THE IMPACT OF HIATUS HERNIA ON THE RADIOLOGIST.

by

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Hiatus Hernia.

The purpose of this thesis is to discuss the broad aspects of hiatus hernia with a more detailed study of the symptomatology where it may be confused with the symptoms of cardiac ischaemia. In this connection I have, along with Dr. McGuinness, studied twenty patients in which we have made electro cardiographic tracings while the gastric pouch was distended with barium. I have also drawn on my radiological observations in some five hundred other patients, with their clinical and operative notes where available, and have studied twelve patients by means of an image amplifier and cine radiography.

As a result of some problems which arose in the course of this study, I decided to examine three other groups of case.

(1.) Thirty patients in some detail in order to see whether one could prevent oesophageal reflux where it was known to exist.

(2.) Fourteen patients who had had an operation designed to repair a hiatus hernia.

(3.) Forty-one patients who had a Vagotomy and Gastroenterostomy, carried out together, for duodenal ulcer. The object of this test was to assess the effect of the Vagotomy on the question of regurgitation.

Akerlund of Sweden gave a comparatively full radiological account of the condition of hiatus hernia in 1926 when he recognised the value of examining patients in the recumbent position after swallowing barium. From his writings however, it is not clear that he recognised that most of these herniae are what is now called "sliding". That is to say they reduce themselves in the vertical position, and only occur in the horizontal, or even head down or stooping position. Akerlund collected ninety-five cases from the literature and from his own practise, and quotes Healy

of the United States as having collected fifty-three cases in 1925.

Before that date hiatus hernia was considered to be rare but Akerlund also quotes Loder as having reported a case in 1784, Von Gaedeckens one case in 1837 and in 1920 Quenus of Paris as having found only two cases of hiatus hernia in one hundred and sixteen cases of diaphragmatic hernia.

Bright of London reported a case of "remarkable displacement of the stomach" in 1836

Hiatus hernia is a fascinating subject. There is now a voluminous literature relating to it and much work has been done since about 1940.

It is, of course, a herniation of a portion of the stomach, usually quite small, but commonly some 4-5.cms. in diameter, often only half this size and occasionally double. The main diagnostic point, which is that the oesophagus is entirely within the thorax and must enter the thoracic portion of the stomach at its highest point, can only be demonstrated pre-operatively by radiology. Recent work by Atkinson and others suggests, however, that it may be possible to predict a hiatus hernia or at any rate chalasia, by changes in the intraluminal oesophageal pressure recorded at the diaphragmatic hiatus. At present radiology remains a simpler and easier method of investigation, not least for the patient.

In this type of sliding hernia the oesophagus must be shortened since it enters the apex of the herniated sac in the thorax. Another type of hernia through the hiatus is the "rolling" or paraoesophageal, where the oesophagus traverses the hiatus and the stomach herniates through the hiatus beside the oesophagus. There are intermediate varieties.

The two types form an interesting contrast not only diagramatically and radiologically where they are easily

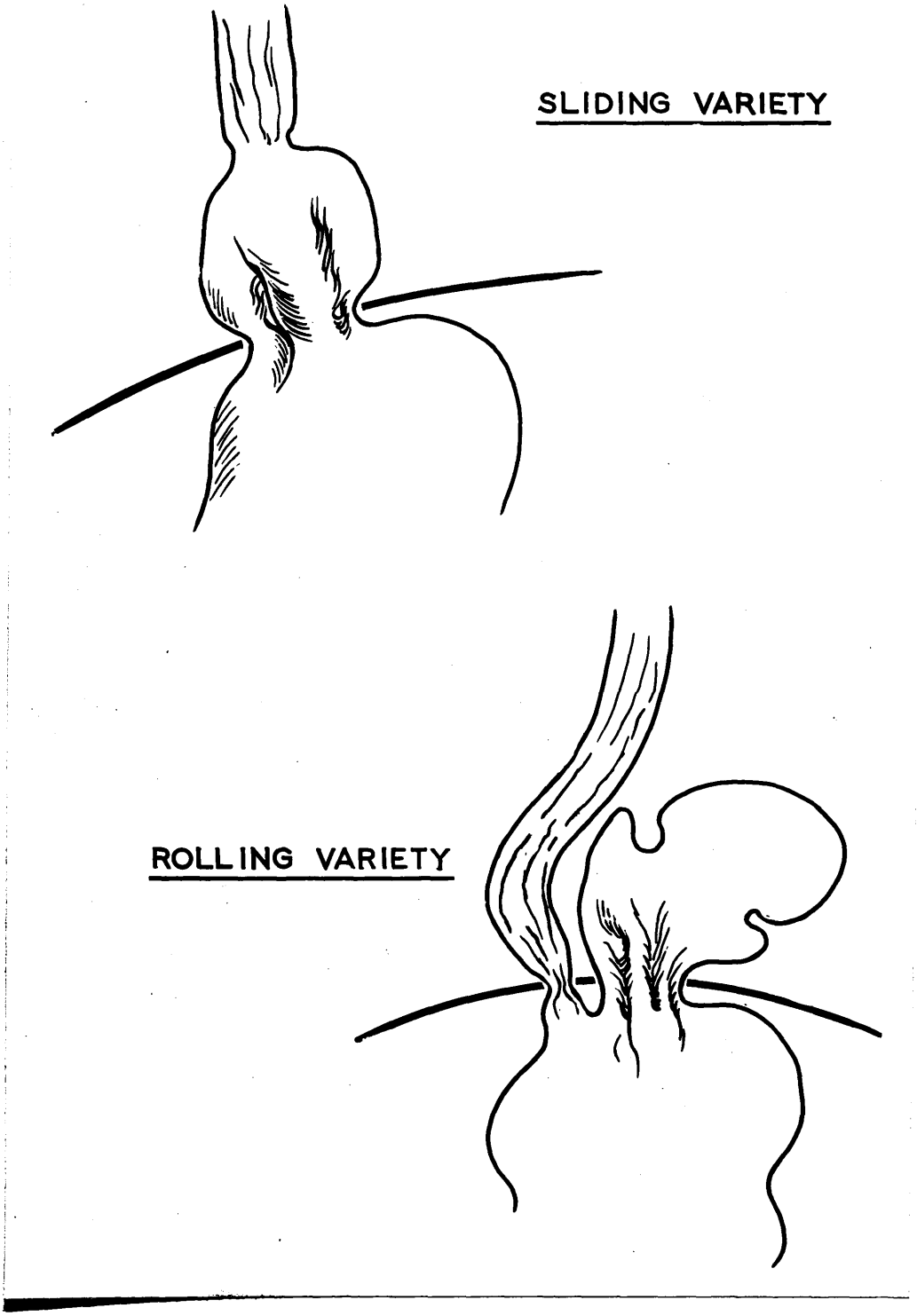


Fig. I.

Fig. II. Sliding Hernia

Fig. III. Same Patient - No Reflux After Prostigmin.

Fig. IV. Paraoesophageal Hernia.

recognised, but clinically. The former even where small and indeed difficult to demonstrate may give rise to clamant symptoms while the latter, even when large may be silent.

The fact that the oesophagus is short in the sliding type and that the condition is not only recognised but can be associated with severe symptoms in infants in the neonatal period was responsible for the view that the condition was congenital and for some years the syndrome was known as a "congenitally short oesophagus". Leonard Findlay and Brown Kelly in 1931 published their findings in a series of nine young children in whom regurgitation developed shortly after birth or when solid food was first taken. With barium swallowed in the horizontal position they found oesophageal stenosis well above the diaphragm, and below the stenosis a pouch in which they noted rugae passing through the diaphragmatic hiatus and continuous with those of the stomach. Confirmation that this pouch was in fact part of the stomach they obtained from oesophagoscopy and biopsy. (This suggests that the stenosis was spasmodic)

Findlay and Kelly regarded the oesophageal shortening as congenital although later (1936) Kelly came to the view that in some cases the shortening was cicatricial and secondary to peptic oesophagitis.

The opinion that the oesophagus may be congenitally short is based not only on the recorded observation that the condition may be and is found on occasion in the new born, but also on the development of the foregut.

The diaphragm is formed by a number of elements. The antero-central and antero-lateral parts are formed by the septum transversum and fused ventral mesentery. The septum transversum is mesodermal, arising from the upper part of the cervical region and in the 5.6.mm. embryo it is at the level of the fifth cervical segment, whence it is innervated.

It gradually descends and in the 7mm. embryo it lies below the level of the lung buds, and the anlage of the stomach, lagging behind, begins to descend more rapidly.

The rest of the diaphragm is formed by the fusion of the dorsal mesentery with the mesoderm from the Wolffian Body and subsequently with postero-lateral pleuro-peritoneal folds. The medial part, containing the aortic and oesophageal openings, is derived from the dorsal mesentery.

Normally the stomach reaches the abdomen first and during its descent two bursae from the pleuro-peritoneal recess are formed and surround the cardia. The left bursa disappears but the right may persist as an infra cardiac bursa and by stretching the hiatus may dispose to hernia.

Should the stomach lag in its descent, then the oesophagus is short. A lesser degree of this more commonly occurs where the stomach at first lags and the posterior part of the lumbar diaphragm is initially imperfect. The hiatus in consequence is formed round the gastric fundus and is enlarged. The stomach may then descend, but the hiatus remains enlarged.

The modern view, and all the recent literature supports it, is that a true congenitally short oesophagus must be extremely rare. (Smithers 1945, Allison 1951, Smellie 1952, Astley, Hodson, Johnstone 1954.)

I personally, in a paper published in 1949 in collaboration with J.B.Rennie and F.T.Land, came to the conclusion that we were dealing with acquired shortening of the oesophagus. We reviewed dysphagia over a ten year period in the Western Infirmary, Glasgow, and found in that period thirty-one cases in whom the dysphagia was attributed to hiatal hernia and shortening of the oesophagus accompanied by stricture or simple ulcer at

the lower end of the oesophagus. Two of these patients died and were examined post mortem. In neither was there a short oesophagus or gastric hernia at necropsy. Other observers Tanner (1954) Smithers (1945) have recorded similar observations and generally speaking in those cases which come to operation the surgeon also fails to find a short oesophagus.

From the radiological point of view the literature has been well reviewed by Smithers (1945) and he concludes, since "thoracic stomach (and short oesophagus) is never found at post mortem" the longitudinal muscles of the oesophagus must shorten, due to spasm induced by peptic oesophagitis, and the oesophagus in consequence recoil.

This view accords well with my own observations since in the vast majority of my cases the hiatal hernia is a true sliding one, reducing itself in the erect position and I have yet to find the surgeon who, at operation, or the pathologist who, at necropsy, has found a true congenitally shortened oesophagus.

Permanent shortening can occur due to fibrosis and cicatricial contraction secondary to long standing peptic oesophagitis or ulcer. This complication is, however, not common.

One other point in the argument against congenital shortening of the oesophagus is the well established fact that other congenital lesions of the oesophagus such as stenosis, atresia, fistula and reduplication are found while I have been unable to trace any record of a reported case of verified congenital shortening.

In this connection an interesting suggestion has been put forward by Waterston (1954) that the normal position of the foetus in utero in the last months of pregnancy might well incline towards gastric hernia through the oesophageal opening, since the foetus is inverted and there is some gastric secretion.

Anatomy.

This section is taken almost entirely from the standard text books on anatomy and from the papers of Low of Aberdeen (1907), Allison of Leeds (1951) and Collis et al of London (1954).

The oesophageal hiatus in the diaphragm is formed by the right crus of the diaphragm. The fibres of the right crus arise from the main tendon and in varying degree from the median arcuate ligament, and it is the fibres of the right crus which decussate to form the boundary wall of the oesophageal hiatus. According to Allison they constitute a type of sling, which holds the oesophagus, and is largely responsible for the oblique entry of the oesophagus into the stomach. In Allison's view and that of many other observers this sling effect, the oblique entry of the oesophagus, and the relatively tight opening in the right crus are responsible for the prevention of regurgitation of gastric content into the oesophagus.

In those cases with a lax hiatus the sling is lax, and on the bare area of the stomach the retroperitoneal tissue is in continuity with the crural canal and the fat here may be the starting point of weakness, allowing a portion of the cardia to slide up. The oesophagus shortens and enters the stomach horizontally. Allison and those who follow his reasoning regard the tightness of the sling of the decussated right crural fibres, and the acute angle of entry of the oesophagus into the stomach as very important factors in the prevention of reflux. If there is no acute entry, and no "pinch cock" action, gastric contents can flood back easily into the oesophagus.

To continue with the anatomy. The deep fascia on the under surface of the diaphragm extends up through the

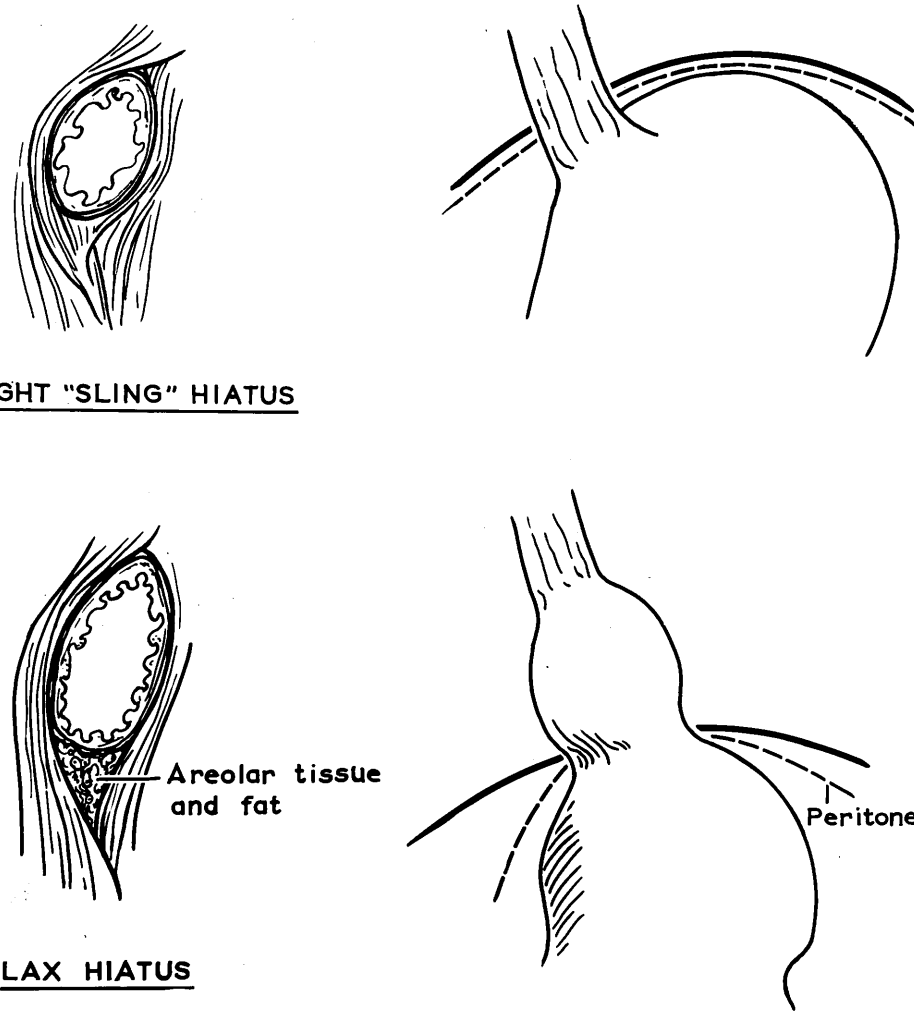

TIGHT "SLING" HIATUS
Areolar tissue
and fat
LAX HIATUS
Peritoneum

Fig. V.

hiatus and becomes continuous with the fascia propria of the oesophagus. The part between the deep fascia and the fascia propria is the phreno oesophageal ligament. It is separated from the diaphragmatic oesophagus in the middle and the peritoneal reflexion below by the left gastric vessels, para cardiac lymph glands and some cellular tissue which forms a bursa-like cushion to the diaphragmatic portion of the oesophagus.

This continuation of the diaphragmatic fascia into the fascia propria ensures a more even pull on the oesophagus by the diaphragm during respiration.

When the area is dissected one can appreciate that the decussation of the crural fibres anteriorly are soft and muscular on the diaphragmatic side, while on the upper surface they are tendinous and form a firm edge against the oesophagus.

According to Low and Collis the right crus of the diaphragm is large, and the left small, and the latter plays no part in the formation of the hiatus. The fibres of the right crus arise from the main tendon and in varying degrees from the median arcuate ligament. Some of the fibres may, in fact, arise from this ligament to the left of the mid line but they can always be easily separated from the fibres of the left crus. Collis states that there is no decussation, but an overlap. The fibres on the extreme left of the right crus, which originate in the median arcuate ligament pass up and below the other fibres and cross over to form the right margin of the hiatus, while conversely those fibres from the right side of the right crus pass above the fibres already described and form the left side of the hiatus. The effect is like the action of a double breasted coat.

There is also a transverse inter tendinous muscle which lies on the upper diaphragmatic surface and passes transversely behind the oesophageal orifice.

Like Allison, Collis and his co-workers believe that these muscles exercise a pinch cock action and when they are well developed and supple there is no regurgitation. Regurgitation in their view occurs when the muscles are under developed or are weak. They believe that all these muscles, when they contract satisfactorily, pull the diaphragm and the oesophagus down during respiration or straining and so maintain a narrow hiatus and also ensure an oblique entry of the oesophagus into the stomach.

Collis, Satchwell and Abrams of London studied the nerve supply to the diaphragmatic crura (1954) and quote Schlaffer who in 1926 sectioned the left phrenic nerve in dogs and found as a result that the left half of the diaphragm became fibrosed and atrophied. Strauss in 1933 showed that this finding applied to man.

The authors sectioned fourteen cadavers and found that the right phrenic nerve pierces the central tendon of the diaphragm to the lateral side of the inferior vena cava. At the level of the under surface of the diaphragm the nerve divides and the posterior division passes down and medially, under the central tendon, to the right crus, which it supplies. It also supplies that part of the diaphragm which is attached to the lumbo costal arches.

The left phrenic nerve pierces the diaphragm one centimetre to the left of the pericardium and three centimetres anterior to the central tendon. The posterior division of this nerve passes backwards, downwards and

medially under the central tendon to supply all the crural fibres of the diaphragm arising from the lumbo costal arches. The left phrenic nerve supplies those fibres of the right crus which are on the left side of the oesophagus, as well as the left crus proper.

An important point in the anatomy of the phrenic nerves is that they are unlikely to be accidentally cut at operation, since the right is close to the inferior vena cava and the left to the pericardium.

General.

It is not surprising that there is now a copious literature on the subject when one considers the chain of symptoms associated with hiatus hernia, and those which simulate this disease. Again there are theories regarding the cause of hernia and its production, and theories relating to the mechanism of oesophageal reflux.

If one deals with the subject systematically taking symptomatology first and the others in order one will appreciate why there is so much writing on the subject.

Essentially the symptoms are those of retrosternal pain or discomfort associated perhaps with heartburn or sour mouthfuls. If there is acute pain this may be sudden in onset and it probably represents a stretching of the tissues at the hiatus by the first appearance or thrust of the hernia. This may be dramatic or gradual with all shades of pain and discomfort in between the two extremes. As I have mentioned the large herniae may not cause much pain, and are often symptomless. This suggests that when the hiatus has been widely stretched the reason for the pain has disappeared.

The presence of sour mouthfuls or heartburn is readily explained by the presence or absence of associated reflux and by the chemical composition of the regurgitated gastric juice. Heartburn is often worse at night and Aylwin (1953) put forward the view that during the day the acid reflux is neutralised by the patient swallowing alkaline saliva, whereas when sleeping one does not swallow. If the reflux is continuous and the contents highly acid then a peptic oesophagitis may be produced which will give rise to discomfort after meals, perhaps even on swallowing. The pain after meals will be aggravated if a large or unsuitable type of repast is taken, and the patient stoops or lies down.

The oesophagitis may progress to ulceration when a more acute type of pain is noticeable. If there is an ulcer there is usually some spasm and the patient may then complain of dysphagia. It is usual for peptic ulcers to have remissions, and oesophageal ulcers are no exception, so that eventually the spasm, usually perhaps 1.cm. above the actual ulcer, becomes a genuine fibrous stricture. When this happens the dysphagia becomes more pronounced. Prior to this there may be haemorrhage, which might be sudden and severe, in the form of a haematemesis, but is more commonly a slow seeping and is really only noticeable as a secondary anaemia. It may be that the symptoms of weakness, breathlessness on exertion and tachycardia predominate, and it is for these the patient seeks advice, the hernia and reflux being discovered in the course of the subsequent routine search.

Thus we already have a large number of associated symptoms, but there are more to follow, which simulate other diseases. The discomfort may simulate gall bladder disease by taking the form of a full, blown out, or distended feeling. Sometimes the pain resembles that of duodenal ulcer by being epigastric and coming on fairly regularly after meals and waking the patient at night. Again the pain may radiate and simulate that of cardiac ischaemia or coronary artery disease. It is not uncommon for a patient to state that the pain may radiate to the shoulder and down the arm, or along the jaws.

In fact probably the commonest cause for anxiety to the clinician in these cases is the similarity of the pattern of pain, and a patient may require a repeat electro cardiographic examination and one or more repeat radiological examinations before a firm diagnosis can be made. Masters writing in 1949 on this subject referred to the

difficulties in diagnosis and found the two step exercise test followed by electro cardiography useful in distinguishing the two conditions. He remarked on the fact that coronary artery disease and hiatus hernia could afflict the same patient, but made the point that the mere presence of a hiatus hernia would never alter the electro cardiograph tracing unless the coronary arteries were already diseased.

Leather, in 1955, reviewing seventy patients remarked that the two types of pain could never be confused if a careful case history were taken. This accords with the findings of McGuinness and myself when we examined twenty patients this year. The six patients who had both conditions were aware of two distinct types of pain.

I have mentioned how a stricture can form at the lower end of the oesophagus, and the symptoms arising from this, especially if there is an associated active oesophagitis or ulcer, may simulate neoplasm. The age of the patient, the inability to swallow and the loss of weight all suggest carcinoma. Occasionally it is difficult for the radiologist and the oesophagoscopist to be certain of the diagnosis. My experience in these doubtful cases is that the radiologist is able to form a better judgement than the oesophagoscopist until biopsy is done.

Land, Rennie, and myself, in 1949 remarked on the number of patients who, in the preceding ten years, had erroneously received Xray therapy in the mistaken belief that a hiatus hernia with stricture was a carcinoma.

Hiatus hernia may be associated with any one of the conditions listed above and indeed on occasion with more than one of them.

Kaplan, writing in 1951, stated that the presenting symptoms might be

presenting symptoms might be:-

(1.) Dyspepsia.
(2.) Myocardial.
(3.) Massive Haemorrhage.
(4.) Anaemia.

He added that concurrent disease might include oesophageal, gastric or duodenal ulcer, cholecystitis with or without calculus, diverticulitis, basal lung conditions such as fibrosis, consolidation or bronchiectasis, and neoplasm.

The Electrocardiograph Tracing in Hiatus Hernia.

Gastro intestinal symptoms have for many years been associated with disease of the cardiovascular system. Thus in 1897 Osler noted that as an attack of angina pectoris ended the patient might belch quantities of wind or pass flatus from the bowel both with apparent great relief. This association has received comment over the years, by Verdon in 1920 who wrote that angina pectoris was due to acute unco-ordinated spasmodic contraction of the oesophagus and stomach. Jackson and Jackson concurred. Wood in 1954 spoke of "oesophageal arrhythmia."

For some years I have remarked on the number of requisition cards coming to the Xray department with this type of remark under the heading "Clinical History" "Chest pain on exertion or stooping, ? hiatus hernia" and how frequently these patients were examined radiologically with negative results.

Obviously there is some difficulty in a busy out patient department in distinguishing between the symptoms of hiatus hernia and those of ischaemic cardiac disease. Both conditions give rise to chest pain and flatulence and both conditions occur mainly in the older age groups, between forty-five and seventy-five (Edmunds : Wood).

Since the management and prognosis of the two groups are so different it is important to recognise them and one would think that electrocardiography and radiology would make this simple and straightforward. However, reports from various sources serve to confuse this apparently simple issue.

J.R.McGuinness and I therefore decided to study a group of persons known to have hiatus hernia and to investigate under fluoroscopic control the effect on the

electrocardiograph when the hernia was distended by a barium suspension.

We selected twenty random patients as they presented themselves at the out patient department. Sixteen were female and four were male, their ages varying from forty to seventy years (Table 1.)

Materials and Method.

Details of the history and physical examination of each patient were recorded and particular attention was paid to chest pain when present. If this was of the pattern originally described by Heberden it was called angina pectoris.

Haemoglobin and packed cell volume were estimated and any level below 80% Hb. was considered abnormal and was corrected by oral iron before the investigation proper was undertaken. This was considered important in view of the effect which anaemia has been shown to have on the electrocardiogram Ref: (Master & Others).

Weight and height were measured and we found that six patients exceeded the optimum weight by more than 10%.

The preliminaries completed, the patients attended for screening after a fast of at least four hours. An electrocardiogram was taken with the patient in the supine position on the Xray table, before and after drinking a pint of fairly thick barium emulsion, twelve leads being recorded on each occasion. These leads were 1, 11, 111, aVR, aVL, aVF, V1 to V6.

After the emulsion was swallowed, the hernia was screened and dilated to the maximum size possible by simple abdominal pressure, aided if necessary by

tilting the patient head down while in the left lateral position. If this position had to be adopted, the table was returned to the horizontal and the patient was laid supine before the second electrocardiogram was recorded. The size of the hernia was measured and its type noted on the patient's record.

The skin of the chest was marked to ensure that the position of the chest leads remained constant throughout.

In eight patients, chosen at random, Trinitrin (glyceryl trinitrate 1/120 gr.) was administered at this stage. The behaviour of the hernia was watched and after a minimum of two minutes a third electrocardiogram was taken.

Results.

Of the twenty patients studied, a history of angina pectoris was obtained in six, one male and five females. In each case the patient was aware of having two pains which behaved in different manners, occurred under different circumstances and were relieved by different means.

For the purpose of the trial, any hernia which projected more than five centimetres above the diaphragm was considered to be large. There were eleven large herniae.

Six of the twenty herniae were seen to vary in size during the screening and presumably while the tracings were being recorded.

Eight patients had a haemoglobin of less than 80% when first seen. Repeat estimation before examination confirmed that treatment had brought it above this level.

Careful study of the initial electrocardiograms showed that six were abnormal before the hernia was distended.

The abnormalities were myocardial ischaemia in three, low voltage tracing in two and potential (Grade 1.) heart block with myocardial ischaemia in one. Cyclical variations in the T. waves were noted in seven patients, these variations being present before and after visual-:isation of the hernia.

These T. wave changes were not seen in any patient who was found to have variations in the size of the hiatus hernia.

Changes were found in six electrocardiograms following distension of the hernia. In the original tracings, four of these were normal and two were abnormal.

No changes were observed in rate, rhythm, P. waves and P.R. interval or S.T. segment.

The herniae were large and fixed in two, and small in four. Four of the six received trinitrin which produced no alteration in the electrocardiagraphic pattern seen in the second tracing.

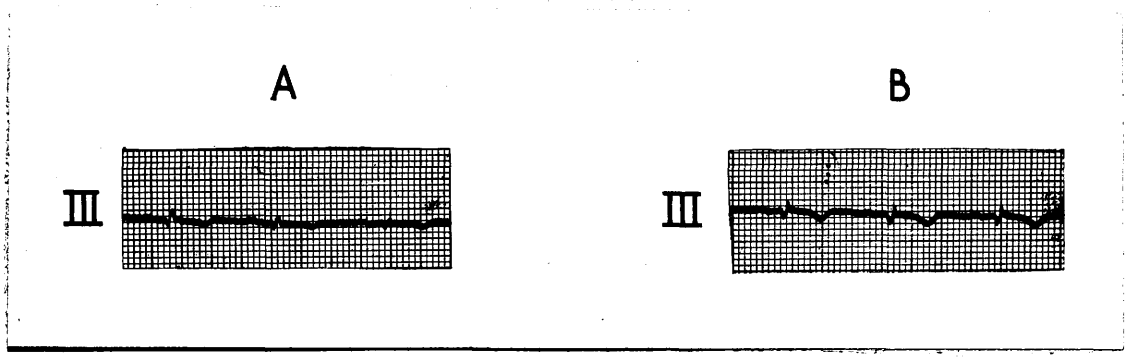
These findings are fully set out in Table 11, and the changes illustrated by Figs. 6 and 7.

Although not the aim of the trial, the patients were asked to comment upon the sensation produced by the procedure of filling the hernia with this heavy barium emulsion. Every one was aware of a sensation of fullness in the epigastrium, or behind the foot of the sternum, but in only one was pain produced.

This patient, whose initial electrocardiogram showed myocardial ischaemia, received trinitrin; following this she developed very free oesophageal reflux, well seen on screening, and accompanied by loud eructations and severe retrosternal pain which caused considerable distress. No electrocardiographic changes were observed in this case following

These tracings in Figs. VI. and VII. are typical of the six patients who showed changes. All the other fourteen patients showed no change.

Fig. VI.

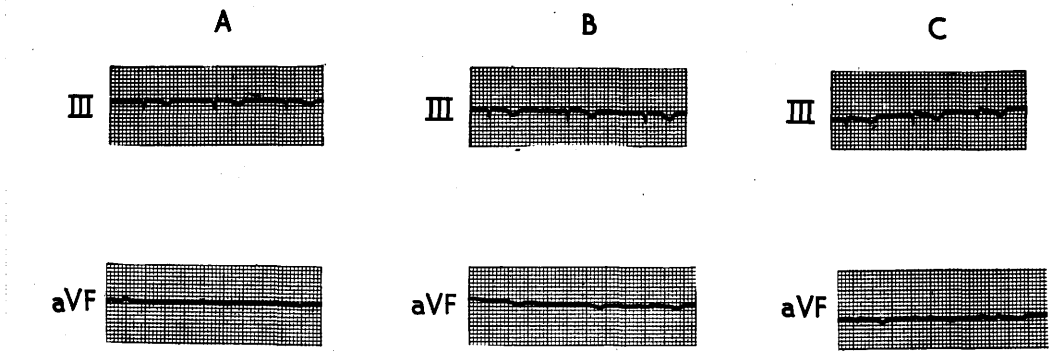


Tracing from Lead III under normal conditions - before swallowing barium.

Tracing from Lead III of same patient - after swallowing barium and with the hernia distended.
Note the deep inversion of the T. wave as compared with A.

All the other Leads showed normal tracings.

Fig. VII.



A. is Lead III & Lead aVF before barium drink. B. after barium drink and C. is with hernia still distended thirteen minutes after glyceryl trinitrate.

A. in Lead III shows T wave inverted.
B. in Lead III shows T wave more deeply inverted.
C. in Lead III shows no change after glyceryl trinitrate.

A. in Lead aVF - T wave flat.
B. in Lead aVF - T wave deeply inverted.
C. in Lead aVF - T wave essentially unchanged from B.

All the other Leads show no change.

Since there is no change in the tracing in Lead III after trinitrate and there is no essential change in the tracing from Lead aVF after trinitrate one would presume the alteration in Lead III to be positional and not vascular.

barium, and while the severe pain was present her tracing improved remarkably.

Discussion.

Various observers have applied different stimuli to the stomach and oesophagus and noted the electrocardiographic effects. Morrison and Swalm tried the effect of distending balloons in the oesophagus, and obtained sinus block in one patient, recorded while the patient was fainting. Johnson and Laing found a deepening of the Q wave with alteration in the T wave in lead lll, and Kohli and Pearson recorded coronary insufficiency.

Brotmacher, investigating post-gastrectomy patients, produced electrocardiographic changes in nine out of ten of them after they had drunk 800.ml. of cold water. In six of these T wave inversion in lead lll was found.

Bloom and Gubbay used barium to distend the hernia, and could demonstrate no electrocardiographic changes which could be attributed to hernia.

In deciding on a method for our investigation, the barium method was chosen in preference to the others, since it was considered less likely to be upsetting to the patients and would allow the investigation of a larger number.

Our findings in this series appear to follow the general pattern already described, five of the six changes affecting lead lll, one of these involving lead aVF.

This type of change has been produced before by altering the position of the patient (Goldberger) and it would seem reasonable to attribute the changes in our patients to slight alteration in the position of the heart in the presence of a distended hernia, or merely to distension of the gastric fundus by a large quantity of

barium.

The cyclical changes in T wave which we observed in six instances have of course been found by others but serve to illustrate once more the possible danger of comparing very short strips of electrocardiographic record.

Again, although not our aim to reproduce symptoms, it was interesting that the only patient in whom this was achieved had very free oesophageal reflux following trinitrin. Assuming that in some patients chest pain is due to oesophageal spasm, trinitrin might be expected to bring relief by relaxing this, as was observed here to a marked degree. It is not surprising that this type of pain might be confused with that of myocardial ischaemia.

Our results in this small series lead us to the conclusion that occasionally the presence of a hiatus hernia will cause changes in the electrocardiogram but that these are likely to affect lead III in particular and are unlikely to be confused with the changes of ischaemic heart disease.

TABLE 1

AGE	40-49	50-59	60-69	70-79	TOTAL
MALE	3	1	-	-	4
FEMALE	1	6	8	1	16

TABLE 11

AGE	INITIAL E.C.G.	E.C.G. AFTER DISTENSION	E.C.G. AFTER TRINITRIN	HERNIA
65	T Inverted 111, Flat aVF	T 111 more deeply inverted	-	Small and variable
58	T Inverted 111, & aVF, Flat 11	T 111 & aVf more deeply inverted	No change	Small
45	Normal	T 111 & aVF less tall. S 111 & aVF much deeper	No change	Large
65	T Inverted 111	T 111 more deeply inverted S, aVF deeper	No change	Small and variable
64	Myocardial Ischaemia Potential Heart block.	Occasional Ventricular Extrasystoles	No change	Large
47	T Inverted 111	T 111 more deeply inverted. T, aVF flatter.	-	Small.

ALL WERE FEMALE EXCEPT THE LAST

Efforts to Prevent Regurgitation.

Acting on the suggestion by Johnstone in Avery Jones' text book "Modern Trends in Gastro Enterology" that something might be gained by trying to prevent reflux by the action of drugs I read the paper by Robins and Jankelson published in 1926 in the Journal of the American Medical Association. In this they stated that 1.mg. of physostigmin salicylate, acting on the para sympathetic nervous system caused closure of the sphincter (sic) at the lower end of the oesophagus, and prevented regurgitation in the majority of cases. They stated that atropin, conversely, acting on the sympathetic nerves relaxed the sphincter and encouraged regurgitation. Since I was interested in preventing regurgitation I arbitrarily selected thirty patients who I knew had regurgitation or who from previous reports were said to have regurgitation and asked them to co-operate. Since these were all middle aged, or preponderantly old or elderly, who all had symptoms and who all were undergoing treatment or were under observation I put the matter fairly to them that this was experimental. I was surprised at the ready response to the test by the patients. I also, of course, asked the permission of the physician or surgeon in charge of the case to proceed and was gratified by the whole-hearted and unanimous co-operation I received.

The patients selected for this test are listed and summarised in Table III.

The method I adopted was simple. I merely gave the patients half a pint of fluid barium to drink, observing this in the upright position. When sufficient barium had been swallowed I gave a sip of water in order to clear the oesophagus. I first tried to elicit reflux by asking the patient to stoop.and touch his toes. I tilted him to the horizontal position and

generally found the hernia by this manoeuvre. I found it confusing in trying to assess regurgitation if barium remained in the oesophagus from the initial drinking in the upright position, hence the necessity for first clearing the oesophagus by a sip of water. I now tried to initiate regurgitation, if necessary by abdominal pressure, by the Valsalva test, by tilting head down and by turning the patient prone with a cushion under the abdomen and tilting head down. Sometimes swallowing saliva in this position would elicit regurgitation, occasionally a sip of water would, and sometimes a bolus of barium would. I tried to avoid a barium bolus as much as possible because of the confusion caused by the radio opaque residue left in the oesophagus. It must be remembered that people do not generally, or naturally, swallow food or fluid tilted head down and my experience is that barium when it is swallowed horizontally, tends to remain in the oesophagus for some time, often two or three minutes and sometimes for as long as ten minutes.

It is interesting to note that two of the patients, both males, who I already knew had hiatus hernia and in whom I had graphic radiological evidence of this disability, refused to oblige by regurgitating or showing a hernia at this examination. The inconstancy of the appearance of hiatus hernia, especially when it is small, is of course recognised and has been remarked on by various authors. Eight patients showed hernia, but no regurgitation.

Perhaps my most striking example of the erratic behaviour of hiatus hernia occurred in the case of a Merchant Navy Captain in a well known line. In New York he experienced sudden severe chest pain and was taken to Hospital. There they suspected a cardiac lesion but blood pressure and electro cardiography being normal he was told

the pain might be due to hiatus hernia. The doctors in the Xray department failed to produce a hernia and so took the patient, to whom they explained the procedure reasonably since I am indebted entirely to him for my information of this account of the proceedings, to the top storey of their Hospital, some ten or twelve storeys high. There they placed him, stomach full of barium, on a trolley and put blocks under the foot end so that the patient was lying head down, in an express lift. The lift was then made to descend at express speed, the doctors telling the patient that this manoeuvre seldom failed to produce a hernia if one were present. He was then wheeled into the Xray department, having experienced the correct "sinking feeling" and was examined with negative results.

The patient was dismissed with no diagnosis having been made and told he should report to his doctor when he arrived home. This he did and was referred to my department for investigation. He walked in, a sturdy well built intelligent man who gave his story while he was being examined. On tilting the patient to the horizontal position the hernia slid up easily causing no discomfort and giving no trouble whatsoever.

This may seem a long digression but I give it in order to stress the variability of the appearance of hiatus hernia and the action one must take in excluding its presence. This tale also emphasises the point that one should always accept a negative Xray finding with considerable reserve.

Having established regurgitation and hernia in the patient I made an Xray exposure for record purposes and proceeded with the next part of the test which was to inject 1.mg. of physostigmin salicylate intramuscularly. In all, two of the thirty patients, both males, produced no hernia

or regurgitation at my examination in spite of repeated manoeuvring, and of the twenty-eight who had a hernia eight at my examination had no reflux, four males and four females.

Thus of the thirty patients I examined ten had no regurgitation and twenty had. The accompanying Table lll summarises the findings.

One other complication arose. My first eight patients were given lmg. of physostigmin but I found that there were too many side effects with this, mainly a feeling of giddiness and flushing but two cases, M.J. and J.S., examined on the same day, had more severe reactions, with nausea, vomiting and a small pulse. I decided it was unfair to subject mainly elderly patients who were not by any means in extremis to what was after all an enquiry, if not an actual experiment. I therefore changed the drug in the remaining twenty-two patients to prostigmin and reduced the dose to .5mg., but later I increased this dose to .75mg. in sixteen patients.

Having given the injection I then waited for thirty minutes and re-examined the patient. The results are set forth in Table lll. From this one can see that of the twenty patients who had regurgitation before the injection of prostigmin or physostigmin six had none after the injection. The injection of prostigmin had no effect on the hernia in any of the patients.

Thus thirty per cent of the patients I examined seemed to show an improvement after the injection. This result must, however, be accepted with considerable reserve bearing in mind the fact that regurgitation in the same patient can be so variable.

For example, ten of the patients that I examined and who had reflux had been examined before and had

shown no reflux although they had a hernia. A further two who gave reflux with me had been variable at previous examinations having exhibited reflux on one occasion, and not on another.

The converse is true and eight of the patients I examined showed no reflux, although they were all reported as having had reflux on some previous occasion. The remaining patient had never exhibited regurgitation.

A most important point in the radiological investigation must be interpolated here and that is that it is necessary to clear the oesophagus of barium before examining for regurgitation. In a busy Xray department a hurried glance at the patient after he or she has been tipped into the horizontal position may show barium in the oesophagus and this column may move up and down. It may readily be assumed to be the result of regurgitation when in fact it is still a residue from the original meal. This may explain at least some of the cases where I found no regurgitation who had previously been reported as having reflux.

Taking everything into account, however, I feel that prostigmin has an influence on the musculature of the oesophagus at the level of the diaphragmatic hiatus, presumably on the intrinsic circular fibres of the oesophagus. The tests I applied to produce regurgitation were severe and the criteria I adopted were strict. In fact three of the patients who still exhibited reflux after the prostigmin injection showed an appreciable diminution in the amount, or frequency of barium reflux. Moreover in the past, in my experience, where a patient shows reflux this usually, but not invariably, continues in the same volume throughout the one examination, however prolonged. I must admit, however, that I do not recollect ever having sent a patient with

regurgitation out of the room for thirty minutes and then re-examining her.

Corroboration that vagal stimulation closes the gastro oesophageal opening comes from Botha G.S.M. in a paper published in 1958. Botha puts forward the novel view that the fibres of the muscularis mucosae play the most important part in the closure mechanism at the cardia. These folds, he claims, occur below the change in the histological cell structure from stratified to columnar epithelium and below the "empty" segment or cardiac antrum. The folds relax at death or after too vigorous palpation. Using radiography he demonstrated them in living foxes, ferrets, pigs and various other animals. While palpation relaxed the folds vagal stimulation tightened them thereby effectively plugging the orifice.

Having in mind the effect of an astringent such as alum or lemon in puckering the buccal mucosa, I gave ten of my patients with regurgitation the juice of half a lemon to drink. This had no effect whatever in preventing or diminishing oesophageal reflux.

Although I believe that stimulation of the vagus nerve by prostigmin has the effect of abolishing regurgitation in a small proportion of cases, and of diminishing it in a slightly higher proportion, I think the results are too variable for the method to have any practical value at present.

In this connection Edith Bulbeing writing in the section on gastro intestinal motility in Avery Jones text book "Modern Trends in Gastro Enterology" has this to say "The nature of gastro intestinal motility is thus very complex but it can be understood if one realises that it is the result of the integrated activity of three different regulating mechanisms. Two are nervous:-

"(1.) Extrinsic sympathetic and para sympathetic innervation.
(2.) Intrinsic nerve plexus.
(3.) Smooth muscle, whose behaviour seems to be due to the instability of its membrane.

" This leads not only to auto-rhythmicity but enables the muscle to react, like a continuously discharging receptor organ to various stimuli, chemical or mechanical, by changing its rhythm. At a low membrane potential the cells are automatically very active, where at a raised membrane potential they are less ready to discharge impulses. Thus it depends very largely on the initial state of the muscle cell whether a stimulant or inhibiting nerve impulse will be effective. It is therefore not surprising that in different experimental conditions various workers have obtained contradictory results and that they could not distinguish strictly between purely muscular responses and those involving nervous interaction, particularly as the transmitter substances merely increase or decrease the automatic motility and modify its pattern" (Edith Bulbeing).

The situation is complicated by the fact that the vagus contains cholinergic and adrenergic fibres. When impulses come along the vagus all the cells are exposed simultaneously to the depolarising effect of the para sympathetic transmitter, since at the nerve endings acetylcholine is liberated.

When sympathetic nerve impulses arrive at all the nerve endings adrenaline and nor-adrenaline are liberated and all the cells are exposed simultaneously to the hyperpolarising effect of the sympathetic transmitter, and cholinergic and adrenergic ganglia are both present in the wall of the intestine. We cannot predict which ganglia will be stimulated at any one time. The response is complicated also by the fact that smooth muscle contracts in response to stretching. The chance effect of a bolus

of wind, or a relatively large bolus of barium might precipitate contraction.

It therefore seems that in the present state of our knowledge we cannot expect to achieve any uniform benefit from the routine exhibition of sympathetic or para sympathetic stimulant drugs.

When one analyses the tables three points stand out immediately. The first is the preponderance of females in the ratio of twenty-one to nine. The second is the average age of the patients, namely sixty-two, the youngest being a male of thirty-nine. Twentyone patients are in the seventh decade and six are seventy or over. The third point is the excessive weight of most of the females. Their average height is 5ft. 2½in. and their average weight is 10st. 7lbs. as opposed to a small male group whose average height and weight are 5ft. 7in. and 10st. 12lbs.

Other points that emerge from the survey are:-

(1.) The comparatively long history. Fourteen of the patients had symptoms for over five years and one of these for ten years or more.

(2.) Not one of the patients gave any history of vomiting in infancy or childhood, a point greatly against the theory of congenital shortening of the oesophagus.

(3.) Few of the patients in this series could pinpoint a cause for the onset of their disability. I had thought that more than two would be able to date the onset from pregnancy or an operation, as Land, Rennie and myself found in our series of cases.

(4.) In most of the patients symptoms conformed to type, namely a pain or burning sensation, mostly epigastric and spreading retrosternally. The majority, twenty-six, experienced this when lying or stooping and almost half experienced the pain or exertion.

(5.) Twenty-eight experienced the pain after food, but seven qualified this statement by saying that food did not necessarily or invariably produce pain.
(6.) Ten of the patients obtained relief by vomiting, three by belching and eleven by alkalis but it is fair to assume that a good many of those using their doctor's "pills" or "powders" would, in fact, be taking alkalis.

It is interesting also that six of the nine males in this series had no reflux at this examination.

A further interesting point in this curious syndrome is that while all the patients had pain, presumably related to their hernia with or without reflux, none in this series produced the pain during my examination. This might be accounted for by the fact that the regurgitation, during an Xray examination, is of barium which probably has a soothing effect on the mucous membrane. But this brings in another argument, suggested by Professor Alstead in a personal communication. I had mentioned to him that in my experience the large paraoesophageal or rolling hernias were often symptomless, and that it was the much smaller sliding variety which was associated with pain or discomfort. Professor Alstead put forward the argument that if the hernial orifice is large then there is a small or diminished risk of pressure or trauma. If on the other hand the orifice is small there is greater risk of pressure or trauma if the viscera or tissues are forced through. When this occurs there may be some trauma to the oesophagus and as a result some oedema or irritability of the adjacent oesophageal mucosa, which will in turn be further irritated by the reflux of gastric contents.

This is a reasonable hypothesis and I can only reiterate that I failed to produce the pain, when I was able to induce even a small and difficult hernia to manifest

itself.

Some of the patients complained of a retrosternal sensation which might even go so far as to be described as discomfort. One patient in the series investigated, along with Dr. McGuinness, by electro cardiography did complain of pain.

I shall have more to say on this aspect when I discuss the findings in my small post operative group.

TABLE 111 FEMALE PATIENTS

CASE	AGE	WEIGHT St.Lb.	HEIGHT	TYPE OF SYMPTOM	DUR-ATION	RELIEVED BY	HERNIA AND REFLUX	EFFECT OF PROSTIGMIN	REMARKS FOLLOW UP
1. S.C.	78	9.	5'5"	Epigastric pain p.c. Not on lying or stooping	4yrs	Pills	Both	No effect	High E.S.R. and T.B.lungs pro-bably active.
2. H.P.	76	10.	5'1"	Reflux on stoop-ing and after food.	9yrs	Vomiting	Both	Both remain	A large hernia and reflux. After prostigmin fleeting
3. M.W.	71	10.11	5'5"	Throbbing and gurgling.	7mths	Pills	Both	No effect	Diabetic and hypertensive
4. E.P.	70	11.	5'3"	Retrosternal choking. Nausea on stooping.	12yrs	Hot water to induce vomiting	Both	No effect	
5. C.R.	70	11. 3	4'10"	Pain right hypochondrium p.c. Nausea on stooping	20yrs	Does nothing	Both Large hernia	Hernia unchanged No reflux	Operation for crystocele a possible cause
6. M.H.	69	13. 4	5'1"	Retrosternal and abdominal pain p.c. and on exertion.	20yrs	Rest	Both Huge hernia	No effect.	10/4/58 cholecyst-ectomy. 19/8/58 - diverticulitis. Postural hyper-tension. Jan.1959 still symptoms
7. A.C.	68	9. 3	5'3"	Epigastric pain spreading over chest in morn-ings.	10yrs	Powders to in-duce vomiting	Large mixed hernia No reflux	No effect	

TABLE 111 FEMALE PATIENTS

CASE	AGE	WEIGHT St. Lb	HEIGHT	TYPE OF SYMPTOM	DUR-ATION	RELIEVED BY	HERNIA AND REFLUX	EFFECT OF PROSTIGMIN	REMARKS FOLLOW UP
8. U.G.	67	8. 9	5'2"	Retrosternal fulness and burning p.c, on stooping and on exertion.	10yrs	Pressing on sternum and belching	Both	Hernia unchanged. No reflux	Has a cervical disc with posterior root pressure.
9. I.K.	66	11. 7	5'3"	Epigastric pain	1yr	Sodium bicarbonate.	Hernia No reflux	No effect.	Symptoms improved temporarily after cholecystectomy Recurred.
10. M.J.	66	9.13	5'3"	Epigastric pain on stooping.	10mths	Rennie's tablets.	Both	Hernia unchanged. No reflux	
11. J.S.	65	9.12	5'3"	Retrosternal pain p.c., on exertion, lying and stooping.	2yrs	Sodium bicarbonate.	Both	Hernia unchanged. No reflux	Patient fainted after physostigmin injection.
12. S.H.	65	11.	5'4"	Severe retrosternal pain p.c. and on stooping and lying	15yrs	Powders from doctor	Both	No effect	

TABLE 111 FEMALE PATIENTS

CASE	AGE	WEIGHT St.Lb.	HEIGHT	TYPE OF SYMPTOM	DUR-ATION	RELIEVED BY	HERNIA AND REFLUX	EFFECT OF PROSTIGMIN	REMARKS FOLLOW UP
13. G.J.	65	9	5'2"	Retrosternal pain radiating down right arm	10yrs	Prodexin and vomiting	Both	No effect	
14. M.G.	64	13. 7	5'5"	Retrosternal lump and burning 2hrs.p.c. and on lying and stooping	20yrs	Stretching arms turning body and belching	Both. Easy to elicit	No effect on hernia. Fleeting regurgitation	
15. S.W.	61	12. 7	5'4"	Retrosternal pain while eating and on exertion and stooping.	6mths	Bismuth	Both	No effect	The pain radiates over chest down arms and to face
16. I.McD	61	11. 2	5'2"	Epigastric pain p.c. on exertion on stooping and on lying	10mths	Rennie's tablets (doubtful)	Both	Hernia unchanged. No reflux	Worked as a packer.Heavy lifting. Had to give this up.
17. M.S.	60	9.12	5'2"	Burning epigastric and retrosternal pain p.c. and on lying and on stooping	9mths	De Witt's powders.	Both	No effect	

TABLE 111 FEMALE PATIENTS

CASE	AGE	WEIGHT St.lb.	HEIGHT	TYPE OF SYMPTOM	DUR-ATION	RELIEVED BY	HERNIA AND REFLUX	EFFECT OF PROSTIGMIN	REMARKS FOLLOW UP
18. A.D.	58	10.	5'½"	Tight pain across chest on exertion and on bending.	4yrs	Codein	Both	No effect	
19. L.J.	58	9.13	5'1"	Wind & fluttering in epigastrium ½hr p.c. and on stooping. Sometimes on exertion	11yrs	Vomiting	Both	No effect	
20. C.W.	56	8. 3	5'1"	Retrosternal pain and feeling of food lying there p.c. and on exertion and on lying and stooping.	1½yrs	Vomiting	Hernia No reflux	No effect	Dates symptoms from husband's death. Is now a housekeeper
21. A.C.	53	12.9	5'4"	Food sticking retrosternally and epigastric pain aggravated by exertion. Tachycardia.	2yrs	Vomiting	Hernia No reflux	No effect	

TABLE 111 (continued) MALE PATIENTS

CASE	AGE	WEIGHT St. Lb	HEIGHT	TYPE OF SYMPTOM	DUR-ATION	RELIEVED BY	HERNIA AND REFLUX	EFFECT OF PROSTIGMIN	REMARKS FOLLOW UP
22. P.D.	74	8 8	5' 5½"	Food sticking retro-sternally p.c.	7mths	Vomiting	Both 25/9/58	Hernia unchanged. No reflux.	This patient thought clinically to have oesophagitis. Put on treatment for this. Symptoms still present.
23. G.L.	66	9 0	5' 7"	Retro-sternal pain p.c. and on exertion, stooping, and lying.	8yrs	Milk of Magnesia. Vomiting	Hernia No reflux 2/10/58	No effect	Has duodenal ulcer Jan. 1959. Feels tired Hb.75%. put on oral iron.
24. J.R.	61	10 4	5' 2"	Constrict-ing pain across chest p.c. and on exertion	8mths	Stout "breaks it"	Hernia No reflux 6/10/58	No effect	Pain occurs even when shaving in morning. In spite of angina of effort E.C.G.negative, Had left parotid calculus removed Aug.1958. Very small sliding hernia .
25. AMcL	60	12 7	6' 0"	Dull upper abdominal pain 3 hrs p.c. and on exertion	2yrs	Vomiting	Both 2/10/58	No effect.	Has old fibrosis and retraction of left upper lobe. Symptoms Jan.1959. I.S.Q.

TABLE 111 (Continued) MALE PATIENTS

CASE	AGE	WEIGHT St.Lb.	HEIGHT	TYPE OF SYMPTOM	DUR-ATION	RELIEVED BY	HERNIA AND REFLUX	EFFECT OF PROSTIGMIN	REMARKS FOLLOW UP
26. J.S.	56	11 0	5'11½"	Retro-sternal pain p.c. and on exertion stooping and lying	2yrs	Lying down	No hernia No reflux 15/9/58	No prostigmin given.	Long history of bronchitis. Has emphysema and pain on moderate exertion. But pain still present on stooping and lying. Put on propantheline 15mg. t.i.d.
27. AMcD	55	8 8	5'4"	Vomiting and heart-burn p.c. and on exertion	3yrs	Powders	Hernia No reflux 10/10/58	No effect	Jan.1959. Symptoms ISQ.
28. G.S.	54	12 5	5'10½"	Epigastric pain p.c. and on stooping and lying	3yrs	Standing or sitting up	Hernia No Reflux 12/9/58	No effect.	This patient has a typical sliding hernia and radiological evidence of peptic oesophagitis.
29. J.H.	47	10 7	5' 0½"	Epigastric pain p.c. on exertion and on lying and stooping	5mth	Glyceril Trinitrite	No hernia No reflux 10/10/58	No Prostigmin given	Jan.1959. ISQ.
30. C.F.	39	13 0	5' 10½"	Epigastric pain and heartburn p.c. and on stooping and lying	10yrs	Milk of Magnesia	Both 2/10/58	Hernia and reflux remained but reflux less	Had sinusitis 1956. Cleared. Jan.1959 Symptoms unchanged.

Investigation by Cine Radiography.

I examined fourteen patients by means of the image amplifier with a 35.mm. cine camera.

The method I adopted with each patient was similar. I gave them barium to drink and exposed a short burst of film - six seconds - recording the fluid barium entering the stomach.

I then tipped the patient horizontally or head down and recorded a further six seconds with the barium entering the hernial sac in this position.

Finally, after observing regurgitation on the screen, I recorded a further six seconds while the patient was regurgitating.

The screening current I used was .75.ma. at 65.K.V. using a filter of .5.mm. of copper + 1.mm. of aluminium, in addition to the inherent filtration of the tube of 2.mm. Al. equivalent.

The cine exposure current and voltage were 10.ma. at 85.K.V. Where the tissues were thick and dense, as they were in these stout elderly patients, I found that these factors were essential. The screening factors of ¾.m.a. at 65.K.V. gave a dose received on the patient's skin, over a 5" diameter circle, of .6r per minute.

The radiographic factors of 10.ma.. at 85.K.V. gave a dose rate on the skin of 38r. per minute. The patients each received therefore a total dose of about 15 or 16 röntgens on a small area of skin.

The actual cine radiography exposure was limited to a six second burst by the limiting factors of the capacity of the Xray tube. Anything over six seconds at 10.ma. exceeded the maximum rating of the tube, thereby causing over-heating and the danger of melting the anode. Therefore the manufactures incorporated an automatic time device

which cut off the power at the end of six seconds.

In practise this proved to be a decided drawback since the exposure might stop at a critical moment of reflux.

One other extremely irritating factor in the purely mechanical design of our particular image amplifier and cine recorder was, and still is, a time lag of five seconds between changing from the screening current to radiography in order to allow time for the filament current to exert its influence in giving the extra heat to the filament to permit radiography. This meant that in practise one had to focus on the diaphragm, ask the patient to swallow, judge the time and then hope that the six second exposure would cover all one wished to record.

The judging of reflux was even more difficult since one had to start exposing after some manoeuvre which one knew from experience would produce reflux.

In practise, to catch reflux, I had often to make more than one six second exposure - hence the figure of about 15 röntgens per patient.

Each six second exposure produced about five feet of film and on projecting this one had about five seconds viewing time of that particular phase. I therefore duplicated, and on occasion triplicated each strip in order to achieve a longer viewing time. In addition I had to reduce the 35mm. size film to 16.mm. size in order to project it on my own and on the hospital projector.

Even so the results were disappointing from the clinical viewpoint. Technically I recorded what I wanted, but I obtained no more information from this method than from conventional screening. I had hoped that the procedure might throw some light on whether there

is or is not a sphincteric action about the oesophago gastric junction. From radiographic screening, like Shanks and others, I think there is, but cine radiography at present does not add to one's knowledge.

The apparatus as designed at present is too cumbersome and has too small an effective field, five inch diameter, to locate what one wants quickly. Furthermore the limitation in exposure time necessary to safeguard the tube from over heating and the patient from over exposure to radiation is altogether too great.

It is plain from the figures that there is a real danger of over exposure to radiation from cine radiography. The dose rate actually approaches that given in some forms of superficial therapy in certain dermatological conditions. It is nevertheless well within tolerance limits, and is permissible in the case of older patients, although undesirable. It is not permissible in the younger potentially procreative group even although the amount of actual radiation reaching the gonads from scatter must be extremely small. I doubt if in practise it could actually be measured, although it could be estimated.

Vagotomy and Gastroenterostomy.

One other aspect of the syndrome that I decided to investigate was the effect of vagotomy on regurgitation. If, I thought, vagal stimulation has the effect of effectively preventing reflux of gastric contents into the oesophagus (Botha, 1958) then complete elimination of the effects of the vagus nerve ought to promote reflux. With the co-operation of Mr. A. P. Forrest of the Department of Surgery who supplied me with the names, the operative findings and type of operation of fifty patients. I asked these fifty patients to report to the Xray department. Forty-one of them reported, and these patients I examined. The examination was the routine one with fluid barium to drink and examination in the Trendelenburg position. Not one showed reflux.

There is, however, a fallacy in the argument. The vagotomy, as carried out at the time of the operation, consists of severing the fibres of the vagus nerve at the level of the hiatus, and for two inches above this. It may be therefore that the ganglion cells responsible for contraction are innervated by fibres from higher up, which have not been resected at the time of the operation.

Even if the conclusion that vagotomy does not dispose to reflux is invalid the test series brought out one other point, which is this. It is generally assumed that hiatus hernia occurs through a wide lax hiatus, and the operation of vagotomy should produce this requisite. The surgeon stretches the hiatus as much as he possibly can in order to identify as many fibres of the vagus nerve as possible. I am assured that the effect is the same as if as much stretching and damage to the hiatus as possible had been done. The phreno oesophageal ligament is, also, considerably

stretched and damaged.

One would think, therefore, that if any one thing were calculated to produce a hiatus hernia this operation would. The fact remains, nevertheless, that not one of the patients showed a hernia.

The conclusions to be drawn from this are quite simple:-

(1.) Vagotomy as performed at this operation does not affect reflux of gastric contents into the stomach.

(2.) The degree of trauma to the oesophageal hiatus at the operation for vagotomy does not dispose to hiatus hernia.

These conclusions are surprising when one considers the various theories which are put forward as to the etiology of the reflux and hernia. Taking reflux first there is one big school of thought which considers that there is a sphincteric action in the oesophagus at the level of its passage through the decussated fibres of the right crus of the diaphragm.

Atkinson and his colleagues Edwards, Honour and Rowlands in 1957 carried out a valuable piece of research designed to prove that there is a sphincter at the lower end of the oesophagus. Briefly they carried out a series of tests on fifteen normal control subjects, and on eighteen patients who had oesophageal reflux with or without hernia.

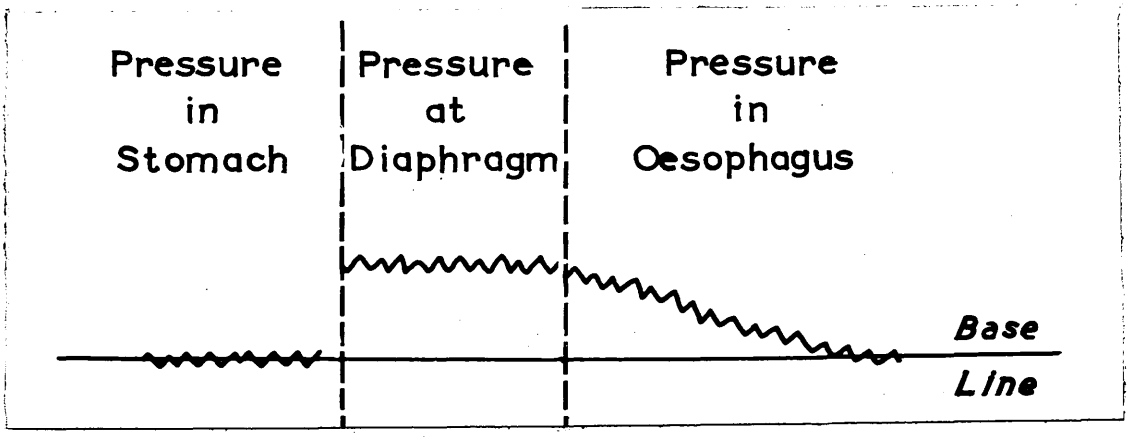
The method used in both series was the same. They introduced three radio opaque air filled polythene tubes into the stomach. Each tube projecting from the mouth was attached to a metal capsule optical manometer which transmitted the pressure recordings to photographic paper. The recording end of the tube, in the stomach, was open ended and consisted of radio opaque rubber 4.cm. long and 2mm. internal diameter.

The placing of the tubes in the stomach and the subsequent manoeuvres were controlled by radiographic screening. Pressures were recorded simultaneously from the three tubes whose tips in the stomach were placed 4.cms. or 5.cms. apart. The tubes were then withdrawn in 1.cm. steps until the distal tube was 3.cms. above the oesophago gastric junction. Pressure recordings were made for fifteen minutes at each cm. step during the withdrawal while the patient was breathing quietly.

This procedure was repeated at least twice and then pressures were recorded in the same way, but with the patient swallowing dry, or with a sip of water. Initiation of swallowing was recorded by changes in pressure in two small tambourines placed over the thyroid cartilage.

Finally, the patient swallowed barium to see whether a hernia was present or not during the test.

In the fifteen control patients they found the pressure recordings followed a pattern.



There was a zone of high pressure at the diaphragm and at the lower end of the oesophagus, gradually dropping back in the oesophagus to the same level as in the stomach. The pressure gradient varied from 6.cms. to 15.cms. of water, with an average of 9.cms. The average pressure in an older

group was 11.cms.

When these patients swallowed, the pressure in the segment from about 1.cm. below the diaphragm to 2.cms. to 3.cms above fell to the general oesophageal level.

In the hiatus hernia group of patients the pressure recorded at the diaphragm and in the hernial sac (Checked by the size of the sac and recorded respiratory waves) was the same as in the stomach. The authors were actually able to predict two cases of hernia by this means and to demonstrate one which had been missed radiologically.

The next stage in the test was to withdraw the recording tips from the stomach to the sac, and then from the sac to the oesophagus, again recording the pressures in 1.cm. steps.

They found that a zone of high pressure occurred at the gastro oesophageal junction, well above the diaphragm, in thirteen of the eighteen patients. This zone of high pressure fell to normal on swallowing.

In the remaining five patients there was no zone of high pressure. The authors found that patients with a pressure zone of 3.cms. or less showed reflux.

The authors also found that in five patients with large fixed hernias extending to 5cms. to 6cms. above the diaphragm the high pressure barrier zone, relaxing on swallowing, occurred here. In two cases there was no high pressure zone at the diaphragm level, but in three there was some rise in pressure in the stomach where it passed through the hiatus. This suggests that the right crus plays some part in the dynamic mechanism in the normal case.

The authors end their excellent paper by citing the case of a patient who had a Heller's operation for cardio-spasm. They found no zone of high pressure in this patient, who subsequently developed peptic oesophagitis.

The point made by the authors that there is a sphincter at the oesophago gastric junction seems to me to be established beyond any reasonable doubt.

Creamer and Pierce in 1957 designed an experiment by pressure recordings somewhat similar to that of Atkinson and his colleagues to demonstrate whether liquid barium was held up at the gastro oesophageal junction by a pressure barrier. They concluded that it was, for about twelve seconds, after which the pressure relaxed, and the barium passed through. They found that the pressure dropped before a peristaltic wave reached the junction. They also found that there was a difference in the behaviour pattern of the pressure barrier when a patient drank barium, as opposed to when the patient swallowed a bolus already in the mouth. When the patient swallowed a bolus the pressure at the junction rose until the bolus reached it, when it fell by some 6.cms. to 10.cms. of water. When the patient drank barium the diaphragm descended as the glass was raised to the mouth, and began to rise at the moment of degludition; so that by the time the head of the barium reached the junction, the diaphragm was about half way between full inspiration and full expiration. At this moment the "barrier" was wide open with a low pressure and barium streamed through. They concluded that there is a sphincter, independent of the diaphragm and that swallowing is a simpler process than drinking.

It is probable, however, that the explanation is more complex, and is related to the view put forward by Bulbeing, that the ganglia respond to local and hormonal stimuli.

The view that there is a pinch cock action by the right crus can hardly apply in the forty-one patients that I examined in my gastroenterostomy and vagotomy series since

the surgeon stretches the hiatus and generally does so much damage that this action cannot reasonably be expected to be effective after the operation.

The second point, that no hernia is produced, is perhaps more difficult to explain. The generally accepted view is that laxity of the hiatus, with or without weakness of the right crus and a poor phreno oesophageal ligament all favour herniation of the stomach. Here we have the surgeon actually creating these ideal conditions.

When one considers this in conjuction with my findings in those patients who had had a repair operation of the hiatus carried out with sutures designed to strengthen the right crus, it would appear that we shall have to reconsider the underlying cause of hiatus hernia. It seems to me that the stucture of the hiatus itself must play an important part in preventing herniation of the stomach, but there must be some other important factor at least equal to this. I suggest that the problem is complex and that the other important factors are the inherent tone of the stomach and the tone of the structures which, within limits, anchor the stomach. These are the folds of peritoneum which go to form the greater omentum, the lesser omentum (and that part of it known as the hepato gastric ligament) the gastro splenic and the gastro phrenic ligaments. If this view is correct then we must assume that there is a general laxity of the structures in the upper abdomen in addition to a locally poor lax hiatus. This would explain why a local repair is often ineffective and why, when the hiatus is disrupted, hernia need not occur.

Incidentally, I found that this operation of vagotomy and gastroenterostomy for duodenal ulcer gave excellent results. Only four patients out of forty-one had any residual symptoms

and all the others spoke enthusiastically of the operation. A typical answer to my routine question asking about their progress might be "I have never been better" "This has opened a new life for me" "Smashing."

Testimonials like the above were entirely unsolicited.

Surgery.

I decided to examine a group of cases who had had a major surgical operation designed to overcome the symptoms associated with hiatus hernia and regurgitation in the Western Infirmary over the past five years. That is from and including the year 1954 until the present year - March, 1958. I was only able to trace thirteen patients on whom operation had been performed. I include one extra case, who was operated on in London, and who reported to the Western Infirmary with dyspeptic symptoms.

The small number of cases suggests that surgery is not regarded favourably as a method of treatment for these unfortunate patients, when one considers the number who have clinical symptoms of hiatus hernia. The main symptoms are heartburn or epigastric pain after food, and on stooping and lying down and it is beyond doubt that many of these patients prove, on radiological examination, to have a hiatus hernia and regurgitation. Edmund's V. (1957) in a clinical study of two hundred patients states that the policy at the Central Middlesex hospital is to treat these patients medically and the few failures are dealt with surgically. Although not a prescribed policy in the Western Infirmary it seems that we have been pursuing much the same course. The medical treatment is designed to reduce the weight, correct any anaemia from bleeding if such be present and overcome the effects of gastric acidity by antacids, and sleeping propped up with extra pillows. Table IV shows the cases, the main symptoms, the type of operation, and the post operative Xray appearance.

It will be seen that approach by the abdominal route, reduction of the hernia and repair and strengthening of the hiatus by posterior sutures aimed at strengthening the crura is the favourite method of treatment. The crura were often

found to be weak, lax or imperfectly developed. It is interesting to note that it is precisely this operation which gives the worst radiological and clinical results.

The numbers I have available for analysis are too few to permit any dogmatic statements being made, and perhaps even too few on which to base any reasoned opinion. However they are all the cases I have, and they may perhaps serve as a basis for discussion. The majority of the patients still had a hernia and reflux postoperatively and a still greater proportion continued to have reflux. Almost all these patients, however, subjectively stated that they felt better, and that the operation had been worth while. Psychology no doubt enters into this. A patient who has made up her mind to have a major surgical operation and who has it, with all that this implies, is going to hope and wish that the operation will be a success. For this reason she will put up with discomfort for some time before she is prepared to admit, even to herself, that the operation has been a failure.

The surgeon who advises the operation and carries it out also probably subconsciously emphasises in his own mind the favourable points in the patient's replies to his questions at the follow up clinic. In fact he and the patient tend to re-assure each other. I think that an independent person, such as the radiologist, in what is more like a casual conversation on symptomatology may obtain a truer picture.

As an example M.Mack,, Number 5 in the table, aged fifty-eight had retro sternal pain and loss of weight for one year prior to her operation. Latterly she was never free from pain. She had her operation, a reduction and repair on 15th July, 1954.

When I examined her on 30th October, 1958 she exhibited a hiatus hernia and reflux, and said that she was a great

deal better. In conversation however, and without any undue amount of prompting, she divulged that she suffered from acid; that this was worse when she stooped and that in fact she usually had sour mouthfuls and a feeling of compression going on to pain on stooping.

She had to give up her work in a grocer's shop because of this chain of symptoms. Now she takes half a glass of milk if she experiences the pain, and that puts it right. If she does not eat she says she feels "fine". And this was the patient who at first said that the operation had been successful!

On the other hand M.D., Number 1 in the table, had improved. She experienced no discomfort, slept well with one pillow, took no alkalis and did her housework, stooping and polishing, with ease. Radiologically she had reflux but no hernia. It must be noted however that she was Xrayed twice pre-operatively, on the first occasion showing no hernia, and on the second occasion a small fleeting one. At the operation the surgeon "found it difficult to be absolutely sure that there was a hernia, but it seemed likely, to the extent of about two inches".

Having regard to the limited number of operated cases, and the variety of operations performed, it is difficult to draw any conclusions but on analysing the table we see that six patients had a straightforward repair and of these three continued to have clinical symptoms and radiologically a hernia and reflux.

One continued to have symptoms and reflux radiologically but no hernia, and one remained well clinically and had no hernia and no reflux.

One was re-admitted to hospital six months after the operation with vomiting and dysphagia and at that time she was found to have a carcinoma of the breast.

Three patients had excision of the stricture and repair. Of these one continued to have symptoms and radiologically a stricture, hernia and reflux.

One continued to have symptoms, a stricture, hernia, reflux and ulcer.

One was symptomatically well. She had put on one and a half stones in weight and was able to eat anything. She had no hernia or stricture but continued to have reflux.

Two patients had gastrectomies. Of these one gave a very good clinical result and had slight reflux but no hernia. One gave a very bad clinical result, deteriorated and died.

Two patients had vagotomies and gastroenterostomies. One of these had very severe postoperative symptoms and continued to have free reflux but no hernia. The bther continued to have pain, more diffusely spread over the abdomen and spread down to the pelvis, not at all typical of the usual hiatus hernia type. Nevertheless he deteriorated and stated that he was unable to sleep at night. Radiologically he had a small hernia but no reflux.

One patient operated on in London had a repair. She continued to have symptoms but these were complicated by anginal pain with electrocardiographic changes and a multiplicity of complaints. She was regarded clinically as a confirmed neurotic, and radiologically she continued to have reflux postoperatively. On one occasion, four years after the operation, she also showed a sliding hernia but on a second occasion, five years after, no hernia.

TABLE IV.

NAME	PRE-OPERATIVE FINDINGS	TYPE OF OPERATION	POST OPERATIVE FINDINGS
M.D. (F) 1. 45	Hysterectomy 1947. 3yrs. story of heart-burn. 18mths. vomiting. 2mths vomiting 10mins. after every meal. Haematemesis. Sliding hernia and reflux.	26/8/58. Difficult to be certain that there was a hernia. Cardia stitched to oesophagus to reform acute angle. Posterior sutures.	18/10/58. Free reflux. No hernia. Note that at first Xray in 1957 no hernia and at second in May 1958 only a small fleeting one. Symptomatically good. Works in house. Stoops, polishing floors. Takes no alkalis. 25/1/59. Still symptomatically well.
A.H. (F) 2. 54	Sliding hernia and reflux. 15yrs.story.	Abdominal approach. Posterior repair 29th June, 1956.	March,1957 dysphagia and vomiting. Not Xrayed. Oct.1958 carcinoma breast. 7/10/58 mastectomy.
I.Y. (F) 3. 68	Symptoms began in 1953. Hernia and reflux;stricture. Treated with bougies but symptoms remained.	Histology inflamed and ulcer. Wt.6st. 7/8/58. thoracotomy. Stricture identified and resected. Anastomosis stomach and oesophagus.	14/12/58 stricture, hernia, reflux, radiological ulcer. Wt.5st.8½lb. 21/1/59 wt. 5st.8lb. She is still having bouginage for dysphagia.
E.B. (F) 4. 54	Incarcerated hernia and reflux. Gastric ulcer. Vomiting 1yr.	15/3/56 wt. 11st.8lb. Billroth gastrectomy for G.U. and posterior repair	11/11/58 wt. 9st.10lb. No hernia, very slight reflux. On the whole well, but has occasional bouts of vomiting. Jan.1959 condition unchanged.
M.Mack (F) 5. 58	Hernia and reflux 1yr. epigastric pain. No response to treatment.	15/7/54. Abdominal approach. Crura sutured.	Hernia and reflux. Still has pain and had to give up work. Carpal tunnel syndrome Dec. 1955. Psoariasis eare 30/10/58. Jan. 1959 still symptoms.

TABLE IV.

NAME	PRE-OPERATIVE FINDINGS	TYPE OF OPERATION	POST OPERATIVE FINDINGS
M.O. (F)6.57	Hernia (small and intermittent) 30yrs. Reflux 17yrs. Hiatus hernia, vomiting, no blood. Pain stricture.	27/1/55. Abdominal approach. Very lax hiatus. Six sutures.	March, 1955 wt. 9st.11lb. 2/11/56 wt.10st.4lb. 10/1/57. Strict. Xray and oesophagoscopy Bougies. Reflux, no hernia. Still has pain and acid for which she takes alkalis. Sleeps propped up. 11/12/58 a good deal better; vomits a large meal.
M.S. (F)7.62	Stricture and hernia.	31/7/58 thoracotomy. Stricture resected. Vagotomy Wt. 10st. 13lb. 12/9/58 - abdominal approach for recurrence. Hernia noted. Gastroenterostomy. Hiatus left.	3/10/58 oesophagoscopy. Still stricture, not Xrayed, still had hernia. 21/11/58 wt. 9st.4lb. 9/1/59. ISQ.
A.R. (M)8.35	Hernia, reflux, ulcer 3yrs. Dyspepsia. Vomiting occasionally blood streaked.	12/3/57 thoracotomy. Vagotomy and gastro-enterostomy.	23/10/58 free reflux but no hernia. Symptoms very bad. Pain in back, off work, chest pains. Mixed salivary tumour excised 30/7/58. Has old juvenile osteo-chondritis.
B.R. (M)9.69	Hernia, reflux and duodenal ulcer. 6mths. history.	Vagotomy, gastro-enterostomy and repair of crura. 24/9/58.Wt.8st.8lb.	14/12/58. Hernia and reflux, but much smaller, and a good "pinch cock". Symptomatically poor. Wt. 8st.11b.

TABLE IV.

NAME	PRE-OPERATIVE FINDINGS	TYPE OF OPERATION	POST OPERATIVE FINDINGS
A.M. (F)10. 61	Small sliding hernia and reflux.6/11/57 5yrs. story. Pain and vomiting. No haemopt. or melaena. 24hr. vomit residue.	Abdominal approach. Crura sutured. 6/11/57 no ulcers.	30/10/58 hernia and reflux. Takes Morelands. Pain. Sleeps propped up. 8/12/58 still has pain when she stoops but does not now stoop so often.
I.G. (F)11. 69	Paraoesophageal hernia 3mths loss of wt.(3st.) and loss of appetite.	Thoracotomy. Abdominal approach. Hernial sac excised. Posterior repair. Gastrotomy to anchor stomach. 29/4/58 wt.9st.11lb.	Fixed hernia. Not paraoesophageal. Free reflux. Symptomatically greatly improved. 15/12/58 wt. 9st. 8lb.
M.McD (F)12. 66	Hernia, stricture, ulcer. Dysphagia 10yrs.	7/4/58 wt.10st. Thoracotomy Stricture could not be excised. Stomach brought up and anastomosed to oesophagus above stricture Partial gastrectomy and gastroenterostomy.	Recurrence of stricture at anastomosis. Wt.7st.7lb. Had to have repeated dilatations post operatively deteriorated and died in 8mths.
M.McC (F)13. 54	Incarcerated paraoesophageal hernia. Many years history. Worse past year. Much vomiting and haematemesis. Wt. 12st. Has osteoarthritis.	17/5/56. Abdominal approach. Crura sutured.	Oct.1958 free reflux and hernia. Still has "bile" and regurgitation and sleeps propped up. Feels better. Had post operative emboli. Jan.1959. Still has symptoms.
M.K. (F)14. 54	Sliding hernia. Retrosternal pain 3yrs.	Dec. 1953 operation Middlesex Hospital. Posterior repair.	Oct.1957 reflux and hernia. Nov.1958 reflux. Continued to have symptoms, angina E.C.G. changes and neurosis.

Summary.

1. Hiatus hernia is a clinical entity, occurring mainly in females in the proportion of about two to one, and mainly in the middle aged or elderly.
2. There are a great variety of symptoms, likely to be confused with upper abdominal conditions and ischaemic heart disease.
3. Radiology will differentiate the former, and radiology and electrocardiography the latter. It is emphasised that in the latter, confusion will rarely arise if a careful case history is taken.
4. Because of the complex neuro muscular mechanism, drugs cannot be relied upon to prevent hernia or, more important, its common complication reflux.
5. Cine radiology at present gives no added information as to the mechanism of hiatus hernia and reflux.
6. The results of surgical repair are disappointing.
7. The operation of vagotomy does not dispose to hiatus hernia or reflux, and, incidentally, in my series proved to be an excellent operation for duodenal ulcer.
8. I suggest, in view of the last two findings that upper abdominal tone plays a part, at least equal to a tight hiatus and strong crura, in preventing hernia and reflux.

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