. In our experience once the baby has become ill and required readmission to hospital the mother is reluctant to continue to attempt to breastfeed.

There continues to be confusion regarding the best way to manage this problem.¹⁻⁵ It should be remembered that these babies have normal guts and are suffering from starvation. If the infant is not shocked, rehydration can occur safely using enteral fluids: expressed breast milk or a breast milk substitute. Serum sodium should be measured six hourly initially and the volume of milk altered to ensure a slow return to normality.

We believe that we need to foster a greater awareness of this problem and weigh the babies at risk around day five if we are to prevent tragedies resulting from a common condition affecting otherwise well babies.

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Hypernatraemic dehydration: excess sodium is not the cause

I am grateful to Laing and Wong for raising once again the issue of hypernatraemic dehydration in the first few days of life.¹ However, I think it is important to remember that hypernatraemic dehydration, like anaemia, is a sign of disease and not a diagnosis in itself. A low haemoglobin concentration in blood can be caused by a large number of different pathological and physiological processes. Hypernatraemic dehydration should be seen in the same light.

Laing and Wong's article describes two situations in which a child can be found to exhibit the typical biochemical and clinical features of hypernatraemic dehydration, that is—weight loss and hypernatraemia. The first mentioned is associated with gastroenteritis in a bottle fed infant, commonly a few weeks old and the second is seen in "breast fed" infants in the first few days of life. The hypernatraemia associated with these situations is caused by different problems with water balance—in neither is the problem an increased intake of sodium. In the infant with diarrhoea there is an excess loss of water and in the "breast fed" baby an insufficient intake of water.

In discussing hypernatraemic dehydration in association with diarrhoea in young infants, Laing and Wong refer to a paper by

Chambers and Steel, where attention is drawn to the slightly increased concentration of sodium in artificial milk mixed incorrectly by parents.² This is a red herring. The excess sodium concentration of the artificial milk mixed incorrectly by the mothers reached a maximum of 59 mmol/l with a mean of 37.2 mmol/l. Those who believe that this concentration of sodium could be responsible for hypernatraemic dehydration should remember that the concentration of sodium in the standard oral rehydration solutions in use in the UK is either 60 mmol/l (Dioralyte, Dioralyte rebel, Diocalm junior) or 50 mmol/l (Rehidrat, Electrolade) and that the WHO formulation for oral rehydration solution contains 90 mmol/l of sodium.

In fact the cause of this association of hypernatraemic dehydration with diarrhoea is the continued feeding with artificial milk after the onset of diarrhoea. The intestinal hurry associated with gastroenteritis results in the delivery of a solution rich in protein and complex carbohydrates to the colon which, after digestion by colonic bacteria, produces a considerable osmotic load in the colon, which in turn results in the production of voluminous stool low in sodium.³ The result is hypernatraemic dehydration due to excessive water loss. Those who require further discussion of this hypothesis are advised to read the excellent paper by Hirschhorn.³

The second situation relates to the title of the piece, namely hypernatraemic dehydration in the first few days of life in association with "breast feeding". Though the breast milk produced, in very small quantities, by the mothers of these children is often found to contain a high concentration of sodium, this has nothing to do with their babies' hypernatraemic state. As Jack Newman puts it so eloquently in his electronic response to Laing and Wong, these babies are not dehydrated because they are breast fed but because they are only pretending to breast feed. They are, in fact, starving. This is amply illustrated by the case described in Oddie *et al* of a "bottle fed" baby admitted at 6 days of age with hypernatraemic dehydration whose dehydration had nothing to do with the bottle milk being "fed" to her but was caused by the fact that she had oesophageal atresia.⁴ Hypernatraemic dehydration is frequently seen in the elderly and the mentally handicapped when their need for basic care, and presumably a regular intake of water, is neglected.⁵⁻⁷

Hypernatraemic dehydration is a sign of illness not a diagnosis. It is commonly caused by excess water loss or by insufficient water intake, either alone or in combination. It is almost never the result of excess sodium intake, which would result in retention of water and an increase in body weight, though this would obviously require intact thirst mechanisms and access to sufficient water.

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Hypernatraemia in the first few days: a tragic case

Laing and Wong¹ highlight the fact that hypernatraemic dehydration can be difficult to recognise and may have serious consequences. We describe an extreme case.

An 8 day old infant was admitted to hospital with a small haematemesis. She had lost 19% of her birth weight and her plasma sodium was 173 mmol/L. She had renal and hepatic impairment and was found to have a thrombosis of the descending aorta. In spite of rehydration, thrombolysis and full intensive care support, she died the following day from progressive subdiaphragmatic ischaemia and multiorgan failure. A postmortem revealed no underlying abnormalities. Parental thrombophilia screens were normal.

The maternity notes revealed that she was born at term weighing 3.18 kg after an uneventful pregnancy. She was breastfed from birth. On day 3 she had five wet nappies and appeared to be feeding well. She was allowed home, with a discharge weight of 2.77 kg, and visited several times by the community midwives. On each occasion she seemed contented and was thought to be feeding well.

We agree with Laing and Wong that health professionals may fail to realise how dehydrated some infants have become until they are dangerously unwell. Our infant had lost 12.8% of her birth weight at initial discharge. The severity of this weight loss was not recognised and no further attention was paid to the weight as the baby was considered to be feeding well. To target at-risk infants, we consider that clinical skills need to be supplemented with a clear policy of regular weight monitoring with defined thresholds for intervention.

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Systemic and cerebral blood flow

We read with interest the findings of Evans *et al* concerning the changes in middle cerebral artery (MCA) haemodynamics over the first two days after birth in preterm infants.¹

Evans *et al* demonstrated a significant change in several Doppler velocity measures in the MCA, including systolic, diastolic, and mean velocity, pulsatility index and velocity time integral, during days 1 and 2 after birth

in a cohort of preterm infants. Blood flow (F), blood pressure (P), and vascular resistance (R) are closely related ($F = P/R$), and resistance changes are a function of changing vessel calibre. The authors attempted to measure MCA diameter, but because measurements were inaccurate the authors did not attempt to calculate values for MCA blood flow.

Our own studies of cerebral haemodynamics, using near infrared spectroscopy, supports the view of Evans *et al* that there are significant changes in cerebral blood flow over the first 36 hours after birth. The demographic details of our cohort were similar to the one studied by Evans *et al*, with 36 infants of mean (SD) gestation 26 (2) weeks and mean birth weight 929 (250) g. We found that a significant increase in cerebral blood flow between days 1 and 2 was accompanied by a significant decrease in cerebral fractional oxygen extraction (FOE). High cerebral FOE may protect the brain from hypoxicischaemic injury, a potential consequence of reduced cerebral blood flow.

The results presented by Evans *et al* give an insight into the complex relationships that exist within a dynamic fluid system. Systemic blood pressure was closely related to MCA mean velocity, but not estimated MCA diameter, thus implying that cerebral blood flow would vary independently of systemic blood pressure because of changes in cerebrovascular resistance. Our own observations have produced similar results. Cerebral FOE, which is expected to increase as cerebral blood flow decreases, is not related to mean arterial blood pressure.² There is, however, a significant relationship between cerebral FOE and left ventricular output, which is a major determinant of central blood flow.² This latter finding is in agreement with the observation of Evans *et al* that superior vena cava flow, also related to central blood flow, was significantly associated with estimated MCA diameter.

Evans *et al* remind us that velocity is not the same as flow. Their observations, and our own, stress the importance of vascular resistance in mediating the relationship between blood pressure and blood flow. In the sick preterm infant, the presence of an adequate mean arterial blood pressure is often achieved using pharmacological methods. Although this is reassuring, it does not guarantee the presence of good central blood flow, nor the adequate perfusion of the end organs, including the brain.

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Transcutaneous bilirubin measurement in newborn infants: evaluation of a new spectrophotometric method

Transcutaneous bilirubinometry in jaundiced newborns has been extensively evaluated in the literature.¹ Information about the new

BiliCheck (TcBC) device has appeared in recent years.^{2,3} Because of the high correlation coefficient, most studies conclude that transcutaneous bilirubinometry could possibly replace the laboratory measurement of serum bilirubin (TSB). However, the number of infants with serum bilirubin levels > 257 $\mu\text{mol/l}$ (15 mg/dl) is too small to assess the accuracy of TcBC for such infants.^{4,5}

We tested the clinical usefulness of this new device and compared it with the established Jaundice Meter (TcBM) by evaluating the levels of agreement, with TSB as the clinically used standard. Bilirubin levels were measured by TcBC, TcBM, and TSB in 122 healthy newborns (gestational age 34–42 weeks, mean 39 weeks; mean (SD) birth weight 3187 (582) g) during the first days of life. For TSB the Vitros 250 BuBc slide was used. For statistical analysis, the correlation coefficients were calculated and the difference plots determined by the Bland-Altman method.⁶

TSB levels ranged between 13.7 and 339 $\mu\text{mol/l}$ (0.8 and 19.8 mg/dl) (mean 186.6 $\mu\text{mol/l}$ (10.89 mg/dl), median 193.6 $\mu\text{mol/l}$ (11.3 mg/dl)). The correlation coefficient, *r*, was 0.92 between TcBC and TSB, and 0.85 between TcBM and TSB. The plot of differences of TcBC against TSB yielded a maximum range of -109.9 to $+56.5$ $\mu\text{mol/l}$ (-6.3 to $+3.3$ mg/dl), and TcBM against TSB -123.9 to $+107.9$ $\mu\text{mol/l}$ (-7.23 to $+6.3$ mg/dl). The 95% limits of agreement were between -61.5 and $+55.3$ $\mu\text{mol/l}$ (-3.59 and $+3.23$ mg/dl) for TcBC, and -93.4 and $+80.2$ $\mu\text{mol/l}$ (-5.45 and $+4.68$ mg/dl) for TcBM.

Although the correlation coefficients give the impression that TcBC and TcBM provide an accurate estimate of TSB, the differences between transcutaneous measurements with either device and TSB values were often quite large. In particular, if TSB levels were higher than 188 $\mu\text{mol/l}$ (11 mg/dl), the measurements of both instruments progressively deviated to lower bilirubin levels than TSB. This leads to a dangerous underestimation of TSB, especially if a decision on treatment has to be made.

We conclude that transcutaneous bilirubinometry with the BiliCheck can be used as a screening tool in the evaluation of hyperbilirubinaemia, but it cannot replace laboratory measurements of serum bilirubin. Given the wide limits of agreement, we recommend that a venous blood sample is taken, if the difference between a potential phototherapy limit and the TcBC level falls below 60 $\mu\text{mol/l}$ (3.5 mg/dl).

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Hyponatraemia as a consequence of serial liquor punctures in preterm infants with a ventricular access device after posthaemorrhagic hydrocephalus

We observed hyponatraemia in several preterm children treated with a ventricular access device (Rickham reservoir)¹ after intraventricular haemorrhage (IVH)² and serial liquor puncture to drain liquor. To rule out a connection, we retrospectively investigated the data of all preterm children (23–32 weeks of gestational age) treated at the University Children's Hospital of Cologne with a ventricular access device during 1996–1999 (n = 16).

Sixteen of 480 preterm infants (3.3%) of less than 1500 g birth weight (430–1500 g) developed an IVH and required treatment with a ventricular access device. The mean gestational age of these children was 27 weeks (range 23–32).

Twelve of them (75%) developed hyponatraemia (< 130 mmol/l). The minimum serum sodium of all patients was 110–136 mmol/l (mean (SD) 125.8 (6.3) mmol/l). The maximum amount of liquor tapped a day was 3–34 ml (mean 15.6 ml). The resulting daily loss of sodium in the tapped liquor was 0.4–3.7 mmol/kg/day (mean (SD) 1.98 (0.94) mmol/kg/day). The extent of the hyponatraemia (minimal serum sodium) correlated significantly with the maximum daily sodium loss in liquor ($r = 0.78$, $p < 0.001$, fig 1).

Further analysis of the use of drugs—for example, thiazides—did not contribute to this correlation. Two children with hyponatraemia

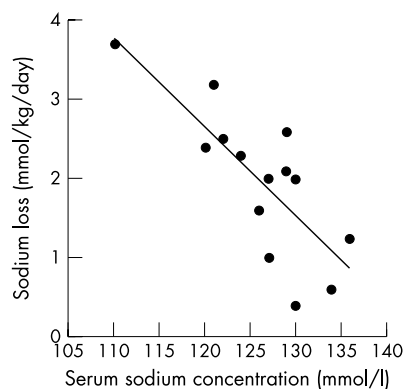


Figure 1 Relation between serum sodium concentration and maximum sodium loss in the liquor ($r = 0.78$, $p < 0.001$).

developed general hypotonia with poor feeding; this prompted further diagnostic measures to exclude syndrome of inappropriate antidiuretic hormone (SIADH) or excessive sodium loss in urine. The investigations were negative. Both children showed normal neurology after adequate replacement of the sodium lost. No child with hyponatraemia developed other acute neurological symptoms such as seizures.

This is the first report of hyponatraemia as a consequence of serial liquor punctures with a ventricular access device in children.³ The sodium loss was sometimes as high as the normal sodium requirement per day (3–5 mmol/kg/day).

Hyponatraemia in children caused by the use of a ventricular access device should be managed carefully and the sodium replaced promptly. Loss of sodium by serial liquor tapping must be taken into the differential diagnosis of hyponatraemia in preterm infants.

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Use of animal surfactant: should we seek consent?

Animal derived surfactants such as Curosurf (porcine) and Survanta (bovine) are the commonly used surfactants in the United Kingdom. Involvement in a trial of a new artificial surfactant, and the specific information on the origins of the surfactant in the patient information leaflet led us to review our practice. Two families declined to participate in the trial. A Hindu family wished to avoid use of Survanta, as cows are considered sacred in Hindu religion. A Moslem family preferred to avoid porcine products. Having reviewed our own practice, we were unsure as to the practice of other units.

We telephoned the second on call doctors in 42 teams providing newborn resuscitation and initial surfactant therapy to preterm infants in England and Wales. Respondents were asked about which surfactant was available and whether the constituents were usually discussed with parents.

Only nine of 42 respondents said that they would routinely discuss the constituents of the surfactant with the families and could remember having done so in the recent past.

Twenty two respondents in England said that their units only stocked Curosurf, two units stocked Survanta, and three units stocked Curosurf and Survanta. One did not

know which surfactant was available. In Wales, 13 units had only Curosurf and one unit stocked both Curosurf and Survanta.

We were surprised by the number of people who had thought about this being a possible problem. With many units choosing to stock only one surfactant, we think that it is important to keep all parents fully informed both of the importance of early administration and the nature of the available surfactants.

We suspect that, when fully informed, most parents would agree to a life saving medicine. However, we are not sure if this consent should be presumed where there are grounds to wonder if this may be a problem. Individual families still need to make their own decisions to avoid the perception that the medical profession has a patronising attitude.

We hope to generate a discussion to see if a consensus can be evolved.

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Intravenous propacetamol overdose in a term newborn

Following a prescribing error, a term female infant was given two intravenous doses of 900 mg propacetamol (307 mg/kg/dose) at 6 hour intervals, which is 10 times the routine dose used in our unit (120 mg/kg/day, 30 mg/kg/dose). When the error was noted, immediately after the second dose, the plasma paracetamol level was 165.8 mg/l. N-Acetylcysteine was given as follows: 150 mg/kg (430 mg) after 15 minutes, 50 mg/kg (145 mg) after four hours, and 100 mg/kg (290 mg) after 16 hours. Plasma paracetamol levels were checked: 119.9 mg/l five hours later, 61.4 mg/l 11 hours later, 28.8 mg/l 16 hours later, and finally 1 mg/l 24 hours after the second dose (fig 1). Liver function and clotting factors were normal. The infant was discharged on day 7.

Paracetamol poisoning in newborn babies is usually due to either maternal absorption of high doses of the drug just before birth¹ or oral absorption of an inappropriate dose.² Reports of propacetamol overdose are unusual, and so far the overdose has only been by intramuscular injection.³ As far as we know, this is the first report of intravenous propacetamol poisoning in a newborn. This reason may be the rare use of this drug during the neonatal period, the pharmacokinetics having been

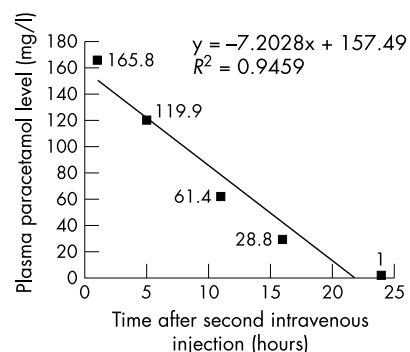


Figure 1 Serum paracetamol levels after administration of N-acetylcysteine.

published in only one study for this stage of life.⁴ However, as with other routes of administration described in the literature, no adverse effects were seen in this case. The administration of N-acetylcysteine following guidelines given for older patients proved efficient. The elimination of the drug seems to be linear. Although drug overdose should be carefully avoided, intravenous propacetamol is probably safe in term newborn babies.

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Endotracheal tube fixation in neonates

A stitch in time saves nine. But not all neonatal units believe in this saying and use different methods to secure oral endotracheal tubes in neonates who require ventilatory support. Success in stabilising a premature infant is best achieved by least intervention and good ventilatory support. A stable oral endotracheal tube will help. A naso-oral endotracheal tube is extremely easy to stabilise; however, stabilisation is not routinely performed in the United Kingdom.

Three commonly used methods are: (a) stitching the tube to a plastic flange; (b) fixing a premeasured and cut tube in a flange with adhesive tape; (c) fixing a premeasured and cut tube into a tight fitting flange. In all three methods, the tube is secured by tying it to the baby's hat.

Normally, weight or foot length is used to determine endotracheal tube size, and this is quite reliable. However, head movement, suctioning, and patient care can all cause instability and displacement of the tube.^{1,2} If the tube is too short, there will be ineffective ventilation. If the tube is too long, it may collapse resulting in selective ventilation. A precut tube is difficult to manipulate if the positioning is not satisfactory. This is not a

major problem in a stitched tube. There are pros and cons to each method.

There are no comparative studies from the United Kingdom to evaluate the benefits and disadvantages of each method. A search through the databases found no randomised trials comparing various techniques, except one study which compared an umbilical clamp with the routine fixing method.

Accidental extubation or unsatisfactory positioning of the tube may influence the reintubation rate. Securing and properly stabilising an endotracheal tube can solve this problem to a large extent.

A prospective randomised trial evaluating each method against reintubation criteria will help neonatal units to adopt the correct policy for their own situation.

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Hypernatraemia in the first few days: a tragic case

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