patient, presumably because of secondary arteriolar damage to the contralateral kidney.

The association between mitral stenosis and hypertension has been the subject of controversy for many years but the opinion has prevailed until now that there is no causal connexion between the two disorders.

However, a recent paper by Obeyesekere et al. (1965), based on case records and necroscopy findings, showed that their patients with mitral stenosis had a mean diastolic pressure higher than a normal population sample. The incidence of systemic hypertension was significantly greater in those patients who had atrial fibrillation compared to those who were in sinus rhythm. Ninety-seven per cent of their patients with renal infarcts at post-mortem had had atrial fibrillation during life.

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Three Familial Cases of Adult Idiopathic Steatorrhoea
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Case 1 G D, woman, aged 50
History: First seen in November 1963 because her doctor thought she was anemic. Three years previously she had attended the surgical department of another hospital because of anemia and had had a hemorrhoidectomy.

It was difficult to obtain a satisfactory history because she appeared to be an immature woman who was uncommunicative and dependent upon her mother. For years she had been subject to attacks of diarrhea; she was always liable to anæmia and for twelve months she had experienced increasing lassitude and pains in the thighs which made it difficult for her to get home from work. Her bowels were opened eight or nine times daily and her stools were watery and pale. She refused to be examined.

Investigations: Hb 39%, ESR 5 mm in 1 hour (Westergren). PCV 28%, MCHC 20%, MCV 83 cu.μ. Blood urea 26 mg/100 ml. Three stool examinations for occult blood: (1) trace, (2) positive, (3) negative. Barium meal and follow through showed clumping; fecal fats averaged 8.5 g per day. Serum calcium 8.4, phosphorus 2.9 mg/100 ml; alkaline phosphatase 26.1 K-A units/100 ml. Jejunal biopsy showed villous atrophy with chronic inflammation typical of gluten sensitivity enteropathy.

The patient was put on a gluten-free diet. All her symptoms have disappeared, her haemoglobin has risen to 86% and her weight has increased from 8 st 3½ lb to 9 st 5 lb.

Case 2 J D, woman, aged 80. Mother of Case 1
History: 1954: Referred because of pain in the neck; X-ray showed a cervical disc lesion at C4-7 and she was treated with physiotherapy. 1960: Attended Gastro-enterological Department because of epigastric pain. Barium meal showed a large gastric ulcer on the lesser curve, for which she was treated medically. The symptoms disappeared and a follow-up barium meal showed no evidence of ulceration. At that time her haemoglobin was 69% with a normal film; after treatment it was 84%. December 1965: Admitted to the Gastro-enterological Department at the request of the Department of Physical Medicine because of three years' increasing backache. On clinical examination she was a thin, pale woman. X-ray of spine showed collapse of vertebrae and appearances suggestive of osteoporosis.

Investigations: Hb 65%, PCV 32%, MCHC 30%. ESR 10 mm in 1 hour (Westergren). Blood urea 39, serum calcium 9-5, phosphorus 3.3 mg/100 ml; alkaline phosphatase 40 K-A units. Bone marrow showed no megaloblastic change. Barium meal normal. Urinary calcium 6 mg/24 hours.

The history of backache, the raised alkaline phosphatase and the fact that her daughter was known to suffer from adult idiopathic steatorrhoea suggested that this woman might suffer from the same complaint. The fecal fat was found to be 9.5 g per day; jejunal biopsy showed complete villous atrophy and changes characteristic of long-standing gluten sensitivity enteropathy.

She has been treated with a gluten-free diet and is improving.

Comment: Had her gastric ulcer been treated surgically, her malabsorption would have been attributed to that.

Case 3 E S, woman, aged 25
History: 18.2.64: Admitted as an emergency because of anæmia, lassitude and vomiting. The patient was twenty-three weeks pregnant; she had had 5 previous pregnancies but only one child had survived. She believed that two of her sisters and one cousin had been treated for celiac disease as children in Dublin. In view of the pregnancy no barium meal was done.

Investigations: Hb 40%, MCV 91.5 cu.μ. Serum iron 196 μg/100 ml (on oral iron). Sternal marrow
showed megaloblastic hyperplasia with giant metamyelocytes. Serum calcium 9-2, inorganic phosphorus 3·6 mg/100 ml; alkaline phosphatase 9 K-A units. Figlu 8 mg per hour. Fæcal fats 8·5 g per day. Serum vitamin B₁₂ 580 μg/ml.

In view of the pregnancy no jejunal biopsy was done, but in response to a gluten-free diet and folic acid there was a marked reticulocyte response of 18%.

The patient went to full term, was delivered of a live child, and has since gained 12 lb in weight. On a gluten-free diet she enjoys good health and looks after her family.

A subsequent attempt at jejunal biopsy failed and has not since been reattempted as she is again pregnant.

Dr E E Doyle of the Adelaide Hospital, Dublin, has informed me that one of this patient's sisters was treated in the hospital from 21.9.45 to 29.12.45 for celiac disease; there was proved steatorrhœa and hypochromic anaemia. The other sister has not yet been traced.

Discussion

Reference to the familial incidence of celiac disease and adult idiopathic steatorrhœa has frequently been made in the literature since 1923, when Hablutzel-Weber reported celiac disease occurring in twins.

Thompson (1951) reviewed 119 cases of celiac disease and found an incidence of 13% in siblings, 6% in parents and 1% in grandparents. In 1953 Cooke et al. found, in 100 cases of adult idiopathic steatorrhœa, that there were ten families with the condition occurring in more than one member and five further families in which it was suspected to occur in more than one member.

Boyer & Andersen (1956) reported that of the 100 parents of 50 patients with celiac disease 43 gave a history of diarrhoea or intolerance to certain foods compared with only 9 of the 100 parents of normal controls. In 1959 Carter et al. calculated that the proportion of celiac disease in sibs attending the Hospital for Sick Children, Great Ormond Street, was 1 in 50, compared with an incidence of about 1 in 3,000 in the general population.

The diagnosis in the present 3 cases can be made with confidence but none was diagnosed on first hospital attendance; in Case 2 the condition would not have been suspected had there not been a family history of the complaint.

In our experience these patients may present in almost any hospital department with diarrhoea, lassitude, osteomalacia, tetany, anaemia, spontaneous fracture, mental changes or hypothermia. The diagnosis is therefore not easy, and a family history may be a valuable diagnostic aid.