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Brittle or battered?

Sir,

My attention has been drawn to correspondence about this subject. There is reference to an article in which I reported study of a series of children with osteogenesis imperfecta with special reference to the occurrence of metaphyseal fractures.¹

In his letter Dr Blumenthal states that 'metaphyseal fractures are a feature of both abuse and brittle bones'.² This is true but needs qualification. In non-accidental injury the bones appear grossly normal and the metaphyseal fractures are often numerous. In osteogenesis imperfecta fractures of this type are infrequent and rarely number more than one or two. They were only seen in my series where there was *gross abnormality of the skeleton* and the diagnosis of the presence of a systemic bone disease was apparent at a glance.

To quote my last sentence, 'Confusion with non-accidental injury did not occur'.

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Somatostatin analogue in short term management of hyperinsulinism

Sir,

We are interested by the report of Kirk *et al* of the use of somatostatin analogue for the short term management of hyperinsulinism in which a fall in glucose requirement of 4-6 mg/kg/minute was seen.¹ We have previously reported similar success, in the short term control of hyperinsulinism in an infant with nesidoblastosis, using growth hormone twice daily by subcutaneous injection.² A fall in glucose requirement of 5.5 mg/kg/minute was seen. The effect of growth hormone in the treatment of hypoglycaemia is reported elsewhere.^{3,4}

At the time of our report further studies of this use of growth hormone were precluded by the withdrawal of pituitary growth hormone after reports of Creutzfeldt-Jakob disease. Now that a growth hormone produced by recombinant DNA technology is available, however, the choice of growth hormone over a somatostatin analogue has advantages.

Somatostatin has a very broad range of endocrine activity,¹ with unwanted effects upon other endocrine axes that do not appear to be produced by growth hormone. Furthermore, on simply practical grounds growth hormone, unlike somatostatin, is available in most regional centres.

In the infant with hyperinsulinism, when it is necessary to temporise before surgery, we believe that growth hormone may be preferable, and its use certainly merits further study.

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Disialotransferrin developmental deficiency syndrome and olivopontocerebellar atrophy

Sir,

I read with great interest the paper of Kristiansson *et al* about disialotransferrin developmental deficiency syndrome.¹ I would like to draw attention to the remarkable similarity between this syndrome and a condition recently reported in two siblings as olivopontocerebellar atrophy with neonatal onset.² Common features included failure to thrive, hypotonia, developmental delay, joint restrictions, pericardial effusions, mild non-progressive liver disease, retinal dystrophy, and cerebellar hypoplasia.

Moreover there was thyroxine binding globulin deficiency in the patients reported by Harding *et al*,² and although serum concentrations of thyroxine binding

globulin were not reported in the paper of Kristiansson *et al* thyroxine binding globulin deficiency was a constant feature in our patients who presented with a closely related condition.³ The only difference was the clinical course of the condition, which was fatal in both patients (at 7 and 23 months) of Harding *et al* whereas the patients of Kristiansson *et al* and those reported by us were all alive (age 3 to 21 years).

I therefore recommend a search for defects in glyco-protein metabolism (particularly in the processing and synthesis of the carbohydrate moiety) in patients with olivopontocerebellar atrophy. A valuable screening method for this purpose is the carbohydrate deficient transferrin test devised by Stibler *et al*.⁴

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Clostridium difficile in an oncology unit

Sir,

Brunetto *et al* describe a cluster of patients with *Clostridium difficile* and conclude that an outbreak had occurred.¹ However, I wonder whether this might, at least in part, have been a pseudo-outbreak. A previous paper reported a cluster of cases, but serotyping of the isolates showed that four strains were circulating and other investigations showed the cause of the outbreak to have been a virus rather than *C difficile*.²

In the incident reported by Brunetto *et al* only two of 21 patients had severe symptoms and in only 18 of 31 (58%) episodes of *C difficile* associated with diarrhoea was toxin detected. Colonisation rates almost as high as 40% have been reported for *C difficile* in oncology units in the absence of overt problems,³ hence positive cultures in the absence of detectable toxin is of questionable significance.

Although the epidemic curve in the current incident appears most impressive, could the rise in incidence merely represent an increased level of investigation for *C difficile*

rather than a genuine increase in prevalence? Typing the isolates by one or more methods might have strengthened or refuted the interpretation the authors place on their data.

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Dehydration fever in the neonate—a common phenomenon?

Sir,

Rises in temperature are seen occasionally on the third to fourth day of life in infants who are otherwise well, and overheating and sepsis as causes of fever must be excluded.¹ Dehydration is perhaps an infrequently recognised cause of fever in the newborn period.

Case report

A boy was born at term after a normal pregnancy and labour weighing 4000 g. The initial examination at 36 hours of age showed a temperature of 37.7°C but he was normal otherwise. He was breast fed and remained well until the age of 3 days when he developed a tachycardia and irritability and was found to have a temperature of 38.1°C that rose rapidly to 39.2°C. Investigations showed a serum sodium concentration of 153 mmol/l and a metabolic acidosis; plasma glucose and creatinine, full blood count, a chest radiograph, and cranial ultrasound scan were normal. Urine and blood cultures were sterile. After resuscitation with plasma the temperature fell to 37.9°C. The infant fed ravenously from the bottle. At discharge the plasma sodium concentration and urine osmolality had returned to normal and his weight had risen from 3620 g to 3810 g (figure).

The clinical features and results of investigations supported dehydration and excluded sepsis and overheating as the cause of this infant's fever. The dehydration was due probably to an inadequate intake of milk. Dehydration fever is seen most commonly in large breast fed babies whose milk intake is poor or in infants exposed to high environmental temperatures, with rises in temperatures to between 37.8 and 40°C.² Infants appear unhappy and may lose approximately 10% of their birth weight. Diagnosis