

Symptomatic hiatus hernia:

A study of the pyloro-duodenal region and the rationale of vagotomy in its treatment

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The association of duodenal ulceration and symptomatic hiatus hernia, with or without peptic oesophagitis, is well recognized. Casten, Bernhang, Nach, and Spinzia (1963) found it in 50% of their cases.

Pyloric channel disease is, in our experience, more frequently associated with symptomatic hiatus hernia than is duodenal ulceration. Unfortunately, this benign disease at the pylorus has in the past been frequently overlooked, both on radiographic examination and at operation.

In 1935 Winkelstein described peptic oesophagitis for the first time in medical history. Of his five patients, three had associated duodenal ulceration and one had a gastric ulcer. If, as we believe (Burge, Gill, and Lewis, 1963), lesser curve gastric ulcer is caused by antral retention secondary to pyloric channel or duodenal disease, then four of his five patients had pathology at the gastric outlet.

Twenty years later Winkelstein, Wolf, Som, and Marshak (1954) described 20 patients with peptic oesophagitis; 16 had associated duodenal ulceration and three had gastric ulceration; 19 of their 20 patients had, on the same reasoning, duodenal or pyloric disease. Since pyloric channel disease was not generally recognized in the past—or indeed even to-day—it is possible that all these 25 patients did in fact have disease either in the duodenum or at the pylorus.

Palmer (1952) suggested that peptic oesophagitis was prone to occur in patients with pyloric stenosis. Both pyloric channel disease and duodenal ulceration may give rise to recurrent transient gastric retention. Especially is this so in pyloric channel disease, for an acute ulcer on an otherwise normal pyloric ring will produce intense pylorospasm and, from this, gastric retention and vomiting.

Dr. J. D. Stewart, taking part in a discussion following Herrington's paper on vagotomy and pyloroplasty in the treatment of hiatus hernia with oesophagitis, said: 'We have been impressed by the frequency with which evidence of pyloric hypertrophy can be found in these patients. In many cases the pyloric ring is hypertrophied, much as it would be found in the infant with congenital hypertrophic pyloric stenosis. I believe that this is a very important consideration in the operation' (Herrington, 1960).

Wieser, Allgöwer, Flury, and Markoff (1963), describing the radiological appearances of what they regarded as pyloric hypertrophy in the adult, noted that 20% of their 44 patients with this hypertrophy also had hiatus hernia.

The association of congenital hypertrophic pyloric stenosis with hiatus hernia in babies is well documented. Roviralta (1952) described this association as the phreno-pyloric syndrome. He believed that the raised intragastric pressure secondary to the obstructive pyloric lesion forced the stomach into the chest.

In this connexion, the paper by Johnston (1960) is important. He wrote: 'Some of the clinical features of infantile hiatus hernia, even when there is no associated hypertrophic pyloric stenosis, also suggest the presence of a gastric emptying disorder. . . . Interference with gastric emptying is suggested by the frequent clinical finding of visible gastric peristalsis and by the fact that the vomiting itself . . . is usually forcible or even projectile. . . .'

Winkelstein (1935) found high fasting gastric acid levels in all his patients with peptic oesophagitis. Fasting free HCl is almost entirely cephalic-phase in origin, and this phase is abolished by vagotomy. Casten *et al.* (1963) found, on average, that fasting gastric acid levels were higher in patients with symptomatic hiatus hernia

without oesophagitis than in an asymptomatic group.

Both duodenal ulceration and pyloric channel disease are best treated surgically by vagotomy and pyloroplasty. This operation cures both these conditions in spite of the fact that pyloric channel disease is frequently associated with hyposecretion or absence of HCl in the fasting stomach and a low response to histamine. This state of affairs might well be called a vagal paradox. We have yet to explain it.

To treat hiatus hernia by any operation without at the same time attempting to cure the more distal lesion, whether it be duodenal ulceration or pyloric channel disease, seems irrational.

THE PRESENT STUDY

When, in 1962, we found not only duodenal ulceration but also pyloric channel disease associated with symptomatic hiatus hernia, we began to study the duodenum and the pylorus in all patients with hiatus hernia subjected to operation. This association of symptomatic hiatus hernia and pyloric channel disease has already been reported by one of us (Burge, 1964). In our present study we made use of the following:

HISTORY This we regard as most important. Almost invariably in this series of patients the onset of dysphagia and heartburn from reflux has been preceded by a history, often long, of pain one to two hours after food, typical of duodenal ulcer or of pyloric channel disease. In many patients there has been a history of intermittent vomiting of food which had clearly entered the stomach one or more hours previously. We presume this vomiting was caused by intermittent pyloric or duodenal disease.

PRE-OPERATIVE RADIOLOGY It is essential that the radiologist should be familiar with all the appearances found in pyloric channel disease. There must be excellent pictures of both the pyloric ring and the prepyloric stomach, and careful studies should be made for evidence of gastric retention. Figures 1 to 7 are examples taken from our series.

EXTERNAL EXAMINATION OF PYLORUS AND DUODENUM AT OPERATION Peritoneal scarring in duodenal ulceration is frequently found, and the typical stippling is produced by rubbing the surface of the duodenum with a swab. Occasionally, scarring exactly at the pylorus is found, but this is rare because the thick pyloric muscle separates

the peritoneal surface from the underlying mucosal disease.

INTERNAL EXAMINATION OF PYLORIC RING THROUGH A PREPYLORIC GASTROTOMY INCISION It is important to understand the normal state when the pyloric ring is examined digitally in this way. Considerable experience is necessary.

EXAMINATION OF MUCOSA FOR ULCERATION AND MUSCLE FOR HYPERTROPHY WHEN THE GASTROTOMY INCISION IS CARRIED ACROSS THE PYLORUS IN MAKING THE PYLOROPLASTY Pyloric and prepyloric muscle hypertrophy is commonly found in pyloric channel disease. Again the normal must be known.

RESULTS

Since we first appreciated the association of pyloric channel disease with hiatus hernia, 44 consecutive cases have been treated by vagotomy and pyloroplasty together with repair of the hernia. Thirty (67%) of our patients gave a history of intermittent vomiting of food, which had clearly entered the stomach before the onset of dysphagia and reflux; 12 (27%) showed evidence of gastric retention on radiological examination.

Concomitant duodenal ulceration was present in 22 (50%) and pyloric disease in 34 (77%). This apparent discrepancy is due to the not infrequent association of basal duodenal disease with pyloric channel disease in the same patient. When duodenal ulceration has been the associated disease, the lesion has almost always been in the base of the duodenal cap, where it can easily (from oedema, spasm, or fibrosis) obstruct the pyloric canal.

Lesser curve gastric ulcer was found in six patients (14%). In each of these either pyloric channel disease or duodenal ulceration was present.

In only one patient in this series was there no clinical, radiological, or surgical evidence of pyloric or duodenal disease. This patient had atypical symptoms and has had severe psychiatric illness since the operation. We believe she was a case of asymptomatic hernia unwisely treated by operation.

There were seven patients in this series with peptic oesophageal stricture from peptic oesophagitis. In five of these gastric acid studies could not be made because a naso-gastric tube could not be passed. In the other two there was con-

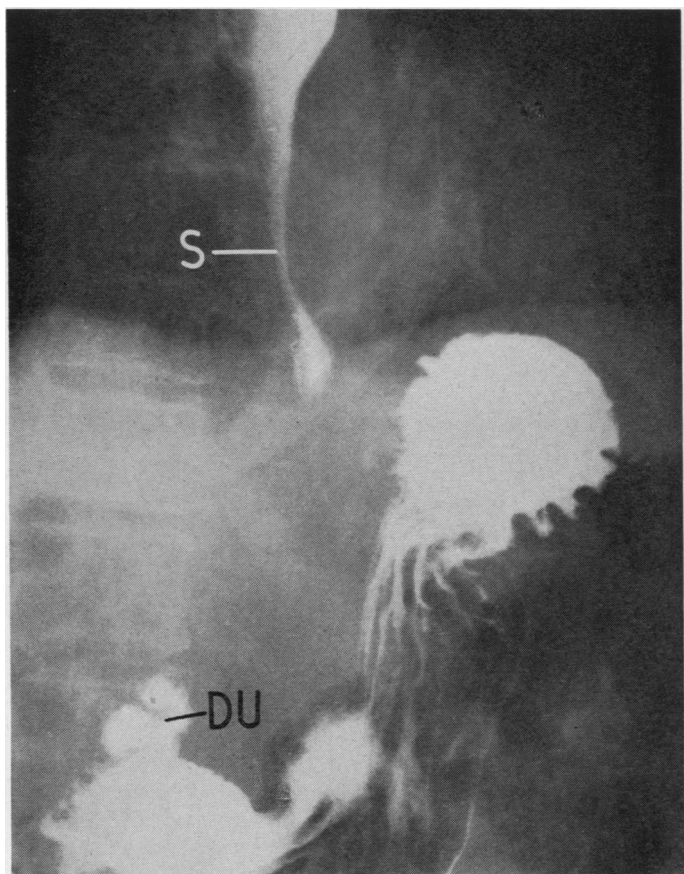


FIG. 1. *Associated duodenal ulceration. Shows a fibrotic oesophageal stricture (S). The duodenal cap is deformed (DU) from chronic ulceration. Barium meal showed gastric retention in the two-hour film.*

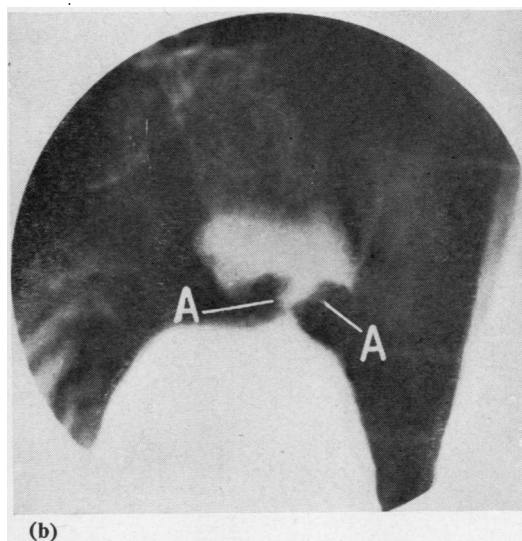
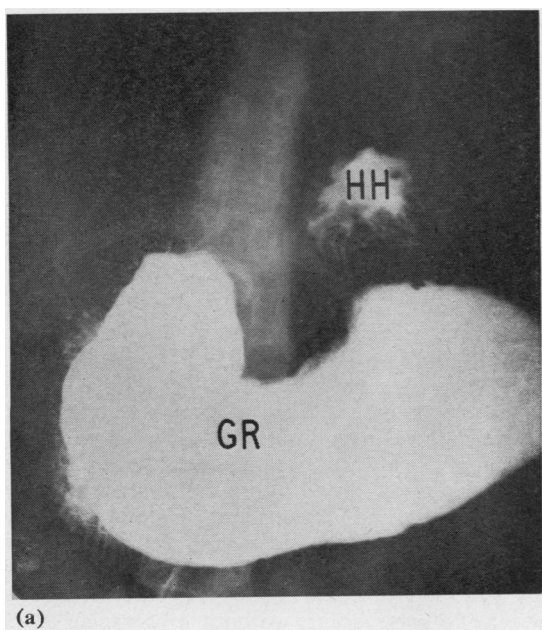
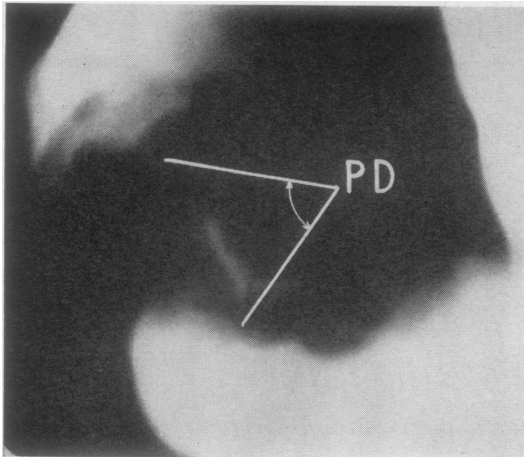
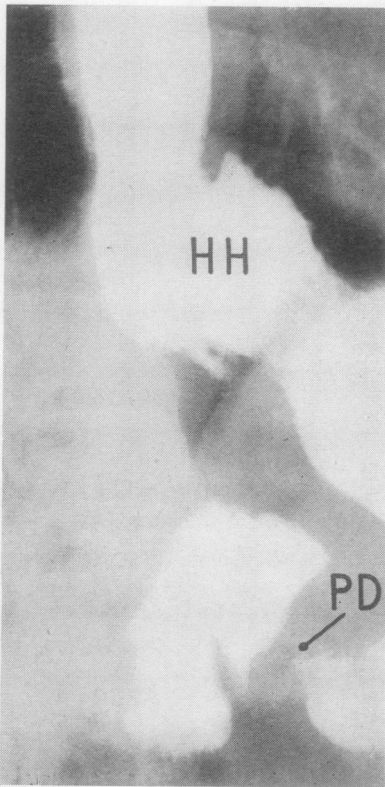


FIG. 2. *Associated pyloric channel disease: (a) hiatus hernia (HH) and gastric retention (GR); (b) an irregular pyloric canal (AA). Internal digital examination revealed a severe mucosal stenosis at the pylorus.*



(a)



(b)

FIG. 3. Associated pyloric channel disease: (a) there is gross radiological abnormality from pyloric channel disease (PD); (b) the hiatus hernia (HH) and the pyloric channel disease (PD).

siderable hypersecretion of free HCl in the fasting stomach and in response to histamine. In one the fasting secretion was 10 mEq/hr., and in the other 9 mEq/hr. (normal=1.4 mEq/hr.). The maximal histamine test in these two patients gave 39 mEq/hr. and 36 mEq/hr. (normal=21 mEq/hr.). In one of these two patients, nine months after vagotomy and pyloroplasty and repair of the hernia, the fasting acid secretion was 1.7 mEq/hr. and, after maximal histamine stimulation, 9.1 mEq/hr.

In those patients without peptic oesophagitis the acid levels have either been normal or have shown a picture of hyposecretion, often with absent free HCl in the resting juice and a low response to histamine.

The incidence of gall-bladder disease is always of interest in symptomatic hiatus hernia because of the significant frequency with which it is reported. Gall-stones were present in five (9%) of our patients.

In the first nine patients we used the Allison type of repair of the hernia, done from below the diaphragm; the results were disappointing in terms of radiological evidence of recurrence, and occasionally symptoms of reflux persisted. Our earlier experience of the Allison type of repair through both the abdomen and the chest, together with a study of the long-term results, influenced us considerably. We have since used the Nissen technique of fundoplication together with snug closure of the crura around the oesophagus. This operation has been associated with bilateral selective vagotomy and preservation of the left gastric artery. Preservation of the artery is important when the Nissen method is used. Completeness of nerve section has been proved by the electrical stimulation test in every case.

DISCUSSION

The incidence of associated duodenal ulceration and symptomatic hiatus hernia in this series agrees with the findings of Casten *et al.* (1963).

Since we have recognized pyloric channel disease, both on radiological examination and at operation, we have found it to be even more commonly associated with symptomatic hiatus hernia than duodenal ulceration. Frequently the two co-exist, and especially is this so when the duodenal ulceration is in the base of the cap where it lies up against the pyloric ring; then both diseases seem to be one. Usually, however, pyloric channel disease occurs without evidence of duo-

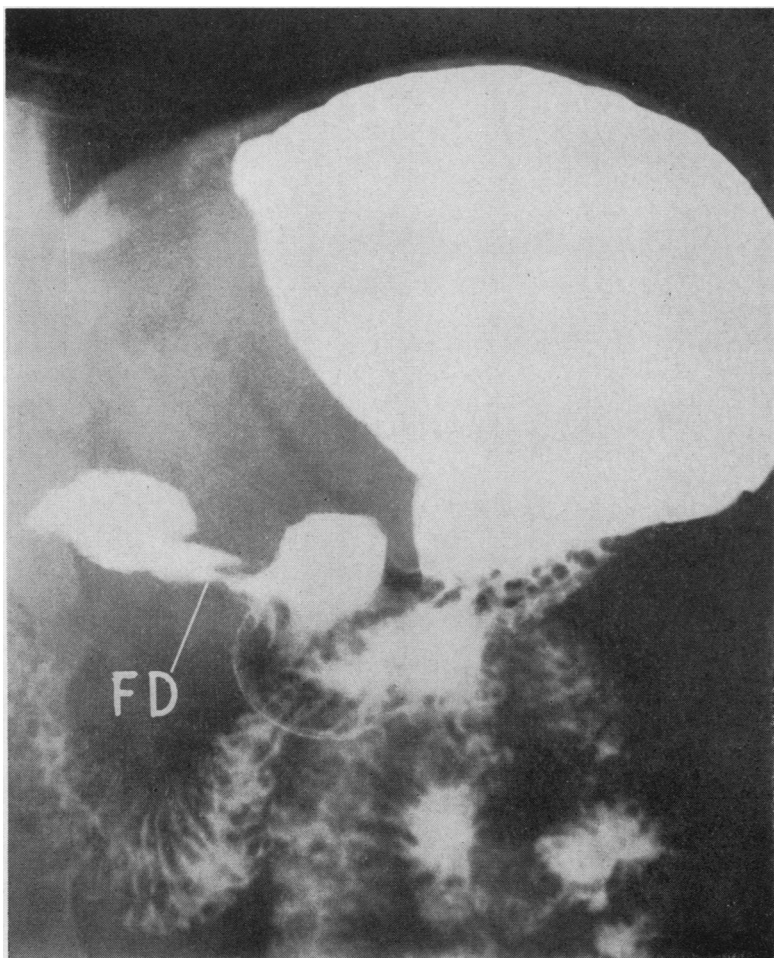
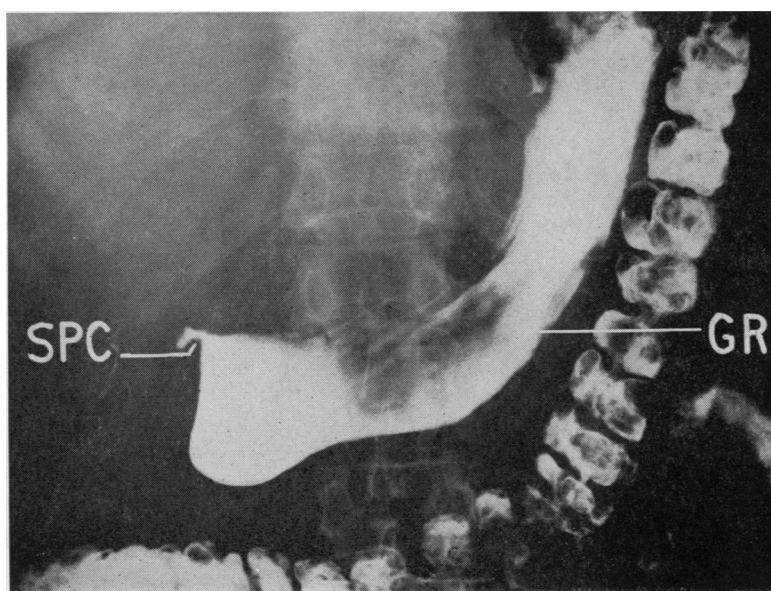
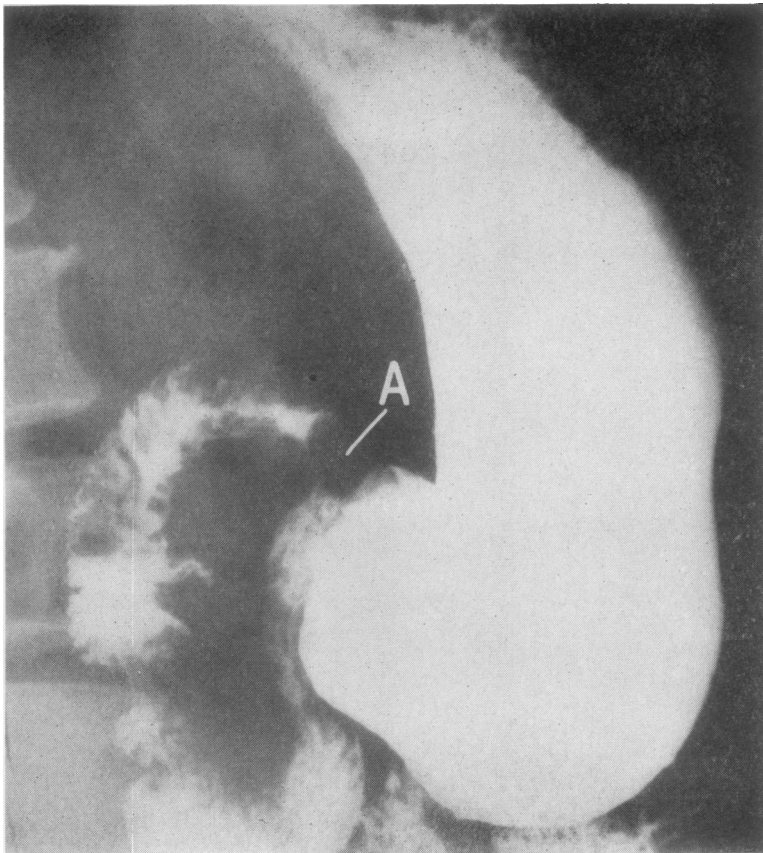


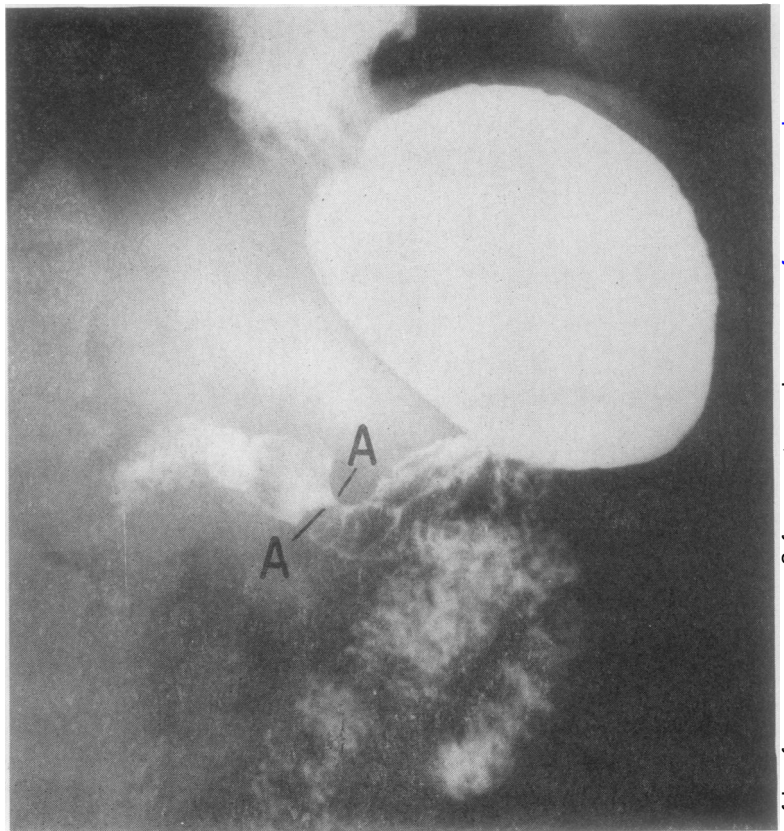
FIG. 4. *Associated pyloric channel disease. The barium meal showed a persistent filling defect which was thought to be malignant (FD). This was in fact benign pyloric channel disease.*

FIG. 5. *Associated pyloric channel disease. In this case there are a stenosed pyloric channel (SPC) and gastric retention (GR).*





(a)



(b)

FIG. 6. Associated pyloric channel disease: (a) the pyloric canal appears elongated (A) and shows the radiological appearances which have for years been associated with idiopathic adult pyloric hypertrophy; (b) the pyloric hypertrophy is well shown by the curves (AA). At operation there was considerable hypertrophy of the pyloric ring.

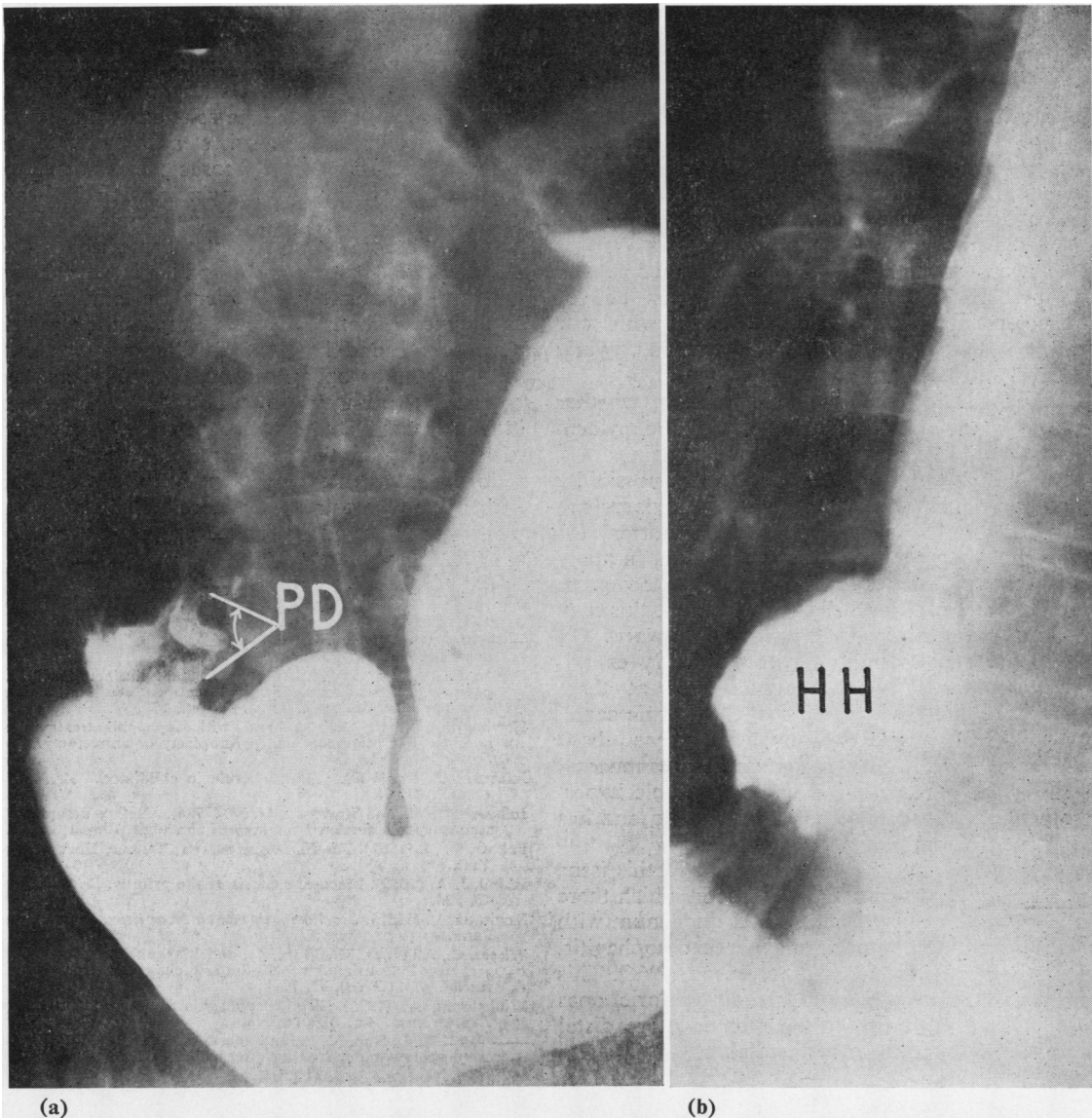


FIG. 7. Associated pyloric channel disease: (a) the typical prepyloric changes of pyloric disease (PD); (b) the hiatus hernia.

denal ulceration, and both radiologically and at operation there is not only pyloric ring change but also a definite prepyloric component, as shown by a contracted and deformed prepyloric stomach on radiography and by muscle hypertrophy at operation. On internal examination of the pyloric ring with the finger, every degree of narrowing may be found, from the earliest submucosal fibrosis to the most advanced fibrotic mucosal obstruction. This is the condition which Rhind describes so well (Rhind, 1959).

As has already been emphasized, associated duodenal ulceration has commonly been situated in the very base of the cap, where it can obstruct the pyloric canal; indeed, the whole picture of the associated distal gastric lesion seems to be mainly one of intermittent pyloric ring stenosis.

A long history of pyloric or duodenal disease with pain one to two hours after food seems almost invariably to have preceded the symptoms of hiatus hernia. This agrees with Winkelstein's (1935) observations. The fasting free HCl in the

two patients with peptic oesophagitis and stricture in whom acid estimations were done was high, and this finding, too, agrees with that of Winkelstein. The remarkable long-term results in the seven patients with peptic stricture make it clear, in our view, that no major operation should be undertaken in the treatment of benign oesophageal stricture, no matter how severe, until vagotomy and pyloroplasty has been tried.

The gastric acid levels in the patients without evidence of oesophagitis have been either normal or low. The picture has generally been one of hyposecretion. These findings agree with our earlier observations of acid levels in pyloric channel disease (Burge *et al.*, 1963).

When duodenal ulceration is present, whether it be juxtapyloric or not, vagotomy and pyloroplasty should be added to the hiatal repair.

We have given much thought to the possibility that our findings with regard to the pylorus are false and that we have mistaken the normal for the abnormal; we do not think this is so. In many cases the pyloric changes have been too gross to allow error. Nevertheless, we feel that this may be one criticism levelled against the work. On many occasions visiting surgeons, greatly experienced in gastric work, have assisted at these operations and have agreed that the pyloric canal, on internal digital examination, has been abnormal. Pyloric channel disease with its mucosal stenosis and pyloric and prepyloric muscle hypertrophy, as well as its mucosal gastritis, is, and has long been, a well-recognized lesion.

It is interesting, therefore, to read in this issue of *Thorax* the description of a case in which there is a mucosal stenosis of the distal duodenum with gastric retention, hiatus hernia, peptic oesophagitis, and stricture (Johnston and Stevenson, 1966). This case is basically the same as our pyloric ones except that the obstructive lesion is more distal and is considered to be congenital.

SUMMARY

The literature concerning the association of symptomatic hiatus hernia with duodenal ulceration is briefly reviewed. The relationship of hiatus hernia and congenital pyloric stenosis in the infant is discussed. This paper records the incidence of duodenal ulceration and of pyloric channel disease in 44 consecutive cases of symptomatic hiatus hernia with or without peptic oesophagitis and stricture. Fifty per cent. of patients were found to have concomitant duodenal ulceration and 77% pyloric channel disease. Lesser curve gastric ulcer was found in 14%. Of the seven patients with peptic oesophageal stricture, very marked hyperchlorhydria was found in the two patients in whom acid studies were possible. The effect of vagotomy in these patients is recorded. The rationale of vagotomy and pyloroplasty together with a hiatal repair is considered. Bilateral selective vagotomy and the Nissen repair have been used in all patients.

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