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CARCINOMA OF THE STOMACH*

BY

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(WITH SPECIAL PLATE)

I need not apologize for discussing the problems of cancer of the stomach, for the stomach is by far the commonest seat of cancer in the body. It is true that in the female population the incidence of cancer of the breast is slightly greater; but taking males and females together, in 1934, the last year for which official figures are available, there were in England and Wales 12,269 deaths from cancer of the stomach, as against 6,687 from cancer of the breast. This figure is certainly an understatement, since there were some 2,400 deaths notified as from cancer of the liver in the same year, and a large proportion of those must have arisen from a latent primary growth in the stomach. In the 12,269 deaths from cancer of the stomach approximately 6,600 were in men and 5,600 in women—a discrepancy between the incidence in the sexes less marked than is usually supposed. One-third of all cancers in men and one-fifth of those in women are in the stomach.

The problem of aetiology remains as baffling in cancer of the stomach as in the wider field of cancer in general. One thinks of the bright hopes engendered by the discovery of Fibiger in 1913 that a form of cancer of the stomach in rats was caused by a nematode worm. This worm infests the cockroach in Copenhagen, and rats fed on a diet of infected cockroaches developed first a gastritis, then a polyposis, and finally a cancer of the stomach, capable of giving rise to distant metastases. Although the worms were found in the primary gastric neoplasm they were absent from its metastases. These early experiments, though they aroused a keen interest in the parasitic theory of cancer, would seem to have no bearing on the aetiology of cancer of the stomach in man.

Nor can it be said that the more recent work of Gye on the filterable virus of the Rous chicken sarcoma has yet solved the riddle of malignant growths in man, though the solution may well be found on the lines indicated by Gye. On the causation of the commoner types of cancer in man we must still admit a large degree of ignorance. On certain occupational cancers of the skin and the urinary tract some light is beginning to dawn, and much knowledge has been gleaned concerning the experimental tar cancer of animals, but when we come to consider cancer of the stomach and intestinal tract, the breast, and the uterus the darkness is still profound, and we can only hope that it precedes the dawn.

* A lecture delivered to the Birmingham Branch of the British Medical Association, March 18, 1937.

Predisposing Factors

While we admit our fundamental ignorance as to causation, we must consider briefly what we know of the predisposing factors in cancer of the stomach. Though it occurs rarely under the age of 40, it is in exceptional cases seen in the early twenties, and the youngest patient I have had was a girl of 22. As in other organs, it holds in the stomach that the younger the patient the more virulent is the malignancy. In sex incidence the males preponderate, but only in the proportion of six to five.

The factor of heredity is more difficult. The work of Maud Slye (1927) has established belief in a hereditary factor in the cancer of mice, and there is a growing tendency to believe that heredity may have a place in the aetiology of human cancers. But it is not proved, and is certainly not the most important factor.

The Relation of Ulcer to Cancer

Of all the predisposing factors in the causation of cancer of the stomach the tendency of chronic gastric ulcers to undergo a malignant transformation is the one on which opinion has fluctuated most widely, and controversy on this point has been most keen. That we should know the truth about this relation is of obvious importance, for the conclusion to which we come must colour our whole attitude, both to the treatment of chronic gastric ulcers and to the prophylaxis of cancer of the stomach.

In 1909 Wilson and MacCarty of the Mayo Clinic startled the medical world by claiming that 68 per cent. of apparently simple gastric ulcers showed early malignant changes on microscopical examination, and that 71 per cent. of carcinomas of the stomach provided histological evidence of a previous simple ulcer. This view was severely criticized by many, both from the pathological and the clinical point of view. Spilsbury (1921–2) pointed out that there are commonly found at the edge of a gastric ulcer gland cells which have penetrated deeply into the scar tissue and are cut off from the regenerating glands. It was these cells, detached from the regenerating epithelium and buried in the fibrous tissue of the ulcer, which were sometimes described as precancerous, or even as the malignant transformation of an ulcer. He regarded this as the error which had led the Mayo Clinic pathologists to the conclusion that 68 per cent. of the peptic ulcers removed at operation showed microscopical evidence of

malignancy, and set forth his conclusion that malignant transformation of a peptic ulcer is an uncommon event. Dible (1925), in a series of 126 resected gastric ulcers with no clinical suspicion of malignancy, found no microscopical evidence of cancer. In cases in which a clinical suspicion of malignancy was entertained 4 per cent. showed carcinoma. Stewart (1931) found an incidence of 6.1 per cent. of carcinoma in a series of 277 chronic ulcers removed at operation, and in 109 operation specimens of carcinoma of the stomach he found 15.6 per cent. showing evidence of having originated in a pre-existing simple ulcer. Newcomb (1932) in another careful pathological investigation found that 3.75 per cent. of the gastric ulcers showed malignant change, and that 13 per cent. of cancers gave evidence of previous peptic ulceration. I would remark that it is much more difficult to be sure on histological grounds that a cancer has been preceded by an ulcer than that an ulcer is showing an early cancerous change.

There are, however, considerations other than the histological evidence to be taken into account, and not the least important is the marked difference in the prevailing sites of ulcer and of cancer. I have reduced Stewart's statistics on this point, which are based on a large number of cases, to the form of diagrams (Figs. A and B). You

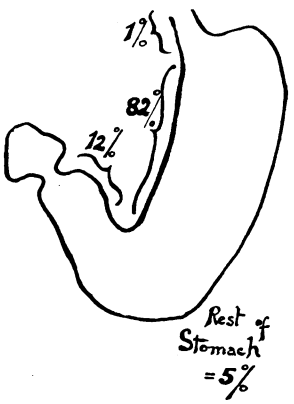


FIG. A.

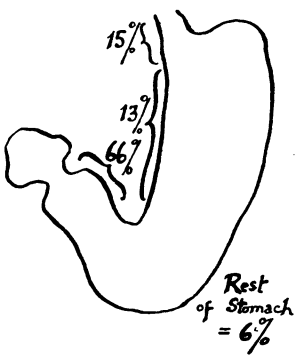


FIG. B.

will see that whereas in chronic ulcer 82 per cent. occur on the middle two-thirds of the lesser curvature and only 12 per cent. in the prepyloric region, in cancer 66 per cent. are found in the prepyloric region, and only 13 per cent. on the portion of the lesser curvature that is the favourite site of ulcers. These figures speak most eloquently against any close causal connexion between ulcer and cancer.

Another important piece of evidence on this point is found in the hour-glass stomach. 90 per cent. of these occur in females, and they result from lesser-curve ulcers of extreme chronicity, with an ulcer history commonly extending back some twenty or thirty years. Surely if the irritation of gastric ulcer commonly gave rise to cancer, it is in these hour-glass ulcers that the incidence of cancer should be the highest. Yet we find that such a change is of extreme rarity. Thurstan Holland (1921) in a radiological and clinical investigation of 128 cases of hour-glass stomach (120 females and eight males) found that only one female and two males proved to have cancer. To suggest, as Sir James Walton (1936) does, that the ulcers giving rise to hour-glass contraction are different from ordinary gastric ulcers seems to me unwarranted. There is no histological basis for the suggestion, and the preponderance of female cases is simply due to the different shape of the long narrow J-shaped stomach in the female, in contrast with the shorter and broader stomach of the male.

Again, the clinical evidence derived from a study of the duration of gastric symptoms provides a powerful argument against a close connexion between the two lesions. I pointed out some years ago (1923) that in fifty-four cases of proved cancer of the stomach the average duration of gastric symptoms before examination was 12.7 *months*, whereas in forty-five cases of proved simple chronic ulcer the average duration of symptoms was 10.3 *years*.

I should be the last to deny the possibility of a carcinoma originating in an ulcer, but I hold that the malignant transformation of ulcers is less common than many authorities would have us believe. It sometimes happens that a carcinoma originates in the same stomach as an ulcer at some point remote from the ulcer. In one of my cases, that of a woman with fifteen years' history of ulcer and six months' symptoms of pyloric obstruction, I found an early annular carcinoma of the pylorus, and an inch away from it, and separated from it by healthy mucosa, the crater of a chronic ulcer. Had that stomach been removed a little later, when the malignant growth had encroached upon the ulcer crater, one would have been tempted to believe that the cancer had started in the ulcer. I have seen three cases of old duodenal ulcer in which, some years after gastro-enterostomy for the ulcer, cancer developed independently in the body of the stomach. Had these ulcers not been proved to be duodenal by the previous operation they would probably have been held to be the precursors of the malignant growths.

It is probable that many of the cases diagnosed on clinical history as ulcer-cancer are really cases of primary cancer of unusually slow growth. We are familiar with the very slowly progressive atrophic scirrhus in the breast. It is not uncommon to see them after four or five years of growth, and even longer, still without widespread or obvious secondaries, and comparatively small in size. It is not sufficiently realized that while most cancers of the stomach run a course of under two years from the first symptom to the fatal end, yet there is a type of cancer, not very uncommon, which may arise in any part of the stomach and is so slow in growth that the total duration of the symptoms may be three, four, or even five years from start to finish. This type of growth may simulate simple ulcer clinically to a remarkable degree, and yet be a cancer from the beginning. Beyond doubt, in my view, these slow-growing ulcer-simulating cancers have unduly swollen many surgeons' lists of cases of ulcer-cancer.

The Relation of Gastritis to Cancer

But if I have convinced you that the danger of malignant degeneration of a simple gastric ulcer is to a great extent a bogy, we have not yet exhausted the possible predisposing causes of gastric cancer. In 1929 A. F. Hurst in his Schorstein Lecture lent all the weight of his great authority on this subject to the view that diffuse atrophic gastritis associated with achlorhydria is an almost universal precursor of cancer of the stomach, and that the chief hope we have for the prophylaxis of this dread disease lies in the recognition and treatment of the gastritis. He holds that "the achlorhydria so commonly associated with cancer of the stomach is a result of chronic gastritis which precedes the onset of the carcinoma and is in fact the most common predisposing condition." He says, "I have never seen a case of carcinoma in which free hydrochloric acid was present at an early stage and disappeared as the disease advanced." When a physician of Hurst's great experience of gastric disease makes such a categorical statement we

are tempted to infer that such cases never occur. Hurst says further: "I only know the history of two cases in which a test meal had been given before as well as after the development of carcinoma of the stomach associated with achlorhydria, and in both achlorhydria was already present on the first occasion." In opposition to Hurst's contention that achlorhydria always precedes a primary cancer of the stomach, Robertson (1935) has reported two cases of cancer of the stomach in which considerable free hydrochloric acid was found in the earlier test meals, while succeeding tests showed a diminution of the acidity parallel with the progress of the cancer, until finally complete achlorhydria was found. I reproduce one of Robertson's charts (Fig. C). Robertson quotes other similar cases from the literature.

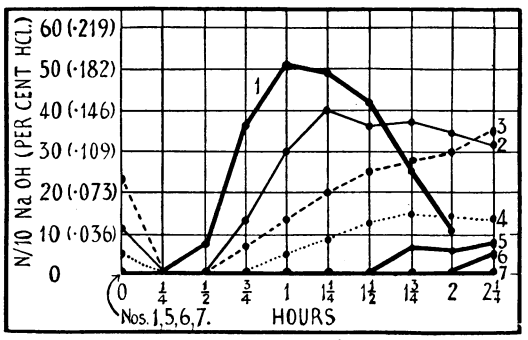


FIG. C.—Chart of Dr. Robertson's case. Curves of the secretion of free hydrochloric acid from November (No. 1) to the following August (No. 7).

Dr. J. F. Wilkinson has kindly given me details of a case of proved cancer of the pyloric end of the stomach at which two test meals at an interval of four months showed a similar change from a low normal acidity to complete achlorhydria (Fig. D). He has also provided

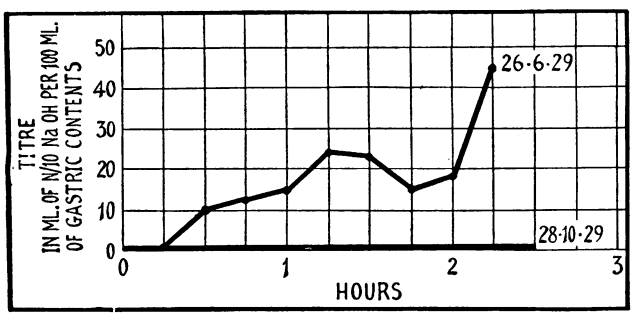


FIG. D.—Chart of Dr. Wilkinson's case.

me with test-meal charts from fourteen other cases of proved cancer of the stomach, all of them, judging by the short history, primary cancers. Eight of these charts showed normal acid curves and six hypochlorhydria, but not achlorhydria.

We know that free hydrochloric acid is present in the gastric juice of about 30 per cent. of cases of cancer of the stomach. Hurst regards this group of patients as suffering, not from primary cancers, but cancers originating in ulcer. This view, however, involves a belief in a frequency of ulcer-cancer far greater, for reasons I have already given, than is warranted by the clinical and pathological data available. He takes a history of over eighteen months' gastric symptoms as a sufficient criterion of ulcer-cancer. I hold that this is far too short, and that many primary cancers of the stomach run a much longer course than eighteen months.

One difficulty in considering the validity of Hurst's theory lies in the impossibility of recognizing the atrophic gastritis without a fractional test meal. "Chronic gastritis, with complete achlorhydria," states Hurst, "is often completely latent; probably the majority of cases give rise to no symptoms of any kind." As patients are rarely submitted to a test meal without previous symptoms, and post-mortem changes in the stomach are a great obstacle to histological studies, it is difficult in most clinics to find first-hand evidence as to the frequency of atrophic gastritis, and we must fall back on the few workers who have carried out extensive researches on this matter. Faber (1900), to whom in great part we owe the conception of chronic gastritis as a pathological entity, made a great advance in the study of this problem when he introduced the method of injecting 10 per cent. formalin into the stomach immediately after death in order to fix the mucosa for histological study and so obviate the difficulty of post-mortem changes. Freshly removed gastrectomy specimens have also been studied extensively, though in these there is usually (and should be always) a gross lesion such as an ulcer or cancer already well established, and opinions differ widely whether the associated gastritis is a primary lesion or a secondary effect.

The fractional test meal augmented by the injection of histamine, that powerful stimulant of the secretion of hydrochloric acid, has also been used in the study of achlorhydria. By this means Faber (1927) has classified achylia as follows: (1) False achylia with a scant and tardy secretion of hydrochloric acid. (2) True total anacidity, but with preserved pepsin secretion and chloride elimination. (3) True anacidity with more or less impairment of pepsin and chloride production. (Faber never found complete absence of pepsin formation.) Faber states that there may be complete anacidity for a long time, though the glands of the mucosa are still intact and apparently capable of being restored to function, but he finds various stages from diffuse round-cell infiltration of the mucosa with intact glands to complete atrophy of the mucosa, with destruction of the entire secretory apparatus, though this last is a rare event. He gives us valuable information as to the great frequency of true achylia, a frequency increasing with age (Table I). Of 1,000 consecutive cases admitted to hospital there were no fewer than 306 with true achylia, and you will note how the percentage increases with each decade.

TABLE I.—*Incidence of True Achylia (Faber)*

Age	Patients Admitted	Achylia	Percentage with Achylia
0–20	102	7	6.9
20–29	224	28	12.5
30–39	221	51	23
40–49	190	81	42.6
50–59	182	95	52
60–69	78	40	51
70 and over...	5	4	80

Faber considers that achylia is not a constitutional and hereditary condition, but an acquired condition due either to some remote infective cause, possibly in childhood, or to the irritation of food or drink, and notably alcoholic drinks. He contrasts this diffuse atrophic gastritis with a form of gastritis localized to the prepyloric region and associated with normal or excessive acidity, which he regards as the main predisposing cause of gastric and duodenal ulcers. It must be noted that Faber himself made no suggestion that he found any evidence that atrophic gastritis with achylia is a predisposing cause of cancer of the stomach.

Bloomfield and Polland combined the investigations of a number of observers who used the histamine test, and

found that in a total of 5,204 people the incidence of anacidity was as follows:

TABLE II

Age	Number of Patients	Percentage of Achylia
20–29	866	5.3
30–39	1,354	9.5
40–49	1,299	16.7
50–59	1,043	24.0
60 and over	642	35.4

It is noteworthy that achlorhydria is more frequent in women than in men in the ratio of 5:4 or 4:3, whereas the ratio of cancer of the stomach is six males to three females. In Polland's series there were 17.4 per cent. of females with anacidity, as against 12.9 per cent. of males.

It would appear from these tables that among the general population of the usual cancer age some 20 to 50 per cent. of individuals have true achlorhydria without necessarily showing any gastric symptoms. While there is good pathological evidence for the view that this achlorhydria may result from a clinically unrecognizable atrophic gastritis, I can see no convincing evidence in favour of Hurst's theory that the gastritis is a common predisposing cause of cancer. It is a theory of great interest, and it demands careful criticism in the light of all the clinical and pathological material that we can study, but the evidence against it is considerable and that in favour of it as yet slight.

Classification of Cancer of the Stomach

The usual classification of cancers of the stomach is based on their gross pathological features, and we describe fungating, ulcerating, colloid, and leather-bottle cancers. Unfortunately, there is a good deal of overlapping between these types, and the classification is of more interest to the morbid anatomist than to the clinician. I prefer to take a simple anatomical basis for our classification, and to divide them, according to the part of the stomach affected, into: (1) cancer of the cardiac end; (2) cancer of the body of the stomach; and (3) pyloric and prepyloric carcinoma.

SYMPTOMS

The first symptoms to be noticed in the great majority of cases are a vague discomfort after meals and an unaccountable failure of appetite. The patient is usually of the cancer age, and has never suffered from indigestion before. With the failure of appetite there is a steady loss of weight, and as a rule a progressive development of anaemia.

We may now consider certain features that depend upon the site of the cancer.

1. *Cancer of the Cardiac End.*—In this type there is commonly, but not always, an early onset of dysphagia, due to stenosis of the cardiac orifice. The condition can only be diagnosed from carcinoma of the lower end of the oesophagus by radiological examination, which shows a filling defect extending into the upper part of the stomach.

2. *Cancer of the Body of the Stomach.*—In this position any considerable degree of obstruction is most unusual, and consequently the symptoms are vague. In some cases we find no specifically gastric symptoms beyond an unaccountable loss of appetite. In others there is a feeling of flatulent distension after meals, and the patient complains of offensive eructation of gas. Pain is, however, slight or absent in the early stages of the disease, and the most noticeable features are a suspicious pallor of the face, some shortness of breath on exertion, due to a rapidly developing anaemia of the secondary type, and progressive loss of weight. Vomiting is unusual and only occasional, owing to the absence of obstruction.

3. *Pyloric Cancer.*—This is both the commonest type and the most hopeful. It is hopeful because obstruction often arises early, and because the growth is accessible for surgical removal. Pain is the most marked feature when pyloric stenosis is present, and it may closely simulate that of ulcer. It often begins an hour or so after meals; it may occur in periodic attacks lasting a few days, with some days of freedom between attacks, though these remissions soon disappear, and are seldom as long as in cases of ulcer. Relief is obtained by vomiting, which may be self-induced, and articles of diet taken the day before may be recognized in the vomited material. If the patient is examined after a meal we find splashing in a large dilated stomach, and may see waves of visible peristalsis rolling across the stomach from left to right.

A mobile palpable tumour with very little tenderness may be detected early or late according to the thickness of the abdominal wall and the position of the growth. While a tumour is as a rule a late sign, it is certainly not true to say, as some do, that by the time the tumour can be detected the growth is inoperable.

Late Signs in Cancer of the Stomach

These consist in the detection of outlying secondary deposits in the liver or peritoneum, the umbilical scar, the lymph glands of the left supraclavicular fossa, or the ovaries. The pouch of Douglas should be examined in both sexes for hard secondary nodules, and in the female pelvic examination may reveal the large secondary ovarian masses known as Krukenberg tumours. When the peritoneum is widely invaded we find, in addition to secondary masses in the omentum, a more or less considerable ascites. Massive secondary deposits in the liver soon give rise to some degree of obstructive jaundice, and are often associated with a slight irregular pyrexia and a moderate leucocytosis.

Differential Diagnosis

(*a*) *From Pernicious Anaemia.*—The non-obstructive type of cancer of the body gives rise to symptoms closely simulating those of pernicious anaemia. There is the same lemon-yellow pallor, shortness of breath, and loss of energy. In pernicious anaemia, however, there are rarely such pronounced feelings of epigastric discomfort after food, and hardly ever is there the rapid loss of weight that occurs in cancer. Yet cases frequently occur in which the diagnosis can only be settled by *x*-ray and blood examinations.

(*b*) *From Simple Ulcer.*—We have seen how closely cancer may simulate simple ulcer, particularly when it is slow-growing and at the pyloric end. If an *x*-ray shows that the lesion is on the duodenal side of the pyloric ring we can dismiss the fear of cancer, for cancer hardly ever occurs in the first part of the duodenum, and gastric cancers never spread over the pyloric barrier into the duodenum. The differentiation of cancer from simple gastric ulcer is, however, much more difficult. In view of the site of election of the two conditions, an ulcer in the prepyloric region must be regarded with considerable suspicion, and if after two or three weeks of strict medical treatment occult blood is still found in the faeces it is not wise to delay surgical measures. Even on the part of the lesser curvature specially liable to simple ulceration the *x*-ray appearances may be most misleading, as in the following case.

A lady, aged 60, consulted her doctor with vague indigestion pains of very short duration and some loss of weight. She had never suffered from indigestion before. A radiograph

showed a penetrating ulcer on the middle of the lesser curvature (Fig. 1, Special Plate). The picture was typical of simple gastric ulcer, and the usual medical treatment for that condition was advised. Six weeks later she was rather better and had put on a few pounds in weight. A second radiograph taken now showed the ulcer crater nearly filled up (Plate, Fig. 2). After a further four months she was again losing weight and feeling rather more discomfort in spite of medical treatment. A third *x*-ray taken some six months from the onset of symptoms showed a definite carcinoma at the site of the original ulcer crater (Plate, Fig. 3). I explored the abdomen, but found early secondary deposits scattered through the peritoneum, so that gastrectomy was out of the question.

It is quite plain that, in spite of the deceptive *x*-ray appearance at first, this was a carcinoma from the beginning, and it affords eloquent testimony to the fact that the clinical history is more important than the radiological signs. I do not wish to belittle the value of an *x*-ray examination. It is by far the most important diagnostic weapon in our hands, and it is imperative, if cancer of the stomach is to be diagnosed earlier, that every patient over 40 who complains of persistent indigestion for the first time in his life should be examined by *x* rays. But cases do occur, not uncommonly, in which the most skilled radiologist cannot be sure whether the ulcer is simple or malignant, and it is in these that the clinical history is all-important.

Whether the use of the flexible gastroscope will prove of great value in the differentiation between simple and malignant ulcers is doubtful. Sometimes we meet with ulcer in which a careful naked-eye examination after gastrectomy does not enable us to make a positive diagnosis, and we have to wait for the result of a microscopical section. I conceive that the gastroscope will never be of great use in this not uncommon type, however valuable it may prove in the more usual cases that present no difficulty in radiological diagnosis.

Treatment

Some form of gastrectomy is, of course, the only treatment that offers any hope of cure. Fixation of the growth to the transverse colon or mesocolon does not necessarily rule out gastrectomy. Fig. 4 (Plate) shows a stomach removed by gastrectomy in which the greater part of the transverse colon had to be removed as well. The patient made a good recovery and returned to work, though the growth recurred later.

Where a cure is plainly impossible a palliative gastro-enterostomy is only of value if there is definite obstruction at the pylorus with either vomiting or delayed emptying of the stomach shown by the radiograph. If there is no such obstruction a case that is inoperable by reason of secondaries is better left alone. In cancer of the cardiac end radical operation is hardly ever feasible, and a palliative gastrostomy has rarely in my experience afforded much relief.

I can best illustrate the question of treatment by summarizing a series of 207 cases under my care (Table III).

TABLE III.—*Showing Surgical Procedure in 207 Cases of Cancer of the Stomach*

Gastrectomy	58
Palliative operations	60
Inoperable but explored	37
Inoperable	52
	207

Gastrectomy was performed in fifty-eight cases, with an operative mortality of fourteen, or 24.1 per cent. Most of them were done by the Schoemaker method, for which I have an increasing preference, since it is possible to remove at least as much of the lesser curvature by this operation as by the more usual Polya gastrectomy. The high mortality is in striking contrast with that of gastrectomy for simple gastric ulcer. It is due in part to the poor condition of these patients by the time they come to operation, and in part to the fact that one feels it a duty to attempt the one hopeful procedure wherever it is reasonably possible, in spite of the risk involved.

Palliative operations, chiefly gastro-enterostomy, were done in sixty, thirty-seven were explored but found to be inoperable, and in fifty-two operation was contraindicated, as radical measures were plainly impossible and there was either no obstruction to relieve or no reasonable hope of relieving it.

In the palliative medical treatment I would stress the importance of treating the anaemia. Many of these patients have a profound secondary anaemia, and can only be made fit for gastrectomy by a short course of iron in massive doses. This may be given as ferrous chloride or 30- or 40-grain doses of iron and ammonium citrate. A fortnight devoted to this treatment before the operation will often bring about a remarkable improvement in the blood picture, and transform an almost prohibitive risk into a reasonable one. Even in the hopeless cases it is often possible to mitigate the distressing effects of anaemia in this way, and it should never be neglected.

As to the end-results of gastrectomy, of forty-four patients who recovered after the operation one is alive and well after thirteen and a half years; one after eleven years; one after nine years; one after eight years; two after seven years; two after three years; and six under two years, making a total of fourteen still surviving. Twenty-eight have died from recurrence, usually within eighteen months, and two are untraced. These results are not materially different from those of other surgeons. Partial gastrectomy will only cure a relatively small proportion of our cases, but in the present state of our knowledge it is the one hopeful method we have. The results can be improved only by earlier diagnosis, and the main purpose of these remarks is to insist that all our efforts should be directed to recognizing the condition at the earliest possible stage, and acting with equal promptitude.

REFERENCES

Dible, J. H. (1925). *Brit. J. Surg.*, **12**, 666.
Faber, K., and Bloch, C. E. (1900). *Z. klin. Med.*, **70**, 98.
—— (1927). *Lancet*, **2**, 901.
Holland, C. Thurstan (1921). *British Medical Journal*, **1**, 6.
Hurst, A. F. (1929). *Lancet*, **2**, 1023.
Morley, J. (1923). Ibid., **2** 823.
Newcomb, W. D. (1932). *Brit. J. Surg.*, **20**, 279.
Robertson, J. D. (1935). *Lancet*, **1**, 1495.
Slye, M. (1927). *J. Cancer Res.*, **11**, 135.
Spilsbury, B. (1921–2). *Proc. roy. Soc. Med.*, **15** (Sect. Med.), 6.
Stewart, M. J. (1931). *Lancet*, **2**, 565.
Walton, Sir J. (1936). Ibid., **1**, 1101.
Wilson, L. B., and MacCarty, W. C. (1909). *Amer. J. med. Sci.*, **138**, 846.

From dissections of 100 cases, B. Chlyvitch and A. Kozintzev (*Ann. Anat. path. méd.-chir.*, June, 1937) conclude that the so-called intrapancreatic portion of the common bile duct lies really in a canal formed by a fold in the gland which can easily be opened without damaging the gland substance by blunt dissection along a cleavage plane of connective tissue, which is indicated by a fine groove on the posterior surface of the head of the pancreas. This method of approach, besides avoiding damage to the pancreas, also exposes the distal part of the duct of Wirsung and the vessels crossing the intrapancreatic part of the common bile duct.

JOHN MORLEY: CARCINOMA OF THE STOMACH

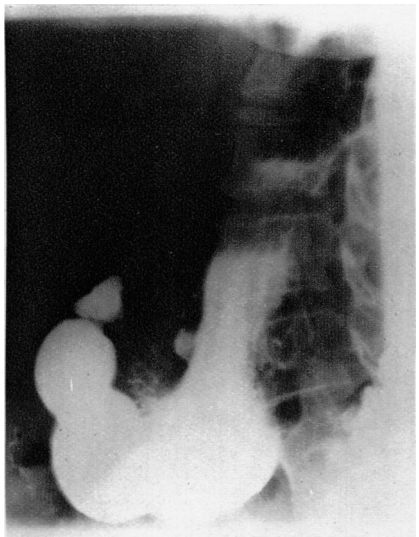


FIG. 1.—Radiograph showing penetrating ulcer on middle of lesser curvature.

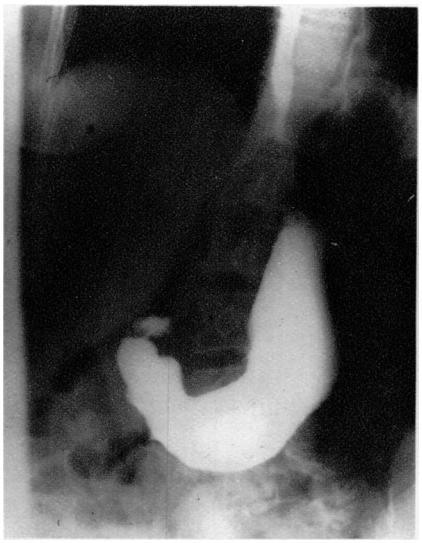


FIG. 2.—Radiograph taken six weeks later, showing the ulcer crater nearly filled up.

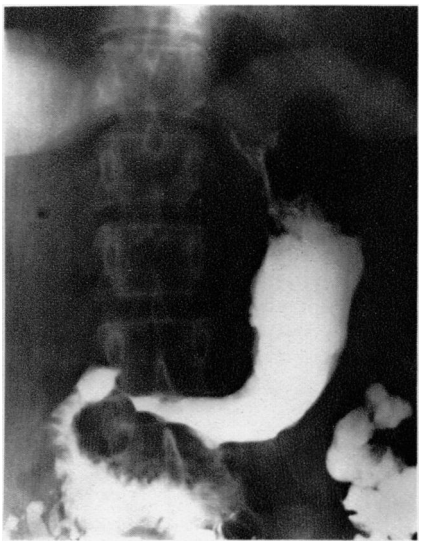


FIG. 3.—Radiograph taken six months from onset of symptoms, showing a definite carcinoma at the site of the original ulcer crater.

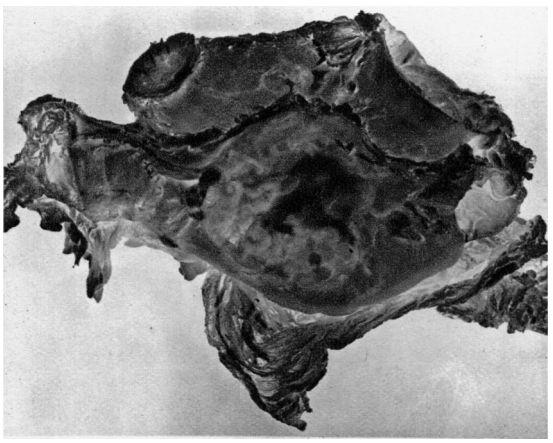
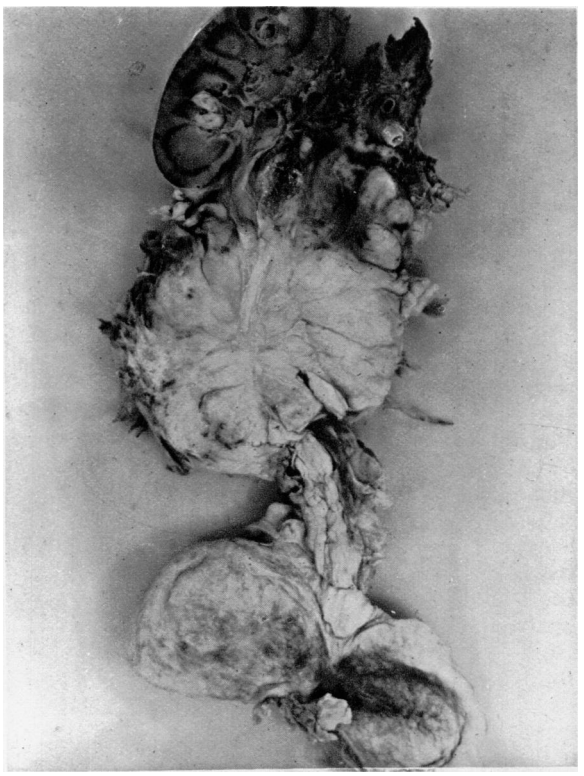


FIG. 4.—Showing a stomach removed by gastrectomy in which the greater part of the transverse colon had also to be removed.

ALEXANDER LYALL: CARCINOMA OF URETER



Tumour of the ureter, showing radiating appearance and also growth of tumour in a downward direction along the ureter.