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HIATUS HERNIA*

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Hiatus hernia is a common serious condition that affects the upper alimentary canal; and yet in 1940 nothing important was known about it.

Some surgeons, regarding it as a bizarre internal abdominal hernia, recognized the "paraoesophageal" variety, and cases of volvulus of the stomach had been described. Radiologists did not use the "head-down" position to test competence of the cardia, though Soresi (1919) had described this technique more than 20 years before. And endoscopists "peering through a glass darkly" (Allison, personal communication) had noted obscure inflammations and strictures that were "idiopathic." Morbid anatomists destroyed the evidence by eviscerating the parts and by cutting the oesophagus away from the stomach.

Thus, in spite of the intensive study of "indigestion" that occupied the physicians, and of countless laparotomies at which the appendix and the gall-bladder were hopelessly removed, sliding hiatal hernia remained unknown and its complications arose without cause.

It was an Englishman, Philip Allison, who strung the isolated threads into one story. He defined hiatus hernia and pointed out how it differed from abdominal hernias in general, and from other diaphragmatic hernias in particular. His ideas, simple and correct, set research and treatment advancing upon the road of progress, and innumerable patients have benefited as a result.

In a number of papers published after 1940 Allison and many others have hammered the following things home.

Varieties of Hiatus Hernia

1. There are three principal varieties of hiatus hernia. These should be differentiated, not for anatomical reasons, but because they produce different pathological changes.

Paraoesophageal hernia, in which the cardia is normally placed, and the greater curve of the stomach together with the gastro-colic omentum herniates into a peritoneal sac in the mediastinum and through the oesophageal hiatus. This hernia scarcely concerns otolaryngologists, because the oesophagus is normal. It produces few symptoms in some cases, but may cause cardiac pain or arrhythmias, and the stomach in the mediastinum may develop a gastric ulcer or a carcinoma.

Sliding hiatal hernia is ten times more common than the paraoesophageal variety. It is a hernia *en glissade*, but the sac is unimportant because viscera from the abdomen do not get into it. This hernia, which occurs at all ages (Belsey (1952) reported one case in a child aged 3 weeks), matters for one reason only: it produces incompetence of the cardia, and this allows reflux of acid pepsin into the lower part of

the oesophagus. It is a strange fact that the oesophagus should be vulnerable to the secretions of the stomach; but so it is. Reflux produces digestion of the gullet (oesophagitis), and this leads to a series of important pathological complications. The clinical story is typical—the principal symptoms being heartburn that occurs when the patient stoops or lies down, dysphagia of varying degrees, and symptoms that result from anaemia. The diagnosis of incompetence and sliding hernia is made radiologically, using the postural techniques that are now well known; and oesophagoscopy confirms the issue. There are many difficulties—mentioned below—associated with unravelling the causes of pain, anaemia, and dysphagia in these cases. The rational treatment is to make the cardia competent, and the simplest way of doing this is to reduce the hernia and fix the oesophago-gastric junction below the diaphragm.

"Mixed" or "rolling" hiatus hernias have features of both the above varieties.

2. Sliding hiatal hernia produces a short oesophagus, not vice versa. The sequence of events is hiatus hernia, reflux inflammation, spasm or fibrosis, shortening, stricture. "The term 'short oesophagus' is used to describe a condition in which the oesophago-gastric junction lies above the diaphragm and cannot be restored to its normal position below the diaphragm." (Ellis *et al.*, 1958).

3. Hiatus hernias are generally acquired, but may be congenital (see below): the term "congenital short oesophagus" is loosely and improperly used.

4. Gastric heterotopia and islands of ectopic gastric mucosa play no part in this story. These occur most commonly at the upper end of the gullet and are not known to be harmful.

In the remarks that follow I express my personal opinions and direct your attention entirely to matters that concern an otolaryngologist. What more should he know about hiatus hernia? How can he help in diagnosis? and, What are the limitations of endoscopic treatment? I shall assume that he will not be anxious to do a thoracotomy, or a laparotomy, except in critical circumstances.

Congenital Short Oesophagus

Before embarking on the subject of hiatus hernia the position as regards "congenital short oesophagus" must be clarified. This name is commonly and erroneously applied to sliding hiatal hernia in which the oesophagus is at first normal and a part of the stomach ultimately moves into the lower mediastinum. My own views about congenital short oesophagus have already been recorded (Barrett, 1956, 1958).

It would have been better if the term had never been introduced, because it has led to wrong thinking. I have never seen a patient in whom all the coats of the

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oesophagus, normal in other respects, were congenitally short, and in whom a part of the stomach, as a consequence, lay in the mediastinum. Moreover, the fact that an infant is found to have a sliding hiatal hernia does not mean that the abnormality is congenital. But a condition exists to which the term congenital short oesophagus can legitimately be applied; though some consider that the anomaly is better described as the "lower oesophagus lined by columnar epithelium." In this we find that the gross anatomy of the gullet and of the stomach is normal to external and radiological appearances; and, in consequence, pathological changes that are detected are apt to be recorded as being "in the oesophagus." And here the question of nomenclature intervenes, for even among those who are well aware of the facts there are some who call the abnormal lower segment of the gullet "stomach" (as I was wont to do in times past) and others who refer to it as "oesophagus lined by columnar epithelium."

This confusion of terms matters because (1) the segment of the gullet lined by columnar epithelium is liable to the diseases that occur in the stomach, as opposed to oesophageal anomalies, and that is why some surgeons call it "stomach" in spite of its anatomy. (2) Allison has pointed out, and I confirm, that in a proportion of cases in which the lower oesophagus is lined by columnar epithelium there is, in addition, a sliding hiatal hernia. Such a patient has, from above downwards, oesophagus lined by squamous epithelium, oesophagus lined by columnar epithelium, and then a sliding hiatal hernia above the diaphragm.

The explanation of how such a deformity occurs is not understood; but let us be clear that it has nothing to do with "islets of ectopic gastric mucosa." In my view it represents a failure in the developmental evolution of the foregut that occurs when the embryo is between 3 and 100 mm. in length (Johns, 1952). This area is at first lined by columnar epithelium that proliferates exceedingly until ultimately the lumen becomes obliterated. After a short time the channel is re-established by the formation of numerous clefts that fuse into one pathway. It is only at the conclusion of these complicated metamorphoses that the oesophagus acquires its "normal" squamous epithelium. I believe that when the lower oesophagus is lined by columnar epithelium this cycle may not have been completed.

A surgeon using an oesophagoscope for diagnostic purposes must be aware of this developmental fault, because the treatment of the diseases that are associated with it differs from that appropriate to sliding hiatal hernia. There is, for instance, no question of replacing the segment lined by columnar epithelium below the diaphragm; it has always been in the mediastinum, and it is anchored there by its segmental aortic blood-supply.

A lower oesophagus lined by columnar epithelium may cause oesophagitis and its complications, and it is the usual source of columnar-cell carcinoma that originates in the gullet as opposed to the stomach. In the lower segment typical gastric ulcer occurs.

Diagnosis

With these facts in mind it would be nice to be able to tell you that diagnosis of these cases is easy. Unfortunately this is not the case; but there are pointers that may help.

1. The majority of sliding hiatal hernias (from which this condition must be distinguished) are small; in them

the hernia seldom extends upwards more than 5 cm. above the hiatus. If the change from squamous to columnar epithelium occurs higher than this in the mediastinum—a point that can be suggested radiologically but can be verified at endoscopy—the condition should be suspected.

2. If there is doubt the surgeon should put one or more silver clips (such as are used by the neurosurgeons to secure haemostasis) upon the point of transition of the mucosa. Special forceps are available that enable this to be done through an oesophagoscope. These clips can then be seen in radiographs and their position relative to the apparent oesophago-gastric junction (the cardia) noted.

3. When a biopsy specimen is taken from the oesophagus at endoscopy, an attempt should be made to get a piece of the mucous membrane below the lesion. In the case of simple sliding hiatal hernia the fact that this mucosa proves to be gastric does not come as a surprise; but if the lesion under investigation happens to be situated in the middle or the upper part of the gullet, and if, in addition, the gut below it has the typical radiological appearances of "oesophagus," the information should transform the picture of diagnosis and treatment.

Sliding Hiatal Hernia

Aetiology and Pathology of Oesophagitis

Since the pathology and the treatment of most hiatal hernias hinges upon the failure of the barrier to reflux that should exist between the stomach and the gullet, you must be familiar with some aspects of this subject.

Wangensteen and Leven (1949) and Ferguson *et al.* (1950) have shown that all the changes we now recognize as "oesophagitis" can be produced by perfusing the lower part of the gullet with acid-pepsin. Within a few hours the mucosa is destroyed, and eventually perforation occurs. In man the process is less drastic because the insult is not continuous. They also showed that hydrochloric acid alone, in physiological strengths, is not harmful, but that the secretions normally present in the duodenum are dangerous. Anybody who has treated a patient with a duodenal fistula knows the damaging effects upon the skin of the abdominal wall and can visualize what goes on in the oesophagus.

In 1953 Aylwin did experiments that showed how the gullet of a normal individual is protected by day against the ravages of reflux. Not only is the cardia competent, but the lower oesophagus is coated with mucus and bathed by alkaline saliva. When a patient who has a sliding hiatus hernia stoops down or lies flat at night, these defences become inefficient. The most damaging secretions may come from the mucous membrane of the pouch that lies above the diaphragm, and not from the main part of the stomach in the abdomen.

The histological changes that occur in the lower oesophagus have been studied at operations by surgeons and in necropsy specimens by Peters (1958). The salient features of oesophagitis are:

It affects the oesophageal mucosa alone at first, and is sharply limited by the gastric mucosa below. It is most pronounced at the point where the epithelium changes from squamous to columnar. One reason that recognition of "oesophagitis," as a real entity, was so long delayed was that the red, angry surface that the endoscopist sees fades completely as soon as the tissues are removed from the body, and there may be little to see at necropsy.

The microscopical changes exceed those seen with the naked eye.

The pathology is of inflammation. There are elements of acute and chronic type, implying repeated clinical episodes.

Each layer of the oesophageal wall protects for some time the one external to it, so that after an acute exacerbation there is a terraced destruction.

Shallow epithelial ulcers occur and may heal completely or lead to leucoplakia.

After the submucosa has been destroyed the lesion generally progresses through the muscle coats, producing, not a penetrating ulcer, but a tumorous mass of inflammatory tissue that becomes fixed in the mediastinum by perioesophageal adhesions and adenitis.

Inflammatory oedema, congestion and thrombosis of blood vessels, fibrosis, and scar play their parts.

The time taken for these changes to occur varies within wide limits, and the reasons for this are obscure.

Barrier Against Reflux

This subject has become confused because two different types of information have been overlaid.

On the one hand, physiologists, studying the mechanisms of swallowing and vomiting, have worked out certain pressure barriers, pressure changes, flap valves, and mucosal rosettes that exist and operate in normal subjects.

On the other hand, surgeons know that when a patient has a sliding hiatal hernia reflux occurs spontaneously into the lower oesophagus. Moreover, they know that reflux is immediately controlled by replacing the oesophago-gastric junction in the abdomen. They infer that competence of the cardia depends upon physical conditions (such as the difference in pressures in the lower mediastinum and in the abdomen, in the angle at which the gullet joins the stomach, and in the pressure of a length of oesophagus below the hiatus) rather than upon physiological sphincters, pinchcock effects of the diaphragm, or aberrations of nerve supply to the cardia. The idea that the hiatus is a pinchcock dies hard with otolaryngological surgeons; the time has come when they must accept that this hiatus in the diaphragm is not a sphincter, but simply the point at which the pressure upon the gullet changes from relatively negative to positive.

Relation Between Symptoms of Oesophagitis and Presence of a Sliding Hernia

The importance of oesophagitis as a cause of a special kind of dyspepsia has been proved beyond dispute. But, whereas in the past this explanation was not known, it is possible that nowadays too much blame is laid at this particular door. In my experience it is common for bleeding into the bowel of unknown cause to be attributed, without sufficient reason, to oesophagitis.

Peters (1958) says that 29% of the cases he examined at necropsy had lesions such as gastric ulcer, duodenal ulcer, or cholecystitis, as well as oesophagitis. This statement underlines the necessity for making sure not only that an individual patient has a hiatus hernia but that the symptoms of which he complains are due to this condition.

It is certain that not all those who have a hiatus hernia are ill, though they stand in danger of becoming so. Occasionally heartburn occurs in normal individuals, and we must remember that the cardia becomes

physiologically incompetent whenever we vomit. Some people who have symptoms such as heartburn have no demonstrable hernia; others have reflux that can be demonstrated radiologically and complain of nothing relative to the oesophagus. The size of a hernia that a radiologist sees bears no relation to the severity of the pathological changes or to the symptoms it can produce. Small damaging hernias require treatment as urgently as those that are larger.

There is a small but important group of patients whose oesophago-gastric junction is apparently below the diaphragm and competent, but who have oesophagitis. These are the cases in which the lower oesophagus is lined by gastric epithelium (congenital short oesophagus). And yet another group, emphasized by Lortat Jacobs, of Paris, have no abdominal oesophagus, and therefore have reflux without obvious cause. Creamer *et al.*, (1959) at St. Thomas's, have shown the importance of having a length of the oesophagus that is subjected to positive abdominal pressure below the diaphragm.

Reflux oesophagitis waxes and wanes, and there are patients whose symptoms are of long duration who have limited destruction in the gullet. On the other hand, oesophagitis that has permeated and invaded the full thickness of the gullet can occur in a few weeks or months; and one of the outstanding problems at the moment is to assess the severity and the permanence of the damage before embarking upon treatment that can be difficult and dangerous.

Endoscopic Appearances

The endoscopic appearance of oesophagitis are well known; but even experienced operators may be uncertain of what they see upon occasion. Hence the necessity for biopsy in all cases.

An important reason for this difficulty in interpretation is that in acute inflammation there will certainly be some bleeding beyond the point of the oesophagoscope, and this obscures the view. This condition bleeds more easily than does a malignant growth, but oesophagitis and a neoplasm may coexist. A normal oesophagus should be empty. Retained secretions or food suggest a hold-up at or above the cardia. The reaction of the oesophageal contents should be tested with Congo red paper: an acid reaction suggests reflux. An incompetent cardia is patulous and easily passed with the tip of the oesophagoscope (the instrument must be long enough to enter the stomach) and drops of gastric juice can often be seen welling back as the patient breathes. But all these things may be wanting and oesophagoscopy may be negative in a patient who yet needs surgical treatment: therefore, do not be dogmatic in your report. It is a serious mistake to miss a carcinoma of the stomach or of the lower part of the oesophagus. A malignant growth near the cardia can cause incompetence and reflux, leading to oesophagitis above it. Growths in this position are notoriously difficult to show radiologically. For this reason it is wise to get a biopsy not only of the inflamed oesophagus but blindly of the stomach beyond. Where doubt persists laparotomy is mandatory.

A hiatus hernia that is causing symptoms is generally suspected clinically, diagnosed radiologically, and confirmed at thoracotomy. The importance of endoscopy is to get extra evidence, to assess the extent and position of lesions such as oesophagitis, and to eliminate malignant disease.

In the case of paraoesophageal hernia the endoscopist confirms that the cardia is below the diaphragm, and that it is competent. The former can be done only in a patient who is breathing naturally, because it depends upon noting the absence of respiratory oesophageal hiatus. The oesophagus itself is normal in these cases.

Ulceration

Apart from carcinoma, there are several distinct types of ulceration that occur in these cases. The first is ulceration situated in the squamous epithelium of the oesophagus itself above the oesophago-gastric junction. These ulcers are generally small, discrete, and shallow; they tend to heal; they cannot be shown radiologically; and they never perforate.

When the ravages of penetrating oesophagitis have transgressed the coats of the gullet and formed a mass of contracted muscle and scar (see below) the epithelium that lined the affected area disappears for most of the length of the stricture, and the lumen that remains is ulcerated—granulating, septic, and tortuous. The lesion that results is a stricture that may be short or long; but at oesophagoscopy one gets an entirely false impression of what has happened because only the entrance to the lesion can be seen, and the mucosa in the vicinity is often remarkably normal. Nor is it possible to assess the length or the penetration of the pathological process at oesophagoscopy.

Ulcers that occur in the pouch of stomach that forms a sliding hiatal hernia are true gastric ulcers (Barrett, 1950) and have nothing to do with reflux. They were originally described by Rokitsanski, though he did not understand their aetiology. They are difficult to see at oesophagoscopy because there is often oesophagitis or a stricture at the oesophago-gastric junction above the ulcer. Allison and Johnstone (1953) described the radiological appearances. Many such ulcers are diagnosed for the first time at operation, and sometimes they cause massive or fatal bleeding by penetrating the heart or the aorta.

Gastric ulcers and carcinoma also occur in the stomachs of those with paraoesophageal hernias, but these are beyond diagnosis endoscopically.

Bleeding and Anaemia

At a conservative estimate 10% of patients who have oesophagitis have anaemia severe enough to affect the symptoms. Considering how easily the inflamed mucosa bleeds at oesophagoscopy, it is surprising that massive haematemesis and melaena are uncommon. The fact is that occult blood in the stools is often difficult to detect, even when the haemoglobin has been severely reduced; this is probably because the bleeding is intermittent. Haematemesis is rare. The anaemia that occurs in these cases is hyperchromic and microcytic; it responds to cure of the oesophagitis and to the administration of iron.

It is a mistake to attribute anaemia of unknown intestinal origin to oesophagitis unless there is an obvious inflammation in the lower oesophagus, and unless all the usual causes have been systematically eliminated.

It is useless and meddlesome to treat bleeding, caused by oesophagitis, by local applications of caustics or styptics.

Stricture of the Oesophagus

Stricture is at once the most crippling and the most dangerous complication of oesophagitis: crippling because the patient ultimately starves; dangerous because treatment can only alleviate, and that at a risk. It is my opinion that even when a stricture has been wisely and successfully treated the patient will never be quite normal again. The normal oesophagus is irreplaceable.

Apart from malignant disease and the effects of swallowed corrosives, oesophageal strictures are, in practice, due to reflux. Congenital, idiopathic, and specific strictures are so rare as to be suspect. Do not make these diagnoses, and do not act on them, unless you are sure of your ground.

When a patient complains of dysphagia and a radiologist reports incompetence of the cardia and a hiatus hernia, and when, in addition, there is a stricture in the lower 2 to 3 in. (5 to 7.5 cm.) of the mediastinum, one can assume that the gullet below the stricture is lined by columnar epithelium whatever its external shape or radiological appearance. But if the stricture is due to carcinoma or to a swallowed corrosive the epithelium will be squamous, above and below the stricture. This fact is important.

A stricture that is causing dysphagia is nature's way of protecting the lower oesophagus against further episodes of digestion. This truth was first impressed upon my mind in the following circumstances.

A child was admitted to Horton War Hospital suffering from dysphagia since birth. A barium-swallow examination showed a long, narrow stricture in the lower part of the gullet. At that time neither the pathology of these lesions nor their treatment was clear. At oesophagoscopy the lower oesophagus contained residue, and the entrance to the stricture was of pinhole size. But the mucous membrane above the stricture was normal; there was no ulceration or inflammation. The stricture was dilated with appropriate skill, and the immediate result was that the child "could swallow anything." A few days later, with the design of completing the good work, another oesophagoscopy was done, and the appearance of the mucosa above the stricture was dramatically changed. It was fiery red and haemorrhagic.

The third thing about a stricture due to reflux is that neither the clinical story nor the radiological appearances nor the findings at oesophagoscopy enables us to be certain of the precise extent of the lesion. There are a variety of strictures associated with oesophagitis, and they may be combined or separate.

The radiological appearances of stricture can be produced by muscle spasm. This happens in most cases of acute oesophagitis, and is especially deceptive when there is a small carcinoma high on the lesser curve of the stomach. It has been said that spasm may follow vagotomy; but I doubt this, and believe that the explanation of the recorded cases is that the dysphagia was secondary to the trauma of the operation.

Spasm due to the irritation of oesophagitis may be complicated by vascular congestion and oedema, and these can reduce the lumen of the gullet to a chink, and cause serious dysphagia.

"Strictures" due to oedema or vascular congestion vary in intensity and are not permanent. As the acute episode of oesophagitis wanes they disappear and the patient may swallow again. But when the destructive changes that follow repeated episodes of oesophagitis extend through the submucosa and the muscles, the

lesion becomes permanent. And the fact that this final phase has been reached can be verified only at operation, though it will have been suggested by the story, the x-ray films, and oesophagoscopy.

The steps by which the gullet is destroyed have been studied at St. Thomas's Hospital by making histological sections of every stricture we have excised; certain problems have been revealed (Barrett, 1958). There has been a tendency to assume that fibrosis and scar accounts for all that occurs; but this is not so. Nor can it be assumed that an oesophageal stricture is comparable to a gastric ulcer or an hour-glass stomach. Here are the facts.

After repeated episodes of oesophagitis in some patients, and soon after the first clinical trouble in others, fibrous tissue begins to accumulate in the submucosa. It is vascular, young, cellular, and oedematous at first. It may have intact squamous epithelium upon its surface, and it produces a palpable hard lump that generally surrounds the gullet. So hard may this tumour be that carcinoma may be confidently diagnosed in error.

In a few cases the process stops at this stage: the oedema settles, and a ring of fibrous tissue in the submucosa is the result. A stricture of this type is usually sited where the mucous membrane changes; but it may be lower down, and, in this event, it has columnar epithelium above it.

"Oesophagitis" is a progressive disease in most cases, and sooner or later the inflammation invades the muscle coats of the gullet. When this has happened sections show a fibromuscular lump about the size of a chestnut. Both the muscle layers are strikingly thicker than normal and the submucosa is completely replaced by scar. The mucosa may or may not be deficient, and the lumen is greatly narrowed throughout the length of the lesion. Swallowing is seriously obstructed.

At this stage the lesion is different from a peptic ulcer in the stomach or the duodenum. It is not an ulcer but a mass of tissue that feels very hard. The fibroblasts do not destroy the muscle, but grow in strands between the bundles. When the inflammatory reaction has reached the mediastinum most of the original muscle is still extant, though the nuclei are abnormal in places and some of the vessels are thrombosed.

We believe that the accretion of muscle that forms so much of the bulk of these strictures is due to a concertina effect. As the scar in the submucosa contracts it heaps the muscle bundles together on its outer surface, thus adding to the bulk of the tumour and the severity of the dysphagia.

Once the inflammation reaches the outer side of the gullet the tissue planes in the mediastinum become oedematous and the adjacent lymph nodes enlarge. The strictured area is now adherent to the pericardium anteriorly, to the aorta at the back, and to the pleura and pulmonary ligaments laterally. There is one other factor that is relevant. As the blood-vessels in the submucosa are replaced by scar, the life of the muscle depends upon vessels that enter from the surface and run towards the lumen. In advanced cases recovery becomes impossible because these vessels thrombose, the muscle atrophies, and nothing but scar remains.

Treatment

Considering the histological changes, one may venture an opinion on treatment. Once a fibrous stricture has

begun to form, medical treatment is unlikely to relieve dysphagia. Two things must now be done: (1) prevent any further reflux of acid-pepsin, and (2) re-establish the lumen of the gullet. Neither alone yields more than passing benefit; and simply to dilate a stricture of this kind is like blowing up a tyre that has a puncture.

When the clinical history is short and dysphagia not serious, when the narrowing is not gross, and when repeated barium-swallow examinations show variations in the outline and size of the narrow segment, one may hope that congestion, spasm, and oedema will subside after surgical reduction of the hiatus hernia. If the patient happens to have a lower oesophagus lined by columnar epithelium (as opposed to a hiatus hernia) surgical reduction is not feasible, and more drastic measures are necessary.

If reflux has been controlled, and an annular stricture remains in the submucosa, some say that the lumen can be most effectively re-established by excising the scar. This has been done by exposing the mass at thoracotomy, incising the muscle coats down to the scar in the line of the bowel, and cutting out the ring of scar. The mucosa in these circumstances regenerates quickly. The merits of this procedure, as opposed to repeated dilatations from within, are matters for individual decision. So, too, are plastic operations to enlarge the lumen.

But if the scar has penetrated through and beyond the muscle and fixed the tumour in the mediastinum, nothing short of excision of the abnormal segment gives a permanent result. After excision the question of how to bridge the gap looms large, and is debatable. My own preference, at the moment, is to transpose an isolated loop of small bowel or colon, and to place this between the proximal part of the oesophagus and the stomach.

It is fair to underline these points: that, before operation, nobody as yet knows how to diagnose the extent and permanence of the destructive changes in the gullet; that the possible benefits of corticosteroids at certain phases in the disease are under debate; and that, once a permanent stricture has formed, the best operation to perform remains a matter of personal opinion.

In the past there has been a tendency to dodge these issues, chiefly because exacting surgical procedures are necessary, and to devise less difficult operations. This would be praiseworthy if the rationale of the new procedures did not defy existing knowledge.

The problems involved in treating stricture of the oesophagus due to oesophagitis are technically more involved than those that occur in the management of carcinoma. Do not embark upon resections without thought; and learn from the mistakes of those who have travelled the road.

Conclusions

I recall some advice that William Mayo, that wise man of surgery, gave me 25 years ago. "Do not," he said, "attempt an important lecture unless you have at least one thing to say." Here, then, is the message that I hope you will think about and accept:

Do not dilate strictures due to oesophagitis unless you can effectively and permanently prevent reflux of acid-pepsin at the same time. In infants, and in old people, it may be justifiable to temporize upon occasion; but the issue is clear in most cases.

"Why not dilate strictures?" you ask. "The operation is safe in competent hands; the immediate results are gratifying; the risks of alternative operations are considerable; and histological preparations have shown that, even in patients who have had a long clinical history of dysphagia, muscle may predominate over scar in the 'tumour.' Moreover, many patients have been jockeyed along from year to year by repeated endoscopies."

The case against dilatation is this. Immediate relief of dysphagia is no criterion of cure. By stretching a stricture of this sort nature's barrier against reflux is broken down, and the inexorable complications of oesophagitis are rekindled to flame again. With each exacerbation the scar, that remains for ever, penetrates the muscle of the gullet more deeply and rises upwards in the mediastinum. The end is worse than the beginning.

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The number of emergency calls received by the London Ambulance Service continues to increase. In a report to the Health Committee Dr. J. A. SCOTT, the medical officer of health, says that 104,983 calls were received in 1959; in 1958 the number was 99,188 and in 1957 97,535. Calls to street accidents in 1959 numbered 21,701, 2,519 more than the previous year. Despite the extra journeys the average time taken by ambulances to reach the scene of accidents was only a fraction of a minute longer than 1958—6.6 minutes instead of 6.5. On average, patients were admitted to hospital 20.9 minutes after the calls for help were received. There were the usual peak hours for the different kinds of call—maternity cases between 3 and 4 o'clock in the morning, home accidents at midday, street accidents in the late afternoon and early evening, and cases of assault towards midnight. Maternity cases, however (27,429 of them), continued to outnumber all others.

ON VIRUS METABOLISM AND
NEOPLASIA

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The term "virus" covers a range of organisms varying widely in physical and biological properties. One common feature is that all are obligate intracellular parasites. No valid evidence has been produced that any virus—bacterial, plant, or animal—possesses any equipment which would allow it metabolic activity in its own right, and it is generally assumed that virus multiplication is dependent on the metabolism of the host cell. The study of "virus metabolism" therefore resolves itself into the search for modifications of the metabolism of the host cell in the course of viral infections. With phage-infected bacteria this has been most profitable, but with infected animal tissues has so far only scantily illuminated the problem of virus multiplication.

Several limitations must be imposed on the interpretation of biochemical changes in virus-infected tissues. When only a small proportion of cells in the tissue are infected compensatory changes may occur in adjoining cells, so that a composite picture is presented of cells under the influence of two stimuli. This applies not only to complex tissues *in vivo* but also to tissue cultures containing only one cell type. The effects of infiltration with inflammatory cells can usually be excluded, either by adequate histological control or by the nature of the host tissue—that is, in tissue culture. Alterations in the overall metabolic activity of a tissue may occur owing to necrosis of some cells. It is important not to confuse the changes secondary to cell death with those causing cell death. For this reason, studies on tissues late in the course of viral infection (and damage) must be interpreted with great reserve. In general, the earlier a change is observed the more likely is it to be primarily induced by the virus.

Substances other than virus contained in the inoculum may also produce metabolic alterations, and this possibility must be excluded by inoculation of controls with material of similar origin not containing virus. Finally, where single enzyme activities are measured, alterations may be due not only to an absolute increase or decrease in the quantity or activity of apoenzyme or coenzyme, but also to a "diluted" or "concentration" effect of an increase or decrease in the amount of fat or water in the individual cell, or the amount of extracellular fluid in the tissue. Estimation of water and lipid in the tissue and calculations of activities per unit dry or lipid-free dry weight deals satisfactorily with this potential course of error.

Ectromelia virus multiplying in mouse liver forms a virus-host system for which these objections and limitations are not serious.

The activities of various enzymes in this system have been estimated and in part reported (de Burgh, 1957; Nelson and de Burgh, 1958, 1959). These have been compared with similar estimations in regenerating mouse liver. In this case new normal liver is rapidly reproduced and cell division occurs; this may be taken