A British Medical Association Lecture ON SOME POINTS IN THE SURGERY OF GASTRIC AND DUODENAL ULCERS.

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OF all the advances which have been made in surgery in the last twenty years none are more remarkable than those in the diagnosis and treatment of chronic ulcers of the stomach and duodenum. Not only has surgery provided a complete cure for one of the most crippling conditions which can affect a man in the full vigour of life, but surgeons have actually supplied physicians with a precise system of diagnosis in a field which was formerly most obscure. Routine explorations of the stomach and constant contact with the physical facts of its diseases have enabled surgeons to reveal its living pathology, and to build up a clinical picture remarkable for its precision.

Before discussing the purely clinical aspects of the subject it will be well to note its extent, and to form a clear idea of the pathological entity we are discussing. The extent can be fairly gauged by the admissions to a general hospital. In the three years 1923-25 close upon 30,000 patients were admitted to the surgical wards of the London Hospital, and of these no fewer than 1,275 were admitted for gastric and duodenal ulcers. It would appear, therefore, that over 4 per cent. of the cases admitted to the surgical beds of a large general hospital are suffering from this condition. Remembering that the hospital admissions form only a small fraction of the actual cases, it will be seen that the disease is one of considerable frequency, and must result in a large amount of ill health and incapacity. Moreover, the average individual who consults a surgeon for chronic gastric ulcer probably only does so after years of intermittent pain which has made life almost intolerable. He is in the most active period of life, and many patients undoubtedly owe their trouble to the strenuous lives they have led, and the irregular meals which these have involved. Perhaps this is an explanation of the greater incidence in men. Of the 1,275 London Hospital cases above referred to 660 were gastric ulcers with a male incidence of 70 per cent., and 615 were duodenal ulcers with a male incidence of no less than 86 per cent.—figures which agree closely with those found elsewhere by both English and American surgeons.

PATHOLOGY.

There is no more concrete pathological entity in surgery than a chronic gastric ulcer, and its discovery at operation can never be a matter of doubt. These ulcers occur most frequently upon the lesser curvature near the pylorus, and on the posterior wall. At the cardiac end of the stomach they are rare, and on the greater curvature they are practically never seen. Their actual cause is unknown, but they probably commence as small acute lesions which gradually develop into chronic ulcers. Indeed, there are some grounds for supposing that the extension of a chronic ulcer is due to the formation of acute ulcers on its margin, and this would account for the very marked periodicity which is so characteristic of the symptoms of the disease. In the early stages the mucous membrane alone is involved, but the exposed submucous and muscular coats are rapidly attacked by the gastric juices and a funnel-shaped ulcer is produced which gradually penetrates the entire wall. Where the ulcer occurs on the lesser curvature the inflammatory process extends into the lesser omentum, which becomes thickened and distorted, a huge sheaf of lymphatics surrounding the coronary vessels. If the ulcer is on the posterior wall it may become adherent to the pancreas or the liver, and finally burrow deeply into its substance. A posterior ulcer will often involve the transverse mesocolon, and this is a matter of extreme practical importance

since it exposes the middle colic artery to considerable risk of injury in the course of an operation.

It is characteristic of these ulcers that the tissues around should become densely infiltrated, and as contraction is thus interfered with the natural healing of the ulcer which might otherwise occur is prevented. Occasionally, however, healing does occur, and it may then be accompanied by gross constriction at the site of the ulcer, as is most strikingly seen in the hour-glass stomach produced by the contraction of a saddle ulcer. In a similar manner the healing of a pyloric ulcer may be accompanied by such contraction as to produce severe organic obstruction of the pylorus.

The pathology of duodenal ulcers is very similar to that of gastric ulcers, but they never reach such large dimensions and are never accompanied by the same degree of induration. They are most common on the posterior surface, and at the junction of the first and second parts of the duodenum. It is quite common to find two ulcers opposing one another, one on the anterior and one on the posterior wall; the latter, from its relationship to the gastro-duodenal artery, is liable to produce severe haemorrhage, whilst the former may readily perforate. When an ulcer on the anterior wall is healed its site may often be recognized as a thin, white, radiating scar, whilst in the active state the peritoneal surface becomes stippled with red points in a characteristic manner if it is gently rubbed with a swab.

The relation of gastric ulcer to cancer is still a matter of acute controversy, but it would seem that although carcinoma may develop on the basis of a chronic ulcer this is by no means a common event. The fact that in every chronic ulcer considerable displacements of the epithelium are liable to occur probably led to the idea that these displacements were malignant downgrowths. From the practical point of view, however, it is sometimes difficult to determine clinically whether an ulcer is or is not malignant, so that where there is any doubt its resection in a suitable case would always seem to be a wise precaution.

The actual causation of chronic gastric ulcers is quite unknown. In animals they are only produced with difficulty, and the most successful method has been that of feeding with food grossly contaminated with *B. coli*. On the one hand, it seems certain that infection plays some part in their production, but on the other it is obvious that, once the mucous membrane has been destroyed, the gastric juice can exert a very destructive action upon the exposed tissues. The fibrosis which naturally results prevents that contraction which is an essential process in the healing of any ulcer, and the continued action of the gastric juice is a constant irritant favouring its extension. The fact that gastric ulcers are so commonly associated with hyperchlorhydria, and that in duodenal ulcers this is almost invariable, suggests that the acidity of the gastric juice has an important bearing on the etiology of both conditions.

SYMPTOMS.

A chronic gastric ulcer usually presents a group of symptoms so characteristic that the diagnosis can be made upon the history alone. It occurs most often in men, and usually between the ages of 40 and 50, although no period of life is entirely free. The most characteristic feature of the symptoms is their periodicity, for they tend to come in attacks lasting, perhaps, for a few weeks, with longer intervals of complete freedom. The cause of this periodicity is unknown, for in general the ulcer does not heal between the attacks. It has been suggested that during attacks an extension of the ulcer is occurring, and in certain cases small acute ulcers have been noted on the margins of the chronic ulcer itself. As time goes on, however, and the ulcer becomes adherent to the surrounding structures, the interval of freedom becomes shorter, until finally the symptoms may become continuous.

The primary symptom is pain in the epigastrium, coming on at a definite interval after a meal. The interval may be anything from ten to ninety minutes, but it is usually constant for a given patient. The pain may be very severe, and it has the characteristics of a colic varying in intensity from moment to moment. It can be entirely relieved by emptying the stomach or by neutralizing its contents with alkali. The pain appears to be due either

to spasm or tension of the muscle wall of the stomach, the spasm of the muscle wall being in some way due to the presence of the ulcer. The acidity of the gastric contents would seem to play an important part in its production, since it is often most severe when the acidity is at its height and is immediately relieved by the ingestion of alkalis or other substances which neutralize the acid.

The pain is, however, seen in its most typical form in cases of duodenal ulcer, and here its mechanism is more easily understood. Here it comes on an hour and a half after food, when the acidity of the gastric contents is at its height, and it is evidently due to a violent spasm of the pylorus, which endeavours to protect the ulcerated surface of the duodenum from contact with the acid chyme. The relationship between the acidity and the pain is here clearly marked, for although small portions of the liquid stomach contents are being passed into the duodenum as a rule from an early stage, their degree of acidity is insufficient to excite the characteristic spasm.

As time goes on pain of an entirely different character may appear—a fixed burning pain in the back. This pain always indicates that the ulcer has penetrated the posterior wall of the stomach, and has eaten its way into the structures on the posterior abdominal wall, of which the pancreas is the one most often affected. It is apparently due to the direct irritation of nerve endings, and although it varies with the acidity it bears no relation to muscular spasm.

Vomiting is sometimes a prominent feature, and it may be welcomed by the patient for the sake of the relief which it brings; many of these patients, indeed, induce vomiting as the surest way of abolishing their pain. The vomiting may be intensely acid, and this is more likely to be the case if it occurs at a late period of digestion. It never has the persistent character by which it is marked in cases of carcinoma.

Haematemesis is seen in cases of gastric ulcer in two forms. In the one the bleeding occurs from small vessels of the mucosa, and although it may be severe it will always respond to treatment and is never fatal. The other form occurs at a late stage and is due to the erosion of a large artery by the ulcer. Its treatment forms as difficult a problem as any in surgery, and it is often fatal.

The story of a typical case of chronic gastric ulcer will thus be as follows. For many months the patient has suffered from attacks, which lasted for a few weeks, in which he has had epigastric pain, coming on an hour or so after meals, and relieved by vomiting. As time has gone by the attacks have become more severe and the interval of freedom shorter, till for some months he has not known what it was to be free from pain after meals. A constant boring pain in his back may have added to his troubles. He has been losing weight, and on one or two occasions he may have vomited blood.

The symptoms of a duodenal ulcer strongly resemble those of a gastric ulcer, but they present certain important differences. The pain appears at a later period—from one and a half to two hours after a meal. It often awakens the patient in the early hours of the morning when his stomach is actually empty of food, and it is often relieved by taking more food. This hunger pain, as it has been called, is the most characteristic feature of a duodenal ulcer. The periodicity of the attacks is very marked, and although it is not always the case the patient often presents a thin, drawn appearance, which is very characteristic. If haemorrhage occurs it is likely to be very severe, but as a rule blood is passed by the bowel and is not vomited. At the moment when the bleeding occurs the patient may become very pale and faint, but it may be only two days later that he passes the black tarry stool which indicates the presence of altered blood.

In cases both of gastric and duodenal ulceration we are unlikely to gain much information from a direct examination of the patient. Where the history is prolonged and the symptoms have been severe there may be marked loss of weight; where haemorrhage has occurred there may be definite anaemia, and sometimes the patient presents a marked pallor from a continuous loss of small quantities of blood, although no obvious haemorrhage has occurred. Examination of the abdomen may show a definite point of

tenderness in the epigastrium, possibly accompanied by slight rigidity, and where the latter chiefly affects the right rectus it is suggestive of pyloric or duodenal ulceration.

Examination of the stomach contents by means of a test meal will enable us to discover the degree of acidity. There are two standard methods, those of Ewald and Rehfuß. In the former the patient is given, on an empty stomach, a slice of dry toast and a pint of weak tea, the contents of the stomach being withdrawn at the end of an hour. In the second, or fractional method, he first swallows a small tube, and then takes a basin of thin gruel, samples being withdrawn by means of the tube every fifteen minutes. The advantage of the second method is that the gradual rise of the acidity in the stomach contents can be noted, but it is doubtful whether the information obtained is of more practical value than in the simpler method of Ewald. Although the information obtained is not absolutely conclusive, it is certain that a high acidity of the gastric contents is almost unknown in carcinoma of the stomach, and from this point of view it may be of diagnostic value in a doubtful case of suspected ulceration.

Radiographic examination has in recent years become of increasing value, and in expert hands is capable of giving results of great reliability. The patient takes a meal containing three ounces of barium sulphate suspended in some thick medium so as to form a viscous emulsion. The shadow of the stomach contents is observed on a screen, and the contents are displaced by the hands of the radio-

FIG. 1.—Small ulcer of lesser curve; interval of twenty minutes. Note persistence of niche and opposing indentation from spasm of muscle.

grapher so as to see if any of the opaque material remains behind in the crater of an ulcer. Should an ulcer be present some of the barium emulsion will be caught in its crater, and the persistence of this little fleck of barium as the stomach is moved about is the characteristic feature (see Fig. 1). Opposite this small niche there is often an indentation due to spasm of the stomach wall at this point, and the combination of the niche with the indentation is conclusive evidence of the presence of an ulcer. Rapid emptying of the stomach, distortion of its wall, and distortion of the duodenal cap, are all suggestive of ulceration of the stomach or of the duodenum, but their interpretation must be left to the expert.

COMPLICATIONS.

The clinical picture of uncomplicated gastric ulceration is thus exceedingly well defined, but it is liable to considerable modification if certain complications occur. Of these the most important are:

1. Involvement of the surrounding organs, especially the liver and pancreas.
2. Pyloric obstruction.
3. Hour-glass contraction.
4. Carcinoma.

All these complications are accompanied by gross interference with the mechanism of the stomach, and it is a striking fact that they are all associated with loss of periodicity in the symptoms.

Take the first of these cases, where the ulcer penetrates into the liver or pancreas, or, more rarely, into the anterior abdominal wall. Not only will the patient have no periods of relief, but the pain itself, though rendered worse by food, will never disappear. It acquires a burning,

penetrative character, and in those cases where the pancreas is involved it characteristically radiates to the left shoulder. It is evident that such ulcers may open up large arteries, such as the splenic, or pancreatico-duodenal, and lead to immediately fatal haemorrhage.

Where the contraction of an ulcer has led to pyloric stenosis the periodicity of the symptoms will again disappear. The pain will become more constant, but will be due rather to the chronic gastritis which is present than to the ulceration, and it will therefore lose its acute

FIG. II.—Hour-glass stomach. Note small active ulcer, and level of fluid in distal sac.

character. The most striking feature, however, of pyloric stenosis is the vomiting of large amounts of food, possibly the contents of several meals. Often the stomach is enormously dilated, and active fermentation may add to the distension and discomfort of the patient.

Hour-glass contraction is almost limited to women, its production being probably conditioned by some degree of visceroptosis. It occurs most commonly in women of middle age, with a history, extending over ten years or more, of chronic dyspepsia. The pain is always severe, and vomiting may suggest obstruction. As there is, however, a marked degree of chronic gastritis the appetite will be less, and there is always gross loss of weight. The fragility of these women is often extreme. They have suffered years of misery as chronic dyspeptics, and one would imagine that their gastric mucosa could never recover its vitality. There are, however, no cases which may astonish the surgeon, and perhaps still more his medical colleague, with more brilliant recoveries. (See Figs. II and III.)

The relation of carcinoma to chronic ulcers is still a matter of dispute, but there can be little doubt that it is an uncommon occurrence. It should be suspected where the pain becomes more continuous, although usually less severe, where vomiting results in the evacuation of foul material with little relief, and where rapid loss of weight occurs. Gross loss of appetite and a positive distaste for food are common, but again the most striking feature is the loss of periodicity in the symptoms.

TREATMENT.

It was for a long time held that chronic ulcers of the stomach and duodenum would yield to medical treatment, and that complete healing of the ulcer would in time occur. Surgical explorations have, however, shown that this view can no longer be maintained. It is true that if the patient is placed in bed on a restricted diet all his symptoms will disappear, but if an abdominal exploration is undertaken it will often be found that the ulcer itself is still present, apparently entirely unaffected by the treatment which has been so successful in abolishing symptoms.

It would appear, indeed, that a chronic ulcer gives rise of itself to no symptoms at all, and it has been suggested

that the periodic attacks which are so characteristic a feature of the disease are due to the formation of acute ulcers on its margin, probably as a septic process. However that may be, it is certain that a fully formed chronic ulcer can only be cured by surgical means, and it is on the face of it unreasonable to expect that a deeply excavated ulcer, fixed in indurated tissues, and bathed by active gastric juice, should be affected by any alterations in diet.

FIG. III.—Hour-glass stomach, complicated by extensive active ulceration involving pancreas and liver.

Of all surgical procedures for the treatment of these ulcers, gastro-enterostomy stands out as the first, and indeed there are few operations in any field of surgery which have met with such success. Introduced in Billroth's clinic forty-five years ago, and only modified in detail by succeeding surgeons, it can still claim absolute success in an overwhelming majority of properly selected cases.

The mechanism by which it acts is still a matter of doubt, but the most important change which it produces is neutralization of the gastric contents by the bile and pancreatic secretions which are thus brought into the stomach. When this neutralization is obtained gastro-enterostomy is completely successful in dealing with ulcers of the duodenum and of the pylorus, whilst it even succeeds in those ulcers of the lesser curve which lie in the neighbourhood of the pyloric antrum. It succeeds, in fact, wherever an opening can be made to the left of the ulcer, and only fails in those ulcers of the lesser curve which lie so far to the left that this condition cannot be complied with.

In a small percentage of cases, however, for causes which are quite obscure, neutralization is never completely effected, and the patient is then exposed to the possibility of a jejunal ulcer starting from the line of anastomosis. Moreover, gastro-enterostomy fails to give relief when the ulcer is so far towards the cardiac end of the lesser curve that it is impossible to place the opening to the left of it,

whilst its success is uncertain in those cases where the ulcer has involved the liver or pancreas, and its application is often difficult in those cases where the contraction of a chronic ulcer has given rise to the gross deformity of hour-glass contraction.

In the last two conditions, where the surrounding organs are involved or the stomach itself is grossly deformed,

there is no question that the most satisfactory solution is partial gastrectomy. The method of Polya, as so largely developed by Moynihan and Sherren, has met with a success in these complex cases which rivals that of gastro-enterostomy for the simpler conditions. A Y anastomosis should not be performed, for the largest jejunal ulcer we have ever seen occurred after this operation. (See Fig. IV.)

FIG. IV.—Huge jejunal ulcer, following Y gastrectomy. Cured by Polya's operation.

The small ulcers occurring high up on the lesser curve still, however, present an unsettled problem. The patient can be cured, it is true, by partial gastrectomy, but the

FIG. V.—Schoumaker gastrectomy. Result after six months.

large section of stomach sacrificed seems scarcely justifiable. In the ingenious method of Schoumaker the portion of stomach excised can be greatly reduced, and the operation is so performed that a stomach is left which is almost normal in appearance and function. (See Fig. V.) The operation, however, is technically difficult, and an equally good result is more easily obtained if the stump of the

FIG. VI.—Gastrectomy clamp.

stomach is anastomosed to the jejunum, as in Polya's operation.

To facilitate this procedure I have devised a clamp based on that of Schoumaker, but of simpler construction. (Fig. VI.) It consists of two members joined to one another, which can be forced together with great power by a toggle lever. On the front of each member is a

FIG. VII.—Gastrectomy with special clamp. 1, Application of clamp. 2, Stomach excised, front members of clamps removed, crushed edges sutured. 3, Clamp removed, Lembert sutures inserted, forceps applied to stoma. 4, Operation completed. New lesser curve protected by jejunal loop, which is itself protected from angulation.

detachable face. After crushing the stomach, amputating, and removing these faces, a fringe of crushed tissue projects which can be sutured with great facility, and in almost perfect asepsis. The clamp is used as follows.

After dividing the duodenum and freeing the stomach the clamp is so applied as to allow of the removal of the ulcer with a minimum of stomach. The rejected tissue is now cut away, and the fringe of tissue in the clamp is

touched with pure carbolic. The front plates of the clamp are removed, the upper two-thirds of the crushed tissue edge is sutured, and the lower third is caught in forceps. The clamp is removed and the suture line is invaginated. A loop of the jejunum is now brought up to the stomach and attached all along the suture line, whilst an anastomotic opening is formed in the usual way between the jejunum and the portion of the stomach stump held in the forceps. (See Fig. VII.)

The operation has the advantages that it is very tidy and clean, that very little stomach need be removed, that the alignment of the jejunum along the suture line prevents this from leakage, and protects the jejunum from kinking, and that the outlet from the stomach is reduced to dimensions approaching the normal. On the other hand, the dotted line shows how the same method can be applied to the most extensive gastrectomy for carcinoma. The operation is followed by a convalescence remarkable for its smoothness, and so far the after-results have been unexceptionable.

HEREDITARY FAMILIAL CONGENITAL HAEMORRHAGIC NEPHRITIS.

BY

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THE question of heredity as an etiological factor in nephritis has been studied and reported upon from time to time. Fortunately the condition is rare, and the literature shows only half a dozen families affected in this way. As far back as 1875 Dickinson¹ published a family tree showing eleven cases of albuminuria in a total of sixteen persons in three generations. Attlee,² Guthrie,³ and later Hurst,⁴ Pel,⁵ and Kidd,⁶ and Eason, Malcolm Smith, and Buchanan⁷ have also described the disease and noted its familial and hereditary tendency.

The cases which have come to my notice belong to a later generation of the series studied by Leonard Guthrie and Hurst, and it is with this family I propose to deal. Guthrie designated the condition "idiopathic or congenital hereditary family haematuria," and used the word "haematuria" rather than nephritis. He gave as his reasons for this that it is "persistent for many years, but may vary in extent; liable to paroxysmal exacerbations with influenzal-like symptoms; not marked by oedema, ascites, or cardio-vascular disturbances; not due to uric acid or oxalates, and is not accompanied by urine of low specific gravity."

The part of the same family described by Hurst showed definite nephritis with low specific gravity of urine and the presence of albumin, some blood, and granular and hyaline casts; the blood pressure was increased, and towards the end anaemia, cardio-vascular changes, and oedema were marked features; these cases terminated in death from uraemia. Hurst thus named the condition "hereditary familial congenital haemorrhagic nephritis," and this terminology is accurate for most of the affected members of the later generations of the family.

Genealogical Tree (Hurst).

The genealogical tree is taken from Hurst's study of the family records, with the addition of the children of the third generation, born since his original communication appeared.