

LETTERS TO THE EDITOR

Carbohydrate intolerance after rotavirus gastroenteritis: a rare problem in the 1990s

EDITOR,—During the first three months of 1993, 32 children with rotavirus gastroenteritis were admitted to our unit (37% of admissions with acute gastroenteritis). Standard treatment was given, that is oral rehydration therapy for 24 hours followed by immediate return to full strength standard formula milk in those under 1 year and light diet for 24 hours followed by reintroduction of 'doorstep' milk in those over the age of 1.¹

Carbohydrate intolerance (>0.5% reducing substances in the stool) was seen in 16 (50%) of the children admitted with rotavirus gastroenteritis; all had watery explosive diarrhoea. Monosaccharide intolerance was present in 11 (10 aged >1 year), lactose intolerance in four (three aged <1 year) and one child had glucose polymer intolerance.

Children were diagnosed as monosaccharide intolerant when after six hours of oral rehydration therapy they had persistent loose watery explosive stools with reducing substances present. Lactose intolerance was defined as the appearance of loose watery stools with positive reducing substances after reintroduction of milk.²

Children with monosaccharide intolerance were given a 12 hour period of carbohydrate free electrolyte solution followed by 24 hours of a glucose electrolyte solution before returning to full strength milk. Secondary lactose intolerance after milk reintroduction was managed by returning to oral rehydration therapy for 24 hours followed by a 12 hourly regrade back on to full strength milk. In all cases the carbohydrate intolerance was transient, resolving after 24 hours in 8/16, after 72 hours in 13/16, and in all by five days. No child developed a prolonged intolerance requiring further investigation and a change of milk. Nevertheless, the short term changes made in the carbohydrate content of the feed resulted in rapid reduction in stool output and relief of acute symptoms.

In 1985 Trounce and Walker-Smith reported carbohydrate intolerance in 15/45 (33%) children admitted to our unit with rotavirus gastroenteritis with rapid resolution of symptoms in most cases.³ In common with others,⁴ we have seen this problem much less frequently in more recent years. Prolonged carbohydrate intolerance after acute gastroenteritis is now considered to be a rare event. Indeed a retrospective review of cases admitted to our unit with rotavirus gastroenteritis in the first three months of 1989, 1990, and 1991 showed carbohydrate intolerance to be present in 5%, 5%, and 0% respectively.

What is the reason for this surge in cases of transient carbohydrate intolerance during 1993? There were only two cases during the first three months of 1994. There has been no recent change in amount of carbohydrate in feeds or in the management of gastroenteritis. No particular rotavirus serotype was identified in the children seen. It is possible that there was a short term change in the pathogenicity of the organism which was expressed in association with carbohydrate intolerance.

The assumption is that monosaccharide intolerance and lactose intolerance coexist reflecting the severity of small intestinal mucosal damage.^{5,6}

Our experience reinforces the importance of prompt testing of loose, watery, explosive stools for reducing substances in children with acute gastroenteritis.

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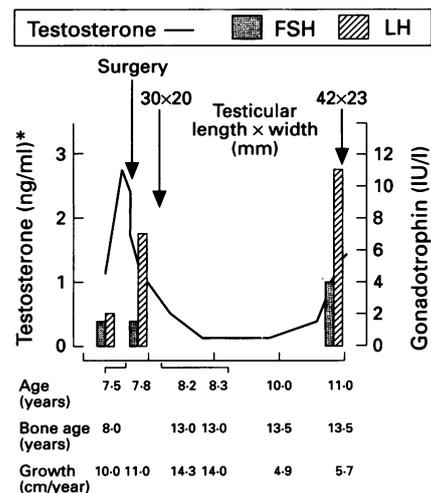
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Transient activation of the hypothalamo-pituitary-testicular axis by testosterone

EDITOR,—Jakacki *et al* showed that the gonadotrophin secretory pattern in early puberty results from amplification of an existing circadian pattern of gonadotrophin secretion.¹ This is caused by an increase in the frequency and amplitude of pulsatile gonadotrophin releasing hormone secretion. The way in which this activates the hypothalamo-pituitary-gonadal axis at puberty is unknown. Androgens may accelerate maturation, as in patients with poorly controlled adrenal hyperplasia and advanced bone age, who may present with central precocious puberty.²

We report a boy who was first seen when he was 7.5 years old. He had pubic hair and accelerated growth velocity due to the testosterone secreted by a left adrenal cortical adenoma (2 cm diameter). The plasma concentrations of the other adrenal metabolites (including oestradiol) were normal. The testicular volume and the gonadotrophin response to gonadotrophin releasing hormone (100 µg/m² intravenous) were prepubertal. A catheter was inserted to find the origin of the testosterone secretion; the plasma testosterone concentrations were 2.9 ng/ml (10.1 nmol/l) in peripheral blood, 7.4 ng/ml (25.7 nmol/l) in the left adrenal vein, and 4.2 ng/ml (14.6 nmol/l) in the spermatic vein (Dr F Brunelle). The tumour and the homolateral adrenal gland were surgically removed (Dr D Jan and Dr C Fékété), but the plasma testosterone remained raised. The testicular volume increased and the gonadotrophin response became pubertal (figure). As plasma testosterone decreased later, no additional treatment was given. This decrease occurred in spite of advanced bone age, no change in the body mass index and persistent increased growth rate.

This case shows that an isolated, moderate increase in testosterone may induce maturation of gonadotrophin secretion to a pubertal



Evolution of testosterone concentrations and the peak luteinizing hormone (LH) and peak follicle stimulating hormone (FSH) response to gonadotrophin releasing hormone. *Testosterone conversion from ng/ml to nmol/l: $\times 3.467$.

level. This phenomenon regressed after removal of the source of testosterone in spite of advanced bone age. This suggests that testosterone may alter the modulation of gonadotrophin releasing hormone secretion by the neurotransmitters.

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Cost savings from 'expert' direction of paediatric interfacility transports

EDITOR,—In his personal view of paediatric intensive care transport, Macrae,¹ in keeping with other reports^{2,3} — correctly indicates that an interfacility transport service requires coordination and direction by an individual (paediatric intensivist) familiar with the complexities of the transport process and environment. He also recognises that there are important differences in the needs and resources of the communities that utilise a transport service. One consistent concern common to both the administrators expected to fund the transport programs and the physicians designated to direct these services is the cost involved (time and money) of having an experienced coordinator continuously available.

In our experience, providing a paediatric transport service for an area of over 370 000 square miles (Province of British Columbia, Canada) and transporting more than 2000 newborns and paediatric patients per year, the individual coordinating each transport now proves to be highly cost effective. After the inevitable lead time to establish the credibility of the service, we have found consistently for the last 11 years that at least 10% of patients referred for transport to tertiary care facilities can continue to be managed in the referring

hospital with appropriate discussion, advice, and shared management responsibility between the transport coordinator and the referring physician. Although the time commitment involved in this process is high (we include this as a responsibility of the on-call intensivists) the costs saved by obviating the need for transport are considerable. These cost savings include direct costs for transport – that is, personnel, vehicles, aircraft and fuel – and indirect costs re utilisation of more expensive tertiary care beds. Humanitarian cost savings are relevant too, although they are rarely recognised. When a child needs care in another geographic location, the uprooting of family members from their community is highly disruptive to their social support structure (spouse, extended family, other children, employment, and accommodation). Cost savings from these 'non-transport' are difficult to quantify and will vary from centre to centre; we fly more than 750 000 air miles per year. However, for the majority of teams, combined direct and indirect savings will likely be more than adequate to balance the projected costs of providing appropriate direction of a transport team's services.

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Systemic vasculitis complicating infantile autoimmune enteropathy

EDITOR,—We read with interest the case reported by Jenkins *et al* in which they highlight the association of autoimmune enteropathy with sideroblastic anaemia.¹ We too do not know of any reported cases of these two associations and believe that they are most likely to be separate clinical entities. The sideroblastic anaemias are a heterogeneous group of disorders that result from different pathophysiological mechanisms impairing haem synthesis. In young children the inherited forms of sideroblastic anaemia of which X linked transmission is the commonest, account for the vast majority of cases when lead toxicity had been excluded. Defining the type of sideroblastic process from blood indices and bone marrow morphology can be difficult as the vast majority of cases have variable hypochromia with microcytosis and less frequently a dimorphic blood picture. In cases where the

blood is macrocytic and the anaemia is refractory to pyridoxine then Pearson's syndrome should be considered in the differential diagnosis.² This is a multisystem disorder characterised by refractory sideroblastic anaemia, with or without vacuolisation of bone marrow precursors, and varying insufficiency in exocrine pancreatic (malabsorption), hepatic (fibrosis, steatosis), renal (proximal tubulopathy), and gastrointestinal (watery diarrhoea, partial villous atrophy) function.^{3,4} Like other mitochondrial cytopathies, Pearson's syndrome is characterised by mitochondrial dysfunction occurring secondary to deletions/rearrangements of mitochondrial DNA (mtDNA). It is usually fatal within the first three years of life, the majority succumbing to end organ failure. Clearly the case reported has not inherited her sideroblastic anaemia in an X linked manner as she is female. It would be interesting to know whether lead concentrations were established, what the red cell indices were at presentation and if postmortem tissue was stored as mtDNA analysis could be carried out. We suggest that deletions/rearrangements of mtDNA should be sought in all young children presenting with anaemia secondary to a sideroblastic process.

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Dr Jenkins and coauthors comment:

We thank the authors for their letter regarding our case report highlighting the association of sideroblastic anaemia and autoimmune enteropathy. Like them we too had considered a wide variety of differential diagnoses and can confirm that lead deficiency had been excluded (normal lead concentrations were found on several occasions) as had Pearson's syndrome. As we stated in our report, there was normal pancreatic function on formal testing and blood had already been sent for mtDNA analysis to two different centres (one in the UK and one in the USA). Neither centre found any abnormality in mtDNA (in particular no deletions) and further specimens were sent to a laboratory in New York which carried out extensive sequence

analysis of the child's genes and found no abnormalities (in particular no abnormality of erythroid ALA synthetase). We can therefore reassure the correspondents that deletions or rearrangements of mtDNA were excluded in our child and agree with them that this should be undertaken in any case of sideroblastic anaemia.

Potentially dangerous sleeping environments and accidental asphyxia in infancy and early childhood

EDITOR,—Byard and colleagues are right to draw attention to the sleeping environment as a cause of accidental death in childhood.¹ I have analysed the Department of Trade and Industry's Home Accident Deaths Database (HADD) for England and Wales for the most recent available year, 1992. There were 10 comparable cases for this single year. The children were aged between 6 months and 2 years and were all found dead in sleep settings (table). These deaths took place despite the existence of safety standards (BS 1753, Cots and BS EN 747 1993, Bunk Beds) or safety regulations (Bunk Bed Entrapment Hazards 1987) designed to minimise such incidents.

Circumstances of deaths in 10 cases of childhood asphyxia (HADD 1992)

Age (months)	Sex	Circumstances
11	F	Cord in cot tangled round neck
10	M	Cord of blind wrapped round neck in cot
17	M	Hooked shirt on cot
9	M	Cardigan caught in cot side
9	M	Found hanging over side of bed
12	M	Cord with mittens at each end caught round neck and part of cot
6	M	Head and chest in 5 inch gap between mattress and cot
11	M	Slipped between bed and wall
14	M	Neck trapped in cot
24	M	Slipped from top bunk, wedged chin between bunk bed and other furniture

I would endorse the view of Byard *et al* that parents should receive advice about the appropriate sleep environment for their infants, including avoiding the use of clothing that could be snagged. However, individualised advice delivered on a one to one basis, perhaps by a health visitor or midwife, is more likely to be effective than the display of pamphlets.

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