- ² Stark, J E, British Journal of Hospital Medicine, 1972, 8, 241.
- ³ Sherwood Jones, E, Proceedings of the Royal Society of Medicine, 1971, 64. 1151.
- Crompton, G K, and Grant, I, British Medical Journal, 1975, 4, 680.
- ⁵ Rebuck, A, Read, J, American Journal of Medicine, 1971, 51, 788.
- ⁶ Karetsky, M, Medicine, 1975, 54, 471.
- 7 Rees, H, Millar, J, Donald, K, Quarterly Journal of Medicine, 1968, 37, 541.

SHORT REPORTS

Impaired immunoreactive secretin release in coeliac disease

Studies using bioassays have suggested that secretion of enteric hormones may be abnormal in coeliac disease.¹ In children with coeliac disease increased numbers of secretin-producing S-cells have been described.² Using radioimmunoassay we have assessed the plasma secretin response to duodenal acidification in coeliac disease.

Patients, methods, and results

After an overnight fast, eight patients with untreated coeliac disease and 12 control subjects had 100 ml 100-mM HCl infused over 15 minutes into the upper duodenum. The position of the tube was checked radiologically. Peripheral venous blood samples for secretin determination were taken at -5, 0, $2\frac{1}{2}$, 5, $7\frac{1}{2}$, 10, $12\frac{1}{2}$, 15, 20, 25, and 30 minutes after the acid infusion was started. Blood was collected into heparinised tubes, plunged into an ice bath, and plasma separated at 4°C. Plasma secretin was immediately extracted by ethanol and assayed in a sensitive and specific radioimmunoassay.³ The sensitivity of the assay was 6 pg/ml with 95 % confidence. The statistical significance of differences was estimated using the paired t test.

The figure illustrates the results. Fasting immunoreactive secretin (IRS) levels were similar in controls and "coeliacs," all being less than 70 pg/ml.



Mean (\pm SE of mean) plasma secretin (IRS) levels after intraduodenal acid in patients with coeliac disease and in controls.

In controls there was a rapid and significant rise (average P < 0.005) in IRS levels during acid infusion, which fell rapidly on termination of the infusion. In the coeliac group as a whole the rise in IRS was not significant. Some coeliac patients, however, showed a response but this was less in magnitude and duration than in the control group; these patients had less severe mucosal abnormalities than the remainder of the coeliac group.

Comment

Children with coeliac disease have increased numbers of secretincontaining S-cells which appear to be full of hormone granules.² The data presented here indicate that IRS release is impaired in coeliac disease and so suggests that the S-cells are full of hormone

- ⁸ Bruce Pearson, R, Acta Allergologica, 1958, 12, 277.
 ⁹ Macdonald, J B, Seaton, A, and Williams, D A, British Medical Journal, 1976, 1, 1493.
- Williams, M H, and Levin, M, American Review of Respiratory Disease, 1966, 94, 608
- ¹¹ Gregg, I, and Batten, J, British Medical Journal, 1969, 2, 29.

(Accepted 17 January 1977)

because of failure of release rather than as a result of excessive synthesis of the hormone.

The reason for the failure of secretin release is not clear. Possibly the mucosal damaging process, brought about by gluten, also damages the S-cells or their apical projections; or because of the greatly deepened crypts, known to occur in coeliac disease, it may be more difficult for the acid stimulus to reach the S-cells. Gastric acid secretion is reduced in coeliac disease and, possibly owing to the lack of endogenous acid stimulation, S-cell function becomes sluggish and cannot respond even to an adequate exogenous acid stimulus. In view of the partial IRS response in the patients with milder mucosal flattening, it seems likely the degree of impairment of IRS response reflects the severity of the mucosal lesion.

The lack of IRS response to acid may have several consequences. Pancreatic exocrine function may be impaired and this could further aggravate malabsorption. There may also be important metabolic consequences. Secretin has a lipolytic function and during starvation it may play a major part in regulating lipid metabolism.⁴ Secretin may also participate in producing the augmented insulin response to oral glucose as compared with the insulin response to intravenous glucose.⁵ Thus, in coeliac disease where secretin response is abnormal, this may lead to impairment of islet response with consequent widespread effects on glucose, fat, and protein metabolism and also, as outlined, to abnormalities in lipid metabolism directly.

F A O'C and J C McL were both in receipt of a Royal Victoria Hospital Research Fellowship. The work was supported by a grant from the Medical Research Council. We thank Professor A H G Love and Dr J J Connon for their co-operation in the study of their patients.

¹ Wormsley, K D, Scandinavian Journal of Gastroenterology, 1970, 5, 353.

- ² Polak, J M, et al, Gut, 1973, 14, 870.
- ^a Buchan, K D, et al, Clinical Science, 1973, 45, 13p.
 ⁴ Stout, R W, et al, European Journal of Clinical Investigation, 1976, 6, 179. ⁵ Marks, V, and Samols, E, Advances in Metabolic Disorders, 1970, 4, 1.

(Accepted 10 December 1976)

Department of Medicine, Queen's University, Belfast F A O'CONNOR, MD, MRCP, senior tutor J C McLOUGHLIN, MB, MRCP, research fellow K D BUCHANAN, MD, FRCP, professor of metabolic medicine

Biochemical differences between amniotic fluid and maternal urine

Amniocentesis is being used increasingly for the antenatal diagnosis of congenital abnormalities such as open neural tube defects and several chromosomal abnormalities. With this method there is a risk of inadvertently tapping maternal urine instead of amniotic fluid, especially when the placenta is placed anteriorly. Therefore it is important to establish the biochemical differences between these two fluids.