POSTVAGOTOMY DYSPHAGIA*

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Vagotomy associated with a drainage procedure or gastrectomy has become an established combination in the surgical management of peptic ulceration. However, vagotomy may be associated with some undesirable effects.

Postvagotomy dysphagia was first described two decades ago.[12,22] Since then an extensive literature has produced a most controversial picture based upon the findings both in animal research and in man.[18-20,24] Unfortunately there is as yet no agreement as to the innervation of the lower end of the oesophagus[12,19,20,24] and therefore no uniformity in interpreting the effects of vagal denervation. Does the transabdominal or transthoracic vagal resection produce any alteration in the function of the lower end of the oesophagus? Due to the fact that most of the conclusions are based upon animal experimentation, there is still no uniformity on this point.

PATIENTS AND METHODS

The patients who developed transient postvagotomy dysphagia were not referred to the oesophageal clinic. Bank *et al.*,[2] however, have recorded (from this hospital) a 33% incidence of transient dysphagia in 130 cases of vagotomy (31% with selective and 34% with truncal vagotomy).

A total of 21 patients with more persistent transabdominal postvagotomy dysphagia have been fully investigated. Vagotomy and pyloroplasty were performed in 14 and vagotomy and Polya gastrectomy in 7 cases.

The vagotomy and pyloroplasty group consisted of 3 females and 11 males whose ages varied between 31 and 71 years. In the vagotomy and gastrectomy group there were 6 females and 1 male whose ages varied between 39 and 66 years. All these patients were investigated by means of cineradiography, associated with the administration of antispasmodics and parasympathomimetic drugs, manometric studies and oesophagoscopy.

RESULTS

The analysis of the 21 patients in both groups is shown in Table I. There was one patient with postoperative dysphagia in whom the pre-operative barium study had revealed an area of stenosis in the mid-thoracic oesophagus (Fig. 1). This was successfully treated with dilatation.

TABLE I. ANALYSIS OF 21 CASES PRESENTED

	Vagotomy and drainage	*Vagotomy and gastrectomy*
Pre-operative oesophageal pathology		
Pre-operative stenosis	1	0
Incoordination	1	2
Neurogenic		
Oesophageal spasm without hiatal hernia	6	5
Oesophageal spasm with hiatal hernia	4	0
Peri-oesophageal pathology		
Fibrosis	2	0
Total	14	7

*Date received: 12 November 1968.

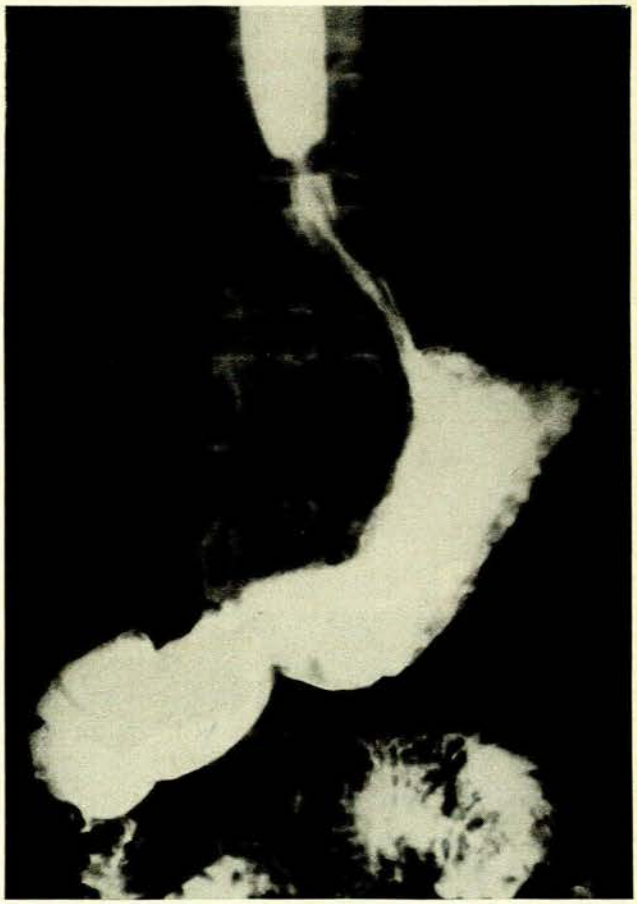

Fig. 1. This indicates prevagotomy oesophageal stenosis.

Incoordination was found to be the cause in 3 cases (1 with vagotomy and drainage and 2 with vagotomy and gastrectomy) but in none was there any reaction to Mecholyl and radiologically they did not simulate achalasia. This group responded to a modified diet and administration of an antispasmodic drug, Buscopan.

There were 15 cases in which spasm of the lower end (sphincteric area) of the oesophagus was the cause of the dysphagia (10 with vagotomy and drainage and 5 with vagotomy and gastrectomy). In this group 4 patients had developed postvagotomy hiatal hernia (Figs. 2 and 3). All patients in this group responded well to antispasmodic therapy and those with hiatal hernia responded to additional conservative management.

Another important aetiological factor seen in 2 patients was peri-oesophageal fibrosis (Fig. 4). This caused severe distress, and a combination of diet and dilatation produced relief in both cases. The true incidence of this group has not been established.

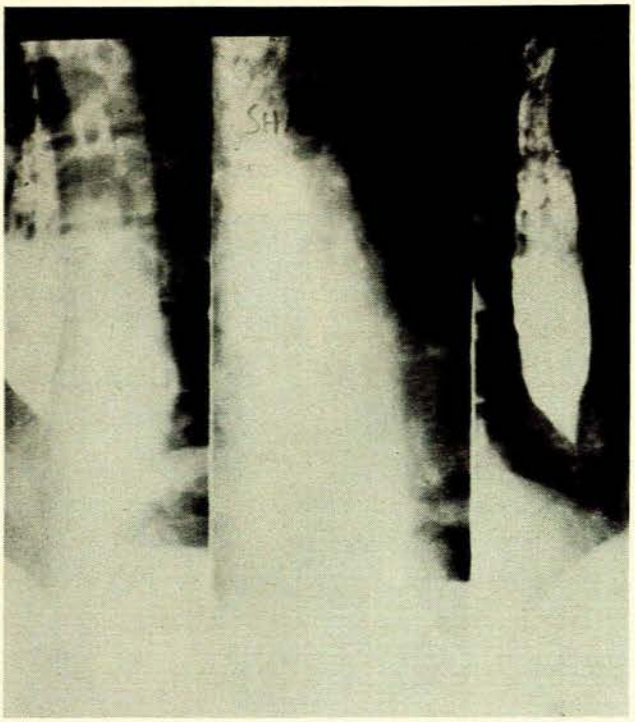

Fig. 2. Postvagotomy spasm of the lower end of the oesophagus.

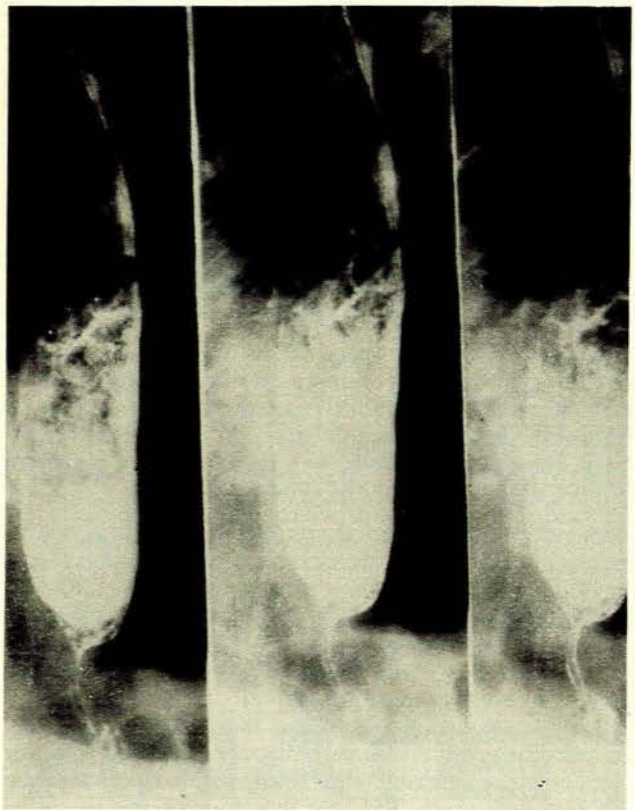

Fig. 3. Postvagotomy hiatal hernia and spasm.

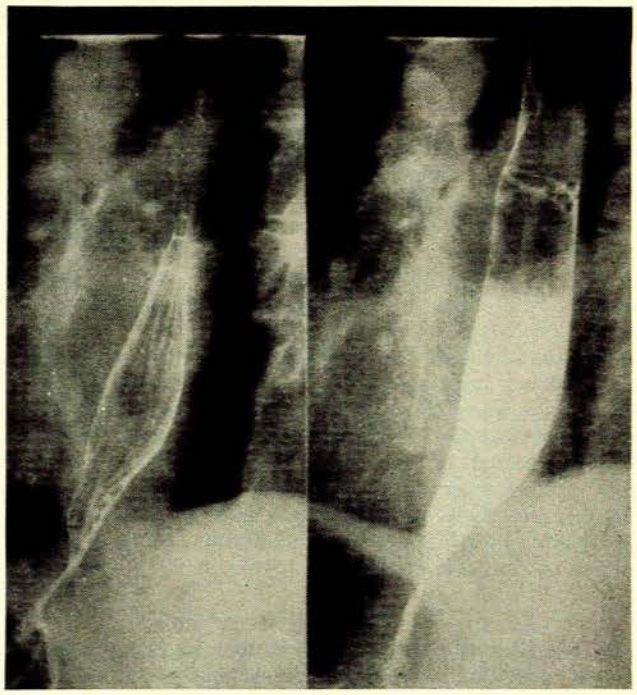

Fig. 4. Postvagotomy peri-oesophageal fibrosis.

DISCUSSION

Postvagotomy dysphagia has been recorded after transthoracic and transabdominal procedures. The incidence has varied between 1 and 40%.[4,6,8,11,13,14,17,23,29] The disability may be transient and usually improves spontaneously while the patient is still hospitalized.[9,28] Some experience more persistent dysphagia,[4] and it is claimed to be more frequent after vagotomy and gastrectomy than after vagotomy and pyloroplasty.[21,30] This disability may persist for weeks, months or years.

Is there a difference in incidence between selective and truncal vagotomy? Bank[2] found no statistical difference in the incidence of transient dysphagia. The extensive mobilization of the lower end of the oesophagus and the phreno-oesophageal ligament in both selective and truncal vagotomy may predispose to the development of a sliding hiatal hernia[1,3,10,26] with resultant reflux, possible spasm and dysphagia.

Patients undergoing vagotomy and associated procedures for the treatment of peptic ulceration should be fully investigated pre-operatively as far as oesophageal function is concerned so that the postoperative status can be clearly assessed. An appreciable number may have a pre-operative abnormality which will, in the postoperative period, be considered to be the effect of vagotomy and the cause of the dysphagia.

It is essential that cineradiography, manometry, assessment of the pH, potential difference and oesophagoscopic studies be performed in all these patients both pre-operatively and postoperatively.

Possible aetiological factors are set out in Table II. It is important to ascertain that the pre-operative oesophageal function is normal. The pre-operative functional ab-

normalities which may produce dysphagia are incoordinations and spasms of the sphincteric area and body of the oesophagus.

TABLE II. AETIOLOGY OF POSTVAGOTOMY DYSPHAGIA

I. *Pre-operative oesophageal pathology*
  Incoordinations
  Spasms
II. *Neurogenic*
  ?? Achalasia
  ?? Reflux oesophagitis; hiatus hernia
  Spasm—localized or diffuse
  Effect of nasogastric tube
III. *Peri-oesophageal pathology*
  Oedema and haemorrhage
  Granuloma/abscess
  Fibrosis
  Stump neuroma
IV. *Idiopathic*

It has been claimed that vagotomy (transthoracic more than transabdominal) produces a transient achalasia[4,14,18,22,23,29] of the oesophagus, the patient experiencing painless dysphagia with solids. This has not been confirmed by my series, and the protocols in the literature do not stand up to critical analysis in favour of achalasia. The radiological appearance of the lower end of the oesophagus simulates achalasia but it is not confirmed by manometric and drug studies.

An entity which definitely occurs and produces persistent dysphagia is spasm of the inferior oesophageal sphincter (vestibule).[6,8,25,28] The radiological appearance is suggestive of achalasia, but manometric studies and the reactions to antispasmodics and parasympathomimetic drugs (Mecholyl) indicate that the narrow segment is in spasm and that the body of the oesophagus is normal. It is interesting to note that Dragstedt *et al.*,[9] on the other hand, have remarked that they have not encountered a single case of vestibular spasm after vagotomy.

The nasogastric tube inserted in the majority of cases postoperatively may be the cause of local temporary spasm with resultant dysphagia. This spasm may be the reaction to the tube or to the resultant traumatic oesophagitis.

Postvagotomy reflux has occurred when a postoperative hiatal hernia develops.[3,16,27,29] This is not directly the effect of vagotomy, although Clarke and others[6,30] found reflux in the absence of a hernia due to decreased sphincteric efficiency after vagal section.

The reflux may in turn produce oesophageal spasm and consequent dysphagia. This is confirmed by the present series.

The incidence of postvagotomy sliding hiatal hernia can be decreased by meticulously resuturing the incised peritoneum at the gastro-oesophageal junction.[9,26,27]

A very important aetiological factor is peri-oesophageal pathology in the form of oedema, haemorrhage, granuloma, abscess and subsequent fibrosis. This is the result of over-zealous manipulation of the lower end of the oesophagus.[4,5] The end-result is an extrinsic obstruction of the oesophagus which produces severe persistent and painful dysphagia and may require persistent conservative treatment or major surgery for its relief.[4]

An occasional cause of dysphagia may be the development of a postvagotomy stump neuroma.[13] There may be an idiopathic group in which no abnormality by any modality is found.[7]

In the majority of instances the dysphagia is not due to the vagal section *per se* but due to pre-operative abnormalities or trauma during the operative or postoperative periods. In attempting to reduce the incidence it may be important to dispense with nasogastric suction and to use gastrostomy instead; to mobilize the vagal trunks with great care and minimal trauma; to create absolute haemostasis, and to resuture carefully the incised peritoneum at the gastro-oesophageal junction and the lesser omentum if this has been mobilized.

SUMMARY

Postvagotomy dysphagia is a not uncommon complication. A series of 21 patients presenting with persistent dysphagia were fully investigated and the results analysed. It is mandatory that all patients undergoing vagotomy should be fully investigated pre- and postoperatively as far as oesophageal function is concerned. It is suggested that vagal section *per se* is not the most important aetiological factor. Meticulous surgery may decrease the incidence.

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