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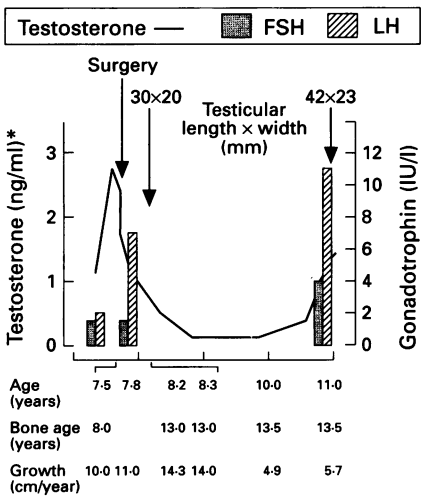
Transient activation of the hypothalamo-pituitary-testicular axis by testosterone

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 public 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 $\mu g/m^2$ 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 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.



*Evolution of testosterone concentrations and the peak luteinising hormone (LH) and peak follicle stimulating hormone (FSH) response to gonadotrophin releasing hormone. *Testosterone conversion from ng/ml to nmol/l: ×3·467.*

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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