Steatorrhea Complicating Diabetes Mellitus with Neuropathy

Report of Cases without Apparent External Pancreatic Insufficiency

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Disturbances of gastrointestinal function have frequently been observed in association with diabetes mellitus. The disorders which have been reported include gastric atony and dilatation, postprandial abdominal cramping, severe constipation, and intractable diarrhea characterized by watery stools, nocturnal exacerbations and fecal incontinence.[2, 10, 14, 15, 17–20] These disturbances have been attributed by many investigators to alterations in gastrointestinal motility secondary to visceral diabetic neuropathy. The purpose of the present report is to call attention to a group of patients with diabetes mellitus and neuropathy who exhibited diarrhea and steatorrhea which apparently could not be attributed to external pancreatic insufficiency.

REPORT OF CASES

Case 1. A white woman, aged 31 years, was first seen at the Mayo Clinic in 1951 because of diabetes mellitus of 17 years' duration. There was a family history of diabetes. Control of the patient's diabetes had been poor. No history of gastrointestinal symptoms was elicited. The patient was dismissed with instructions to follow a program of diet and insulin.

The patient returned in 1953 because of frequent insulin reactions. Symptoms of gaseous abdominal distention and diarrhea without nocturnal fecal incontinence had been noted for the preceding four months. There had been no loss of weight and no pain suggestive of pancreatitis.

On examination the blood pressure was 104 mm. of mercury systolic and 70 diastolic. There was no postural hypotension. The ocular fundi were normal. The results of examination of the abdomen were negative. With the exception of the absence of sweating below the level of the thorax in response to heat (figure 1) the results of neurologic examination were normal. The laboratory findings are given in tables 1 and 2.

Because of the steatorrhea the patient was advised to follow a diet limited in fat to 30 gm. daily. Subsequent correspondence indicated that only a temporary remission of symptoms was noted with the initiation of this diet. Subsequent diarrhea was not improved by therapy with pancreatin.

Case 2. A white man, aged 34 years, was seen at the clinic in 1954 with a history of diabetes mellitus of 14 years' duration. The family history was positive for diabetes. Control of the patient's diabetes had been poor, as evidenced by a lack of dietary regulation and a history of frequent insulin reactions. For six years the patient had had diarrhea with four to eight foamy, greasy stools daily. Nocturnal diarrhea and fecal incontinence were distressing symptoms, and the patient experienced exacerbations of the diarrhea following meals. For five years, impotence had been present. Four years prior to examination subtotal gastrectomy had been done because of a bleeding duodenal ulcer. Postural dizziness had been present for five months. The patient also noted nocturnal paresthesias and shooting pains in his legs. There had been a loss of 20 pounds in the 10 years prior to examination at the clinic. There had been no pain suggestive of pancreatitis.

On examination, the blood pressure was 120 mm. of mercury systolic and 90 diastolic; after the patient stood for 5 minutes, this decreased to 74 mm. systolic and 60 diastolic and the patient felt dizzy. The pupillary reactions and ocular fundi were normal. The results of examination of the abdomen were unremarkable. The tone of the anal sphincter was decreased. Muscle stretch reflexes were absent in the legs and hypoactive in the arms. Vibratory sense was absent in the toes. The sweating pattern is shown in figure 1. The laboratory data are included in tables 1 and 2. During a brief period of observation, a low dietary intake of fat was associated with improvement in

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the gastrointestinal symptoms.

Case 3. A white man, aged 35 years, came to the clinic in 1952 with a history of diabetes mellitus of 5 years' duration. There was no family history of diabetes. Careful control of the diabetes had not been attempted, as indicated by the lack of frequent urine testing for sugar. For 3 years the patient had been impotent and had suffered from diarrhea characterized by the passage of 10 to 12 stools per 24-hour period. The patient noted postprandial exacerbations of diarrhea, with the appearance in the stool of food ingested as recently as 15 minutes previously. Although he passed the majority of his stools during the daytime, he also had nocturnal diarrhea associated with fecal incontinence at least once nightly. There were no gross steatorrhea and no history of pain suggestive of pancreatitis. The patient had lost 44 pounds since the onset of diarrhea.

On examination, the blood pressure varied between 90 and 110 mm. of mercury systolic and between 50 and 60 diastolic when the patient was supine and measured 70 mm. systolic and 56 diastolic with the patient standing. The pupillary reactions and ocular fundi were normal. There were no abnormalities on abdominal examination. The knee jerks and ankle jerks were absent and vibratory sensation was impaired at the ankles. Slight weakness of the muscles of the pelvic girdle and of the anterior and posterior tibial muscle groups was noted. There was no sweating below the waist in response to heating (figure 1). The laboratory findings are listed in tables 1 and 2. Therapeutic trials of large doses of pancreatin, banthine, biohepulin, and cortisone provided no symptomatic benefit with respect to the diarrhea.

Case 4. A white man, aged 31 years, first came to the clinic in 1946 with a history of diabetes mellitus of 10 years' duration. There was a family history of diabetes. Control of the diabetes had been poor in that he had experienced numerous insulin reactions and an episode of coma. For four years the patient had noted diarrhea; this was intermittent initially but had been constant for three years. There were as many as 18 stools during the day and 5 stools during the night, at which time fecal incontinence was frequently present. The stools appeared greasy, floated on water and often contained particles of food that had been ingested as recently as three hours previously. There were no history of abdominal pain and no loss of weight. The patient had been impotent for two years.

On examination the blood pressure was 98 mm. of mercury systolic and 78 diastolic while the patient was supine. These levels fell to 56 mm. systolic and 40 diastolic with the patient standing. The pupils were of the Argyll-Robertson type; the ocular fundi exhibited diabetic retinopathy. The results of examination of the abdomen were unremarkable. The knee jerks were diminished and the ankle jerks absent. Pain and temperature perception was diminished in patchy areas over the entire body. No sweating test was done. Laboratory data are included in table 1. Quantitative studies of fat excretion were not carried out. Therapy with 5 cc. of crude liver extract daily for 10 days provided no improvement of the diarrhea, and further similar treatment following the patient's return home was also ineffective.

Because of persistent, incapacitating diarrhea the patient returned in 1947. Fecal fat and nitrogen during control periods and during periods of therapy with pancreatin while the dietary fat was 100 gm. daily are given in table 3. It is apparent that during therapy the steatorrhea was more severe than during the control periods. All attempts to control the gastrointestinal symptoms were unsuccessful, and correspondence indicated that severe diarrhea persisted until the patient's death in 1948.

Case 5. A white man, aged 36 years, was first seen at the clinic in 1934 with a history of diabetes mellitus of two years'

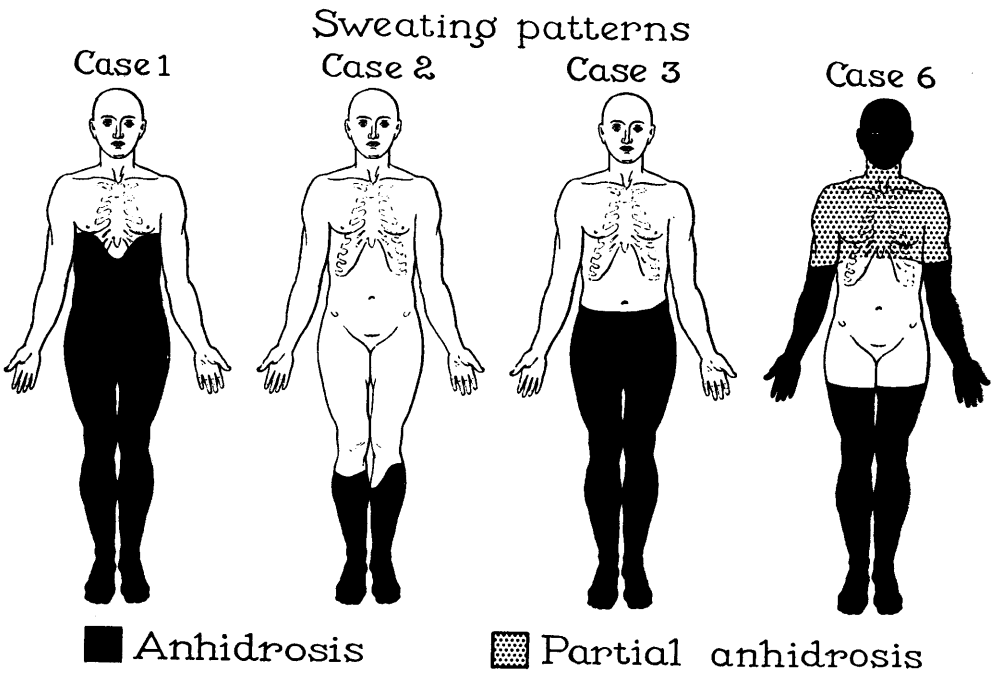

FIG. 1 Sweating patterns in four cases.

TABLE 1

Steatorrhea complicating diabetes mellitus with neuropathy: laboratory data in six cases*

Determination	Case 1	2†	3†	4†	5†	6† 1942	6† 1945
Albuminuria (grade, basis 4)	0	0	1	1	1	0	2
Hemoglobin (gm. per 100 cc.)	12.6	11.6	11.7	13.8	9.3	11.7	12.4
Erythrocytes (million per cu. mm.)	4.45	4.60	4.91	4.31	3.03	4.17	4.31
Blood sugar (mg. per 100 cc.)	440	260	390	208	470	248	63
Macrocytosis	—	—	0	0	0	0	+
Serum protein (gm. per 100 cc.)	5.25	—	5.25	6.80	5.90	4.70	6.20
Albumin-globulin ratio	1.43:1	—	0.86:1	1.44:1	1.82:1	1.58:1	1.80:1
Sulfobromophthalein dye retention (% in 1 hr.)	—	—	—	0	0	0	—
Prothrombin time (normal 19 sec.)	—	—	19	17	20	19	—
Serum calcium (mg. per 100 cc.)	—	9.6	8.8	9.9	9.3	9.5	9.8
Serum phosphate (mg. per 100 cc.)	—	—	3.2	3.9	3.3	—	3.2
Blood urea (mg. per 100 cc.)	—	32	14	—	66	36	50
Serum amylase (normal <320 units)	8.0	—	—	106	160	106	—
Serum lipase (normal <0.3 cc. N/10 base)	0.1	—	—	0.3	0.3	0.0	—
Roentgenographic studies:							
Pancreatic calcification	0	—	0	—	0	0	0
Stomach	—	0	0	atonic	0	0	Retention
Small intestine	Deficiency pattern	0	Deficiency pattern	0	0	0	0
Colon	0	0	0	0	0	—	—
Gastric acidity (units: free/total)	—	—	—	46/60	60/80	38/48	20/34
Cerebrospinal-fluid protein (mg./100 cc.)	—	—	—	60‡	30‡	40‡	—

\+ indicates finding present.
0 indicates normal finding.
— indicates no data.

* Kline test for syphilis negative in all cases.
† Proctoscopic examination negative.
‡ Gold sol and serology negative.

TABLE 2

Data on intake and excretion of fat

Case	Dietary fat, gm. per day	Fecal fat: Gm. per day	Fecal fat: Per cent of dry weight of stool
1	104	69.5	63.3
2	145	66.7	46.5
3	122	51.0	48.0
4	100	24.9	27.5
5	100	40.7	45.4
6	96	13.9	—
Normal	100	<7.5	<30

duration. The family history was positive for diabetes. For two years he had noted an epigastric burning pain with relief on ingestion of food, and for three months he had noted constipation. Examination of the eyes revealed diabetic retinopathy; the pupillary reflexes were normal. The physical examination disclosed no other abnormalities. A duodenal ulcer was demon-

TABLE 3

Fecal excretion of fat and nitrogen
Effect of pancreatin*

Case	Dietary fat, gm. per day	Therapy	Duration, days	Gm. per day: Fecal fat	Gm. per day: Fecal nitrogen
4	100		3	24.9	4.76
		Pancreatin, 5 gm. t.i.d.	3	30.3	6.37
			3	23.8	7.16
5	100		3	40.7	5.20
		Pancreatin, 5 gm. t.i.d.	3	47.9	5.53
			3	39.2	3.83
6	96 (1942)		15	12.1	4.30
		Pancreatin, 10 gm. t.i.d.	10	24.4	5.95
			5	19.6	4.4
		Pancreatin, 6.7 gm. t.i.d.	10	25.6	6.25
			5	13.3	3.9
	100 (1945)		6	18.6	4.7

*Triple-strength, enteric-coated tablets (0.3 gm. each).

strated roentgenographically. The patient was placed on a program of medical treatment for diabetes and duodenal ulcer. He returned to the clinic three months later because of continuous pain in his feet, alternating constipation and diarrhea, and epigastric distress which was not characteristic of either ulcer or pancreatic pain. Neurologic examination disclosed only diminished muscle stretch reflexes and vibratory perception in the legs.

The patient returned to the clinic in 1944 because of intermittent, severe diarrhea of 10 years' duration characterized by the appearance of food particles in the stools as early as 2 hours after meals and by nocturnal fecal incontinence. The patient stated he had had as many as 50 bowel movements in a 24-hour period. There was no gross steatorrhea. The patient had lost 9 pounds during the preceding 10 years. He had noted paresthesias of the feet for four years.

On examination the blood pressure was 115 mm. of mercury systolic and 50 diastolic. The pupils were Argyll-Robertson in type. Diabetic retinopathy was again noted. The liver was palpable two fingerbreadths below the right costal margin. The tone of the anal sphincter was diminished. Knee and ankle jerks were absent and the perception of pain, light touch and vibration was impaired in the lower extremities. There was minimal edema of the ankles. Three hundred cubic centimeters of residual urine were found in the bladder immediately after urination. Laboratory findings are given in tables 1, 2 and 3.

Attempts to study the duodenal contents following administration of secretin were unsuccessful because the tube failed to enter the duodenum. The results of studies carried out while the dietary intake of fat was 100 gm. per day, given in table 3, indicated that the patient had marked steatorrhea and azotorrhea, which were intensified during therapy with pancreatin. Following the patient's return home, severe diarrhea persisted in spite of all therapeutic attempts, including further oral treatment with pancreatin. The patient died in 1946.

Case 6. A white man, aged 35 years, was first seen at the clinic in 1942. At that time there were a history of severe diabetes mellitus of 12 years' duration and a positive family history for diabetes. For the preceding 6 years the patient had noted intermittent diarrhea with as many as 20 stools daily. Food particles were frequently noted in the stools following meals; the patient also experienced nocturnal diarrhea with fecal incontinence. There was no history of pain suggestive of pancreatitis. For four years the patient had been impotent. For one year he had noted paresthesias involving the feet. During the four weeks immediately preceding examination there had been difficulty in urinating. The patient had lost 20 pounds in the 6-month period prior to his coming to the clinic.

On examination while the patient was supine, the blood pressure was 120 mm. of mercury systolic and 80 diastolic and the pulse rate was 80 beats per minute. After the patient had stood for 5 minutes, the blood pressure was 70 mm. systolic and 50 diastolic and the pulse rate was 92. The pupils reacted poorly to both light and accommodation. Diabetic retinopathy was present. There was no glossitis. The results of examination of the abdomen were negative. The tone of the anal sphincter was diminished. The knee and ankle jerks were absent, and vibratory perception was diminished at the ankles. The sweating pattern is shown in figure 1. Neurogenic vesical dysfunction was evident on a cystometrogram. The entire neurologic syndrome was characteristic of pseudotabes diabetica. The results of laboratory studies are given in tables 1, 2, and 3. The patient noted a definite increase in the severity of his diarrhea and abdominal discomfort during the time he was taking pancreatin.

A transurethral resection of the vesical neck was done for control of urinary retention. Following the patient's return home, therapy with liver extract and thiamine was instituted. Intermittent diarrhea with three to five watery stools daily continued although other symptoms of neuritis became quiescent. Further trials of treatment with pancreatin were of no benefit.

The patient returned to the clinic in 1945 because of a protracted episode of malaise, anorexia, and vomiting following a respiratory infection six weeks previously. There had been a loss of 23 pounds during the preceding 4 months. The findings on physical examination were similar to those noted in 1942 except that the liver was palpable one fingerbreadth below the right costal margin. Laboratory findings at this visit are given in tables 1 and 3.

The patient died three months after his return home, a few hours after the onset of upper abdominal pain. At necropsy elsewhere the pancreas weighed 135 gm. and gross evidence of destruction or atrophy was lacking. On histologic study the acini were of usual appearance, although in some areas there was an increase of interlobular connective tissue. The stomach and small intestine were markedly distended but otherwise grossly normal. Other findings were myocardial fibrosis, acute pyelonephritis with minute abscesses of the kidneys, pulmonary congestion, cloudy swelling of the liver, and fibrosis of the spleen.

COMMENT

The symptoms of intractable diarrhea with postprandial and nocturnal exacerbations and fecal incontinence in the foregoing cases (table 4) are similar to those previously reported in cases of diabetic diarrhea in which steatorrhea had not been demonstrated. It should be noted that the diabetes antedated the onset of gastrointestinal symptoms by an average of seven years and a minimal interval of two years. The diabetes was severe in all cases, requiring more than 30 units of insulin daily for control (table 5). Three of the six patients had diabetic retinopathy, and in two there was evidence of nephropathy with renal insufficiency. In all cases there were neurologic abnormalities, which are presented in table 6. The finding of altered sweating patterns (figure 1), postural hypotension, impotence, vesical dysfunction, and pupillary abnormalities provided evidence of widespread autonomic neuropathy, which has previously been reported in association with diabetes mellitus.[6, 9, 11, 16–18, 20, 21]

Although there was no history of bulky, malodorous stools, the laboratory evidence of steatorrhea was striking in all cases (table 2). In addition three cases, in which nitrogen balance studies were performed, exhibited an associated azotorrhea. In all instances the gastrointestinal symptoms were exceedingly disturbing to the

TABLE 4

Steatorrhea complicating diabetes mellitus with neuropathy
Characteristics of the diarrhea in six cases

	Cases
Nocturnal occurrence	5
Fecal incontinence	5
Postprandial exacerbation	5
Associated abdominal cramping	3

TABLE 5

Steatorrhea complicating diabetes mellitus with neuropathy
Clinical data in six cases

		Duration, years	
Case*	Age (years) and sex	Diabetes prior to diarrhea	Diarrhea
1	31F	19	4/12
2	34M	8	6
3	35M	2	3
4	31M	6	5
5	36M	2	10
6	35M	6	9
Average	33.7	7.0	5.5

*Diabetes, grade 4, was present in all cases. Grade 4 indicates that more than 30 units of insulin are required daily.

TABLE 6

Neurologic abnormalities in six cases

Symptom or finding	Cases
Fecal incontinence	5
Ankle jerks absent	5
Abnormal sweating pattern (data in 4 cases)	4
Impotence (data on 4 males)	4
Sensory disturbance	4
Abnormal response of pupils	3
Postural hypotension	2
Vesical dysfunction	2
Neuritic pains	2

patients. Attempts to alleviate the diarrhea by treatment with pancreatin, banthine, atropine, vitamins, cortisone, and parenteral liver extract were unsuccessful. In addition, intake-excretion studies of three patients during periods of treatment with pancreatin revealed an increase rather than a decrease in the degree of steatorrhea and azotorrhea (table 3).

A search for the basis of the gastrointestinal symptoms and the steatorrhea was made in all cases. Coexisting diabetes and steatorrhea due to involvement of the pancreas by chronic pancreatitis or neoplasm were strongly considered in the differential diagnosis. However, the clinical picture was not that usually associated with destructive or obstructive lesions involving the acinar tissue of the pancreas, and in no case was it possible to establish the presence of external pancreatic insufficiency. The lack of response to treatment with large doses of a preparation of pancreatin that has uniformly given satisfactory results in the control of steatorrhea in patients with known external pancreatic insufficiency constitutes strong evidence against pancreatic steatorrhea. Furthermore, in the one case in which necropsy findings are available there was no postmortem evidence of destruction or obstruction of the pancreatic acini. Attempts in four cases to study the duodenal contents after stimulation of the external secretion of the pancreas by the intravenous injection of secretin were not entirely successful. However, in two cases the presence in the duodenum of large quantities of pancreatic enzymes was demonstrated. Indirect evidence against the existence of pancreatic insufficiency was the lack of a history of abdominal pain suggestive of pancreatitis, the absence of roentgenographic evidence of pancreatic calcification, the severe degree of diabetes, and the long interval by which the diabetes antedated the development of diarrhea.

In no case was the clinical picture that of nontropical sprue. Glossitis, edema, tetany, osteomalacia, and hypoprothrombinemia were all lacking, and there were neither spontaneous remissions nor therapeutic responses to diet, liver extract, or vitamins. Among the other disorders considered and excluded with reasonable certainty were Whipple's disease, fistulas or anastomotic operations which bypassed a large segment of intestine, liver disease with jaundice, regional enteritis, amebic colitis, and chronic ulcerative colitis. The lack of evidence of a primary disease process which was responsible for the gastrointestinal dysfunction in this group of cases with steatorrhea is in agreement with the absence of significant gross or microscopic abnormalities in a recent pathologic study of the intestinal tract in eight cases of diabetic diarrhea.[5]

In view of the reported relationship between visceral neuropathy and diabetic diarrhea, the possible role of neuropathy in the pathogenesis of the steatorrhea deserves careful consideration. Although the precise physiologic sequelae of sympathetic and parasympathetic denervation of the intestinal tract are not known, the importance of autonomic regulation of digestion, assimilation, and evacuation is generally accepted.[1,3,4,7,8,12,13] In each case of steatorrhea included in this report there was evidence of altered intestinal motility with a decrease in transit time of food particles, a deficiency

pattern on roentgenologic examination, or both. In each case there was also evidence of autonomic neuropathy. Because of the lack of specificity of many of the subjective evidences of autonomic disturbances, however, the precise time relationship between the onset of neuropathy and the onset of steatorrhea could not be determined. In most of the cases, the diarrhea antedated the subjective phenomena of impotence, postural dizziness, vesical dysfunction, neuritic pains, and paresthesias. However, this observation does not exclude earlier, and otherwise asymptomatic, autonomic neuropathy involving the intestinal tract which was etiologically related to the steatorrhea. Conversely, neuropathy secondary to nutritional deficiencies induced by the steatorrhea has not been excluded, but this sequence of events seems unlikely in view of the not uncommon occurrence of neuropathy in diabetic patients who do not have disturbances of gastrointestinal function.

It is of interest in this regard that in two additional cases steatorrhea was associated with severe diabetes in the absence of definite evidence of diabetic neuropathy. In one of the cases, gastrointestinal symptoms similar to those recorded above occurred in a 31-year-old woman 5 years after the onset of brittle diabetes. Extensive neurologic studies, including postural blood-pressure studies and a sweating test, were normal, and analysis of the duodenal contents provided evidence of adequate pancreatic exocrine secretion. In another case, similar gastrointestinal symptoms appeared in a 31-year-old man 7 years after the onset of severe diabetes. An extensive neurologic investigation was not performed; the finding of diminished reflexes in the lower extremities was not sufficient evidence for a diagnosis of diabetic neuropathy. In these two cases there is no evidence to support a neurogenic origin of the steatorrhea, although functionally significant visceral neuropathy involving only the intestinal tract could not be excluded.

SUMMARY

Six cases of intractable steatorrhea complicating severe, long-standing diabetes mellitus with neuropathy have been reported. The symptoms of diarrhea characterized by postprandial and nocturnal exacerbations and by nocturnal fecal incontinence were similar to those previously reported in cases of diabetic diarrhea. The clinical picture was not that of external pancreatic insufficiency, nor was there evidence of other disorders characterized by steatorrhea, such as nontropical sprue, Whipple's disease, or entero-enteric fistulas or anastomoses. In all six instances there was definite evidence of diabetic neuropathy involving the autonomic nervous system. The possible etiologic role of autonomic neuropathy involving the gastrointestinal tract has been discussed.

SUMMARIO IN INTERLINGUA

Steatorrhea Como Complication de Diabete Mellite con Neuropathia: Reporto de Casos sin Apparente Insufficientia Pancreatic

Es reportate 6 casos de steatorrhea intractabile, occurrente como complication de sever diabete mellite a longe durantia con neuropathia. Le symptomas de diarrhea characterisate per exacerbationes postprandial e nocturne e per nocturne incontinentia fecal esseva simple al symptomas previemente reportate in casos de diarrhea diabetic. Le aspectos clinic non suggereva externe insufficientia pancreatic, e nulle signos esseva notate de altere disordines characterisate per steatorrhea, como sprue nontropic, morbo de Whipple, o entero-enteric fistulas o anastomoses. In omne 6 casos signos esseva presente de neuropathia diabetic afficiente le autonome systema nervose. Es discutite le possibile signification etiologic de neuropathia autonome que involve le vias gastrointestinal.

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DISCUSSIONS

HENRY T. RICKETTS, M.D., (*Chicago*): I wonder whether the possibility has been considered that serum lipids are excreted into the bowel. This is known to occur in the normal bowel; the amounts of fats which are thus excreted are usually quite small. It seems to me unlikely, I must confess, that any excretion or secretion of lipids into the intestine could be of sufficient degree to account for the large amounts of fat found in these stools. I am led, however, to ask the question whether there seemed to be any relationship between the amount of steatorrhea and the levels of serum lipids in these patients.

DAVID ADLERSBERG, M.D., (*New York*): It is of great practical importance to study the diarrhea of diabetes and to separate the entities which are probably included under this term. I believe that I have seen cases similar to those presented by Dr. Berge. I should like to cite one case in which a remarkable therapeutic result was achieved. The patient was a woman of forty-one, who has had diabetes for thirty years, since the age of eleven. For seventeen years she has had diarrhea, mostly in bouts; the last one lasted for over a year, and resulted in loss of seventy pounds in weight. She had steatorrhea with 44 per cent of fat in the dry fecal material and other evidences of nontropical sprue. She was treated with cortisone. The diarrhea stopped; she gained fifty-three pounds in weight. At the same time, her insulin requirement increased considerably; she now has to take instead of 15 units 65 to 70 units of insulin. But she is healthy to the extent that she has one bowel movement a day, and she can work.

May I ask, Dr. Berge, if dilatation and thinning of the walls of the intestine, as occasionally seen in nontropical sprue, were found in the case which came to autopsy.

Secondly, I should like to know what the serum calcium and proteins were.

DR. BERGE: In reply to Dr. Ricketts' suggestion concerning fats in the blood, these were determined in several of the cases; hyperlipemia was not present in any. As he mentioned, the large amount of fat in the stools, in some instances over more than 50 gm. a day, would probably support the idea that this fat does not enter the bowel entirely from the circulation.

In response to the question concerning the use of cortisone in treatment, I should like to say that cortisone was tried in one of our cases, but it provided no benefit whatsoever.

The possibility of coexistent nontropical sprue was strongly considered in each case. The clinical picture, however, was not that of nontropical sprue and, except for steatorrhea, laboratory evidence in support of this diagnosis was usually lacking.

With respect to the necropsy findings, one patient was examined in a hospital in his home town, and tissue for examination has not been available to us. However, it was reported to us in a detailed manner by the pathologist that significant changes were not present in the intestinal tract, including the pancreas.

JOSEPH I. GOODMAN, M.D., (*Cleveland Heights, Ohio*): In view of the unusual rarity of the steatorrhea in the literature, I should like to ask Dr. Berge about the incidence of steatorrhea among the other diabetic diarrheas encountered.

DR. BERGE: Dr. Goodman, I am afraid I cannot give you a definite answer regarding the incidence of either diabetic steatorrhea or diabetic diarrhea. It is our impression, however, that of the two disorders, steatorrhea is the rarer. The incidence of diabetic steatorrhea, I am sure, would depend in part on how careful a search was made for the condition.