erroneous conclusions in the absence of K or Cl determinations. We can now accept that a sweat [Na] of 59 coupled with a [K] of 40 mEq/l (Case 34) is more likely to be indicative of CF than the same [Na] on its own.

Case 26 is indeed regarded to have CF, the absence of steatorrhoea and failure to perform intestinal intubation owing to the patient's poor clinical condition accounting for the question mark in our table. However, the really noteworthy aspect of this case, to which we wished to draw attention, is the variability of the sweat [Na] in competent laboratories from 31 to 81 on different occasions. Cases 12 and 14 also show remarkable variability and here the diagnosis of asthma is based on the consensus of experienced paediatricians on the clinical evidence. Duodenal aspiration on Case 12 showed a pH of 7.0 and normal trypsin and amylase levels. 3-day faecal fat excretion was 4 mmol/day. She had eosinophilia and serum IgE levels in excess of 4000 ng/ml.

Cases 23, 24, and 25 are regarded as CF and the reason for their inclusion in our table is the relatively low sweat [Na] in one test in each case. A value of 71 mEq/l in a 12-year-old child (Case 23) is not only fairly common but compatible with normal, or possibly heterozygous, status which can be seen in the case of a perfectly healthy 12-year-old sibling of a CF child, with sweat [Na] of 70 and 75 (Case 27).

V. SCHWARZ, A. S. AHUJA, and
NAOMI I. M. SIMPSON

Department of Medical Biochemistry,
University of Manchester Medical School,
Stopford Building, Oxford Road, Manchester
M13 9PT

CSF acid-base changes after convulsions

Sir,

In Dr H. Simpson's interesting and detailed articles (*Archives*, 1977, 52, 836, 844) he does not give the exact ages of the children. Many were in the period of active brain growth defined by Ounsted and Taylor (1971), which is earlier for girls but both sexes are at risk from 6 months, and this extends in boys up to 18 months. At this time the vulnerable brain is developing special cells, probably with maturation of specific enzyme systems, and localised damage (for instance to the Sommer sector in the hippocampus) might be caused by chemical poisoning by lactic acidosis to these systems. In a series of 40 children who underwent temporal lobectomy for intractable drug-resistant temporal lobe epilepsy, Davidson and Falconer (1975) found that of the 20 children who had mesial temporal sclerosis affecting the hippocampus, 83% had had prolonged febrile convulsions in infancy. Most had convulsed for much longer than 30 minutes and many had repeated severe convulsions.

Presumably the rise in lactic acid produced by tonic/clonic contractions of nearly all muscle groups is dependent on the severity and duration of the convulsions.

Focal or intermittent seizures might not be so dangerous. In a healthy child seizures are followed by sleep, relaxation of muscles, and rapid metabolism of excess lactic acid. It is reassuring that seizures controlled within 30 minutes are not followed by lasting metabolic upset. In such young children one is always worried about the risk of the next febrile convulsion being prolonged and prophylaxis with anticonvulsants and prompt treatment of fevers is most worthwhile.

SERENA DAVIDSON
Westminster Children's Hospital,
Vincent Square,
London SW1P 2NS.

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Prostaglandin-induced ulcerative colitis in Bartter's syndrome

Sir,

The article by Dodge *et al.* (1977) describing a patient with persistent diarrhoea and raised levels of prostaglandins $\text{PGF}_2\alpha$ and PGE_2 follows a similar report (Barrowman *et al.*, 1975) of a man with medullary carcinoma of the thyroid who had severe diarrhoea, also with high serum concentrations of $\text{PGF}_2\alpha$ and PGE_2 . We report our experience with a similar child with Bartter's syndrome.

The child developed moderate to severe diarrhoea over 3 years of observation. The clinical course and findings on barium enema were consistent with the diagnosis of ulcerative colitis. However, steroid therapy produced no improvement. We recently administered indomethacin as a prostaglandin synthetase inhibitor and this led to a marked resolution of the renal disturbances typical of Bartter's syndrome. Equally interesting was the complete remission of his colitis, with formed stools observed on a dose of 2 mg/kg per day of indomethacin. The steroids were reduced and discontinued without subsequent relapse.

Prostaglandins are known to have a cholera toxin-like action on the gastrointestinal mucosa (Pierce *et al.*, 1971; Bennet, 1971) as well as effecting increases in gastrointestinal motility. These case reports should alert clinicians that perhaps some chronic idiopathic diarrhoeas can be specifically diagnosed and treated as conditions due to prostaglandin excess.

JOSE R. SALCEDO and LEWIS HARDEN
Department of Nephrology,
Children's Hospital National Medical Center,
111 Michigan Avenue NW, Washington,
DC 20010, USA.