Two-thirds of the patients who had strokes were over the age of 50. It is important, therefore, to include the possibility of bacterial endocarditis when considering the differential diagnosis of all patients with strokes.

Even more striking was the diagnostic difficulty encountered in those patients who presented with meningitis or meningoencephalitis. Osler (1885) wrote: "The meningeal complication of endocarditis has not received much attention." A recent case report (Harris, 1965) has also pointed to this association. Our series included seven such patients, in five of whom C.S.F. cultures were sterile. Toone (1941) had noted that bacterial endocarditis should be considered in cases of sterile meningitis. It must also be considered in the differential diagnosis of toxic organic psychoses (Antel *et al.*, 1955), and mental symptoms occurred in 10 of our patients.

In only four patients did their neurological complications develop after antibiotic treatment, and two of these had polyneuritis. This would suggest that the risks of cerebral embolism and meningitic infection are reduced by antibiotic therapy, and further stresses the importance of early diagnosis.

The immediate mortality was high (67%), and was higher than in the cases which had not presented as neurological problems (44%). The difference can probably be attributed to the neurological lesions themselves, and the general features of the bacterial endocarditis did not seem to differ from those seen in cases presenting in other ways. Pearce and Guze (1961) found no difference in cases with or without neurological manifestations.

Two features of the endocarditis were of interest, however. Staphylococci were responsible for a high proportion of cases, and the incidence of staphylococcal infection is higher than in most previous series—for example, Friedberg *et al.* (1961). Staphylococcal endocarditis has been reported to carry a high mortality (Pearce and Guze, 1961), but neurological complica-

tions occurred only rarely in the cases described by Meade (1959). The other striking finding was the high incidence of negative blood cultures, and this general problem has been discussed elsewhere (Hampton and Harrison, 1967).

Summary

The hospital records of 116 patients with bacterial endocarditis have been studied. Thirty-seven showed neurological manifestations, and in 33 these were present at the time of admission to hospital and formed the immediate diagnostic problem. Fifteen presented with strokes, and seven with meningitis or meningoencephalitis. Bacterial endocarditis was unsuspected in five of the seven cases with meningitis, and was discovered only at necropsy.

The importance of bacterial endocarditis in the differential diagnosis of both common and uncommon neurological syndromes is stressed.

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Iron Absorption in Coeliac Disease of Childhood and Adolescence

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Iron-deficiency anaemia is a frequent finding in coeliac disease. The most obvious explanation for the development of this anaemia would be malabsorption of iron. Indeed, malabsorption of iron in idiopathic steatorrhoea of adults has been reported by a number of workers (Badenoch and Callender, 1954; Bonnet *et al.*, 1960; Davis and Badenoch, 1962). There is evidence that idiopathic steatorrhoea of adults and coeliac disease of children are the same hereditary condition (MacDonald *et al.*, 1965). Though a number of radioiron studies in children have been reported (Darby *et al.*, 1947; Sharpe *et al.*, 1950; Schulz and Smith, 1958; Smith, 1964), a study of iron absorption in children or adolescents with coeliac disease has not been found in the literature. The present study of this subject was prompted by the finding, in a few such cases, of iron absorption results within the normal range.

Materials and Methods

Nine children and young adults with proved coeliac disease were studied. Four had been treated by a diet partially or com-

pletely free of gluten at an earlier stage of their illness, but none was receiving dietary or corticosteroid treatment at the time of study. In all these patients the jejunal mucosa was abnormal on both dissecting microscope and histological appearance. Five patients had an excretion of fat in excess of 7 g. per day. All except one showed evidence of iron deficiency with either hypochromic anaemia or low serum iron.

Nine control subjects without steatorrhoea or other evidence of malabsorption were also studied. They were matched as closely as possible with the coeliac group for age, sex, haemoglobin, and serum iron levels. None of the coeliac or other patients had positive tests for occult gastrointestinal bleeding at the time of the iron-absorption test, and none were receiving iron therapy.

Iron absorption was measured by a radioiron balance method. A 3-mg. dose of iron in the form of rabbit haemoglobin was

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given after a standard meal. The meal was calculated to contain 2.34 mg. of iron. The haemoglobin was labelled *in vivo* with ^{59}Fe (Callender *et al.*, 1957), and the dose used was 0.3 to 0.5 μCi . Faecal collections were continued until less than 1% of the dose was present in a single day's collection. This method has been described in detail (Taylor and Gatenby, 1966).

Haemoglobin was estimated by the oxyhaemoglobin method (Dacie and Lewis, 1963), and serum iron was determined by a modification of the method of Peters *et al.* (1956) and Peters and Giovanniello (1958).

Faecal fat was estimated by the method of Cammidge (1914) on a faecal collection of at least five days' duration on a normal ward diet.

Jejunal biopsies were obtained with a Crosby capsule, and they were graded according to their appearance when examined fresh with a dissecting microscope. The grading was an extension of that used by Creamer (1964): grade 3, ridges; grade 4, convolutions without mosaic pattern; grade 5, convolutions with mosaic pattern; and grade 6, flat with mosaic pattern.

Results

The data are given in Tables I and II. Nine control subjects absorbed 36–61% of the oral dose of 3 mg. of haemoglobin iron

TABLE I.—Absorption of 3-mg. Dose of Haemoglobin Iron in Nine Control Subjects

Case No.	Sex and Age	Hb (g./100 ml.)	Serum Iron ($\mu\text{g.}/100\text{ ml.}$)	Faecal Fat (g./day)	Iron Absorption (% of Oral Dose)	Diagnosis
1	M 4	8.8	33	4.2	39	Dietary iron deficiency
2	M 16	12.8	38	5.6	40	Duodenal ulcer
3	F 20	10.4	49	1.6	61	Menorrhagia
4	F 22	12.7	58	3.5	43	Duodenal ulcer
5	F 22	11.6	68	6.3	59	Dietary iron deficiency
6	F 22	10.0	40	4.1	40	" " "
7	F 24	12.8	116	2.4	57	Neurosis
8	F 25	9.8	30	2.5	36	Dietary iron deficiency
9	M 28	15.0	82	—	39	Healthy. Recent blood donation
Mean	20	10.4	57		46	

TABLE II.—Absorption of 3-mg. Dose of Haemoglobin Iron in Nine Coeliac Patients

Case No.	Sex and Age	Hb (g./100 ml.)	Serum Iron ($\mu\text{g.}/100\text{ ml.}$)	Mucosal Grade	Faecal Fat (g./day)	Iron Absorption (% of Oral Dose)	Dietary Treatment Before Study
10	M 5	11.2	48	3	3.8	31	Gluten-free diet, aged 1–4 years
11	M 12	12.3	34	6	6.0	38	None
12	F 12	11.0	17	6	7.5	42	"
13	F 14	11.2	29	6	18.7	9	"
14	F 15	13.5	152	4	2.0	60	Low gluten diet, aged 18–21 months
15	M 18	11.1	24	4	11.7	56	Low gluten diet, aged 3½–4 years
16	F 20	8.4	56	4	8.6	58	None
17	F 21	11.7	29	4	9.3	44	Irregular gluten-free diet, aged 18–20 years
18	F 22	8.6	84	6	5.1	55	None
Mean	15	11.0	53			44	

(mean 46%). Eight of the coeliac patients absorbed 31–60% (mean 48%) showing no significant difference. In one coeliac patient (Case 13) malabsorption of iron was shown. The result for this child (9%) was greater than two standard deviations from the mean for the coeliac group.

No relation was found between iron absorption and jejunal mucosal grade, nor between iron absorption and faecal fat content.

Discussion

The two groups examined were reasonably comparable as regards haemoglobin and serum iron levels. These factors are thought to be of particular importance in relation to iron absorption (Taylor and Gatenby, 1966). The unexpected normal iron absorption in the majority of the coeliac patients might be explained if one regarded them as being "recovered" cases. On the other hand, five of the nine had definite steatorrhoea and all of them had abnormal biopsies with four out of nine being flat (grade 6). The results indicate that the iron deficiency of the coeliac group cannot be due to a continuing malabsorption of iron. It is possible that the absorption of iron in these cases may be defective intermittently and especially during exacerbations of the disease. This concept is supported by the finding that the single patient showing a poor absorption of iron was the most severely affected clinically. However, it is likely that factors other than malabsorption of iron played a more important part in the causation of iron deficiency in these cases of coeliac disease. Inadequate dietary iron to meet the demands of growth is common in Dublin (Powell, 1960; Gatenby, 1963). Excessive loss of iron could result from intestinal bleeding, but no convincing evidence of this was present in this series. The rapid loss of mucosal epithelial cells (Padykula *et al.*, 1961) or of inflammatory cells into the intestinal lumen are other possible causes of iron loss in coeliac disease, but this hypothesis has yet to be proved.

Summary

The absorption of haemoglobin iron was studied in nine patients with coeliac disease and in nine controls. In only one case, a 14-year-old child with coeliac disease, was a low absorption of iron detected. Iron absorption was normal in each of the other eight coeliac patients despite an abnormal jejunal mucosa.

Other possible causes of iron deficiency in coeliac disease, apart from malabsorption, are discussed.

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