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Diabetes and Oesophageal Dysfunction

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Summary: Cineradiographic and manometric studies of oesophageal function were carried out in four different groups of patients (those suspected of having oesophageal disease, with and without symptoms; and in two groups of diabetes, with and without diabetic neuropathy). All groups were found to have abnormal oesophageal function, and this was not commoner in those with diabetic neuropathy.

Introduction

Neurological complications of long-standing diabetes are well known. The effects on the gastrointestinal tract have also been well documented, but not those on the oesophagus. Oesophageal dysfunction in diabetic neuropathy has been specifically investigated clinically and cineradiographically (Mandelstam and Leiber, 1967). Subsequently it has been suggested that further information might be gained by the use of intraluminal manometry (*British Medical Journal*, 1967). It was for these reasons that the present study was undertaken.

Methods and Results

Routine cineradiographic and manometric studies of the oesophagus were performed during the past year on all patients in this series. The patients, who ranged in age from 30 to 75

(mean 38) years, were subdivided into four groups (see Table), each group consisting of 20 cases matched in age and sex. The manometric studies were carried out by means of three water-filled Portex tubes with lateral openings attached to external transducers and a Beckman Dynograph pressure-recording machine.

FIG. 1.—Incoordinated peristalsis. S=Swallow.

Analysis of Findings in the Four Groups

Group	No. of Cases	No. with Oesophageal Symptoms	No. with Abnormal Cineradiographic Changes	No. with Abnormal Manometric Findings
1. Non-diabetic and asymptomatic	20	0	4 (20%)	9 (45%)
2. Non-diabetic and symptomatic	20	20 (100%)	14 (70%)	14 (70%)
3. Diabetic unassociated with neuropathy	20	8 (40%)	5 (25%)	12 (60%)
4. Diabetic with neuropathy	20	8 (40%)	11 (55%)	12 (60%)

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In the control group (non-diabetic, asymptomatic group), who were being investigated for suspected oesophageal disease,

the results showed that 11 patients were normal in all respects. Of nine patients who had abnormalities, two had sliding hiatal

herniae (one with adequate and one with inadequate sphincteric pressures) and seven had abnormal primary peristalsis in the body of the oesophagus. Abnormalities were found on x-ray examination in only four patients (two had sliding hiatal herniae and two abnormal oesophageal peristalsis).

In the second group all the patients had oesophageal symptoms which correlated well with the abnormal cineradiographic and manometric studies. Of 11 patients who had sliding hiatal herniae six had normal oesophageal peristalsis and five incoordinated peristaltic waves (Fig. 1). The inferior sphincteric pressure was normal in three of the patients with herniae. Primary incoordination was found in a further seven patients and diffuse spasm in one (Fig. 2). Typical peristaltic abnormality was also found in one patient with scleroderma. Cineradiography demonstrated sliding hiatal herniae in 11 patients and abnormal oesophageal motility in three others.

In the third group only eight patients had oesophageal symptoms, but 12 had manometric evidence of abnormalities (Fig. 3). One patient had a sliding hiatal hernia with an absent barrier pressure at the sphincteric area, and 11 others showed evidence of incoordination of primary peristalsis. Cineradiographic studies demonstrated hiatal hernia in one patient and abnormal peristalsis in four.

In the fourth group eight had oesophageal symptoms, while 12 were asymptomatic, though two of

FIG. 2.—Spastic oesophagus. Relief with hyoscine butylbromide (Buscopan).

FIG. 3.—Diabetic with normal peristalsis.

FIG. 4.—Diabetic with incoordinated peristalsis

them had sliding hiatal herniae. Of the 12 patients with abnormalities demonstrated by manometric studies, four had sliding hiatal herniae (two with adequate barrier pressures at the inferior sphincter) and eight evidence of incoordination of primary peristalsis (Fig. 4). Cineradiography showed abnormalities in 11 patients, of whom six had sliding hiatal herniae and five abnormal peristalsis. The pharyngeal pressures in all four groups were within normal limits.

Discussion

In considering the abnormalities found in the gastrointestinal system (excluding the oesophagus) in association with diabetes, Katz and Spiro (1966) maintained that the changes could not readily explain the symptoms and often were not associated with any clinical manifestations. Similar views were previously expressed by Rundles (1945), who, in discussing the oesophageal changes, considered that the whole problem was a challenge.

Mandelstam and Leiber (1967) concluded that the oesophageal motor dysfunction was a common finding in diabetic neuropathy-gastroenteropathy, and that on occasion it results in dysphagia. In their group 12 had oesophageal dysfunction but only three had symptoms, while their control group had no changes at all.

The present study indicated that abnormal oesophageal function was not limited to one group and therefore that the abnormality is not specific or exclusive to the diabetic neuropathy group. The chief finding was an incoordination of the primary peristalsis in the body of the oesophagus. In each group there were cases of sliding hiatal herniae associated with both

normal and abnormal sphincteric action and peristalsis. With the exception of the non-diabetic symptomatic group, however, there was no correlation between the clinical picture and the cineradiographic motility studies. Similar findings of oesophageal dysfunction were reported in alcoholic neuropathy and normals (Winship *et al.*, 1968).

Furthermore, there is no explanation of the pathogenesis which could account for these changes occurring in all the groups at all ages. Vagal degeneration and myopathy of the oesophageal muscle have been incriminated, but with no absolute proof. Recently it has been considered that diabetic neuropathy is a metabolic defect predominantly affecting fat metabolism within the cell which is associated with hyperplasia of the basement membrane (Bischoff, 1968). This latter investigation was carried out on peripheral nerves, and not on the oesophagus or on the rest of the gastrointestinal tract.

The conclusion therefore drawn from the present study is that identical cineradiographic and manometric evidence of oesophageal dysfunction can occur in all four groups investigated and not only in those affected by diabetes, with or without neuropathy.

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Selective Vagotomy without Drainage. An Interim Report

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[WITH SPECIAL PLATE BETWEEN PAGES 692-693]

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Summary: The results of surgical treatment for duodenal ulcer were compared in two groups of patients—51 who had undergone selective vagotomy without drainage and 17 who had had selective vagotomy and pyloroplasty. It is suggested that in the absence of organic pyloric or duodenal stenosis the former method seems both preferable and desirable, since postoperatively dumping does not occur and there is a steady improvement in gastric emptying.

Introduction

The addition of a drainage procedure (pyloroplasty or gastrojejunostomy) to a truncal abdominal vagotomy is now standard practice. This was introduced soon after vagotomy was first used in the treatment of duodenal ulcer, in order to relieve the pronounced gastric retention which, it was found, followed truncal vagotomy alone. At that time vagotomy was often performed in patients with advanced duodenal disease with stenosis. We are not aware of published results describing the effects of truncal vagotomy in the absence of stenosis in man.

Total abdominal (truncal) vagotomy (Fig. I) denervates the pylorus, prepyloric stomach, and duodenal bulb, but bilateral selective vagotomy (Fig. II), by virtue of its anterior selective nerve section, preserves the vagal supply of these parts. Our work was started in the hope that this preservation might allow normal gastric emptying without a drainage operation. Dumping, which is more common and more severe than we had earlier thought after vagotomy and drainage, might then be eliminated. Probably dumping is caused not by vagotomy but by excision of the pylorus or by bypassing it as in gastric resection or gastrojejunostomy, or by division of it as in pyloroplasty.

There are other possible advantages of omitting the pyloroplasty. It may be that some postoperative diarrhoea is also due to loss of the normal pylorus, and it seems that steatorrhoea may be caused by the pyloroplasty rather than by the vagotomy (Wastell, 1966). Again, the risk of leakage from the pyloroplasty is eliminated. Soiling of the peritoneal cavity and the wound should not occur.

With these considerations in mind it was decided to perform bilateral selective vagotomy without drainage in a series of patients with duodenal ulcer, some with no duodenal narrowing whatever and some with moderate though not advanced organic stenosis. A preliminary report of this work has already been made (Burge, 1968). The success or failure of this

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