Correspondence

Copper deficiency and non-accidental injury

Sir,

We write to congratulate Dr Jonathon Shaw on his comprehensive and balanced review of copper deficiency in infancy and its relation to fractures diagnosed as non-accidental injury.1 We trust that it will put an end to poorly supported attempts on the part of some expert witnesses to ascribe fractures to copper deficiency in circumstances when it is manifestly not present. These attempts have been the subject of adverse comment in the Appeal Court2 and Family Division of the High Court3 in both criminal and wardship proceedings. The waste of time, money, and emotional damage which results from the protracted and complicated legal proceedings is incalculable.

Dr Shaw together with Dr Carty,4 in the same issue of your journal have established clearly the criteria which should apply before the diagnosis of copper deficiency is considered possible and have shown that proper analysis of clinical, radiological, and laboratory criteria even in the absence of serum copper concentrations are quite adequate in most cases. It is our view, borne out by the data and arguments presented by Drs Shaw and Carty, that infants who sustain fractures but do not have recognised risk factors for copper deficiency, have normal bone structure, and no haematological abnormalities do not need to have copper and caeruloplasmin concentrations measured on clinical grounds. It is questionable whether it would be ethical to do them for purely forensic reasons in the absence of reasonable evidence suggesting that they might be abnormal. We are concerned at the prospect of multiple requests for serum copper determination. It would be unfair both to the infants and the laboratories.

While we accept that premature infants may represent a special risk group because of diminished hepatic copper reserves, many of the references cited by Shaw involve infants given outmoded parenteral feeding formulae or receiving partial enteral feeds. With the use of modern crystalline amino acid parenteral feeding solutions together with a trace element solution (supplying 0.3 μmol/kg/day of copper) we have found copper deficiency rare.

References

2 Regina versus Lees and Lees, Lord Justice Lane 1987.
3 Judgement of the Honorable Mr Justice Hollis in Wardship Proceedings, Middlesbrough 1987.

Urinary creatinine excretion in the newborn

Sir,

Al-Dahhan et al claim that their data are only the second to report the urinary excretion rate of creatinine in preterm infants,1 and are apparently unaware of our 1985 paper which theirs closely mimics.2

We measured the urinary excretion rate of creatinine in babies of similar gestational ages (26 to 40 weeks), weights (640 to 3710 g), and postnatal ages to Al-Dahhan et al, and produced almost identical results and conclusions. We found that the rate varied with weight, surface area, length, gestational age, and postnatal age, but that the relation with body weight was the most powerful, so that there was no correlation between urinary excretion rate of creatinine/kg and most of the other parameters. The only difference between our findings is that we found the urinary excretion rate of creatinine/kg to be slightly higher in the first week of life than subsequently. We presented these results as arithmetic means (SD) in mmol/kg per minute. Converting to μmol/kg daily our values of 104 (24) in the first week of life and 95 (19) subsequently were almost identical to the overall figure of 95.7 (33.3) reported by Al-Dahhan et al.

Al-Dahhan et al argue that a knowledge of the urinary excretion rate of creatinine should allow the urinary excretion rates of other substances to be estimated without the need for timed urine collections; in our paper we showed this to be the case for sodium and potassium. Although the 95% confidence intervals for these estimations are wide at 62 to 161%, we have found them to be narrow enough to provide useful clinical information. Using our data, the daily excretion rate of a substance, x, per kg is 104 Ux/Uc for infants in the first week of life and 95 Ux/Uc for older neonates, where Ux is the urine concentration of the substance in units/l and Uc is the urine creatinine concentration in μmol/l. We proposed that for clinical usage the formula 100 Ux/Uc would be more easily memorable with little loss of accuracy. We feel that the data of Al-Dahhan et al further strengthen our conclusion.

Using the same principle, we also showed that the urine flow rate per kg can be estimated with a similar degree of accuracy from a knowledge of the urinary excretion rate of creatinine/kg and the creatinine concentration of an untimed ('spot') urine sample. The daily urine excretion (ml/kg) is thus 104/Uc in the first week of life or 95/Uc thereafter, or can be approximated to 100/Uc for convenience, but it must be noted that in this case Uc is the urinary creatinine concentration in mmol/l.

References

Patient triggered ventilation in premature neonates

Sir,
Greenough and Greenhall recently reported their experience of the trigger ventilator. They modified our original circuit by switching the inspiration detector from its site on the abdomen to the oesophagus. We had considered this approach but felt that it had a limited role because it could not be used on a routine basis by untrained staff. In their paper they allege that ‘gross body movements’ made the abdominal sensor prone to false triggering. This has never been a problem in my experience over the last three years. While it is obviously theoretically possible to interfere with the signal from the capsule, in practice the design of the circuit largely eliminates this problem. This is best illustrated by the example of an infant on triggered ventilation with an inspiratory time set at its recommended 0.5 seconds. When a breath occurs there is a delay of up to 100 msec before the trigger fires. The ventilator is refractory to further signals from the time of firing, through the inspiratory time setting (0.5 seconds) and for 0.25 seconds after this interval (to allow enough time for expiration). Most infants breathe at 60 breaths (approximately) per minute with this inspiratory time (unpublished work). Thus on average the ventilator is refractory for 0.85 seconds out of the 1 second between breaths and only 0.15 seconds remain as a susceptible period before the next breath is due. The only consequence of such a false signal is to make the next breath occur fractionally early.

I submit therefore that the criticism of our original approach is invalid and that triggered ventilation using an abdominal sensor is an effective means of assisting the newborn’s respiratory efforts. 5

References

4 M G COULTHARD, E N HEY, and V RUDDOCK
Central Sector,
Royal Victoria Infirmary,
Queen Victoria Road,
Newcastle upon Tyne NEI 4LP

Infecctive endocarditis in neonates

Sir,
We read with interest the recent review of five patients with neonatal infective endocarditis by O’Callaghan and McDougall.1 We wish to report a further case where echocardiography had an essential role in the early diagnosis of pericarditis and endocarditis in a newborn infant where clinical signs specific to endocarditis were absent.

A boy weighing 3200 g was born vaginally at term to a primigravida who had loose bloody stools due to Shigella sonnei infection during the last trimester of pregnancy. The infant developed shigella enteritis with intermittent gaseous abdominal distension. Ultrasound examination showed an abdominal abscess but during this procedure the pericardial effusion was incidentally detected. Although the infant was ill and looked septic, arterial pulses and blood pressure were normal. The heart sounds were normal with no evidence of a murmur or pericardial rub. The chest radiograph showed cardiomegaly but the electrocardiogram was normal. A septic spot was noted on the sternum. A peripheral blood count showed chromytopenia but no petechial lesions were present. There was no haematuria.

Cross sectional echocardiography confirmed the presence of a large anterior pericardial effusion as well as a vegetation on the anterior leaflet of the mitral valve. Thirty ml of pus was obtained by needle aspiration of the pericardial space. Subsequent surgical exploration was performed and the pericardial space was left on continuous drainage for one week. An identical strain of Staphylococcus aureus was isolated from the pericardial fluid, blood cultures, and the septic lesion on the sternum. Gentamicin and cloxacillin to which the organism was sensitive were given intravenously for six weeks. After one week of treatment the pericardial effusion had resolved but the vegetation persisted. Umbilical arterial or venous lines were not used at any time during his management. He was discharged from hospital at the age of 3 months. At that time his cardiac examination was normal; in particular there was no evidence of mitral valve insufficiency. Echocardiography confirmed persistence of the mitral valve vegetation which had become more echodense and possibly calcified. After discharge from hospital the infant was lost to follow up.

Clinical detection of bacterial endocarditis in the newborn who is already ill from sepsis is difficult. Embolic phenomena, congestive cardiac failure, and changing heart murmurs are important clues but are infrequently found.2 Cross sectional echocardiography permits earlier diagnosis of endocarditis provided vegetations are at least 2 mm in diameter.3 We surmise that survival of our patient was due to early diagnosis and prompt treatment with appropriate antibiotics in high doses, and the maintenance of normal tissue perfusion and blood pressure in the presence of overwhelming septicemia. We concur with O’Callaghan and McDougall in the usefulness of echocardiography in the diagnosis of neonatal endocarditis. We have shown that with early diagnosis recovery is possible.