possible in such patients and a precise diagnosis may or may not be achieved, even in retrospect. The pathophysiological processes operating in a proportion of infants with protracted diarrhoeal states remain a challenging area for future research, and we look forward to reading the publication which Dr Branski and colleagues have in press.

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Limitations of the sweat test

Sir,
We are in agreement with Schwarz et al. (Archives, 1977 52, 870) when they state that the sweat test has some limitations in the diagnosis of cystic fibrosis (CF) and that the diagnosis is not justified on the basis of an abnormal sweat test unless there is other supporting evidence of the disease. We also agree that there is a 'grey area' of sweat Na and Cl levels which we would class as between 50 and 70 mmol/l (50, 70 mEq/l). However, there are some aspects of their paper which cause us concern.

We feel the summary at the beginning of the paper is somewhat misleading, especially to those who do not carefully read beyond. From the text, one sees that the 30 patients referred to in the summary as giving equivocal sweat Na concentrations represent only 1-5% of patients tested in their laboratory, the total being some 2000. As the remaining 98.5% of patients did not give rise to diagnostic difficulty, it is clear that the 'sweat test' is still highly discriminatory, probably to the degree of any such test.

In our experience a much greater problem of sweat test accuracy occurs in hospitals where the test is carried out on a very much smaller number of patients and where the laboratory has a much lesser degree of experience than that of the authors. We have had a significant number of patients referred to our regional children's hospital who have had abnormal sweat electrolyte measurements elsewhere, but unequivocally normal measurements when repeated by a laboratory carrying out a large number of tests. Anderson and Goodchild (1976), in their recent monograph, stress the difficulties of carrying out an accurate test and describe some of the errors of technique which on inquiry we still find to be common practice, e.g. inexperienced house officers carrying out the collection of sweat rather than experienced laboratory or nursing personnel. We agree with the authors that factors other than technique may affect sweat electrolyte levels but feel that these are not significant when compared with the variation produced by errors in performance of the test.

We have not needed to go to the extent of serial collections of sweat and of giving spironolactone as have the authors. If we obtain a test within the 'grey area' on repeated occasions, we carefully seek other evidence of the disease, particularly evidence of pancreatic disease. This may be obvious from the presence of steatorrhoea of pancreatic type as described by Anderson and Goodchild (1976), but if steatorrhoea is not present (about 10–15% of cases) we should then carry out detailed pancreatic function studies with pancreozymin and secretin stimulation. Hadorn et al. (1968) and others have shown that, although enzyme secretion may be sufficient to prevent steatorrhoea in some patients, the volume of duodenal fluid and its bicarbonate content are always low and neither increases to a normal extent after injection of secretin. This investigation, together with a detailed and critical clinical appraisal, has invariably clarified the diagnosis in our patients with equivocal sweat Na and Cl levels. We do not see evidence that Schwarz et al. have investigated pancreatic function in such detail although they may have done in some of their 'equivocal' patients. We wonder whether in Cases 14 and 26 detailed studies might not show some abnormalities. Case 12, where sweat Na is repeatedly raised, is stated to have normal pancreatic function but no details of the method of assessing this are given. We also wonder why Cases 23, 24, and 25 are included in Table 2 as they seem to have good criteria for the diagnosis of CF at present and even when first seen.

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References


Dr Schwarz and co-authors comment:

We agree with Professor Anderson and co-workers that the sweat test is as good a diagnostic test for CF as any, or better. We also concur with their restatement of the well-known fact that the sweat test is badly performed in many hospitals, so yielding far more unreliable results than those due to uncertainties inherent in the test and discussed in our paper.

We do not advocate the general use of spironolactone; the merit of this drug in our experiments on certain patients was merely to support our suspicion of temporary hyperaldosteronism, a condition which might have led to
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 convolution being prolonged and prophylaxis with anticonvulsants and prompt treatment of fevers is most worthwhile.

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References


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 PGF\(_2\) 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 PGF\(_2\) 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 mobility. These case reports should alert clinicians that perhaps some chronic idiopathic diarrhoeas can be specifically diagnosed and treated as conditions due to prostaglandin excess.

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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 Unsted 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.

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).

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