the Asian influenza epidemic of 1957 was identified and typed within a few weeks after it began in the Far East; but with other virus diseases, such as rubella and hepatitis, we have to report no progress whatsoever. These and other diseases still to be tackled constitute a current challenge.

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FAT-MALABSORPTION IN CONGESTIVE CARDIAC FAILURE

BY

R. VAUGHAN JONES, B.A., M.B., M.R.C.P.

Lecturer in Clinical Pathology, St. Thomas's Hospital Medical School, London

It is a well-recognized fact that many patients suffering from congestive cardiac failure of long duration tend to lose weight, and this has generally been ascribed to anorexia. The possibility that malnutrition has resulted from malabsorption seems to have been largely unexplored, and in this paper an attempt is made to assess the frequency of steatorrhoea in cardiac failure.

Material and Methods

The 20 cases studied were all in-patients of St. Thomas's Hospital, and in each case the diagnosis of the cause of cardiac failure was made by the physician in charge of the case. In 17 of the cases this cardiac failure, as judged by jugular venous pressure, was present during the investigation—in three cases the venous pressure had fallen to normal from three to seven days before the tests were begun.

Patients received a normal ward diet, although in some cases anorexia resulted in a diminished intake of food. An attempt was made to grade the anorexia in each of these cases. After an oral dose of 10–20 μ c. of I-131-labelled triolein given in 10 g. of arachis oil, the faecal output of radioactivity was measured and expressed as a percentage of the dose received by the patient. Stool saves were made for from three to six days, and in most cases the faecal total lipids were measured as well and expressed as grammes per day (Prunty *et al.*, 1959). Results below 3% were regarded as normal for faecal I-131 triolein and those between 3 and 7% necessitated parallel faecal-fat estimation for their proper evaluation. Above 7% the results were regarded as abnormal (Vaughan Jones, 1960). Faecal total lipids above 7 g. a day were regarded as abnormal for persons eating a normal ward diet (Annegers *et al.*, 1948; Wollaeger *et al.*, 1947). Conventional liver-function tests were performed in most of the patients.

Results

The results obtained are shown in the Table. All the cases except Nos. 3 and 18 were receiving treatment with digitalis. It will be noted that Cases 1–5 all had evidence of fat-malabsorption as judged by one or other of the two tests. Only in Cases 1 and 3 was malabsorption demonstrated by both tests. Case 2 had high faecal fat and normal faecal radioactivity; Cases 4 and 5 had high faecal radioactivity and normal faecal fat.

Discussion

In this group of 17 patients in congestive cardiac failure, three had steatorrhoea as judged by chemical fat estimation. One of these cases had normal radioactive fat excretion, but it is generally considered that the correlation between these tests is less than perfect and in particular that the triolein test tends to miss steatorrhoea (Pimparkar *et al.*, 1960; Vaughan Jones, 1960).

In Cases 4 and 5 a high output of faecal radioactivity coexisted with a normal faecal fat. There are two possible explanations. First, the anorexia and consequently diminished food, and therefore fat, intake of these patients may have masked steatorrhoea. Such masking is well documented for low fat intakes (Wollaeger *et al.*, 1947; Frazer, 1949; Weijers and van de Kamer, 1953; Crowe *et al.*, 1956). Secondly, the discrepancy may represent a further failure of the I-131 triolein test. Van Handel and Zilversmitt (1958) have shown that I-131 triolein is absorbed differently from triolein labelled with C-14 and presumably therefore from triolein itself, possibly because of breakdown of the former during absorption. However, in congestive cardiac failure because of anorexia it is often not possible to guarantee a sufficient intake of fat to validate estimation of daily faecal fat as a test of malabsorption, and therefore one may underestimate the occurrence of malabsorption. Perhaps therefore one should not too lightly discard this isotopic evidence of malabsorption in Cases 4 and 5, especially since where discrepancies occur between faecal fat and iodinated triolein estimation they are usually, as already indicated, on the side of normal iodinated triolein and high faecal fat.

Thus it is certain that three and possible that five of 17 patients with congestive cardiac failure showed evidence of fat-malabsorption, clearly a much higher incidence than in the general population.

Examination of the Table indicates that three of these five patients had shown moderate or considerable loss of weight, and, although appetite was classed as moderate in each of the three, the possibility exists that fat-malabsorption as well as anorexia had played some part in the genesis of this loss of weight. However, three other patients with cardiac failure in the present series, in whom steatorrhoea was not demonstrated, lost weight to an equivalent amount.

It remains to be explained why these patients with cardiac failure had steatorrhoea. Digitalis, so commonly prescribed to such patients, was suggested as a cause (A. C. Dornhorst, personal communication, 1958), but one of the patients with steatorrhoea was not receiving digitalis and 14 patients receiving digitalis did not have steatorrhoea. If a cause at all, digitalis is clearly not the only cause nor a very potent one. No obvious correlation emerged between the chronicity of cardiac failure and steatorrhoea, nor between any particular cause of cardiac failure and steatorrhoea.

There appeared to be little correlation, either, with evidence of liver damage as judged by conventional liver-function tests except that the serum bilirubin level was slightly raised in three patients out of the four with steatorrhoea in whom it was measured. It was raised in only one of the 11 patients without steatorrhoea in whom it was measured.

Only one account of the presence of steatorrhoea in patients with cardiac failure has been found: de Langen (1953) states that steatorrhoea is occasionally present—

Table of Results

Case No.	Diagnosis*	Faecal Fat (G./day)	Amount of Administered Radioactivity in Faeces	Weight Loss	Appetite	Duration Present Cardiac Failure	No. of Previous Admissions for Cardiac Failure	Serum Bilirubin (mg./100 ml.)	Thymol Turbidity (units)	Plasma Alkaline Phosphatase (K-A units per 100 ml.)	Serum Protein (g./100 ml.)		
											Total	Albumin	Globulin
Group I: In cardiac failure at time of test													
1	MS	10.1	55.4	Slight: 1-1 st. (3.2-6.4 kg.) in 1 year	Moderate	4 weeks at least	Nil	2.4	3	14	6.8	2.9	3.9
2	MS MI TI	8.4	1.6	Striking: 2½ st. (15.9 kg.) in 5 years	"	Chronic failure	Numerous	1.3	1	27	6.0	3.3	2.7
3	H	8.5	4.3	Nil	Good	4 weeks	Nil	1.2	7	9	6.8	3.8	3.0
4	MS MI TI	2.9	12.6	"	Moderate	4 "	7	"	"	"	"	"	"
5	MS MI TI	6.5	9.5	Striking: 2 st. (12.7 kg.) in 2 years	"	2 months	3	0.6	2	"	"	"	"
6	MS MI TI	3.9	6.3	Nil	Poor	Chronic failure	Several	0.5	3	20	"	"	"
7	AI	6.3	1.0	"	Good	4 weeks	2	0.5	1	19	7.0	4.2	2.8
8	H CP	1.8	2.0	"	Poor	Chronic failure	2	"	"	"	"	"	"
9	MS MI TI	5.2	—	Striking: 4 st. (25.4 kg.) in 7 years	Good	4 weeks	10	0.5	3	17	6.1	4.2	1.9
10	MS MI AI	—	1.0	Nil	Moderate	4 "	1	0.6	1	8	5.8	3.4	2.4
11	MS MI	—	1.0	"	Good	2 "	3	0.5	2	8	6.7	3.3	3.4
12	CP	2.4	0.5	"	"	3 "	2	"	"	"	"	"	"
13	Unknown	2.8	3.7	"	"	10 days	4	0.5	7	12	7.6	3.7	3.9
14	H	5.2	2.1	Striking, but had deliberately dieted	"	2 weeks	1	0.4	2	8	5.7	3.5	2.2
15	MS MI	5.9	1.0	Nil	"	2 months	1	0.7	3	8	6.2	3.4	2.8
16	MS	2.5	1.3	"	"	5 weeks	2	0.9	2	34	8.0	4.5	3.5
17	MS MI	—	1.4	Striking: 2 st. (12.7 kg.) in 2 years	Poor	4 months	2	1.8	1	38	5.7	3.4	2.3
Group II: No longer in cardiac failure at time of test													
18	MS MI	5.4	1.0	Slight	Good	Cured 1 week before test	Nil	—	—	—	—	—	—
19	MI	4.8	0.5	Nil	"	Cured 3 days before test	"	0.8	4	17	7.4	3.4	4.0
20	IHD	3.2	0.9	"	"	" "	"	—	—	—	—	—	—

* MS=mitral stenosis; MI=mitral incompetence; TI=tricuspid incompetence; AI=aortic incompetence; H=hypertension; CP=cor pulmonale; IHD=ischæmic heart disease.

"even in cases of cardiac decompensation with hypertension in the portal area considerable quantities of fat may occasionally be found in the stools."

A recent paper includes a comparison between the plasma levels of radioactivity obtained after an oral dose of I-131 triolein in patients while in congestive cardiac failure with those obtained after recovery (Berkowitz *et al.*, 1959). Few details were given, but low levels of plasma activity were said to revert to normal after recovery from cardiac failure.

It is known that disturbed liver function is often found in patients with chronic cardiac failure. This is especially so in those with constrictive pericarditis and rheumatic valvular disease, in particular mitral stenosis with tricuspid incompetence (Koletsky and Barnebee, 1944; Sherlock, 1951; White *et al.*, 1955). Analogy with infective hepatitis and cirrhosis suggests that the findings of Gross *et al.* (1950) in these diseases may be relevant. Fat-malabsorption, which they assumed to be due to lack of bile salts, correlated moderately well with the serum bilirubin concentration and roughly inversely with faecal sterobilinogen output.

Other possible explanations for fat-malabsorption in cardiac failure are pancreatic and intestinal malfunction. Masoni and Pellegrini (1953) have demonstrated impaired pancreatic function in patients with severe chronic cardiac failure, and Meakins (1940) thought that such patients had an intestinal absorption defect for glucose; de Langen (1953) suggested that intestinal congestion or ischaemia in cardiac failure might explain fat-malabsorption. It is possible that one of these two explanations may apply in the cases described, but the point was not investigated further.

Summary

Fat-malabsorption has been demonstrated in 3 out of 17 patients with congestive cardiac failure and may have been present in two more. No correlation with cause or chronicity of cardiac failure, loss of weight, or digitalis therapy was observed. The possible significance of the raised serum bilirubin in three of the cases of fat-malabsorption is discussed.

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ULCERATIVE COLITIS AND PARATHYROID DYSFUNCTION

BY

I. J. GREK, M.D.

AND

B. SENIOR, M.R.C.P.

*From the University of the Witwatersrand Medical School
and the Johannesburg General Hospital*

A significant proportion of patients with hyperparathyroidism have concomitant peptic ulceration (Rogers, 1946), and a recent survey of a group of 300 consecutive peptic-ulcer patients (Frame and Haubrich, 1960) resulted in the discovery of hyperparathyroidism in 4. The hyperparathyroidism precedes and in some as yet undefined way provokes the ulceration. The association of pancreatitis and hyperparathyroidism is also recognized, but this relationship is more obscure. It has been suggested that the hypercalcaemia causes intraductal calcium precipitation with blockage, but such deposition is not always present, and in one patient the pancreatitis appeared to precede the hyperparathyroidism (*British Medical Journal*, 1959).

Steatorrhoea has been observed in combination with hypoparathyroidism (Jackson, 1957; Williams and Wood, 1959) and more rarely hyperparathyroidism (Davies *et al.*, 1956). Again the link is perceived but the mechanism of association remains to be elucidated.

These examples of the coexistence of gastro-intestinal and parathyroid disorders lend interest to the presence of hypoparathyroidism and hyperparathyroidism, respectively, in two patients with long-standing ulcerative colitis, who were treated with steroids.

Case 1

A 40-year-old white physician was seen in January, 1957, because of cramps in the extremities. His relevant history went back approximately 20 years, when he first experienced attacks of mild diarrhoea which recurred every few months. In 1947 the diarrhoea became more severe and was accompanied by a blood-stained rectal discharge. In 1951 he again had severe diarrhoea and in addition frank rectal bleeding. He was admitted to a London hospital, and after investigation, which included a barium enema and a sigmoidoscopy, the diagnosis of ulcerative colitis was made. He returned to South Africa and a year later developed severe pain and some swelling of the joints of the fingers, wrists, ankles, and knees, for which hydrocortisone was prescribed in a dosage of approximately 60 mg./day. Both the diarrhoea and the arthritis lessened, and this treatment was continued for two years, until 1955, when severe diarrhoea recurred, with marked loss of weight. It was thought that the steroid dosage was inadequate, and prednisone 40 mg./day was substituted. The diarrhoea subsided and the prednisone was slowly reduced to 20 mg./day. He gained weight and felt stronger, but experienced carpal spasms which were ascribed to hyperventilation. He later attended a hospital in San Francisco, where the diarrhoea became worse and was accompanied by severe carpal spasms, paraesthesias of the extremities, and tingling around the mouth.

Laboratory tests at the time revealed: serum calcium, 5.5 mg./100 ml.; serum phosphorus 5.7 mg./100 ml.; serum protein, 6.1 g./100 ml. (albumin 3.6 g., globulin 2.5 g.), and alkaline phosphatase 4.4 King-Armstrong units.

He was told that his symptoms resulted from the steroid therapy, but attempts to reduce the dose provoked such severe diarrhoea that intravenous-fluid therapy was necessary. He was given parenteral calcium and calciferol, 200,000 units/day. Thyroid function was reported as normal. He then visited another clinic, where reduction of