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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.

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. 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 μmol/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.

TSB levels ranged between 13.7 and 339 $\mu mol/l$ (0.8 and 19.8 mg/dl) (mean 186.6 $\mu mol/l$ (10.89 mg/dl), median 193.6 $\mu mol/l$ (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 $\mu mol/l$ (–6.3 to +3.3 mg/dl), and TcBM against TSB –123.9 to +107.9 $\mu mol/l$ (–7.23 to +6.3 mg/dl). The 95% limits of agreement were between –61.5 and +55.3 $\mu mol/l$ (–3.59 and +3.23 mg/dl) for TcBC, and –93.4 and +80.2 $\mu mol/l$ (–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 µmol/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 µmol/l (3.5 mg/dl).

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