

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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J JAEKEN
Department of Paediatrics,
University Hospital Gasthuisberg,
49 Herestraat,
B-3000 Leuven,
Belgium

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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J W HARTLEY
Department of Medical Microbiology,
Withington Hospital,
West Didsbury,
Manchester M20 8LR

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