

IV.—*Region affected.*

Cervical	10	Dorso-lumbar	8
Cervico-dorsal	2	Lumbar	18
Upper ,,	7	3 foci present, 1st D., 9th D.,	
Middle ,,	26	1st L.	1
Lower ,,	29	Not noted	5

V. *Cases with other tubercular affections—14.*

Hip disease (right, 2; left, 4) ...	6	Lupus of face	2
Knee	1	Peritonitis	1
Ankle (double, also had phthisis)	1	Pleurisy (several attacks) ...	1
Glands of neck (caseating) ...	1	Phthisis (one case also had tubercular ankles)	2

VI. *Scoliosis* was present in addition to the angular kyphosis in 4 cases.

VII. *Abscess*.—Present at some time or other in 40 cases (34 per cent.). It must, however, be noted that in 39 of these the abscess was large enough to be observed on palpation or inspection, and that in only one was the abscess detected by X rays (Case 72). It is highly probable that some of the remaining 76 cases suffered from abscess, which, however, never became large enough to be detected by ordinary methods.

These 40 cases may be divided into four groups:—A, Abscess opened before coming to Department—27 cases. B, Patient arrived with abscess present but unopened—10 cases. C, Abscess developed during treatment in Department—1 case. D, No abscess present; case not treated because patient only attended Department once; abscess appeared later—2 cases. Dealing with these four groups in detail:—

Group A.—Of 27 cases in which the abscess had been opened before coming to Department, on arrival 19 had healed and 8 were discharging. Of the 19 which had healed—

14 developed no further abscess while under treatment.

1 developed a fresh abscess a year later while under treatment, which opened and eventually healed (treated at first by jacket) (Case 22).

1 at first not treated; then fresh abscess; died three years later from dropsy (Case 65).

2 were referred to other Departments.

1 died five days later from pneumonia.

Of the 8 which were discharging—

2 healed up.

3 still discharging after 2, 2½, and 3 years respectively.

1 discharged for 3½ years, and died in home for incurables.

1 referred to another Department.

1 not known.

Group B.—Of 10 cases in which patient arrived with abscess present but unopened, in—

5, the abscess disappeared without aspiration or incision, after intervals of 9, 9, 9, not stated, and 33 months respectively.

2, abscess disappeared after aspiration 1 and 4 times respectively.

1, two abscesses present; after two years both incised; 3 months later one healed and one still discharging.

1, "acute abscess," incised, sewn up, broke down, and had healed in 2½ years (no particulars, Case 37).

1, abscess opened and continued discharging until the patient ceased attending, 6 months later (Case 32).

Group C.—One case in which abscess developed during treatment in Department (Case 2). Treated three years by jacket; then developed abscess; treated by two aspirations and Thomas splint 2½ years; abscess gone after 1½ years; has worn jacket last 1½ years.

Group D.—Of 2 cases in which no abscess present; case not treated because only one attendance; abscess appeared later, in—

1 (Case 53), abscess appeared 20 months later.

1 (Case 40), abscess appeared some years later.

VIII. *Compression paraplegia*.—Signs of this were present at some time or other in 27 cases (23 per cent.); in 9 of these abscesses also present.

These 27 cases may be divided into 3 groups: A, Recovered from before attending Department—9 cases. B, Suffering from when attended Department—14 cases. C, Suffered from for first time during treatment in Department—4 cases. Dealing with these groups in detail:—

Group A.—Of 9 cases in which symptoms were recovered from before attending Department,

8 still well at periods varying from 2–6 years (3 of these treated by Thomas splint).

1 referred to another Department and known to be well now.

Group B.—Of 14 cases which were suffering from symptoms when attended Department—

6 treated by Thomas splint; symptoms completely cured.

1 treated by jacket and steels; symptoms completely cured.

5, result not known, because patient ceased attending.

1 not treated, because patient only attended once.

1 transferred to another Department and known to have died from bronchitis.

Group C.—Of 4 cases which suffered from symptoms for first time during treatment in Department—

3 not at first treated by Thomas splint, but by jacket.

1 treated 2½ years by Thomas splint, then 4 months by jacket, when symptoms appeared.

In other words, not one of the four developed paraplegia whilst in the recumbent position. The latter and 2 of the former are cured, and of one no particulars are known.

IX. *Treatment*.—Of the 116 cases, 100 were treated in the Department. The apparatus employed was as follows:—

41 cases treated by Thomas splint.

20 " " jacket.

36 " " jacket and head support.

1 case " " poroplastic case for head and neck.

2 cases " " "kyphotic support."

Note.—Of the 40 cases treated by Thomas splint, 17 were treated either before or after with jacket.

Previous treatment.—Of the 100 cases, 57 had received treatment previously.

IS GASTRIC ULCER A FREQUENT PRECURSOR OF CANCER?

By HERBERT J. PATERSON, M.A., M.C.,
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OF late years there has been considerable discussion as to the frequency with which cancer is grafted on simple ulcer of the stomach. Cruveilhier, in 1839, and Dittrich, in 1848, were the first to point out this possibility.

Zenker, in 1882, went further and put forward the hypothesis that *all* cases of gastric carcinoma have their origin in simple ulcer. More recently Dr. W. J. Mayo and Dr. Christopher Graham have laid great stress on the origin of cancer in ulcer, and Dr. L. B. Wilson and Dr. W. C. MacCarty from the same clinic, have contributed a valuable paper dealing with the problem from a pathological standpoint. These writers state that they have been able to demonstrate a pre-cancerous or non-malignant ulcer in 62 per cent. of their cases. Most observers, however, have placed the proportion at a much lower figure. Thus, according to Sonicksen, 14 per cent. of all cases of cancer of the stomach originate in ulcers; Lebert estimated the frequency at 9 per cent.; Rosenheim at 6 per cent.; Fenwick and Haberlin at 3 per cent.

The possibility that carcinoma may originate in an ulcer cannot be denied; it is, however, very doubtful whether such an occurrence is as frequent as some authorities would have us believe. The problem is a very difficult one, and a dogmatic conclusion impossible, but the evidence available in favour of the view that simple ulcer is a frequent precursor of cancer is not convincing. This evidence is both clinical and pathological.

(a) *Clinical evidence*.—A number of the patients who come under observation with gastric carcinoma give a history of gastric trouble extending over many years. The first step in the argument is the assumption that the gastric symptoms are due to gastric ulcer. When subsequently, on the operation table or in the post-mortem room, the existence of cancer is confirmed, the argument is carried a stage further by assuming that cancer has originated in the ulcer, which from the history is supposed to have existed previously. The argument may be summarised briefly thus:—Cancer is a disease the duration of which is comparatively short. A long history of gastric trouble is evidence of the presence of an ulcer; therefore, when cancer is associated with a long history, the cancer must have been grafted on a simple ulcer. Such an argument is unscientific. It contains two fallacies, and disregards an alternative conclusion at least of equal probability.

The first fallacy is the assumption that we can diagnose a gastric ulcer from the clinical history alone. In duodenal ulcer, it is true, the history often is so typical that from it a diagnosis may be made with confidence. This is not so with gastric ulcer. Usually, the diagnosis of duodenal ulcer is easy, that of gastric ulcer very difficult. From operative experience we have learnt that gastric ulcer is far less common than formerly was supposed. The symptoms attributed to gastric ulcer frequently are due to disease of the appendix or of the gall-bladder, to septic gastritis, or to intestinal toxæmia. Although in some instances, when dealing with a particularly intelligent patient, it may be possible from the history alone to differentiate between these conditions, in the majority of cases it is not so. I maintain, therefore, that because a patient with cancer of the stomach gives a long history of dyspeptic trouble we are not justified in assuming that the symptoms have been due to a gastric ulcer.

The second fallacy is the assumption that the duration of carcinoma is never protracted. It is conceivable that the growth of cancer in its early stages is much slower than we think. At present we have no means of determining the life-history of cancer of the stomach. We do know, however, that in some instances it is of slow growth, and that dissemination is a late manifestation. Most surgeons have met with instances in which, after gastro-jejunostomy for inoperable cancer of the stomach, the patient has lived for years before succumbing to the disease.

Some years ago I performed gastro-jejunostomy on a woman, aged 27 years, who had an extensive pyloric cancer

with such widespread glandular infection that a radical operation was impossible. A year later she had gained nearly 3 st. in weight, and was so well that when the American Society of Clinical Surgery was in this country I showed her to the members as a case in which I had been mistaken as to the nature of the disease. The patient remained well for two years, then exhibited signs of sudden, rapid growth of the disease, and died within a few months.

A more remarkable case is that of a man, aged 52 years, upon whom I performed gastro-jejunostomy in 1908. He had cancer of the pylorus, which had spread in the sub-mucous tissue almost up to the cardiac end of the stomach. Radical operation was impracticable. I removed a portion of the growth, and microscopically it proved to be a spheroidal-celled carcinoma. It is now more than four and a half years since the operation, and the patient states that "he is as well as he has ever been in his life." Until we know more of the natural history of gastric cancer we are not justified in drawing conclusions as to whether it is or is not, greatly prolonged in some instances.

The alternative to the conclusion that carcinoma is grafted on ulcer is that simple ulcer and carcinoma may occur independently in the same patient. That sometimes this should happen is highly probable, for the frequency of the incidence of cancer and ulcer is very similar. Further, it is a hypothesis based on fact. On several occasions, when operating for cancer, I have been able to demonstrate to those present either an open ulcer or the scar of an ulcer quite distinct from the malignant growth. In several, at least, of the cases recorded as instances of *ulcus carcinomatosum* two ulcers were observed—one simple and the other malignant.

A perusal of the records of cases of *ulcus carcinomatosum* reveals one striking feature—namely, the frequency with which there is an interval between the symptoms of supposed ulcer and the onset of the symptoms of gastric cancer. With regard to such cases as these, the assumption that cancer has been grafted on an ulcer is less reasonable than the hypothesis that an ulcer has been present and has become healed, and subsequently cancer has occurred independently of the ulcer.

(b) *Pathological evidence.*—According to Dr. Wilson and Dr. MacCarty, in 71 per cent. of their series of cases there was "a large ulcer with scar tissue centres and overhanging borders, deep in the bases of which cancer is present, which ulcer in almost every instance has unmistakably originated in the lesser curvature of the stomach, the usual site of gastric cancer. Further, almost every case gives a clinical history suggesting gastric ulcer for a long period of years preceding the relatively short period when the history became that of gastric carcinoma." Apart from the assumption that a previous history of gastric trouble is evidence of gastric ulcer—an assumption which I have criticised already—the weak point of this argument appears to me to be absence of proof that an ulcer in which cancer cells are present has ever been other than malignant. The presence of scar tissue is not necessarily evidence of non-malignancy. We know that in cancer of the breast there may be formation of scar tissue, nature's attempt at arrest of the disease. Further, the border line between a benign and a malignant growth is a narrow one, and a given tumour may be inherently malignant before clinically or pathologically we can recognise its malignant character, with the means at present at our disposal.

There are two truths which appear to me to make it very difficult to accept the view that the grafting of cancer on ulcer is of frequent occurrence. First, simple ulcer of the duodenum is, at least, as common as gastric ulcer, and yet cancer of the duodenum is a rare disease. Surely, if simple ulcer be a frequent precursor of cancer, then cancer of the duodenum should be at least as frequent as gastric cancer. Then, again, cancer is commoner in the second than in the first portion of the duodenum. Ulcer in the second portion is the rare exception. If ulcer be the precursor of cancer, then cancer should be far commoner in the first than in the second portion. Exactly the contrary is the case.

Secondly, death from cancer after gastro-jejunostomy for supposed simple ulcer is a rare event. If the hypothesis as to the frequency with which simple ulcers become malignant be correct, then many of the ulcers supposed to be simple should prove later to be malignant, and consequently many of the patients on whom gastro-jejunostomy is performed

should die subsequently from cancer. As a matter of fact, the mistake when made is more often in the opposite direction—it is the supposed malignant growth which proves to be innocent, not the supposed simple ulcer which proves to be malignant. My own experience is that 1 per cent. of the patients on whom gastro-jejunostomy is performed for simple ulcer die later from carcinoma. Professor Kocher has published recently a review of 50 cases of gastro-jejunostomy. The period which had elapsed since operation varied from two to twelve years. In no case was there a recurrence of symptoms, or any suggestion of malignancy. Dr. Gressot states that malignant degeneration of surgical ulcers occurs in only 2.3 per cent. of all cases after gastro-jejunostomy.

In view of such observations it is difficult to accept the view that grafting of cancer on simple ulcer is a frequent event. Of course, I do not deny that a simple ulcer may become malignant. My point is as regards the frequency of such a happening. That it is anything like so common as some authorities teach, in my opinion, is doubtful. The subject requires much further investigation. In the meantime the verdict should be the Scotch verdict of "Not proven."

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ON THE SECRETORY ACTIVITY OF THE STOMACH IN CHRONIC APPENDICITIS WITH GASTRIC SYMPTOMS.

WITH AN APPENDIX CONTAINING CLINICAL DESCRIPTIONS OF 19 CASES.

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SINCE W. J. Mayo drew attention to the association of chronic appendicular disease with gastric ulcer, the combination of these two conditions has attracted the notice of a number of observers, and is now generally admitted (1).¹ Moynihan (2) especially has shown beyond all reasonable doubt that there is a definite relationship between these two pathological states, and has laid emphasis on a constriction of the base of the appendix that is liable to occur in such cases. Of late years, also, many other writers have been inclined to attribute gastric symptoms to chronic appendicular disease, and Fenwick (3) and Paterson (4) have found that a large proportion of cases presenting obscure gastric symptoms are the subjects of chronic appendicitis and in many they have demonstrated hyperacidity.

I have recently had the opportunity of making very detailed analyses of a series of 300 cases presenting gastric symptoms. Most of these cases were subjected to laparotomy. Among them some 19 presented unequivocal signs of appendicular disturbance, and analyses of the gastric contents of these cases are set out in the accompanying table, being divided into two groups according as a peptic ulcer was or was not present. A few lines of explanation must be given of the actual measurements and readings in the table.

The phenolphthalein and dimethylaminoazobenzene readings must not be taken to indicate total acidity and free hydrochloric acid. These readings, as explained elsewhere (6), are dependent on hydrogen ion concentration, and their difference (i.e., phenol minus dimethyl) is a function of the amount of nitrogen present and of the degree of digestion. In an active stomach, therefore, in which all the dissolved nitrogen is present as peptones or similar products of digestion, the fraction $\frac{\text{phenol minus dimethyl}}{\text{nitrogen}}$ is a constant,

provided that a standard test-meal be adopted and removed always at a fixed time after ingestion. A certain multiple of this constant has been designated the nitrogen factor and has been found to nearly approach 2.4 in normal cases. A rise above this number indicates either stasis or impairment of the digestive process.

¹The numbers in brackets refer to the bibliography at the end of the paper.