

Correspondence

Securing haemostasis during umbilical catheterisation in a neonate

Sir,

The most common method of maintaining haemostasis after catheterisation of umbilical vessels is to secure the catheter to the edges of the cord by using a nylon stitch. When the cord is initially cut to insert the catheter there may be some blood loss, and furthermore some blood may ooze at times in spite of a stitch. We recommend tying firmly a single knot at the base of the cord using wet ribbon gauze (15.2 × 1.0 cm). If this is done before the cord is cut, the catheters will pass without difficulty. Moreover should there be oozing from the cord the nursing staff have merely to tighten the knot before summoning medical help.

A. R. GATRAD AND A. H. GATRAD
*Royal Manchester Children's Hospital,
Pendlebury, Manchester M27 1HA*

Henoch-Schönlein purpura after chickenpox

Sir,

Although Henoch-Schönlein purpura has been associated with many viruses, I have been unable to find a report of it after chickenpox.

An 8-year-old boy was admitted with a 36-hour history of abdominal pain, a tender right ankle joint, and a purpuric rash on his legs. 10 days earlier he had been unwell with a runny nose, sore throat, and cough, followed by a typical chickenpox rash, diagnosed by his family doctor.

On admission he complained of abdominal pain. He was apyrexial. There were a few submandibular nodes palpable and the tonsils were inflamed. Blood pressure was 110/70mmHg. There were scattered lesions over his back and anterior chest wall showing crust formation consistent with chickenpox. Over the buttocks and extensor surfaces of the legs he had a macular-purpuric rash and small haemorrhagic lesions. His right ankle joint was tender and slightly swollen. The platelet count was normal. There was a trace of proteinuria on the first day only.

The history, together with the physical findings, was typical of Henoch-Schönlein purpura.

He made an uneventful recovery during the next 4 days and has remained well since.

C. J. HALLE
*Queen's Park Hospital,
Haslingden Road,
Blackburn BB2 3HH*

Incidence of dental caries in coeliac children

Sir,

As the result of comments made by parents at a Coeliac Society meeting to my paediatric colleague, Dr J. Kuzemko, concerning the rate of decay of their children's teeth, I have reviewed the developing dentitions of 12 of the coeliac children under his care at Peterborough during the last 5 years, for tooth quality and incidence of dental caries.

Coeliac disease was diagnosed at ages ranging from 6 months to 13½ years, and the children have been on a gluten-free diet for between 3 and 11 years. This is a small sample, but the caries experience of this group of children was certainly no worse than one would expect in a similar group of normal local children. Only one case of gross caries of the deciduous dentition was found, and the parent confessed to plying the child with sweets to compensate for the restricted diet. Thus a gluten-free diet does not appear to affect the quality of the developing teeth as far as caries resistance is concerned.

What does have an effect is if the condition is not diagnosed correctly soon after its onset in the early months or years, as the severe constitutional disturbance causes hypoplasia of those areas of the crowns of the permanent teeth which are calcifying at that time. During the first year this affects the anterior teeth and first molars, and these hypoplastic areas are prone to decay and spoil the appearance of the teeth.

We should be interested to learn of the experience of others in this perhaps neglected area.

E. D. FULSTOW
*Peterborough District Hospital,
Thorpe Road, Peterborough PE3 6DA*

Atypical PKU with normal phenylalanine hydroxylase and dihydropteridine reductase activity *in vitro*

Sir,

In a recent report (*Archives*, 1978, 53, 674) Schaub *et al.* described a patient with phenylketonuria who had normal hepatic activity of phenylalanine hydroxylase and dihydropteridine reductase. The patient responded to the administration of tetrahydrobiopterin (the natural co-factor of phenylalanine hydroxylase) by a rapid fall in plasma phenylalanine levels, and the authors postulate that their patient has a defect of biopterin synthesis.

Evidence for the existence of such a defect was obtained in two cases (those of Dr F. Rey in Paris and Dr K. Bartholomé in Heidelberg) who showed reduced serum