THE SURGICAL TREATMENT OF DIAPHRAGMATIC OESOPHAGEAL HIATUS HERNIA

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ALL THE REPORTED series of cases of hiatus hernia treated surgically which I have found have shown a failure rate[1], and it is with the failures that I am principally concerned here. I propose to review the history of surgical endeavour and surgical research in this field, and I hope to show how these, together with the limitations imposed by the diagnostic procedures available to us, have led to present techniques with which surgical failure is far too common. I shall then review the results in a series of my own patients, certain aspects of which illustrate the points I wish to make and will, I hope, lead to more success in the future.

HISTORICAL REVIEW

The classical paper of Harrington in 1940[2] is a convenient point to commence the historical review—in fact I can find no earlier reference. Harrington dealt with the aetiology of all types of diaphragmatic hernia and reported a series of 250 cases operated upon between 1926 and 1939, most of which were of hiatus hernia. He considered the aetiology to be a congenital weakness of the hiatus with a superadded congenital or acquired weakness of the 'diaphragmatico-oesophageal membrane', his term for the phreno-oesophageal ligament, as we describe it today. He recognized that there was normally a considerable range of movement of the oesophago-gastric junction relative to the hiatus. He recognized the para-oesophageal hernia in which a pouch of stomach herniates alongside an oesophago-gastric junction normally placed below the diaphragm (Fig. 1*a*) and its more common modification in which the oesophago-gastric junction lies above the hiatus, but not at the apex of the herniated pouch (Fig. 2). Two-thirds or more of his cases were of the para-oesophageal type. What we now call sliding hernia he described as the pulsion type (Fig. 1*b*); he does not seem to have operated upon many of these, but recognized the complications of reflux and oesophagitis and of stricture and oesophageal shortening. He states that 'the para-oesophageal hernias are in reality a type of sliding hernia', as indeed most of them are, but this was the beginning

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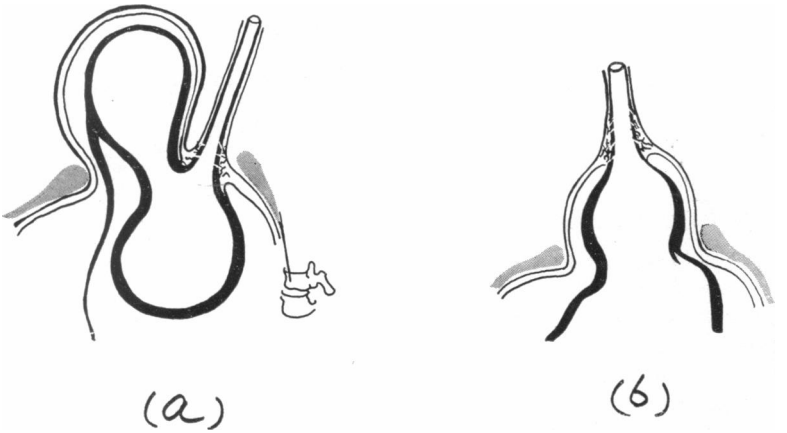

Fig. 1 (after Allison). The classical para-oesophageal hernia (*a*) and sliding hernia (*b*).

of a confusion of thought in this matter of nomenclature which has dogged us to this day.

Harrington's surgical objective was to reduce the hernia below the diaphragm, and it is clear from the diagrams which accompany his paper that he paid no attention to the function of the hiatus. It was probably unnecessary in his cases to do so: the predominant symptoms which he described were those of a large or gross hernia with pressure effects in the mediastinum, and reduction, even if incomplete, would relieve them. However, his work must be regarded as a surgical tour de force, having regard to the problems of anaesthesia and postoperative care at that time, and set the tone for many years thereafter.

The next historical milestone is the paper by Allison in 1951[3]. In this he draws attention to the predominant type of hiatus hernia, the sliding type, in which there is incompetence at the oesophago-gastric junction and of which reflux and oesophagitis and their complications are the main features. Para-oesophageal hernia, with competence of

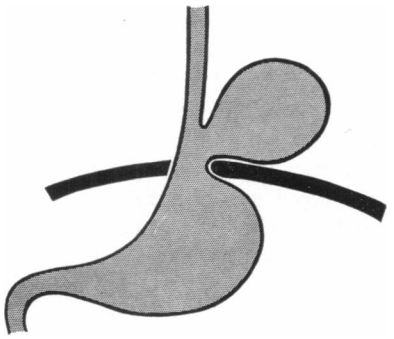

Fig. 2. The common type of para-oesophageal hernia in which the oesophago-gastric junction is displaced above the diaphragm.

the closing mechanism, is contrasted sharply with it. Allison draws attention to the sequelae of oesophagitis—ulceration, stricture, and shortening—but stresses that oesophagitis is consequent upon the derangement of the cardia and its loss of function. At that time the intrinsic sphincter at the oesophago-gastric junction was not known to exist, although it was realized that competence of the closing mechanism could be maintained even when it was displaced from its normal situation below the diaphragm—as in many cases of so-called para-oesophageal hernia. Allison described his surgical technique for repairing hiatus hernia, with much attention to the restoration of normal anatomy, the clear implication being that restoration of normal anatomy would restore function to the closing mechanism. This paper is indeed a true landmark, beautifully describing the clinical syndrome of the sliding hernia and incompetent cardia, but reiterating the fundamental misconception of Harrington that hiatus hernia is a mechanical condition remediable by restoration of normal anatomy. True though this may be of gross hernia producing mediastinal pressure effects, I hope to show that it is untrue of some cases of sliding hernia with incompetent cardia, and I hope to show also that it is from this group that failures of surgical treatment arise.

During the decade 1950–60 much work was done on the detailed anatomy of the hiatus and its innervation by Collis and his colleagues[5, 6], by Peters[4], and by many others with the object of improving the surgical technique of repair. Much of this work was done against a background of suspicion that an intrinsic lower oesophageal sphincter existed, but with the knowledge that no such anatomical sphincter could be demonstrated. It required the work of Code and his colleagues[7-11], Atkinson[12], and Inglefinger[13, 14] to show by manometric methods that such a sphincter does exist and to demonstrate its function in relation to cricopharyngeus function and oesophageal motility in general. These techniques, and that of measuring mucosal potential to identify the oesophago-gastric junction[15], have much value in assisting the diagnosis of difficult disorders of the oesophagus.

The decade from 1960 to 1970 has seen a move away from anatomical repair of the Allison type. Belsey[16, 17] has progressed to an operation in which the abdominal portion of the oesophagus is invaginated into the stomach to prevent reflux. Nissen[18] has described a similar technique combined with fixation of the stomach to the abdominal wall. Borema[19] has described a gastropexy similar to that of Nissen. During this period there has also been an increasing acceptance of the fact that in many cases of hernia with oesophagitis oesophageal shortening and stenosis prevent reduction of the hernia. The problem is one of reconstruction of the lower oesophagus. Collis[20] has described gastroplasty. Merendino[21] described jejunal interposition, and many surgeons, including myself, have used a similar technique with left colon instead of jejunum.

At the other end of the scale the existence of incompetence of the cardia without hernia has been recognized as not infrequent and causing symptoms of sufficient severity to warrant surgical treatment[16].

During these last years there has been an increasing appreciation of the not infrequent association of hiatus hernia with incompetence of the cardia and with duodenal disease, either frank ulceration or the vague entity of duodenitis. This has led to attempts to correlate the level of gastric acidity and the extent of reflux with the degree of oesophagitis, but with little or no success. Once again we are indebted to Collis and his colleagues[27]; and operations in which vagotomy and pyloroplasty are combined with hiatal hernia repair are increasing in frequency.

Recently two papers have appeared which to my mind point in a new and hopeful direction. Heitman[23] has demonstrated three types of sliding hiatus hernia which he classifies as those with competent sphincter, those with incompetent sphincter, and those with hypertonic sphincter, the three types being differentiated by oesophageal pressure measurements; and Castell and Harris[24] have shown variations of sphincter pressure following the instillation of acid, alkali, and betazole (ametazole) into the stomach, suggesting a control of the sphincter by endogenous gastrin secretion.

AETIOLOGY OF HIATUS HERNIA

I have tried to describe 40 to 50 years of endeavour in a brief space. To compress this even further, I think it is true to say that for the first 30 years or more there was a concentration upon the oesophago-gastric junction and the hiatus to the virtual exclusion of distant factors which might provoke the condition. Lip service was paid to gall-bladder disease as a concomitant condition, and cholecystectomy was carried out at the time of hernia repair if gall stones were present. But this removal of the evidence of past disease did nothing to influence the surgical results. The original dictum that hiatus hernia was a mechanical condition held the field—and still largely holds it, in spite of the growing evidence in the past 10 years from pressure manometry, from motility studies, and from the study of gastric secretions other than acid that an incompetent cardia is often a functional disorder rather than an anatomical displacement, and it is my thesis that it is from this group, in which the disorder is functional rather than anatomical, that the failures arise.

Mechanical factors

Allison in one of his papers draws a simile between the oesophagus and its angle of entry to the stomach and the hiatus, and the angle of the ano-rectal junction and the levator ani. One would be inclined to deride the efforts of a surgeon endeavouring to treat diarrhoea by

increasing this angle and strengthening the levator ani; but the surgeon would no doubt counter by pointing to some good results—achieved no doubt in cases of incontinence due to local weakness or paralysis. But if he was unable to distinguish diarrhoea from incontinence—unable to distinguish between disorder of function and disorder of structure and of anatomy—he would be dogged by persistent failure. In the case of hiatus hernia it is more difficult to distinguish these two. Some hiatus hernias—probably almost all of the so-called para-oesophageal hernias—are anatomical hernias and nothing more. Some sliding hiatus hernias are probably anatomical, with consequent derangement of the function of the cardia. But some are functional derangements with consequent anatomical change.

Disorders of secretion

If one suspects that there is a disorder of function, one has next to consider what that disorder is and why it has so far escaped notice. There are only two functions of the oesophagus and stomach, so far as our present problem is concerned—secretion and motility. Secretions of mucus from the oesophageal glands have so far been completely ignored. Some factor in the gastric secretion, or even duodenal reflux, has always been incriminated as the cause of oesophagitis, but it is not beyond the bounds of possibility that a disorder of oesophageal mucus production, in quantity or quality, by failing to give adequate protection to the oesophageal epithelium, is responsible for the development of oesophagitis. The investigation of gastric secretions has been almost confined to estimations of acidity, apart from the recent work on gastric hormones to which I have referred. But here, as with oesophageal mucus, there is a vast unexplored field of investigation into the qualities, both chemical and physical, of gastric secretions—especially the mucus, the most prolific but the most ignored—which must await further developments in protein chemistry before any significant advance can be made. But whatever advances may be made in this field, one cannot consider disorders of secretion from either oesophagus, stomach, or duodenum as a cause of incompetence of the cardia and hiatus hernia unless disorders of secretion give rise to disorders of motility; for oesophagitis follows incompetence and never precedes it. It seems more likely that disorders of secretion accompany or follow disorders of motility rather than cause them. Disorders of both secretion and motility may of course share a fundamental cause. Examples of this are common—the young person with duodenal ulcer in association with hiatus hernia, and, more dramatic, incompetence of the cardia and severe oesophagitis rapidly advancing to stricture formation which sometimes accompanies severe vomiting in pregnancy.

Disorders of motility

Obstructed by the lack of technical methods of investigating secretion

in the oesophagus and stomach and reasonably convinced, I hope, that therein does not lie the key to the problem, we turn to disorders of motility, for what is more likely than that a disorder of motility should give rise to a disorder of position or anatomy.

Here we find another enormous gap in our knowledge and in our technical resources. The work of Code and others, to which I have referred, applies only to the circular muscle coat. The present technique of manometry does nothing to measure longitudinal muscle function in the oesophagus, and radiography and cineradiography provide little help. The oesophageal mucosa is so loosely attached to the muscle coats that longitudinal activity could not be effectively observed by the attachment of radio-opaque clips or markers to it. The longitudinal muscle is the predominant structure in the oesophagus, but I have yet to see any comment on disorders of its function other than spasm accompanying oesophagitis[25]. Pressure manometry and cinefluoroscopy leave us in no doubt of the existence of functional disorders of circular muscle —achalasia, diffuse muscular hypertrophy, cricopharyngeus spasm. Disorders of circular muscle function are now reported in association with hiatus hernia. It is almost inconceivable that the function of the circular muscle of an organ can be disordered and that of the longitudinal remain normal, but because we cannot measure longitudinal muscle function we ignore it and its disorders.

Although I can produce no scientific evidence—no measurements —to show that longitudinal muscle disorders exist, if you will grant that there are good and reasonable grounds for supposing that they do exist, then there is no harm in looking for their effects. These will be due to irregular contractions, excessive contractions, persistent excessive contractions, spasm, weakness, or paralysis. All these types of disorder of function are known to occur in the circular muscle, but it is particularly with excessive contraction, either irregular or persistent, that I am concerned in the longitudinal muscle.

At this point I will ask you to dismiss from your minds the accepted classification of hiatus hernia into the sliding, the para-oesophageal, and the mixed types, and to consider them all as hernias of stomach through the hiatus. They may then be divided into those with a competent closing mechanism and those in which it is incompetent. Most, but not all, of the para-oesophageal type have a competent sphincter, even when it is displaced from the hiatus—the 'mixed' type (Fig. 2). Most sliding hernias (Fig. 1*b*) have an incompetent sphincter; in some it is competent and in some its competence is dubious.

Table I shows my own cases classified as para-oesophageal and sliding and Table II shows the same cases classified as described above. The figures in brackets in Table II are those for sliding hernia (above) and para-oesophageal hernia (below), and they are greater than the

corresponding numbers classified as incompetent and competent sphincter. It is not a simple matter of transferring the excess in each case to the dubious group; many cases of sliding hernia had a competent sphincter and many classifiable as para-oesophageal had an incompetent sphincter, and these to some extent cancel each other out.

The identifying feature of the sliding hernia is that the oesophago-gastric junction lies at the apex of the herniated pouch of stomach (Fig. 1*b*). It has always been assumed that the natural recoil of the oesophagus has kept the oesophagogastric junction at the apex of the pouch as the stomach has been pushed up from below. But if this is so, why does it not do so in the 'mixed' para-oesophageal hernia, where the junction has moved up from the hiatus into the mediastinum? There is nothing to prevent the oesophagus 'taking up the slack', so to speak, and draw-

TABLE I

STANDARD CLASSIFICATION OF 327 CASES OF HIATUS HERNIA

Sliding hernia:	246 (75.4%)	
	Males	73 (30%)
	Females	173 (70%)
Para-oesophageal:	76 (23.1%)	
	Males	13 (17%)
	Females	63 (83%)
Children under 2:	5 (1.5%)	

TABLE II

CLASSIFICATION OF 327 CASES OF HIATUS HERNIA ACCORDING TO COMPETENCE OF SPHINCTER

Incompetent sphincter	233 (246)
	71% (75.4%)
Dubious competence	34
	10.4%
Competent sphincter	60 (76)
	18.3% (23.1%)

ing the oesophago-gastric junction to the apex of the stomach. The oesophagus, freed from its attachment to the stomach and its ligaments, can quickly shrink to a half or a third of its length.

Again, in sliding hernia, why should the oesophago-gastric junction be pushed through the hiatus first, when it is not normally nearest to the hiatal orifice but lies at the lower end of 2–3 cm of abdominal oesophagus with the fundus of the stomach lying at a higher level? Why should the junction take up this distorted position at the apex of the herniated stomach when, as Barrett has shown, even when the stomach and oesophagus are removed from the body, the oesophagus maintains its acute angle of entry to stomach?

There is one sure way of producing a classical sliding type of hiatus hernia, and that is to pull on the oesophagus from above, and this is the central point of my whole case, that the common sliding type of hiatus hernia can hardly be produced in any other way than by excessive irregular or sustained contraction of the longitudinal oesophageal

muscle. If you will grant this possibility—or, as I think, strong probability—a great many other factors fall into place. If there is excessive longitudinal muscle activity you would be entitled to think that there would be concomitant excessive circular muscle activity and a hypertonic lower oesophageal sphincter. This may well exist at the outset; Heitman's work[23] shows that variations of sphincter tone do occur in this type of hernia, but the sphincter is a weak one and the longitudinal pull, dragging up the stomach from its normal situation and attachments—and they are fairly strong—will stretch out the length of this sphincter and destroy its function. The sphincter in this position will be exposed to a direct dilating effect at the apex of the funnel-like pouch of stomach and to the full intragastric pressure, unprotected by a compressed abdominal segment of oesophagus or by the usual angle of entry. The exposure of the herniated sphincter to excessive pressure from below is usually considered to be the sole cause of incompetence. The possibility of a traction effect has been neglected.

I said earlier that in some cases sliding hernia is probably due to anatomical weakness and in some to functional disorder. To qualify this, it is not unreasonable to suppose that an abnormal longitudinal muscle pull by the oesophagus will produce incompetence of the sphincter, with or without a hernia, if it is sufficiently strong, even when the diaphragmatic muscle and its oesophageal attachments are normal—the obvious example of this being the spare, taut young person with duodenal ulcer and hypermotility in the stomach. On the other hand, a normal longitudinal oesophageal pull may be sufficient to produce hernia in an obese middle-aged or elderly person with lax and flabby musculature.

Para-oesophageal hernia cannot be attributed to longitudinal muscle pull, for the oesophagus lies lax and of normal length and yet the whole stomach may be herniated into the chest. This type I attribute to a weakness of the phreno-oesophageal ligament, either local or general, and consequent hernia.

When incompetence has developed oesophagitis may or may not follow. If it does, then this accentuates the oesophageal spasm, both circular and longitudinal: in clinical practice dysphagia due to spasm is frequently encountered in the absence of stricture in patients with oesophagitis. So far as longitudinal spasm or excessive contraction is concerned, all those accustomed to operate on hiatus hernia will know of the strength of this and the difficulty to which it gives rise in reducing these hernias at operation. In this connection attention has frequently been drawn to the lack of correlation between the finding of oesophagitis at oesophagoscopy and the finding of a thickened and somewhat shortened oesophagus at operation, and vice versa. The thickened and shortened oesophagus may well be evidence of longitudinal muscle spasm and hypertrophy, and one would not expect this to be necessarily

commensurate with the degree of oesophagitis if longitudinal hyperactivity was a precursor of hernia and oesophagitis an unpredictable concomitant. However that may be, it is common experience that once oesophageal shortening has progressed beyond a certain point, reduction of the hernia is not possible. This stage is often accompanied by oesophageal fibrosis and frequently, but not invariably, stricture formation. When this occurs some more complex surgical procedure is required to deal with the condition—some form of oesophageal reconstruction by a bowel graft if the stomach is reduced below the diaphragm; my series contains a good number of such cases. But in the vast majority of cases the hernia is readily reduced, although in some it can only be reduced after extensive mobilization of the oesophagus—up to the arch of the aorta—and by exerting considerable traction upon it, and very firm fixation may be necessary if reduction is to be maintained—a tribute, as I think, to longitudinal muscle hyperactivity. This necessity for extensive mobilization of the oesophagus to obtain reduction, and for firmer fixation to maintain it, is the basis of the more recent surgical techniques for repair, though these have been developed without any real reference to the longitudinal muscle as the prime cause of the disease.

Some corroboration for the view that these hernias are pulled up rather than pushed up is to be found by observing the end results of oesophageal reconstruction by colon graft (Fig. 3). The colon graft is a lax structure with a tendency to elongate. Certainly it cannot transmit to the stomach any upward pull by the oesophagus. I have always deemed it inadvisable to close the hiatus around the colon graft, for fear of occluding its blood supply and for fear of producing a mild obstruction, for grafts of colon show little or no peristaltic activity. But although the hiatus is left fully open, with the graft lying loosely through it, I have never observed any herniation of stomach —nor for that matter of any other abdominal contents—through this lax hiatus. This is not because the hiatus becomes obliterated by adhesions. Sometimes I have found it necessary to reoperate in these cases many months later, and one then finds the graft quite mobile in the lax hiatus. There is clearly little or no inherent tendency for the stomach to herniate once the traction of the oesophagus has been removed.

TREATMENT

It may not seem to matter whether these hernias are pushed up or pulled up. It would seem to be only necessary so to fix the stomach and lower oesophagus that the hernia cannot recur from either cause. But if the proper function of the cardia as a whole depends upon a degree of mobility of oesophagus and stomach in the hiatus, and we know that a fair degree of mobility does normally exist, then firmer fixation will destroy this mobility and there will not be full restoration of normal

function following operation. This is not infrequently seen as persistent dysphagia, in substitution for reflux, following over-firm fixation operations.

If one believes, as I do, that excessive pull by the longitudinal muscle is a cause of some hiatus hernias, then one must identify those cases in which it is the causative factor and overcome it by some procedure other than the ordinary reduction and simple repair; or accept that however extensive the mobilization, however firm the fixation, there is going to be a high risk of recurrence in these cases.

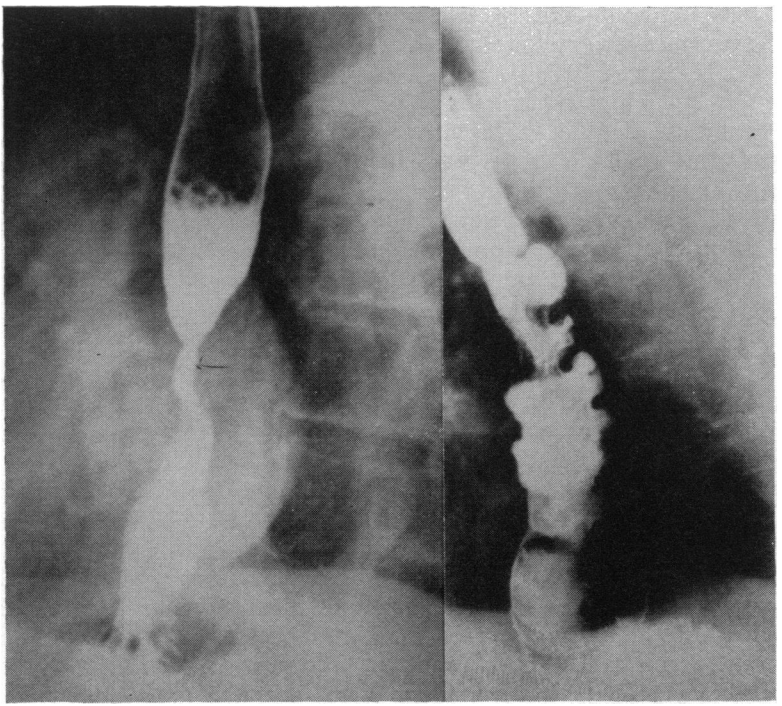

Fig. 3. (*a*) Marked oesophageal shortening with stricture formation (arrowed). (*b*) A similar case treated by excision of the stricture, reduction of stomach below diaphragm, and interposition of a pedicled graft of colon.

In my own series of cases I have taken the view that longitudinal muscle traction is the causative factor in many, but that it is a powerful factor in only a few; that there is no strong inherent tendency of the stomach itself to herniate and therefore that a very simple type of repair is adequate in the great majority of cases. This operation I have called the 'standard simple repair'. It consists in freeing the lower quarter or third of the oesophagus from its attachments in the mediastinum down to the attachment of the phreno-oesophageal ligament or

the peritoneal sac, for the ligament is seldom easily identifiable (Fig. 4). A tape is passed round the oesophagus 2–3 cm above the upper limit of the sac. A stab incision is made in the diaphragm 6–7 cm from the margin of the hiatus, anterolateral to it, and a forceps passed through and guided up through the hiatus to penetrate the sac. The tape is grasped, the forceps pulled back, and the lower thoracic oesophagus drawn below the hiatus, invaginating the sac and the phreno-oesophageal ligament—where it exists—into the abdomen. With gentle traction on the tape to maintain reduction of the hernia, and to maintain 3–5 cm of abdominal oesophagus, the crura of the hiatus are approximated by wide bites, behind the oesophagus, and the oesophagus sutured to the margins of the hiatus with fine sutures which take the thickness of the oesophagus wall down to the mucosa. The tape is removed, the stab in the diaphragm closed, and that is all that

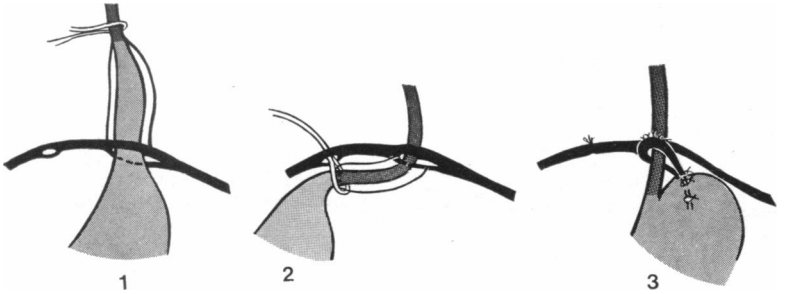

Fig. 4. The standard simple repair. See text.

is done. Nothing could be simpler and nothing—in the view of some —more inadequate, but nevertheless it has proved very satisfactory in properly selected cases.

At the other end of the scale there are those cases in which there is such extensive shortening and fibrosis, usually with stricture formation, that reduction is clearly impossible. In these I have used the colon interposition operation shown in Fig. 3*b*.

Between these two extremes there lies the group of cases in which there is evidence of longitudinal pull by the oesophagus, as shown by some shortening seen on the barium meal, at oesophagoscopy, or at operation, sometimes with oesophagitis and sometimes with evidence of frank duodenal ulcer or duodenal irritability—'duodenitis'. In some cases of this type I have reinforced the simple repair described above with a further ring of sutures between the oesophagus and the under margin of the hiatus, together with suture of the stomach to the abdominal oesophagus and suture of the fundus to the underside of the diaphragm (Fig. 5). In some I have carried out vagotomy and pyloroplasty as well as the simple repair or the simple repair reinforced below the diaphragm.

If I had adhered firmly to my principles I should have interrupted the oesophagus, and thus broken the longitudinal pull, by a colon or jejunal interposition in all those cases in which I thought it a significant factor. But this operation of colon or jejunal interposition carries an operative risk far higher than operations not requiring bowel anastomosis and I have been reluctant to use it except for established stricture or marked shortening.

In searching for some means of interrupting the longitudinal muscle pull I have made a point of taking the full thickness of the oesophageal muscle coat and putting in the sutures very close together when suturing the oesophagus to the margins of the hiatus, as I have already described. When a second row of sutures is similarly placed below the diaphragm I hope that the longitudinal muscle continuity is further interrupted. I hope that vagotomy may have some effect as well as reducing gastric

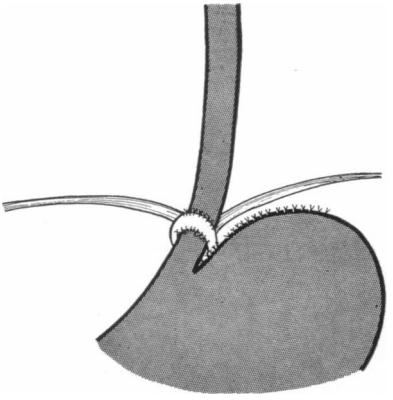

Fig. 5. The standard simple repair reinforced by suture of the oesophagus to the abdominal margin of the hiatus and fixation of the fundus to the oesophagus and abdominal surface of diaphragm. See text.

mobility and secretion. It would be easily possible to interrupt the longitudinal pull by a circular incision analogous to the longitudinal myotomy dividing the circular muscle in the Heller operation for achalasia; but a circular incision would clearly be followed by a disastrous stricture.

Interruption could be achieved by a multitude of small incisions, transverse to the long axis, at different levels, the total effect of which would be to interrupt all the long muscle strands at intervals along them. I do not think this would have adverse effects and I have begun to use this method (Fig. 6).

RESULTS

Between early 1948 and the end of 1969 I have operated on some 700 patients with hiatus hernia. I have discounted those operated on at Harefield up to 1960, for no consistent surgical policy was followed up to that time and they are not now accessible to me for follow-up. I have taken for analysis the 327 patients operated upon consecutively

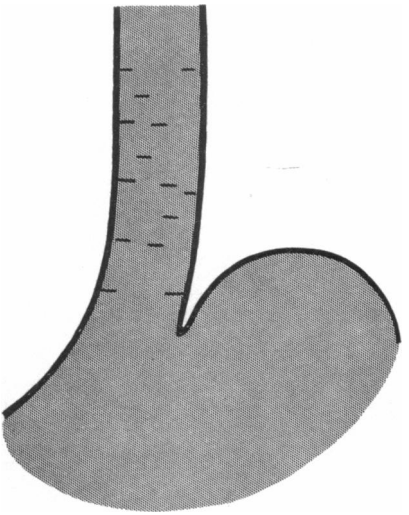

Fig. 6. Multiple transverse myotomy to interrupt longitudinal muscle tension.

between September 1960 and the end of 1967 at Southampton, whom I have treated by the techniques I have described for the reasons I have given. The age and sex distribution is shown in Table III. The period of follow-up averages 3½–4 years and ranges up to 9 years. The usual classification into sliding and para-oesophageal types is shown in Table I. These figures show, I think, the usual age and sex distribution in most series of cases, with perhaps a higher preponderance of the para-oesophageal type owing to the activity of many general physicians in referring these cases for surgery after reviving them from gastric haemorrhage—33 such cases were referred for surgery after one or more haemorrhages.

My suggested classification of cases into those with incompetent sphincter, those with competent sphincter, and those in whom it is doubtful is shown in Table II. Most of the para-oesophageal group are included in the competent sphincter group, but some are transferred to the incompetent group and vice versa; the group of dubious sphincter

TABLE III

AGE AND SEX DISTRIBUTION

Age groups		*Sex*	
0–20	7	Males	87
21–30	6	Females ...	235
31–40	16	Children under 2	5
41–50	35		
51–60	104		
61–70	120		
71–80+	39		
	327		327

TABLE IV

OPERATIVE TREATMENT

Standard simple repair (S.S.R.)	219
S.S.R. + Dacron patch	5
S.S.R. + subphrenic reinforcement	44
Colon or jejunal interposition	41
Anterior transposition	3
S.S.R. + vagotomy and pyloroplasty	6
S.S.R. + vagotomy and gastroenterostomy	2
S.S.R. + partial gastrectomy	3
S.S.R. + cholecystectomy	2
Abdominal repair	1
Partial reduction and dilatation	1
	327

competence comprises some of both types. I have judged whether the sphincter is competent or not on clinical, radiological, and oesophagoscopic grounds. All patients with stricture have been included in the incompetent group, for stricture only follows oesophagitis, although one does not always obtain a good history of persistent reflux preceding dysphagia from stricture.

Table IV gives the various operations. Standard simple repair I have already described. In 5 cases this was reinforced by a patch of Dacron or tantalum gauze sutured across the crura where these were widely divergent or of poor quality. The addition of subphrenic reinforcement has already been described, as has the colon or jejunal interposition operation. Anterior transposition has been used in 3 cases in which there was some oesophageal shortening but no stricture. A new oesophageal hiatus is made in the diaphragm, more anterior in position and nearer the top of the dome, to allow full reduction without tension on the oesophagus. The remainder are self-explanatory.

Causes of failure

The group of 219 simple repairs in Table IV can be broken down as shown in Table V, which also shows the failure rate. I have defined failure as failure to relieve the patient's symptoms, whether the hernia has recurred or not. If one considers only recurrence of the hernia, the rate falls to 8%, and in those with doubtfully competent sphincter to 12%. But the point I wish to make is that, even with the most

TABLE V

RESULTS OF STANDARD SIMPLE REPAIR

	No.	*Failures*	*Recurrences*
Incompetent sphincter ...	136	24 (17.6%)	11 (8%)
Dubious sphincter	25	6 (24%)	3 (12%)
Competent sphincter	58	1 (1.7%)	Nil
Total	219	31 (14.2%)	14 (6.4%)

euphoric interpretation, the failure rate in patients with incompetent sphincter is ten times that for those with competent sphincter. This latter group consists of patients with large hernias with a considerable portion of the stomach, sometimes all of it, in the chest. Those with incompetent sphincter are the great majority, the small hernias. This simple operation with a 1.5% failure rate in cases of competent sphincter has a failure rate of 8–12% in cases with an incompetent or dubiously competent sphincter; there must surely be some special factor present in such cases.

TABLE VI

CAUSES OF FAILURE OF STANDARD SIMPLE REPAIR IN 24 OF 136 CASES WITH INCOMPETENT SPHINCTER

4 died	Pulmonary embolism.
1 insane	Dysphagia attributed to hernia but unrelieved by operation. Repair sound, with no obstruction before or after operation. Patient later developed frank psychosis.
1 anaemia	Anaemia attributed to hernia but unrelieved by sound repair.
3 pain	Pain attributed to hernia but unrelieved by sound repair.
1 persistent vomiting	Vomiting, mistaken for reflux, indicated repair of hernia. Sound repair failed to relieve vomiting.
2 obesity	Hernia recurred in one patient with weight of 400 lb (180 kg) and in another grossly overweight during pregnancy.
1 hernia through dome of diaphragm	A hernia of stomach developed through dome of diaphragm at a distance from repair of hiatus, which remained sound. Further repair satisfactory.
2 dysphagia	Dysphagia attributed to oesophageal spasm secondary to reflux oesophagitis but was unaffected by sound repair. Oesophageal thickening noted at operation in one case. Further repair satisfactory in the other.
2 technical failure	Satisfactory after further repair in one; persistent reflux in the other.
2 persistent incompetence	Incompetence with reflux persisted in spite of sound anatomical repair.
5 oesophageal shortening	Noted at preliminary oesophagoscopy and barium meal in 4 and at operation in one. Repair failed, both anatomically and symptomatically.

Table VI shows a detailed analysis of the causes of failure of simple repair in the incompetent sphincter group. One can exclude 10 of the cases on the grounds of operative death, mistaken diagnosis, or failure to carry out the designed operation. Three others can be excluded on errors of surgical judgement or technical mishaps, leaving 11 patients in whom symptoms persisted with or without recurrence of the hernia, and in 5 of those oesophageal shortening was noted. In the next group, those with dubious competence, one finds the same picture on a smaller scale (Table VII). The single failure in a patient with a competent sphincter (Table VIII) illustrates the effect of longitudinal muscle pull on the lower oesophageal sphincter; for here the sphincter was

competent when the oesophagus lay loose in the presence of a lax baggy hernia, but incompetent after repair owing to the pull of the short oesophagus, and recurrence took the form of a sliding hernia. Examples of this are not uncommon.

TABLE VII

CAUSES OF FAILURE OF STANDARD SIMPLE REPAIR IN 6 OF 25 CASES WITH SPHINCTER OF DUBIOUS COMPETENCE

1 died	Perforated duodenal ulcer in early postoperative period.
1 anaemia	Anaemia attributed to hernia but unaffected by sound repair.
1 persistent vomiting	Vomiting, mistaken for reflux, unaffected by sound repair.
1 dysphagia	Dysphagia attributed to oesophageal spasm secondary to reflux but unaffected by sound repair.
2 pain	Pain attributed to hernia but unaffected by sound repair.

TABLE VIII

CAUSE OF FAILURE OF STANDARD SIMPLE REPAIR IN 1 OF 58 CASES WITH COMPETENT SPHINCTER

1 failure	Short oesophagus detected at operation only.

A similar picture is found in those treated by standard simple repair with subphrenic reinforcement, and is shown in Table IX.

TABLE IX

CAUSES OF FAILURE OF STANDARD REPAIR WITH SUBPHRENIC REINFORCEMENT

	No.	*Failures*	
Incompetent sphincter	43	4	Three noted to have oesophageal shortening and oesophagitis at barium meal and oesophagoscopy. One dysphagia, unaffected by sound repair.
Dubious sphincter	1	Nil	—
Competent sphincter	0	—	—

An unusual feature of this series is the rather high proportion of cases—12½%—in which oesophageal fibrosis, stricture, and shortening required oesophageal reconstruction by colon or jejunal interposition. This high figure is due partly to the popularity which the operation achieved in this area in providing virtually complete relief from the distressing condition of chronic dysphagia and partly to my own preference for it, rather than extensive mobilization and forced reduction, in patients with marked oesophageal shortening, even in the absence of stricture. The results are shown in Table X.

TABLE X

CAUSES OF FAILURE OF COLON OR JEJUNAL INTERPOSITION IN 5 OF 41 CASES

4 died	1 ileus
	1 infection
	1 haemorrhage from suture line
	1 liver failure
1 anastomotic stricture excised; failed. Partial gastrectomy succeeded.	

TABLE XI
MISCELLANEOUS OPERATIONS

	No.	*Failures*
Standard simple repair with Dacron patch	5	0
Anterior transposition	3	0
SSR + vagotomy and pyloroplasty	6	2
SSR + vagotomy and gastroenterostomy	2	0
SSR + partial gastrectomy	3	1
SSR + cholecystectomy	2	1
Abdominal repair	1	0
Partial reduction + dilatation of stricture	1	1
Total	23	5

The results of the remaining miscellany of various procedures are shown next (Table XI) for the sake of completeness, but they clearly have no analytical value.

Oesophagitis and shortening

There is nothing new in the observation that the presence of oesophagitis and shortening is the warning signal of difficulties to come in the repair of hiatus hernia, and great care was taken to detect evidence of these by radiography, oesophagoscopy, and at operation. Table XII shows the findings. Oesophagitis was suspected at barium meal in 72 of 233 patients with incompetent sphincter, from irregularities of mucosal pattern, lower oesophageal spasm, ulcer niches, or stricture. The findings were confirmed at oesophagoscopy in 59 of them and further confirmed at operation in 55. But it has to be remembered that in 41 of these the oesophagitis was gross and combined with shortening and stricture. Oesophagitis was detected at oesophagoscopy, without suggestion of its presence on barium meal, in 44 cases but was confirmed at operation, by evidence of thickening of the oesophagus, peri-oesophagitis, or local lymphadenitis, in only 13—a marked discrepancy. Oesophagitis was noted for the first time

TABLE XII
OESOPHAGITIS

	Suspected on radiography	Confirmed at oesophagoscopy	Confirmed at operation	First detected at oesophagoscopy	Confirmed at operation	First detected at operation
Incompetent sphincter (233)	72	59	55	44	13	5
Dubious sphincter (34)	2	2	1	4	1	0
Competent sphincter (60)	0	0	0	3	1	1

TABLE XIII
OESOPHAGEAL SHORTENING

	Suspected on radiography	Confirmed at oesophagoscopy	Confirmed at operation	First detected at oesophagoscopy	Confirmed at operation	First detected at operation
Incompetent sphincter (233)	72	55	46	16	6	4
Dubious sphincter (34)	0	0	0	0	0	2
Competent sphincter (60)	1	0	0	2	0	1

at operation in 5. The incidence of oesophagitis was very much less in patients with doubtfully competent or competent sphincters.

Table XIII shows the findings for oesophageal shortening. As with oesophagitis, the findings on barium meal and at oesophagoscopy do not correlate well with the findings at operation. But when oesophagitis and shortening are combined, one usually sees the more obvious and severe manifestations and the findings do correlate well. There were 73 such cases (Table XIV). Of these, 41 were treated by colon or jejunal interposition. Among the remaining 32 there were 9 failures (28%).

TABLE XIV
COMBINED OESOPHAGITIS AND SHORTENING

73 cases	41 treated by jejunal or colon interposition	5	failed (Table X)
	32 others — 16 standard repair	5	failed (Table VI)
	11 standard repair + subphrenic reinforcement	3	failed (Table IX)
	3 anterior transposition	0	failed (Table XI)
	1 partial reduction + dilatation	1	failed (Table XI)
	1 standard repair + subphrenic reinforcement + pyloroplasty	0	failed (Table XI)

CONCLUSION

To summarize, there were 39 failures in all types of cases. Nine of these patients have died; of the remaining 30, 12 have failed to obtain relief of symptoms in spite of anatomically sound repair; of the remaining 18 failures, 8 have been due to miscellaneous causes and 10 to oesophageal shortening. It is clear that shortening is an important cause of failure, even in this series in which I have taken great care to look for it and in which I have used colon or jejunal interposition whenever it was present to an extent which prevented easy reduction.

It can be argued that shortening results from spasm consequent upon oesophagitis. This may well sometimes be so but it is certainly not always so. Oesophagitis and shortening do not correlate well. The as-

sumption does not explain the manner in which hiatus hernia recurs. It is common experience that almost any type of repair is satisfactory for a year or two—then the hernia recurs, and then the reflux and the oesophagitis. One cannot blame oesophagitis for the recurrence, for it has all subsided for a year or two before the recurrence took place.

In 10 patients symptoms attributed to a hernia have not been relieved in spite of apparently sound repair; 2 had reflux, 3 had dysphagia, and 5 had pain. It is possible that pain may have been due to some entirely unrelated and undiagnosed lesion; but it is also possible that the pain was due to oesophageal muscle spasm and would not be relieved by repair of the hernia, which was a secondary effect of the spasm. This is more likely in the cases of dysphagia for there was no organic obstruction—no stricture or over-firm repair; similarly in the cases of persistent incompetence. Unfortunately, oesophageal pressure manometry was not available for these patients.

I hope you will accept that I have produced some evidence to support the view that disorder of function of the oesophagus, and particularly of the longitudinal muscle, is at the root of many cases of hiatus hernia and that we must look for a method of assessing it. We must cease to be disappointed that firmer and stronger methods of fixation do not cure disorders of structures which depend upon mobility for their function.

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