T H E S I S

- on -

P E P T I C U L C E R

Submitted by:-

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For the degree of Doctor of Medicine.

OCTOBER 1938.

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P E P T I C U L C E R

I N T R O D U C T I O N

By the term Peptic Ulcer, I mean a non-malignant ulcer occurring in the stomach and duodenum as far as the papilla. I also include in the definition a simple ulcer which occurs occasionally at the stoma of a gastro-enterostomy. I appreciate that these ulcers may be multiple. I do not include erosions.

For the purpose of this thesis I am ignoring the acute ulcer and am proposing principally to discuss the chronic form.

In hospital practice one is impressed by the proportion of cases of the "gastric" type. I persistently have more cases of peptic ulcer than of any other group of diseases. When this is coupled with the number of patients one strongly suspects as suffering from ulcer (but because of many circumstances this cannot be proved) the importance and prevalence of the complaint assumes major importance.

Major catastrophes do occur and are greatly to be feared. More often, however, it is the disablement that is so worrying. In Middlesbrough we have to supply labour for the heavy industries. It is next to impossible to keep a man working at a blastfurnace or

as a steel-maker or in a rolling-mill on a "gastric" diet. Such a patient puts up with the pain and discomfort as long as he can and then either suffers from a major catastrophe or pleads for operative interference. Not only is pain a disabling factor but also there is a general lowering of health through repeated small and often unknown haemorrhages and lack of proper digestion. Again the fear of another attack always looming ahead, even during a quiescent period, must and does constantly irritate a sufferer. In my experience, however, it is the pain that is the direct disabling factor.

Treatment is either surgical or medical. In Moynihan's early days I suppose one could safely say that the treatment of duodenal ulcer was mainly surgical and that but for certain technical difficulties this would have been true of gastric ulcer. Now we have a greater appreciation of dietetics, a greater knowledge of neutralising or buffering gastric juices and a well-founded fear of the failure of many surgical operations. Thus the pendulum has been allowed toswing to medical treatment. There is, however, a more or less general agreement that definite indications for surgical interference exist..

My own opportunities for giving special study to this subject are comprised in the ordinary work of a

general practitioner who has the good fortune to be on the staff of a general hospital of some 140' beds. By means of a Medical Out-patient Department which I started a year or two ago, I am able to follow up a good many cases and thus can sometimes prevent a premature cessation of treatment and can also often check up "cures". In the past I have been compelled to deal with cases in a practical rather than in an academic sense, but I am rewarded by a consistent reference of peptic ulcer cases by my surgical colleagues.

P E P T I C U L C E R (Cont'd)

PATHOLOGY AND ACTIOLOGY

In this section we are confronted with a few facts and an immense amount of theory. Surgery has helped in knowledge pertaining to pathology and also with regard to incidence.

1. Firstly, I propose to deal with the general pathology of peptic ulcer, which will be a more or less straight statement of the macroscopic and microscopic findings. The place of theory is small.

2. Equally there is little of the theoretical in the second part in which I shall deal with the common sequelae. Discussion concerning the superimposition of carcinoma will, however, disclose some differences of opinion.

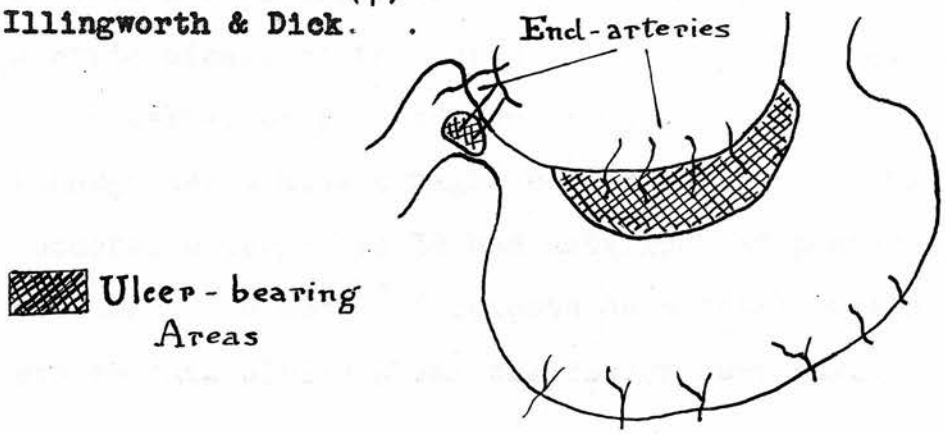
3. When we turn to the next question the difficulties of actiology will be at once very evident. A most amazing point will be early appreciated. There is no agreement (or for the moment no sign of such), as to what constitute the fundamental changes in the body, local or general, in the production of a peptic ulcer. There is little doubt that an excess of acid in the stomach is an essential link in the chain of evidence. The antecedent history is a matter for conjecture. The statement that theories vary from local trauma to damage to the brain exemplifies the astounding fact that one of the most common maladies of our time cannot be adequately explained.

4. Surgeons have added particularly to a knowledge of matters pertaining to incidence. As a rule, they deal with a selected type of case. Their findings obviously will make the statements of pure pathologists seem fallacious for that reason. Probably an average taken from these sources and from clinical experience, will not be far from the truth.

5. There seems to be an inherent power of healing in each peptic ulcer. So much is this the case that it would appear only necessary to remove what fundamental cause there is to have a cure without making any great effort at the local lesion. The final part of this section will deal with the phenomena of healing.

GENERAL PATHOLOGY

Chronic peptic ulcer occurs in 90% (7 & 24) of cases in an area known as the Magenstrasse. The literal translation of this compound German word is the street of the Stomach. The diagram below is taken from Illingworth & Dick.(7)



The Magenstrasse results from the fact that oblique muscle fibres in the stomach mainly pass along the cardia and the greater curvature. When contracted a groove is formed which corresponds very nearly to the lesser curvature. The Grove of Retzius has also been applied to this ulcer bearing area.

Bolton (28) Levine (20) and Stewart (24) are agreed that the initial lesion is an erosion of mucous membrane, and the formation of an ulcer is a persistence and extension of this erosion. Moynihan (26) maintained that from a practical viewpoint chronic peptic ulcer has existed for a long period before a diagnosis should be made.

Chronic Ulcers are usually single.(2) I make free use of Prof. Stewart's statistics[24] in putting forward many of the following statements. In so far that they are compiled from a series of 6,800 consecutive autopsies they carry weight; apart altogether from a consideration of the eminence of their author. In this series there were 200 duodenal ulcers and 150 gastric ulcers of the chronic type. In the gastric ulcer series only 3 had two or more independent ulcers (though acute ulcers might be present). In the duodenal ulcer cases 32 had anterior and posterior lesions. Bolton (28) reports on a fatal case having six chronic ulcers along the lesser curvature.

The size of the ulcer varies as the following table from Stewart shows:-

Size	40 Gastric Ulcers	40 Duodenal Ulcers
Under ½"	3	10
From ½" to 1"	14	19
" 1" to 2"	9	11
" 2" to 3"	6	
" 3" to 4"	5	
" 4" to 5"	3	

From this table the average size of a gastric ulcer would seem to be about 2" which strikes me as rather a high figure. The average size of a duodenal ulcer on the other hand is half this.

In order to show the wide variation of post-mortem statistics I have drawn up a table in which figures published by Sturtevant and Shapiro (34) from the Belle Vue Hospital, New York are compared with these of Stewart from the Leeds General Infirmary.

The figures from the Belle Vue Hospital agree with earlier British figures. Stewart's figures are considered high.

	Sturtevant and Shapiro		Stewart	
	No.	%	No.	%
Number of Autopsies	7.700	100	6.800	100
Cases of Gastric Ulcer	120	1.6	150	2
Cases of Duodenal Ulcer	44	0.6	200	3
Cases of Both ...	5	0.07	3	0.04
Total	159	2.27	347	5.04

It is apparent from these summaries that incidence is dependent in some way on geographical conditions.

The shape of an ulcer is round or rounded. There is often undermining at the edge. Not uncommonly the actual edge is raised and hard while the sides are terraced. The floor is hard and is covered by a slimy layer of mucus. From the peritoneal aspect there is

thickening and opacity of the serosa with congestion and may be areas of haemorrhage. White lines of fibrosis run outward from the margin. Vascularised bands of adhesions to some adjacent structure are not infrequent. In old scarred ulcers this aspect is white, punched out and deformed. The sensation of induration is characteristic.

Microscopically these statements are amplified. With regard to the edge, new epithelial tissue will be flat if the ulcer is callous. In an active ulcer the margins are rounded and overhanging (Stewart [24] Dibble [17] etc.) The walls and deeper edges are seen to be made up of scar tissue which is in section like a wedge.

Without some penetration into the muscular coat it would seem that an ulcer cannot be chronic. It is particularly mentioned by Stewart that complete penetration is the rule. Invasion is prevented by a thick fibrous bar at the very base. There is a varying amount of endarteritis. This may be as much as to result in complete occlusion of vessels.

Karsner[19] calls attention to the fact that though the bases of chronic ulcer are comparatively clean, there is usually a covering of "an adherent granular or slimy, pale, bloody or brown tinged exudate". He also quotes Askanazy that there

is a reduction in the number of ganglion cells and loss of the myelin sheaths of many of the nerve fibres.

COMMON SEQUELAE

It is with diffidence that I mention Haematemesis and Haemorrhage. I agree with much I have read about the vomiting and passing of blood and regard these as symptoms. It is, as I know, looked upon without great distress by some sufferers. Statistics of the frequency are a little misleading. I myself have come into contact with patients who have had a Haematemesis in their own houses.

Old figures vary from 16½% (Miguel) and 84% (Martin) [28] Hurst and Ryle[13] have experienced 32% of their private cases of gastric ulcer associated with haemorrhage compared with 28% in Duodenal Ulcer. My own combined series has 28% associated with haemorrhage. My number is too small to divide into gastric and duodenal ulcers.

The pathology of haemorrhage is simple. The fibrous base of the ulcer is invaded further and ulceration through a vessel ensues.

The second catastrophe is perforation. Here again the fibrous base gives way and allows stomach and

duodenal contents to enter the peritoneal cavity. The complication probably occurs most frequently in a rapidly growing ulcer. Adhesions have not had time to form with any degree of density.

Stewart [24] found that out of his 150 cases of gastric ulcer coming to autopsy, 58 had perforated; of his 200 duodenal ulcer cases, 122 perforated. It may be that the dramatic onset and urgency of treatment is responsible for a rather exaggerated idea of its frequency. Miller [29] had a perforation incidence of 15% in a series of 279 cases. My own figure is 22%.

Perforation may occur into the lesser sac, as well as the greater. I have had surgeons of my acquaintance speak of pin-hole perforations into the lesser sac. I have no data to confirm or refute this idea.

Penetration, as a distinction from what is commonly known as perforation occurs, particularly into the pancreas. This is a slower pathological process than is perforation. In the pancreas a mass quickly forms. This organ is "fairly resistant to the action of gastric juice, but in the liver there is much more likely to be necrosis and abscess formation" (Boyd [22]).

Contracture by scar tissue is a later sequela.

The main results are pyloric stenosis and hour-glass stomach. I have been surprised at the time apparently taken before contraction causes pyloric stenosis. Cases come to the Infirmary with quite long histories of ulcer and yet stenosis is not so common as one would expect. It may be that my surgical colleagues see more of it than I.

The following brief notes provide examples:-

(a) M.G. - midwife, aged 48: 10 years history - operation; large chronic pyloric ulcer with enormously dilated stomach.

(b) S.G. - 5 years history - X-ray showed persistent spasm of pylorus - operation; ulcer with no gastric hypertrophy.

(c) F.W. - 7 years history - operation; stenosis present but no great degree of dilatation.

CANCER, IN RELATION TO GASTRIC ULCER:

The superimposition of cancer on gastric ulcer has been long debated. Ewing[30] in 1918 reported that the Mayo Clinic considered that 68% of gastric ulcers were complicated by carcinoma. He thought it reasonable to choose between 2% & 68% as figures compiled by reputable authorities. He himself thought that 5% was an outside figure. It would seem that too much attention was at one time paid to academic pathology. It was permitted to become a

master rather than a servant. Horsley[3] denied that cytological evidence necessarily implied that cancer was about to develop. He described a case with a 15 years' history of ulcer which showed two supposed malignant acini. Jordan[4] summarised a contribution on gastric ulcer and carcinoma by, "Gastric ulcers need not all be regarded as malignant or potentially malignant". Morley[21] went further. 'He did not think that carcinoma was more likely to develop in a person with ulcer than in a person without'.

Dibble and his co-workers thoroughly investigated the matter. Not only did they judge from cytological grounds but also quite properly from other considerations. These were such as site, length of duration, and statistical information. They took as one of their main guides the formation of scar tissue. Any association would be in a scirrhus type of carcinoma. In their series only 6% could possibly have had pre-existing ulcers. Every allowance had been made in favour of cancer. A little earlier Spilsbury[15] had come to quite the same view as Dibble. He thought that 6% was a very exaggerated figure. Both these authorities showed that many investigators had allowed normal healing processes to be confused with malignant changes. They repeat the statement that I have already made. Histological

grounds alone should not be the final arbiter.

I personally, have come across a possible case of superimposition of cancer in a minor way. My surgical colleague, J.C. Clark had occasion to perform a Laparotomy. He found a soft cancer of the lesser curvature. The patient had a history that for ten years he had suffered from epigastric pain, coming on three hours after food, relieved by alkalies. The time interval was a little long. Histological findings confirmed a columnar-celled carcinoma.

There is no serious suggestion that malignancy follows duodenal ulcer. In reporting on a case of duodenal cancer Deaver and Ravden[32] mention the following facts:- 2.5% (Brill) of malignant tumours of the intestinal tract occur in the small intestine. They further quote Jefferson that of that 2.5% only 3.1% occur in the duodenum. The figure is thus extremely low and is significant in a consideration of such a widespread disorder.

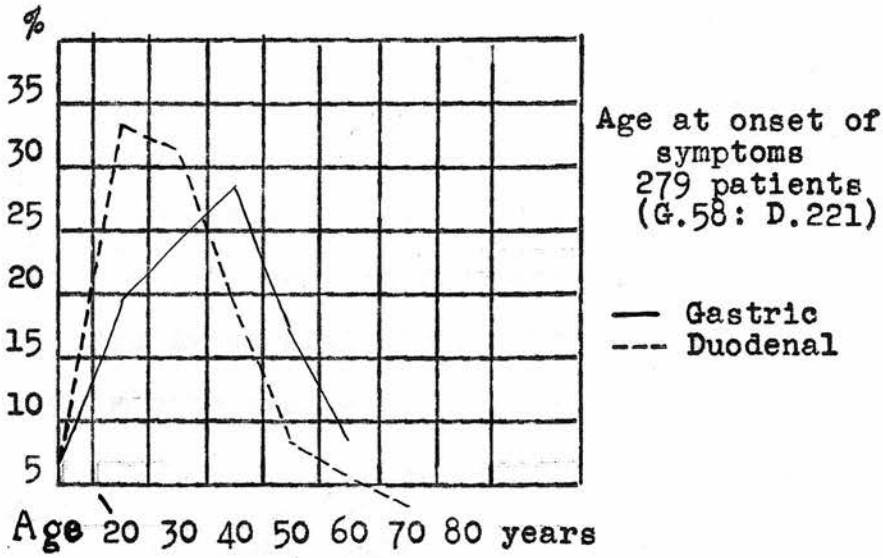
INCIDENCE

Peptic ulcer is probably more common than figures prove. The general public has however a very exaggerated idea of its prevalence. This is fostered by the manufacturers of certain antacid powders.

Wilkie[31] has stated that at the Edinburgh

Royal Infirmary the number of cases of duodenal ulcer rose from 46 in 1906, to 338 in 1926. In the same communication he gives figures showing that the average age for operation in his cases is 45 years.

Miller et alia[29] have published interesting details of 279 patients proved by operation to have peptic ulcer (gastric - 58: duodenal - 221). 90% of gastric ulcers and 85% of duodenal ulcers occurred in males. All women coming for operation for gastric ulcer and 64% with duodenal ulcers were over 40: while respectively 73% and 51% men were over 40. The following chart shows ages at onset:-



These authors show a 4:1 rate of duodenal to gastric ulcers. Then figures regarding sex incidence agree approximately with the 4:1 ratio in favour of males which they quote from Balfour of the Mayo Clinic. Moynihan's[33] figures indicate that he has experienced

a ratio of 3:1 in favour of duodenal ulcer and at 3:1 in favour of males. These surgical figures are challenged particularly in a recent article by Duncan Lees[37]. Though his number of cases is small (58) it is remarkable that there were nearly twice as many gastric ulcers as duodenal ulcers. Ryle[13], however, supports the 4:1 ratio. My own series has 13 with gastric ulcers and 16 with duodenal ulcers.

Illingworth & Dick[7] state that while peptic ulcer is common in Great Britain and N. America, and in certain parts of India, it is extremely rare in China. They suggest that this might indicate a relationship to the dietary of civilization.

CAUSATIVE FACTORS

It seems evident that in the first place a chronic ulcer is derived from an erosion. Acute ulcers also arise similarly. Schematically a triangle may be used:-

Erosion

Acute Ulcer — Chronic Ulcer

Evidence is forthcoming from Illingworth and Dick[7] Mann and Bollman[8] Vines[2] Levine[20] and Bolton[28 & 24] that this is so. Mann & Bollman, however, explicitly exclude the assumption that acute ulcer

as such ever passes into the chronic state.

When the erosion is considered there is such an extraordinary variety of causes blamed that it is only worth while considering many of them in the merest detail. Levine[20] goes into some detail about these and divides the causes into:-

(a) Local Irritants
(b) Circulatory disturbances
(c) Nervous impulses

The first on his list under (a) is food. Presumably he means unsuitable food. This is at once acceptable to commonsense and clinical experience. Frequently the food in itself is good enough. It is the method of cooking, and of eating that are so often at fault.

Levine's List more fully is as follows:-

(a) Local Irritants:

1. Food
2. Trauma (through abdominal wall)
3. Faulty posture such as pressure from corsets
4. Hot meals and drinks
5. Chemicals

It is of interest under (3) to ascertain how many cobblers suffer from ulcer. The pressure on the sternum is so great from pressure of the last that a permanent depression is found. I personally, have only once met with a cobbler, with a questionable ulcer.

(b) Circulatory Theory:

Though admitting the cause he denies that Virchow (as stated by Decker, Bolton and Stewart) ever said that gastric ulcer was preceded by circulatory disturbances in its mucous membrane

(b) Circulatory Theory (Cont'd)

sufficient to deprive the area of alkaline blood, exposing the area to attack by acid gastric juice. It seems that the very deprivation of blood supply would rather devitalise the tissue and allow the digestive juices to destroy that area.

(c) Nervous Causes:

He called attention to the fact that in 1928 Cammerer injured both vagi and both sympathetic nerves so producing gastric ulcer. He also mentioned that Durante also obtained ulcer. He did this by tying both splanchnic nerves. Ivy however, failed when he used careful feeding methods.

This very brief summary of Levine's writing serves as an excellent introduction. It lends itself as a comparison for other theories. In reading about the cause of peptic ulcer I have found it somewhat difficult to appreciate one fact. It is difficult for me to be sure when reference is being made to chronic ulcer and its development or to acute ulcer or even erosion.

Ingested Material:

Experimental proof of the power of ingested material is afforded by reference to McCann.(5) In an artificially produced jejunal limb in the dog which was so constructed that the alkaline duodenal contents were side-tracked, an ulcer developed and perforated. Hairs had been swallowed and some were found actually sticking into the distal part of the

ulcer. Presumably some hairs had passed through the perforation. These hairs were quite fine, yet they were firmly fixed by the force of the blood stream. This is an example of a particular trauma. Boyd[22] reminds us that there are a number of small and large injuries which cause an ulcer of the leg. Similarly it must be accepted that there is a host of local causes of ulcer of the stomach and duodenum. Local irritants do play a part.

Bacterial Theory:

The credit for the proper foundation of the bacterial theory is due to Rosenow[23]. He demonstrated a non-Haemolytic streptococceus with selective affinity for the stomach and duodenum. The finer bacteriological researches do not in themselves require consideration here. Rosenow found that identical strains of the organism present in the ulcer bed as in such foci as teeth, tonsils, appendix or gall bladder. He also produced ulcers in experimental animals by injection of the cocci. His work has been confirmed amongst others by Haden[18].

Circulatory Theory:

(a) The blood supply to the main ulcer bearing areas is derived from end arteries. Wilkie has

demonstrated an end artery supplying the site of ulcer in the duodenum. Reeves has shown a similar arrangement along the lesser curvature (both quoted 7). It is supposed that devitalisation is either due to embolism or spasm.

(b) The second theory is a mixture of nervous and circulatory disturbances. Von Bergman, (quoted Karsner[19]), has suggested that spasm of the vessels results from paralysis of sympathetic nerves or ganglia. Local ischaemia results and so permits of erosion.

Toxic Theory

(a) It is accepted that severe burns occasionally result in gastric ulcer.

(b) Bolton[25] prepared a "gastrotoxic serum" by injecting an emulsion of the mucous membrane of a guinea-pig's stomach into a rabbit. The blood serum of the latter became toxic to the tissues of the guinea pig. He also injected human gastric cells into the rabbit and the domestic fowl. The blood serum in each case resulted in characteristic test tube reactions. He also employed cat into goat and monkey into goat with similar results. By immunising the rabbit with an injection of rabbit gastric cells he produced an "isogastrotoxin". This

was highly toxic to guinea-pig tissue. If a lethal dose ~~is~~ was given which causes death in more than 48 hours, punched-out ulcers ~~are~~ were found in the stomach. This, however, did not take place if the stomach contents were kept persistently alkaline. For the experimental production of ulcer Bolton injected a gastrotoxin locally in the stomach wall. Very accurate artificial ulcers followed.

(c) Rosenow and Anderson(Quoted 22) produced acute ulcers by the local injection of diphtheria toxin.

Cerebral Theory

(This theory is based on a more central part of the nervous system than that quoted from Levine).

Many years ago Rokitansky(quoted 1) suggested a central nervous system cause in the etiology of ulcer. More recently Cushing(10) published rather confirmatory evidence. He had been struck by the similarity of three cases, he had operated on for removal or attempted removal of tumours situated in the "inter brain". Each case died. The first died from a perforated gastric ulcer, the second from a perforated duodenal ulcer, and the third from a perforated ulcer in the lower end

of the oesophagus. Each of these ulcers was of a fulminating character.

As a result of a consideration of these cases Cushing was led to make further investigations. He injected various substances either intramuscularly or intravenously. He found that intraventricular injection of Pilocarpine and Pituitrin caused severe gastric motility with spasm of the pylorus, hyperacidity and even haematemesis.

I do not see that the evidence produced by Cushing and confirmed by Fulton(110) have any great importance. To my mind the fact that a sudden, severe and often fatal ulcer develops following very violent muscular contractions in the stomach, could be expected. The fact that these violent movements follow cranial irritation is, I think, not surprising.

Ulcer Diathesis

In his 1933 Alvarez Lecture Hurst(38) discusses what he terms the hypo- and hypersthenic gastric constitution. By that he means that there are some 20% of individuals who have either a long, low, slowly emptying stomach with hypoacidity or a short, high, rapidly emptying stomach with hyperacidity. He suggests that such irritants as food, drugs and infective material will injure only this 20%. The

more slowly emptying stomach would lead to gastric ulceration while the rapidly emptying stomach would lead to duodenal ulcer. The figure of 20% is based on the results of the investigation of Bennett & Ryle (1921). They examined 100 healthy students; of these 10 were of the one type and 10 of the other.

Hurst's description of these two states reminded me somewhat of Russell's[37] old division. He classified gastric illness according to excess or deficiency of acidity, irrespective of the pathological changes. I think that much more investigation might be made of the future of the 100 medical students. Particularly interesting would be a follow-up of what actually became of the 10%. (I have just found that such a follow-up has been made, but no important conclusions have materialized).

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Diet per se

The general contributions of McCarrison to the study of diet led me to look to him for guidance. Particularly was this so with regard to vitamins.

It is gratifying to find lucid reference to the effect of wrong diet from that point of view in his works. I do not think it of serious importance that one should investigate too closely the scientific explanation of the modus operandi of the deficiency.

It may or may not be that the deficiency results in a slowing up of intestinal movements with consequent excess of putrefaction. Similarly interference with the adrenals (Magee, Anderson & McCallum[40]) may follow and be blamed for the changes.

McCarrison clearly found frank ulcers or conditions leading to them in experimental animals such as monkeys, pigeons and rats, when starved from vitamins in general[44]. In India the "Tapioca" and "Rice" diets have almost a predilection for peptic ulcer. By experiment McCarrison confirmed this by feeding rats on similar lines[45]. In these diets the husk of the seeds is removed by polishing, thus removing the vitamin content. McCarrison thinks that the lack of a properly balanced diet is to blame in the aetiology of peptic ulcer rather than a particular vitamin shortage. He attributes the rise in incidence of digestive disorders in general to the lack of milk products, fresh eggs and fresh garden produce. This point is illustrated by a further experiment of McCarrison. In Travencore peptic ulcer is frequent. The diet is low in fresh foods, including fresh meat. In the Sikh country, however, where good "honest" food is taken peptic ulcer is infrequently encountered. In his experimental rats (of which he is very proud), ulcer never developed when the Sikh Diet was given to them.

With the diet of Travencore 29% of the rats developed peptic ulcer.

HEALING

It is remarkable how constantly one comes across a reference to the innate power of healing of peptic ulcer. There seems to be little doubt that the mucosa of the alimentary canal has an extraordinary power of recuperation. Experimentally McCallum(1) has cut off a portion of the mucous membrane of the stomach and at a subsequent examination has failed to find any sign of damage.

This power of healing has been shown by Caylor(16) to be present in peptic ulcer in man. The first change he found to be the appearance of a tuft of granulation tissue over the base of the ulcer. Sometimes a series of small tufts began. Following this, rather a-typical mucous membrane grew from the edge of the tuft. He studied 30 cases of gastric ulcer removed at operation. Of these, 25 showed such signs as described. He thought that the remaining five were of the perforating type because of the lack of healing processes.

Mann & Bollman(8) passed gastric juices direct to the jejunum by short circuting the duodenum. In practically every case a jejunal ulcer developed. The ulcer persisted but immediately began to heal when normal

mechanics were re-established.

McCann[5] made similar experiments. From them it appears that the contents of the stomach will produce ulcer in any part of the intestine according to the experimenter's wishes. The duodenal juices must be bye-passed. Once the duodenal contents are released the ulcer goes.

Thus it has been shown that the direct action of gastric juice is responsible for ulcer; also that the presence of the duodenal buffer tends to prevent this.

The experiments alluded to deal with acute ulcers. In view of a possible common aetiology this need not exclude their importance. Caylor's work supported by Stewart[24] very definitely proves that a chronic ulcer is constantly making efforts to heal. No doubt steptococci do have an effect in preserving an ulcer. There is enough data here to warrant a belief that an ulcer has more chance to heal when the stomach contents are neutralized or buffered.

P E P T I C U L C E R (Cont'd)

SYMPTOMS DIAGNOSIS & SEQUELAE

A. SYMPTOMS & SIGNS.

Pain is a constant feature of chronic peptic ulcer. It is always somewhere in the epigastrium. In case notes one comes constantly to the "complaint" being transcribed into such words as epigastric pain. One also notes that the pain has gradually become worse. Most of the cases with which I have had to deal have had a reasonably long history, the average being six years. I have always been careful to insist on the first appearance of discomfort. F.F. a woman aged 55 years who was admitted for haematemesis had actually suffered from indigestion for 24 years.

The pain varies a great deal. Frequently it is described as burning, often as boring in character, and not rarely as though a knife were sticking into the stomach. Occasionally the pain is in addition radiated to the interscapular area. Heartburn is another form of pain. One does meet patients who suffer pain also along the lower border of the ribs either left or right.

There are two cycles as regards the onset of the pain:

(a) That in which the patient is most insistive is the relationship between the

taking of food and the onset of the pain. By the time I see a case this has possibly been well brought out by previous examiners. The patient then has his attention well focussed upon it. The relationship may be given as the time period before or after the taking of food. The longer the interval after food the more likely is the cause to be duodenal. Conversely the shorter the time, the likelihood is that there is a gastric ulcer. That this is only a general statement is exemplified by two exceptions:

(1) P.M. 47 years: labourer: admitted for diagnosis and treatment. His pain had come on in the upper abdomen some two or three hours after food, over a period of 13 years. The radiogram revealed a constant ulcer deformity in the lesser curvature.

(2) O.D. labourer aged 50 years: One year's history of pain to the left of the midline almost in the hypochondria He thought the pain came, in about ½ hour after food. The radiogram showed a small but definite ulcer crater in the duodenum.

Moynihan's writings had led me to suppose that pain during the night in nearly every case. When such does happen the time is usually between one and two. In my district the patient states that the pain comes on just after he falls into a good sleep (owing to shift work reference may be made to the sleeping period, rather than to night time). I have not found nocturnal pain to be very common.

Relief from this pain is variously afforded by alkalies by hot drinks, by food or by local heat. In most instances there is a spontaneous gradual diminution, particularly in duodenal ulcer pain may persist until the

last meal. McLean's (proprietary) powder is practically always tried by the patient. To its power of relief allusion is frequently made (Note - in proved ulcer I have found that this powder does not in itself give a great deal of help). The gradual disappearance of pain either following drugs or otherwise is of some importance.

Hurst[48] has shown that pain is most constantly present when the stomach is not quite empty. He quotes such authorities as Palmer, Enriquez, and Wilson. No precise reason is given for the occurrence and disappearance of the usual peptic ulcer pain. It is suggested that achalasia of the pyloric sphincter occurs with an increase in the acid content of the stomach. Pain is not caused by the acid chyme. This fact was proved by Wilson (quoted by Hurst). He forced acid chyme through the pylorus so that the material flowed over a duodenal ulcer. Instead of causing pain relief was felt.

(b) The second periodicity is that of the duration of the attacks of pain. Included in this is the interval between such attacks when there is freedom from pain. It is common to find that the pain appears at its usual time before or after a meal for as long as a fortnight or even a month. More or less complete freedom then follows for a comparatively long period. This may be as long as six months, but may be quite short. There is no definite space of time in this category. It is extremely variable. An example of lack of definition in this periodicity occurred as follows:- A man of 34 years only had attacks of pain when

he did any really hard manual work. If he was unemployed or on any easy task he did not experience any discomfort at all.

VOMITING

Vomiting is quite commonly encountered. Its relationship to the taking of food varies. The patient may find in it quick and effective relief. These remarks also allude to erructations.

Haematemesis:

The most characteristic vomit is of blood. In my series I have met it in 28% of all cases. This is a low figure. The figures of Hurst and Ryle and other writers are considerably higher (see below). The vomited material is unchanged blood, mixed with food, or consists of blood which has been acted upon by the stomach juices, so that it resembles coffee grounds. The haemorrhage may be so great as to cause grave anxiety for the patient's life. Fortunately, I have not yet had a fatal result. There is evidence from my own cases as well as elsewhere to show that it is liable to recur.

Melaena:

It is a simple matter to establish the true interpretation of a black motion in the wards. It is difficult to treat such a motion seriously when given in the history. The use of bismuth is so common that

such a history has to be ignored unless one is completely satisfied about the intelligence of the patient. Melaena occurs following haematemesis. It also occurs when the ulcer is bleeding in a much less violent manner. Its presence is of great importance. Especially is this so when other investigations have not added to clinical judgment.

I have seen copious rectal haemorrhage from duodenal ulcers in two instances. I have to trust entirely to memory. I especially remember the second case. I was called to see a middle aged architect one evening. I found him unconscious with all the signs of a serious haemorrhage. He had been vomiting great quantities of blood and had passed a copious red stool. Ultimately he made a perfect recovery.

Clinical Findings:

Clinical findings are unfortunately vague.

Tenderness:

In association with pain one might reasonably expect tenderness. This is by no means constant. I have examined patients both during an attack of pain and in an interval of freedom. It is more common to find tenderness in the former state. Even so, however, it is not at all a constant sign.

Characteristically one does find in a fair proportion of cases, a point of tenderness, not often severe, in the mid-line between the xyphisternum and the umbilicus. Occasionally the tenderness is a little to the right. On one occasion I found tenderness at the junction of the left rectus abdominis with the rib margin (case

of gastric ulcer). In a few instances I have elicited tenderness in the more common place with the patient sitting up and bending slightly forward. I am not prepared to advance any definite opinion as to the value of this test, as I have not tried it sufficiently long to make a statement. Wilcox(47) considers that tenderness is more common than I have experienced. Absence of tenderness is certainly no help either for or against a diagnosis of peptic ulcer.

Rigidity:

I think that rigidity is rare unless the ulcer is in an active phase. It is then, and then only, that one can expect to find rigidity at all constantly. The sensation is often that of resistance rather than an ~~dual~~ actual rigidity.

Anaemia:

Anaemia is present when there has been any degree of blood loss or when dietetic restrictions have excluded or limited blood forming foods. It is profound after a serious haematemesis.

Miscellaneous:

During an attack of pain the tongue is dry and is covered with a light brown fur. The covering becomes gray-white when milk is used freely.

Loss of weight entirely depends on the amount and detail of diet taken. Persons suffering from ulcer are usually inclined to be thin. When pyloric stenosis occurs to an extreme degree emaciation begins.

METHOD OF EXAMINATION

The history obtained from a case of peptic ulcer is so characteristic that it forges the first link in the chain of evidence. It must not be under-

stood by that as implying that the history of each and every case is equally definite.

When evidence has been obtained that ulcer is likely, I arrange for a Barium meal examination. The diagnostic criteria used in this fundamentally important examination will be found in the section devoted to radiology. Providing the clinical picture is consistent with that of peptic ulcer, I take a positive X-ray report as conclusive. In such circumstances no further investigation may be called for. If the report is negative, or inconclusive, the radiological examination is repeated. It may be that a barium series will suggest itself.

It sometimes happens that a clinically obvious case of peptic ulcer is not confirmed by the radiologist, even after a careful repeat examination. I take this to be sufficient to exclude a positive diagnosis being made. I sometimes treat the case as though it were positive. Not infrequently the case is referred back to his medical attendant explaining the situation. We have at the North Riding Infirmary, a system of "Confidential Letters". These are used to inform the family doctor about his patient and also to ask for his co-operation.

Following the X-ray examination the case may warrant further consideration. The next step is the

performing of a test meal. I use the fractional test and not the Ewald single meal. Even with a raised acid curve, and other test meal findings, I have not included in my series other than radiologically proved cases with some obvious exceptions.

A blood count is made after a haemorrhage. It may not seem necessary to do a full count always. A periodic estimation of the haemoglobin serves as an easy and quick guide to the progress of recovery and as an indication as to whether bleeding continues.

It is necessary to examine the stools for blood. A persistence of a positive reaction obviously shows a persistence of ulceration. Melaena must be proved. It cannot be accepted that a black stool means haemorrhage.

At the end of the appropriate treatment another radiological investigation is undertaken. I use this as a guide as to whether hospitilisation needs prolonging rather than evidence of cure. I feel that much time must pass before daring to use this word in any case of peptic ulcer.

DIFFERENTIAL DIAGNOSIS:

Excluding peptic ulcer, chronic pain in the epigastrium may be due to the following causes:-

1. Extra abdominal causes.

   Spinal Caries
   Pleurisy
   Intercostal Neuritis
   Angina Pectoris
   Epigastric Herma

2. Gastric Disorders:

   Carcinoma
   Hyperchlorhydria
   Visceroptosis
   Gastralgia
   Gastric Crisis
   Reflex pain from appendicitis

3. Diseases of the Gall Bladder and Liver:

   Congestion of the Liver from Hepatitis
   Mitral Disease
   Carcinoma
   Hepatic Abscess
   Gallstones

4. Diseases of the Pancreas:

   Calculus
   Chronic Pancreatitis
   Neoplasm

5. Abdominal Aneurysm:

   Abdominal Angina
   Lead Colic

Haematemesis and/or blood in the stool associated with symptoms of chronic peptic ulcer may be due to:-

1. Diseases of the Stomach:

   Chronic Gastritis
   Gastrotaxis
   Carcinoma

2. Portal Obstruction:

   Cirrhosis of Liver

3. Blood Diseases:

   Pernicious Anaemia
   Scurvy

4. Miscellaneous:

Chronic Bright's Disease
Syphilis

Under these two main headings are grouped those disorders from which uncomplicated cases of chronic peptic ulcer should be distinguished.

1. Extra abdominal Causes:

The characterstic distribution of pain round a nerve belt will suffice to differentiate spinal caries and neuritis. Pleurisy will be found by ordinary clinical methods, but may be an accompaniment of ulcer. A dilated right ventricle will be self-suggestive. The pain in angina pectoris is not characteristically associated with the taking of food. There is a much closer relationship to effort. I have found difficulty in such a differential diagnosis, especially in later life. An electro-cardiograph may be of assistance. Quite often clinical acumen is of more help than the ancillary sciences. The presence of epigastric hernia is not likely to be missed if the possibility is kept in mind.

2. Gastric Disorders:

Carcinoma of the stomach is distinguished by the comparatively short History given. There is progressive loss of weight and relationship to food is not consistent. Radiological findings are helpful. Gastric analysis shows persistent achlorhydria, with the presence of sarcinae, torulae, Boas-oppler bacilli, lactic acid, etc. One rarely seems able to get into contact with a case of carcinoma of the stomach sufficiently soon to be able to advise satisfactory treatment. It is only too frequent that one finds the diagnosis only too obvious and too late.

Hyperchlorhydria per se must only be (see chart 2) diagnosed in the absence of radiographic evidence of ulcer; and in the absence of clinical criteria of ulcer. Visceroptosis can only be diagnosed accurately by radiology. (see plates 25) Tabetic crises affecting the stomach are more likely to be confused with a perforation. The usual signs of syphilis of the nervous system

will be found. If in doubt a complement-fixation test on the cerebro-spinal fluid may be advisable. Reflex pylorospasm from a diseased appendix is shown by a negative X-ray. A barium series may show this organ to be pathological. Clinically an area of tenderness in the right Iliac Fossa would be suggestive. Gastralgia is a condition found in young and badly fed women. It must never be diagnosed unless all other possibilities have been excluded. It is associated with a moderate degree of secondary anaemia.

3. Diseases of the Gall Bladder:

Congestion of the liver from any cause results in an enlargement of the liver. It should be found in clinical examination. The cause and effect should be obvious. Carcinoma, however, is not so straightforward. A secondary growth in the liver will sometimes develop before the primary focus has become suspect. The clinical history with progressive jaundice and enlargement of the liver associated with commencing emaciation will be of assistance. Hepatic abscess will be suspected from the previous history but may only be thought of as a result of a differential blood count. In biliary colic, pain is not so persistently related to food: it tends to begin and end abruptly, and to have waves of intensity. There may be jaundice, and referred scapular pain. The use of a selective dye along with a radiogram is often, but not always, of considerable help. Palpation of the gall bladder is sometimes possible. The difficulty of differentiation is complicated by the fact that peptic ulcer, gall bladder disease and appendicitis frequently co-exist (Wilkie(31)).

4. Diseases of the Pancreas

The diagnosis of calculus is extremely difficult. It will be confused with perforation, but the rigidity felt in perforation is absent and the condition of the patient does not become grave. Chronic pancreatitis may be associated with gallstones and often has transient periods of jaundice. There may be fatty diarrhea glycosuria and a "pancreatic reaction" in the urine. Carcinoma of the pancreas is also difficult to diagnose. I remember having a

patient under my care who gave a history very akin to peptic ulcer. He became very anaemic. He gradually went down and down, and ultimately died. At autopsy I found a carcinoma of the body of the pancreas. There was a palpable tumour for only two weeks before death. Progressive jaundice only occurs when the head of the pancreas is involved.

5. Abdominal Aneurysm is not only clinically demonstrable but very generally is shown radiologically. Angina occurring in the abdominal aorta is very rare and is diagnosed by indirect reasoning. Lead colic should be kept in mind when the occupation is considered. Lead may be found in the urine and the stools. Other evidence such as punctate basophilia should be sought.

6. Of the remaining disorders by far the most important is cirrhosis of the liver. A radiological report that no ulcer could be found, following haematemsis, is the clue to a search for further signs. The liver may be enlarged but not in the early stages. The history should be carefully checked, any evidence of excess in alcoholic intake being sought for. Though no hepatic enlargement is found, the edge of the liver may be palpated because of its firmness. The next stage is enlargement of the spleen, ascites and some jaundice. The Van den Bergh reaction may assist. Chronic gastritis is a common precursor of cirrhosis. This is associated with loss of appetite, flatulence, nausea and vomiting. The vomit consists of much mucus owing to an excess of this substance in the stomach.

Splenic Anaemia is often exposed by a copious haematemesis. In a routine examination the spleen may be felt to be moderately enlarged. There is a persisting secondary anaemia with a leucopenia. The fragility of the red cells is normal or slightly less than normal.

Keys and Walther[52] describe a case in which a diagnosis of duodenal ulcer was made, though without radiological or test meal evidence. Just before death the opinion was changed and at the post mortem lymphosarcoma was found.

SEQUELAE:

1. Haemorrhage

I have already treated this as a symptom.

2. Perforation

In my series I have encountered 22% of the cases where perforation has occurred at some time. At operation each was shown to be due to be the result of a chronic ulcer. Five out of the thirteen cases of gastric ulcer had perforated, while two out of 16 cases of duodenal ulcer had perforated. One of the patients had already had a gastro-enterostomy performed five or six years previously. In that interval he had also suffered from a haematemesis.

The onset of perforation is startling. A man of 55 had been sent to my outpatient department for investigation. As I passed through the waiting hall I noticed him suddenly doubled up with pain. He looked grey, anxious and drawn. His legs were drawn up to his chin. He crouched, unable to speak. He told me a little later that he didn't know how we moved him to an examination couch. He thought he had died. His abdomen was as rigid as a board. His doctor's letter gave his history. At operation a chronic gastric ulcer on the lesser curvature was found to have perforated. In spite of early operation (4 hours after onset) the man eventually died from a sub-phrenic abscess.

After a short interval the rigidity and much of the pain pass away quite considerably but rarely absolutely. Observation, however, reveals a general deterioration in the patient's condition. The pulse rate begins to rise. Pain begins to become more intense and vomiting starts. The abdomen then begins to show signs of general peritonitis. If the perforation passes into the lesser sac, such may not ensure but rather a more localised inflammation and subsequent pus formation.

Gastric Fistula:

This is a localised abscess breaking through the skin so that a communication to the stomach is established.

Gastro-colic Fistula:

A similar happening as above but occurring so that the communication is between stomach and transverse colon. Clinically erructation of obnoxious gasses is experienced. Faecal matter is vomited and undigested food will be passed very shortly after food. The radiograph proves the case.

While perforation is a surgical matter, the physician has to ensure that no tabetic crisis has taken place and that the pain is not due to pleurisy. The surgeon has to decide for or against operation. The interval between perforation and recovery from shock to

permit of operation has also to be considered. The following disorders run through the mind - gallstone colic, acute appendicitis, acute pancreatitis, extra-uterine pregnancy and pyosalpinx. The essential decision resloves itself as to the advisability of immediate operation.

The main step in the operation is to isolate the leaking ulcer and to close the stoma. Whether to do more depends in the first place on the condition of the patient and in the second place on the skill of the surgeon.

3. Perigastric and Periduodenal Adhesions:

In any chronic inflammatory condition adhesions form. It is so in peptic ulcer. Adhesions form between the affected part and adjacent organs. Local kinking may result in interference with the passage of food. It is only when such adhesions are dense that this happens. Most chronic ulcers have some degree of adhesion formation. Clinically the result is in many ways dealt with below.

4. Cicatrical Contractions:

(a) Hour-glass stomach

As a result of contracture two pouches are formed so that the shape of an hour glass is taken up. It may be possible to recognise a sulcus between the two pouches by distending the stomach with gas. X-ray investigation clinches the diagnosis.

4. Cicatrical Contractions (Cont'd)

(b) Pyloric Stenosis:

I would like to suggest that pyloric stenosis is encountered more commonly than hour-glass stomach because of the comparatively narrow canal involved. The relative incidence of duodenal and gastric ulcer is a partial explanation. In the early stages narrowing at the pylorus is suggested by an increase in or the onset of vomiting. It is only late in the progress of the complication that copious vomiting results. The pylorus itself is often palpable. Both (a) and (b) are easily demonstrable by X-ray. One fallacy may be mentioned. It is considered better to re-X-ray after adminstering atropine. This is said to prevent spasm.

5. Carcinoma Supervening in Gastric Ulcer:

Earlier in the thesis I have examined the frequency and incidence in this connection. The conclusion then reached was that carcinoma did not occur with any greater frequency than in patients free from ulcer. One has to bear the possibility in mind in a differential diagnosis.

P E P T I C U L C E R (Cont'd)

RADIOGRAPHY

There are radiological signs common both to chronic gastric and chronic duodenal ulcer. It is, however, more convenient to discuss the X-ray findings under the two headings. In this section I am particularly indebted to Carman's book.(49) The signs alluded to are those following the administration of a barium meal. This may have also been given six hours before examination.

"A". GASTRIC ULCER:

The radiological signs of gastric ulcer may be divided as follows:-

1. Primary Signs:
   - (a) Niche
   - (b) Accessory Pocket
2. Secondary Signs:
   - (a) Spastic Manifestations.
     - i. Incisura
     - ii. Spasmodic Hour-glass stomach.
     - iii. Diffuse spasm.
   - (b) Retention from the six-hour meal.
   - (c) Gastric Hypotonus and lessened gastric motility.
   - (d) Acute fish-hook form of stomach.
   - (e) Alterations of peristalsis.
   - (f) Localised tenderness.

Any one of the two signs in (1) is practically pathognomonic. The signs in (2) are in general fairly conclusive. In my own cases a diagnosis has only been made on signs in the former group. Diagnosis for treatment has been made on less evidence, but no case of this type has been included in the series.

Primary Signs:

(a) Niche - This is a budlike prominence on the peripheral outline of the stomach (see plates 2, 3, 6, & 10). It is variable in size and may be seen in any portion of the stomach. As has been said previously, the majority of ulcers occur on the lesser curvature. The niche is due to the ulcer penetrating through the stomach wall. It is common experience to find a niche disappear with pleasant rapidity. Barclay(50), however, has warned us that a niche may be seen when only an erosion is present. A niche empties and fills with the stomach and accompanies the gastric wall in its movements.

(b) Accessory Pocket - Following the penetrating of the ulcerating process into an adjoining structure an accessory pocket is formed. The characteristic feature of such an occurrence is that the contents are in layers. The lowest portion is opaque, on this is a translucent area which is capped by a small gas bubble.

Secondary Signs:

(a) Spasmodic manifestations - which follow are strong presumptive signs of the presence of ulcer. They are of value when the ulcer is not of a sufficient depth to show a niche. They are also of assistance when the ulcer is not on a visible plane.

i. The Incisura - is an indentation of the gastric wall opposite an ulcer. It is rarely seen on the lesser curvature (see plate 1).

It is probably due to a spasm of the circular muscle fibres. There is some variation in depth and width which also depends on the angle of view. As an incisura may occur fairly high up, the stomach has to be carefully watched during filling. The incisura is constant and stationary both in the prone and standing positions. It also remains after the administration of an antispasmodic. In the absence of these criteria the cause is extra gastric. It may be due to such irritating foci as diseased gall bladder and appendix. It may also be due to a duodenal ulcer.

ii. Spasmodic Hour-glass - In a way this is an extreme form of incisura. It is so extreme that there is a relatively short canal joining the two chambers. The indentation is from the greater curvature, thus giving a "B" shape. The same tests are applied to exclude extrinsic factors. It may be impossible to distinguish radiologically between spasmodic hour-glass stomach and the organic form. This latter is due to adhesions from either an old ulcer or from external adhesions or to actual infiltration by an ulcer.

iii. Other forms of spasm - usually involve the pyloric region, though the ulcer may not be in this area. The whole pylorus is vaguely shadowed and cannot be demonstrated even after manipulation.

(b) A distinct residue in the stomach after a six-hour meal is a common accompaniment: though sometimes the only definite evidence to be found, it does not in itself warrant a diagnosis.

(c) Gastric Hypotonus and hypomobility are only contributing signs. The presence of hypotonus may be more than suspicious if such a sign is not in accord with the habitus of that patient. Lessened mobility may be of no importance at all. It is, however, supremely important if fixation is shown at a definite point. At the same time such fixation may be due to any perigastric adhesion.

(d) Acute Fish-hook Stomach may be an abnormality. It is particularly so when there is displacement downwards and to the left. Per se it is not an important sign.

(e) Abnormalities in Peristalsis

Variations are found in the peristalsis of the stomach. The waves may be excessive or may be diminished. Sometimes reversed peristalsis is found. It must be conceded that these abnormalities are not of great diagnostic aid.

(f) Localised tenderness:

In the section on "Symptoms & Signs" I did not place great reliance on the finding of tenderness in the upper abdomen. Lees(37) agrees on this point. Tenderness elicited by a radiologist over a place where he suspects ulcer is, however, of more importance. The radiologist has a definite point to examine. The finding of persistent tenderness in such a place is of much greater diagnostic significance, than tenderness elicited at a clinical examination.

Differentiation from Carcinoma:

The most important differentiation is that from cancer. In general a simple ulcer projects from the lumen of the organ. A malignant growth encroaches on the lumen. Thus there is a filling defect. Such a

defect is permanent. It cannot be altered by palpation, by antispasmodics or at re-examination. It may be that the radiologist suspects a portion of the stomach and having his attention focussed on this area may detect a tumour mass which has eluded clinical examination. Additional evidence of the presence of a neoplasm consists of:-

(a) Alteration in the pyloric function.
(b) Perversion of peristalsis.
(c) Altered motility
(d) Alteration in the size of the organ.
(e) Displacement of the organ.

(a) The function of the pylorus may be such that the opaque meal flows rapidly into the small intestine. Conversely function may be altered so that retention is observed because of obstruction.

(b) The absence of peristaltic waves over a definite area of stomach contour is a valuable sign of cancer.

(c) When a portion of the stomach cannot be moved by manipulation, suspicion of cancer is warranted.

(d) An invasion of the organ by new growth diminishes its size. The presence of a growth at the lower end of the stomach may lessen the size of that organ. On the other hand a small obstructive lesion would bring about considerable dilatation.

(e) Infiltration by a tumour may press the remaining part of the stomach away from the normal position. The displacement observed is upward and to the left.

"B" DUODENAL ULCER

The radiological signs of duodenal ulcer may be

conveniently subdivided as direct and indirect. The former alludes mainly to appearances of duodenum itself and the latter to changes in the stomach.

1. Direct:

2. Indirect:

   (a) Alteration in gastric tone
   (b) Alteration in gastric peristalsis.
   (c) Alteration in gastric motility.
   (d) Gastrospasm.

1. Direct Signs:

These are shown by deformity of the duodenal contour more especially of the first part. It is known as the duodenum cap. These alterations may be due to organic distortion, organic change plus spasm or to spasm alone.

(a) General distortion - giving the appearance of branched coral, may be seen either over the entire border or over just one border. It is largely due to spasm which is unvarying. It gives a constant picture in a series of plates. The sign is of great value.

(b) Deformity of the Basal Border - Instead of a smooth base line, the cap shows a shaded filling defect. It may be very small (See Plate 13, etc.)

(c) The Niche-type Deformity - corresponds to the niche described in gastric ulcer. It is of great diagnostic importance. The ulcer is visualized as a barium filled recess projecting from the bulb. The size may be quite small and is rarely as large as that found in gastric ulcer. Spasmodic deformity may accompany this organic change.

(d) Incisura Type of Deformity - is a further change corresponding to that found in gastric ulcer. It is spastic, small but sharply defined. As in the case of the incisura found in gastric ulcer a bilateral incisura is sometimes encountered. The resulting condition is termed hour-glass duodenum.

(e) Small Bulb - An abnormally small bulb shadow is produced by an ulcer stenosing the duodenum. In such a case only the proximal portion is filled. Significance should only be attached to this sign providing signs of obstruction are also found. This is because a small bulb is sometimes present in health.

(f) Accessory Pocket - A perforating ulcer which has invaded tissues outside the duodenum shows up as a projection. It is almost a distinct entity and may be layered in a similar manner as that of a gastric accessory pocket.

(g) A Diverticulum is continuous with the bulb. It is usually associated with and proximal to a stenosing ulcer.

The diagnosis of duodenal ulcer from direct signs rests essentially on a constant deformity. Frequently screen examination is adequate. The method of plating a series of exposures is used to demonstrate a persistent alteration in contour.

"In an overwhelming preponderance of cases a constant bulbous deformity means duodenal ulcer (49,p.469). Such a deformity may also be due to adhesions following inflammation occurring in the right upper quadrant of the abdomen, such as cholecystitis. It may also be due to carcinoma. Malignant change in the duodenum

itself is, however, very rare[32]. Extrinsic causes may also occasionally produce a constant deforming of the cap. Such include appendicitis and cholecystitis. This is particularly interesting in view of Wilkie's triad: duodenal ulcer, gall bladder disease and appendicitis being present concomitantly.

2. Indirect Signs:

(a) Alteration in gastric tone:

The rule is to find hypertonicity. This is due to a reflex spasm induced by the ulcer and/or to the effort to overcome commencing stenosis of the duodenum. The latter may be organic from scarring or it may be persistent spasm. It is well to remember that hypertonus may be a normality. When obstruction becomes severe hypotonus follows. The antrum is the first part of the stomach to be affected but soon the remainder dilates.

(b) Alteration in gastric peristalsis:

Normally one or two peristaltic waves are seen in progress at the same time. In hyperperistalsis three or four may appear. The phenomenon is commonly seen even in non-obstructive lesions. Hyperperistalsis may accompany disease of the gall bladder or appendix. It is also found normally in the "steer-horn" stomach.

(c) Alteration in gastric motility:

The barium meal given six hours before screen examination will in these circumstances be seen even as far as the transverse colon, instead of in the caecum. The barium swallow passes quite rapidly through the pylorus. It is almost an evacuation of the stomach. If obstruction is present the meal passes more slowly. In advanced conditions part of the six-hour meal will still remain in the dilated

stomach. Even here hyperperistalsis may be found.

Hypermotility is also present in gastric carcinoma and will be found in any person suffering from diarrhoea.

(d) Gastrospasm:

The usual spastic manifestation is an incisura. It may pass slowly along the stomach or remain stationary. Spasm produced by extra-gastric conditions disappear on re-examination when an antispasmodic is utilized. Duodenal ulcer is, however, an exception to this rule.

The value of these indirect signs is a little variable. The coincidence of hyperperistalsis with gastric retention and a normal stomach outline is strong presumptive evidence for duodenal ulcer. Hyperperistalsis alone is not pathognomonic but is strongly indicative. This is especially so when sound clinical judgment is also applied.

Differentiation from Duodenitis(51)

There are four findings which distinguish this entity:-

(1) Rapid passage of barium through the duodenum.
(2) A coarse reticular pattern.
(3) Absence of ulcer crater and niche.
(4) Absence of gastric retention and obstruction.

The differential diagnosis is, however, of some difficulty.

TEST-MEAL.

"A" The older single test-meal consisted of:-

(a) The administration of a meal consisting of a pint of weak tea without milk. A little sugar was permitted. In addition two slices of toast were given. The meal was taken in the morning before the patient had broken his fast.

(b) An hour later the stomach was emptied and the contents preserved for measurement and analysis.

The drawbacks were four-fold:-

(1) The observer was in ignorance as to the contents of the stomach before the meal was given.

(2) The results of the examination could only be given on empirical grounds.

(3) There was a lack of variety of findings compared with the more modern fractional test-meal.

(4) I personally have found the necessary filtration of gastric contents tedious.

"B" The Factional Test-Meal is more widely used and has become more freely accepted. The method was evolved by Rehfuss and his co-workers in U.S.A. It is necessary that the conditions of the test-meal should be the same for each case.

Nothing should be given by mouth after 9 p.m. Bennett[54], however, advises a glass of milk containing two level teaspoonfuls of powdered charcoal during the evening. The teeth should not be cleaned to avoid accidental buccal haemorrhage.

At 9 a.m. next day an oatmeal gruel is administered. It may be flavoured if necessary. It is an advantage to have this white so that bile or blood are more easily detected. Ryle's modification of the Einhorn or Rehfuss tube is passed so that the marking on the tube shows that the end lies well in the fundus of the stomach. The meal is better swallowed with the tube in situ. Sometimes vomiting and retching take place during its passage. Previous to the meal the total fasting juices are withdrawn. After a quarter of an hour, and at every subsequent ½-hour 10-15 cc. aspirated. Usually the stomach is empty in from 2 to 2½ hours. If not the analysis is continued until there is no more to withdraw.

Bennett & Ryle[53] summarised their findings in an experiment on a hundred medical students. The standard test-meal chart contains a shaded area corresponding to the limits of the results. This has become the normal limit.

The analysis consists of many parts:-

1. $\frac{N}{10}$ NaOH is titrated against 5 cc. of stomach contents. The result expressed in cc. used is plotted and is the Ewald scale. The comparative percentage of HCl is also included in brackets. In the presence of free HCl Topfer's solution imparts a red colour to the specimen which returns to orange-yellow to show an end point. At this stage a few drops of phenolphthalein indicator are added. A second end-point indicating the total chlorides is the appearance of a pink colour. Throughout these procedures the tested solution should be freely shaken more especially when there is any excess of mucus.

2. So long as any of the meal remains in the stomach a few drops of weak iodine solution will turn the sample blue, indicating the presence of starch. The disappearance of this reaction times the emptying rate (see chart 1.)

3. The presence or absence of food, bile and blood are noted. Any excess of mucus is also charted. This latter cannot be accurately measured. The quantity is appreciated by experience.

4. If Bennett's method of giving charcoal is employed the presence of carbon signifies marked Pyloric obstruction.

5. Lactic acid is tested for.

6. A microscopical examination will reveal the presence of starch, bacilli, torulae and sarcinae, and undigested food or foreign matter, as well as blood cells.

The results are plotted on the standard charts.

Ryle states, "The important conclusion to be drawn -------- is that neither hyperchlorhydria nor achlorhydria can in themselves be regarded as pathological findings"(53).

It is only possible to give general principles of use in diagnosis. The classifications to which radiological findings naturally fall have no analagous headings under which to describe chemical investigations.

Peptic ulcer is usually associated with a rising acid curve. This is frequently associated with a fairly rapid emptying rate, (See Chart 1). Even when the meal has passed through the pylorus the gastric

secretion is often high. In addition the amount of fasting juice is excessive. The findings in the investigation of duodenal ulcer are more constant than those of a gastric ulcer.

If pylorospasm is present the resting juice tends to rise. With frank stenosis it becomes copious, e.g. 60 to 120 cc. When obstruction has developed so as to cause gastric dilatation with hypotonus the resting juice is still high but both its acid content and the acid content of subsequent fractions diminishes. A condition bordering on achlorhydria arises. If gastric lavage is performed some acid may be found at subsequent examinations. Hyperacidity does not show such a rapid emptying rate. The chart shows some variation other than a stenognome (Chart 2).

The test-meal is useful and important. It lacks, however, the precise character of radiography.

GASTROSCOPE

There have been numerous attempts at designing a gastroscope. The first to be used in diagnosis was that invented by Mikulicz. In 1932 Schindler perfected a flexible instrument. He was aided by Wolf. This apparatus, known as the Wolf-Schindler gastroscope, is that most generally in use. It consists essentially of

a flexible tube. It is passed through the mouth into the stomach. By means of lenses and electric illumination an image is projected to the eye-piece. The employment of the gastroscope has revealed an undiscovered sphincter. The incisura angularis has been seen to produce a fold on the lesser curvature. Schindler[55] has called it the Musculus Sphincter Antri. It seems to divide the stomach into two anatomical and physiological parts. The precise function is as yet unknown.

The main uses of the gastroscope are (Rose[56]):-

1. An early diagnosis of carcinoma can be made.
2. It serves as a control in the medical treatment of gastric ulcer.
3. It is the only method of diagnosing chronic gastritis, and atrophic gastritis in particular.

I have no personal experience of the use of the gastroscope. It is, however, a valuable adjunct in the diagnosis and control of the treatment of gastric ulcer.

SUMMARY OF CASES

GENERAL STATEMENT

	Male	Female	Total
Gastric Ulcer	13 (41%)	1 (3%)	14 (45%)
Duodenal Ulcer	16 (50%)	2 (6%)	18 (55%)
			= 32

2 (a) HAEMATEMESIS

	Male	Female	Total
Gastric Ulcer	4	1	~~6~~ 5
Duodenal Ulcer	3	1	4
			= 9

(b) HAEMATEMESIS: percentage in relationship to general statement (1)

	Male	Female	Total
Gastric Ulcer	31%	100%	43%
Duodenal Ulcer	23%	50%	22%
			= 28%

S U M M A R Y O F C A S E S (Cont'd)

3. (a) PERFORATION

	Male	Female	Total
Gastric Ulcer	5	Nil	4 = 7
Duodenal Ulcer	2	Nil	2

(b)

PERFORATION: percentage in relationship to general statement (1)

	Male	Female	Total
Gastric Ulcer	38%	Nil	29% = 22%
Duodenal Ulcer	13%	Nil	11%

4. AGE AT ONSET OF SYMPTOMS

Years	Number
10-20	3
20-30	10
30-40	12
40-50	6
50-60	1

5. DURATION OF SYMPTOMS

Years	Number
1	6
2	4
3	1
4	4
5	4
6	1
7	4
8	0
9	1
10+	7

Name and Occupation	Age Sex	Diagnosis	Confirmed	History	Main Symptoms	Treatment
Becks, F Shop Assistant	20 F	Two Gastric Ulcers, pyloric.	X-ray and operation.	Upper abdominal pain for 2 yrs. Haematemesis 1 year after.	Very severe pain an hour after food. Relieved by small meals.	(1) Diet (2) Histidine did not respond. (3) Gastro-jejunostomy & appendicectomy.
Barbour, William Lorry Driver	38 M	Duodenal Ulcer.	Operation	No previous history (say one year)	Perforation	Suture without drainage.
Barnes, Charles. Unemployed	46 M	Gastric Ulcer near pylorus.	Operation	Indefinite dyspepsia (say 5 years)	Perforation	Suture with drainage. (Large chronic ulcer).
Bowman, John Fireman	36 M	Duodenal Ulcer.	X-ray	1927 Perforation. Recurrent attacks of pain since.	Haematemesis	Routine diet and alkalies.

Name and Occupation	Age Sex	Diagnosis	Diagnosis Confirmed by	History	Main Symptoms	Notes on Treatment	Result
Brown, John Unemployed	56 M	Duodenal Ulcer	X-ray (previous)	1926 Appendicectomy. Repeated Haematemesis 3 occasions	Epigastric pain intensified by food. Passes to Shoulder blades	Refused Operation. Routine diet	
Brown, Sidney Roadman	42 M	Gastric Ulcer (lesser curve)	Post mortem	Vague indigestion for 5 years	Suggested Perforation Left lobar pneumonia	Treated as pneumonia P.M. showed both present, i.e., perforation and lobar pneumonia.	died
Broderick, William Chemical Worker	32 M	Duodenal Ulcer	X-ray Test Meal	10 years duration X-rayed at another hospital some years ago. Nil found.	Burning epigastric pain three hours after food. Relieved by vomiting, alkalies and food.	Routine diet and alkalies	Clinically well.

Name and Occupation	Age Sex	Diagnosis	Diagnosis Confirmed by	History	Main Symptoms	Notes on Treatment	Result
Carter, Joseph Erector (unemployed)	39 M	Duodenal Ulcer.	Operation	Vague indigestion some relation to food for 10 years. Haematemesis on three occasions.	Three admissions to hospital.	Treated medically on previous occasions. This time a non-loop gastro-jejunostomy	Relieved
Davies, Owen Steel Straightener.	50 M	Gastric Ulcer.	X-ray	Three years history of pain ½ hour after food. X-rayed at another hospital 1935. Nil.	As history. Also passed blood. P.R. which has been associated with diarrhea. Pain mainly in left hypochondrium.	Routine diet and alkalies.	
Dolan, John Baker's helper	48 M	Gastric Ulcer (lesser curvature) Very fibrosed.	Operation	7 years history of indigestion. More acute in last 3 weeks.	Perforation	Suture and drainage.	

Name and Occupation	Age Sex	Diagnosis	Diagnosis Confirmed by	History	Main Symptoms	Notes on Treatment	Result
Duckling, George Unemployed	41 M	Gastric Ulcer (Pyloric antrum)	Operation	Flatulence and fullness after meals at intervals for 4 years.	Perforation	Difficult access. Suture and drainage.	
Fletcher, Richard Miner	33 M	Gastric Ulcer.		Pain in epigastrium 15 - 20 minutes after food. Duration 4 years with intervals of freedom.	Haematemesis with melaena.	Starvation followed by diet and careful alterative therapy. (under care of medical colleague).	
Fox, Richard Joiner	26 M	Duodenal Ulcer.	Operation	None	Perforation	Suture without drainage.	

Name and Occupation	Age Sex	Diagnosis	Diagnosis Confirmed by	History	Main Symptoms	Notes on Treatment	Result
Galliford, John	53 M	? Gastric Ulcer		Stabbing pain near left nipple for 2 years at intervals. Morning vomiting some cough.	Haematemesis with melaena	Meulengracht's diet.	
Foster Mrs Frances	55 F	? Duodenal Ulcer		Haematemesis 24 years ago. Recurrent upper abdominal pain ever since.	Haematemesis. Pain to right of midline in epigastrium.	Meulengracht's diet.	
Gibson Mrs Agnes Midwife	48 F	Duodenal Ulcer (pyloric stenosis well marked)	X-ray and operation	10 years history of pain in epigastrium two hours after food. Very periodic	Increasing vomiting after meals in last 6 months	Posterior gastro-enterostomy stomach found to be very dilated.	

Name and Occupation	Age Sex	Diagnosis	Diagnosis Confirmed by	History	Main Symptoms	Notes on Treatment	Result
Gibson, Edgar Plater's Help	28 M	Gastric Ulcer (Low on lesser curvature).	X-ray and operation	7 years history of epigastric pain 1½ hours after food. Vomiting present which relieved pain as did the taking of small meals.	After preliminary medical treatment operation had to be performed. Pain persisted. Vomiting increased.	Gastro-jejuno-stomy.	
Gibson, Stephen Unemployed	34 M	Duodenal Ulcer. (large and fibrosed).	X-ray and operation	Pain at varying intervals after food in epigastrium for 5 years. Only affected when doing hard work.	1st X-ray only revealed pylorospasm. Second X-ray showed in addition fair sized ulcer on bace of duodenal cap.	Gastro-jejuno-stomy.	

Name and Occupation	Age Sex	Diagnosis	Diagnosis Confirmed by	History	Main Symptoms	Notes on Treatment	Result
Grimes, Percy Wire galvaniser	37 M	Duodenal Ulcer.	X-ray	Pain for 6 yrs. Has occurred mainly in lower abdomen. Very long intervals between attacks.	Pain 3 hours after food. Commences in right iliac fossa, passes upward across epigastrium to left iliac fossa. No sign of lead poisoning.	Diet and alkalies including oranges and marmite.	
Harvey, Jesse (male) Unemployed	48 M	Duodenal Ulcer.	Operation Confirmed. X-ray negative.	Epigastric pain 2 hrs after food for a few months. No periodicity.	As in history. In view of uncertainty of diagnosis a laparotomy was performed.	Small fibrosed ulcer on antero-superior aspect of duodenum found. Gastro-jejunostomy (oblique) performed.	

Name and Occupation	Age Sex	Diagnosis	Diagnosis Confirmed by	History	Main Symptoms	Notes on Treatment	Result
Jackson, Thomas Miner	59 M	Appendicitis.	Operation and X-ray	Perforation thought to have occurred 6 months previously. No operation notes available.	Profuse melaena.	Operation performed. No stomach lesion found. No cirrhosis. Chronic appendix removed.	
Jones, Joseph Ice Cream Vendor	20 M	Duodenal Ulcer.		Has had indigestion for 7 yrs. Along with it has been lienteric diarrhea	Haematemesis	Two days starvation with salines. Then routine diet and alkalies.	
Kemp, Arthur Cellar man	33 M	Gastric Ulcer	Previous operation and X-ray. Test meal.	Perforation 4 yrs. ago. 1 yr later had haematemesis. Vomiting in last few mths. Previously treated by diet.	Epigastric pain with no relationship to food.	Diet and alkalies X-ray at end of treatment revealed no abnormality.	

Name and Occupation	Age Sex	Diagnosis	Diagnosis Confirmed by	History	Main Symptoms	Notes on Treatment	Result
McPartland, Peter Process Worker	34 M	Duodenal Ulcer.	X-ray	One year's history of pain passing from epigastrium to the left shoulder blade. Treated previously. Then thought surgery would be needed.	Pain 2 hours after food. Recurred 5 months after previous treatment.	Diet and alkalies. Detained for five weeks.	
Molloy, Patrick Labourer	47 M	Gastric Ulcer.	X-ray	History of 13 years of epigastric pain. Attacks last 3 weeks - interval usually 6 months.	Pain 3 or 4 hours after food. Has much vomiting which relieves.	Diet and alkalies. First 3 days treatment prolonged to 6 days.	

Name and Occupation	Age Sex	Diagnosis	Diagnosis Confirmed by	History	Main Symptoms	Notes on Treatment	Result
Mitchell, John Clerk	43 M	Duodenal Ulcer.	Previous operation and X-rays.	Has had treatment before. (1) Gastro-jejunostomy. (2) Histidine injection Pains recurrent soon after both. Just before admission became much worse with vomiting and right iliac pain.	A mass was palpated in and around umbilicus. Surgical opinion agreed that malignancy was present. Not accurately diagnosed.	X-ray treatment. Sent to another institution, died. No P.M.	
Moyce, John Labourer	45 M	Gastric Ulcer high in lesser curvature.	Operation	Pain 2 hours after food in epigastrium for 2 years. The last few months the pain has been excruciating passing through to back.	Seen to perforate by me in O.P.D.	Suture with drainage Sub-phrenic abscess drained.	Died.

Name and Occupation	Age Sex	Diagnosis	Diagnosis Confirmed by	History	Main Symptoms	Notes on Treatment	Result
Puckering, John Shearman (Unemployed)	32 M	Gastric Ulcer. (cardiac)	X-ray and previous operation.	Indigestion for 16 years. Perforated in 1933. Gastro-enterostomy. Haematemesis more recently Treated by Histidine, but no good came of it.	Readmission because of another ulcer. The first ulcer was not defined The second developed beyond gastro-enterostomy opening. The third is high in cardiac end.	A difficult patient. Gave routine diet and alkalies. (Note. since writing this series he has been an inpatient for 3 months. Edentulous and a pauper)	
Robson, Thomas Fireman	39 M	Duodenal Ulcer.	X-ray and operation.	In 1935 seen and ulcer suspected. Not revealed. History of epigastric pain for 5 years. In 1936 came back. X-ray positive. Medical treatment not successful.	Pain persisting in spite of negative X-ray.	Operation performed. Gastro-enterostomy.	

Name and Occupation	Age Sex	Diagnosis	Diagnosis Confirmed by	History	Main Symptoms	Notes on Treatment	Result
Taylor, W.R. Chemical Labourer	36 M	Gastric Ulcer lesser (curvature)	X-ray	Pain in epigastrium for 4 years; intervals of 6 weeks. No relation to food.	Had teeth removed early in history. Diagnosis not at all likely without X-ray.	Routine X-ray negative in 3 weeks.	
Warburton, James Unemployed	29 M	Duodenal Ulcer.	X-ray	Pain in epigastrium for 2 years.	Pain comes in 2 - 2½ hours after food. Relieved by alkalies. Vomiting or four occult blood in stools.	Routine for 4 weeks.	
Ward, Fred Reporter	30 M	Duodenal Ulcer (stenosis)	X-ray and operation.	7 years history. Sometimes pain occurred in back. Thinks he passed blood in stools.	Was first admitted for (1) medical treatment. His pain occurred about 10 minutes after food. (2) Vomiting commenced. X-ray still showed ulcer and residue.	(1) medical (2) Gastro-enterostomy.	

Name and Occupation	Age Sex	Diagnosis	Diagnosis Confirmed by	History	Main Symptoms	Notes on Treatment	Result
Wicks, Ruth Housewife	34 F	? Duodenal Ulcer.		1935 Haematemesis 1935 Haematemesis 1937 Haematemesis.	None of these was very severe clinically. The lowest blood count was: RBC 3,690,000 Hb 78%	Routine ferrous sulphate on first occasion. Had long starvation. Did better on second and third occasions with the increased diet.	

Test meal

Chart 1.

GASTRO-INTESTINAL ANALYSIS.

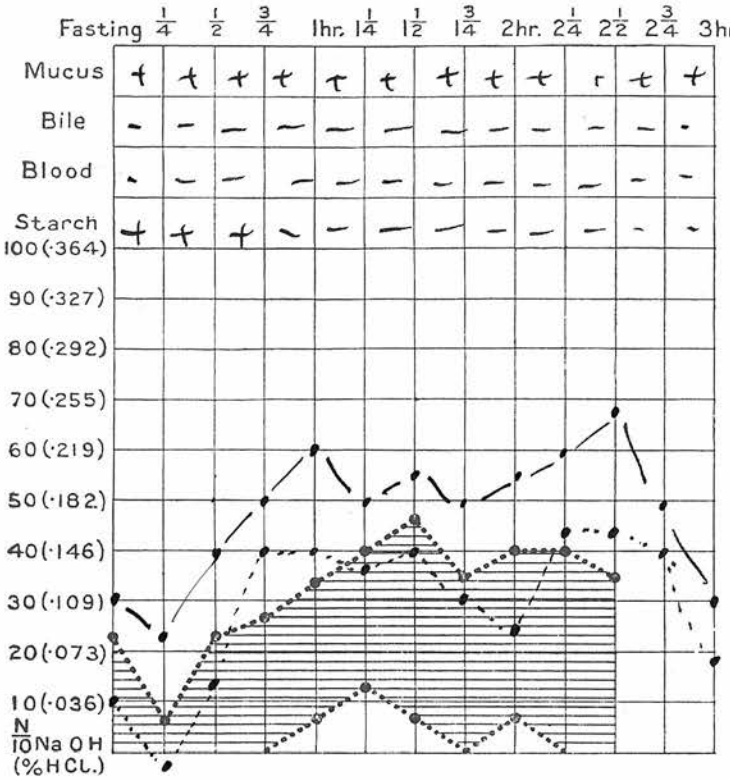
NAME OF PATIENT W. Broderick 21. 1. 37.

1. FRACTIONAL TEST-MEAL. DATE Duodenal Ulcer

Fasting Juice.

Volume. 78cc.

Cells.



One Hour Juice.

Free HCl.

Active HCl.

/

The shaded area represents the limits for free HCl of 80% of normal people

SUMMARY. .....represents free HCl

_____represents total acidity.

No Lactic Acid.

Rapid emptying
increasing acid for 2 3/4 hours.

2. VOMIT.

Date.	Hour.	Volume.	Food.	Mucus.	Blood.	Free HCl.

3. 12 HOUR TEST-MEAL.

Date.	Volume.	Food.	Mucus.	Blood.

4. FÆCES.

Date.	Occult Blood.	Macroscopic Blood.	Mucus.	Pus.	Food Residue.	Bacteriology.

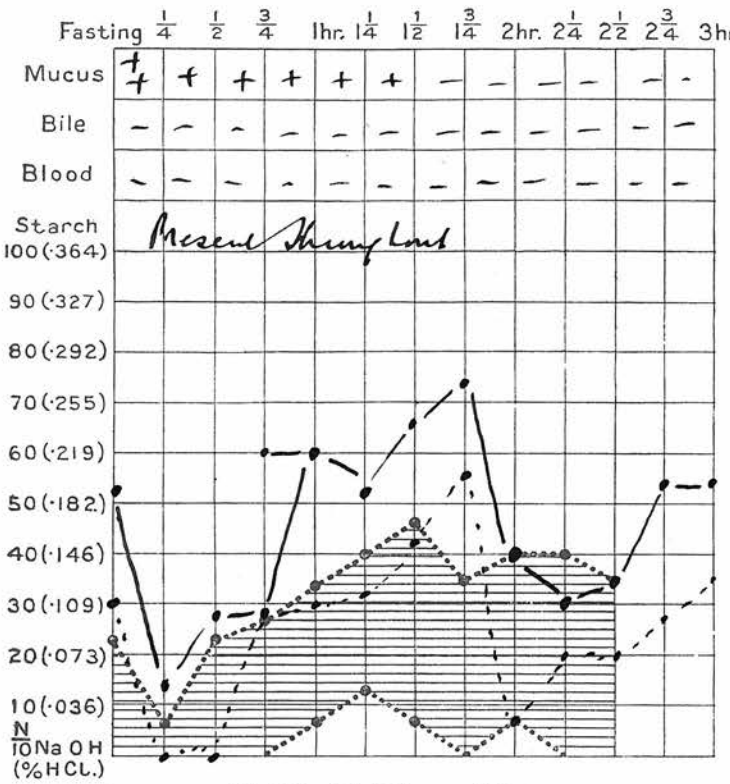
Test-Meal
Chart 2

GASTRO-INTESTINAL ANALYSIS.

NAME OF PATIENT John Chandler

1. FRACTIONAL TEST-MEAL. DATE 5.3.37.

Hyperacidity



Fasting Juice.

Volume. 30cc.

Cells.

One Hour Juice.

Free HCl.

Active HCl.

The shaded area represents the limits for free HCl of 80% of normal people

SUMMARY. .....represents free HCl

_____represents total acidity.

Not a typical chart — Excess of combined acid & free acid.

2. VOMIT.

Date.	Hour.	Volume.	Food.	Mucus.	Blood.	Free HCl.

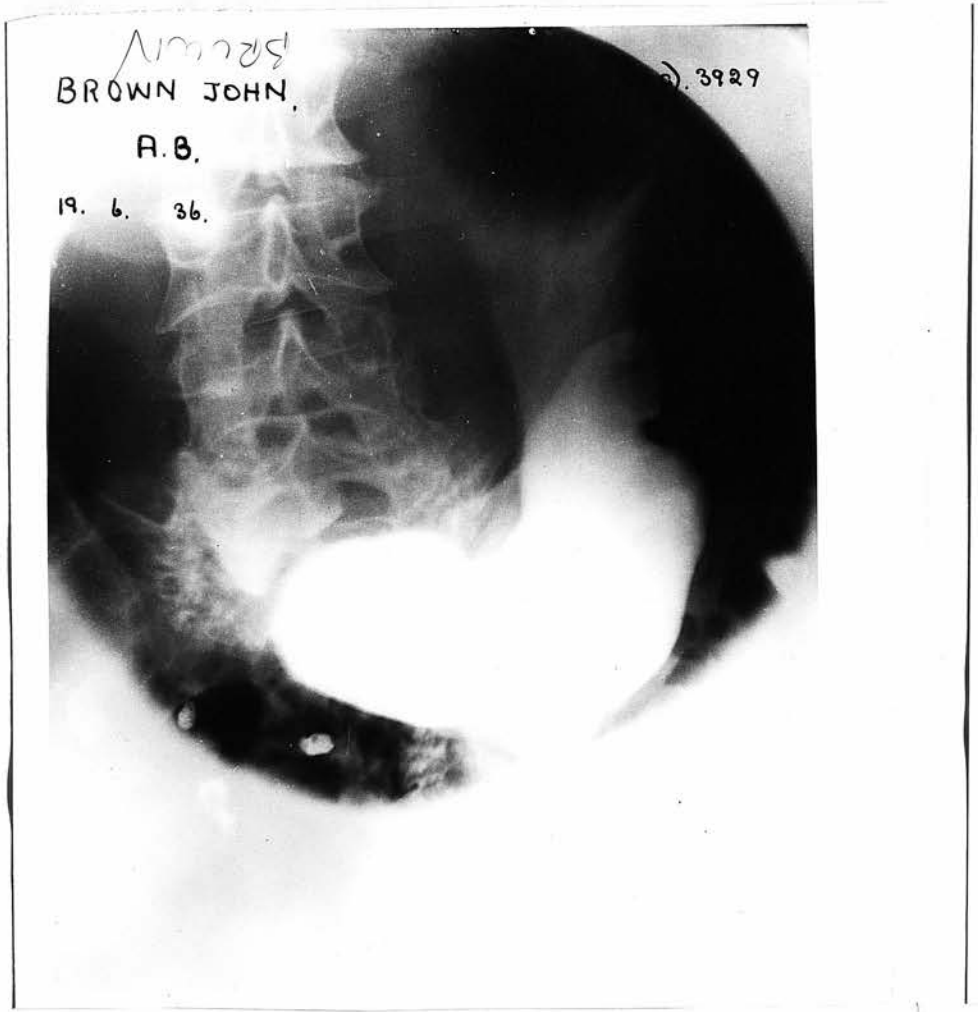
3. 12 HOUR TEST-MEAL.

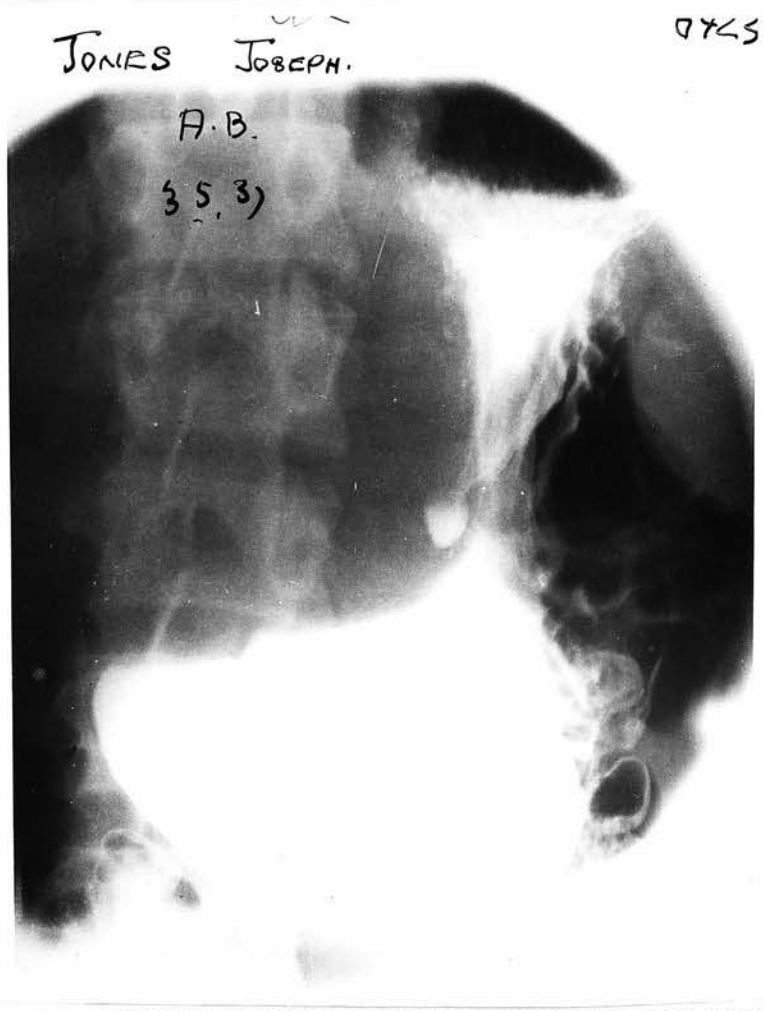
Date.	Volume.	Food.	Mucus.	Blood.

4. FÆCES.

Date.	Occult Blood.	Macroscopic Blood.	Mucus.	Pus.	Food Residue.	Bacteriology.

INDEX FOR X-RAY PLATES





Brown.

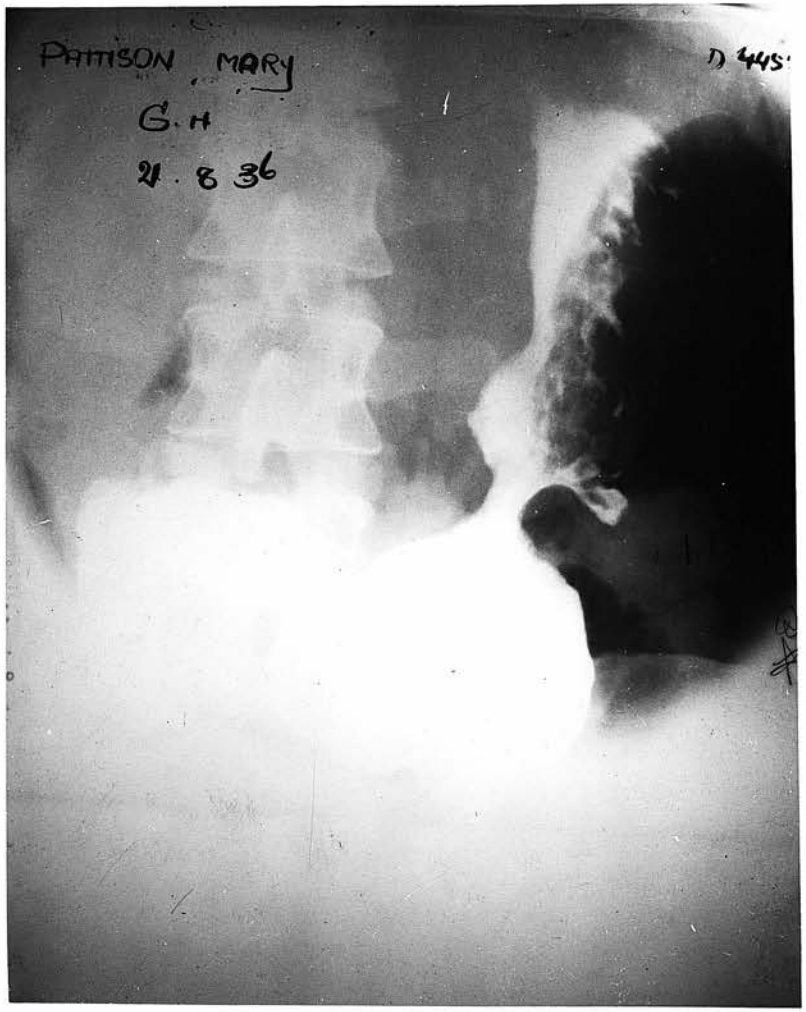
Incisura on cardiac end of greater curvature. Ulcer on posterior aspect.

Plate 1.

Jones.

Large ulcer on lesser curvature. Spasm present.

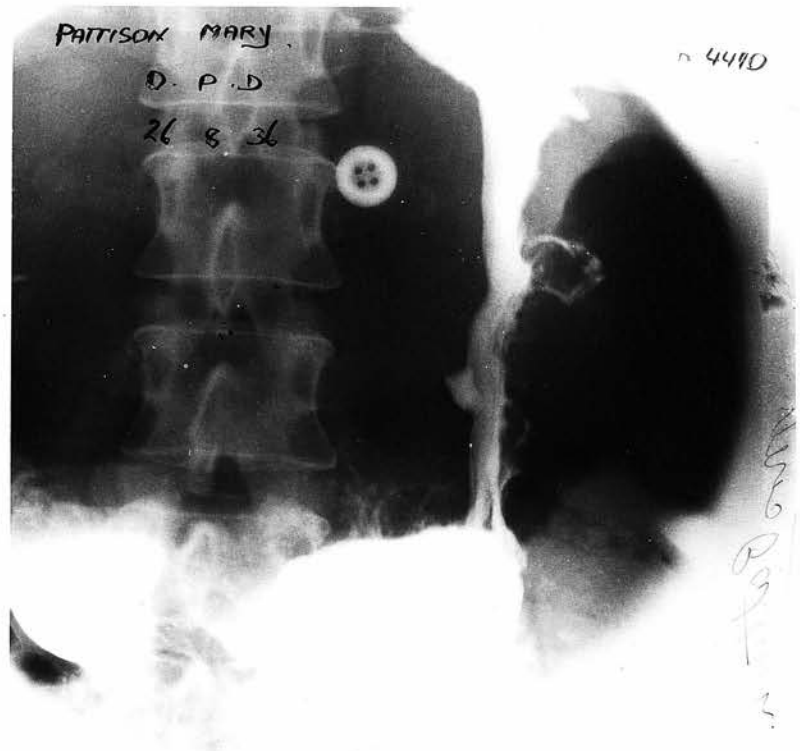
Plate 2.



Patterson Mary.

Large ulcer in cardiac region. Considerable spasm.

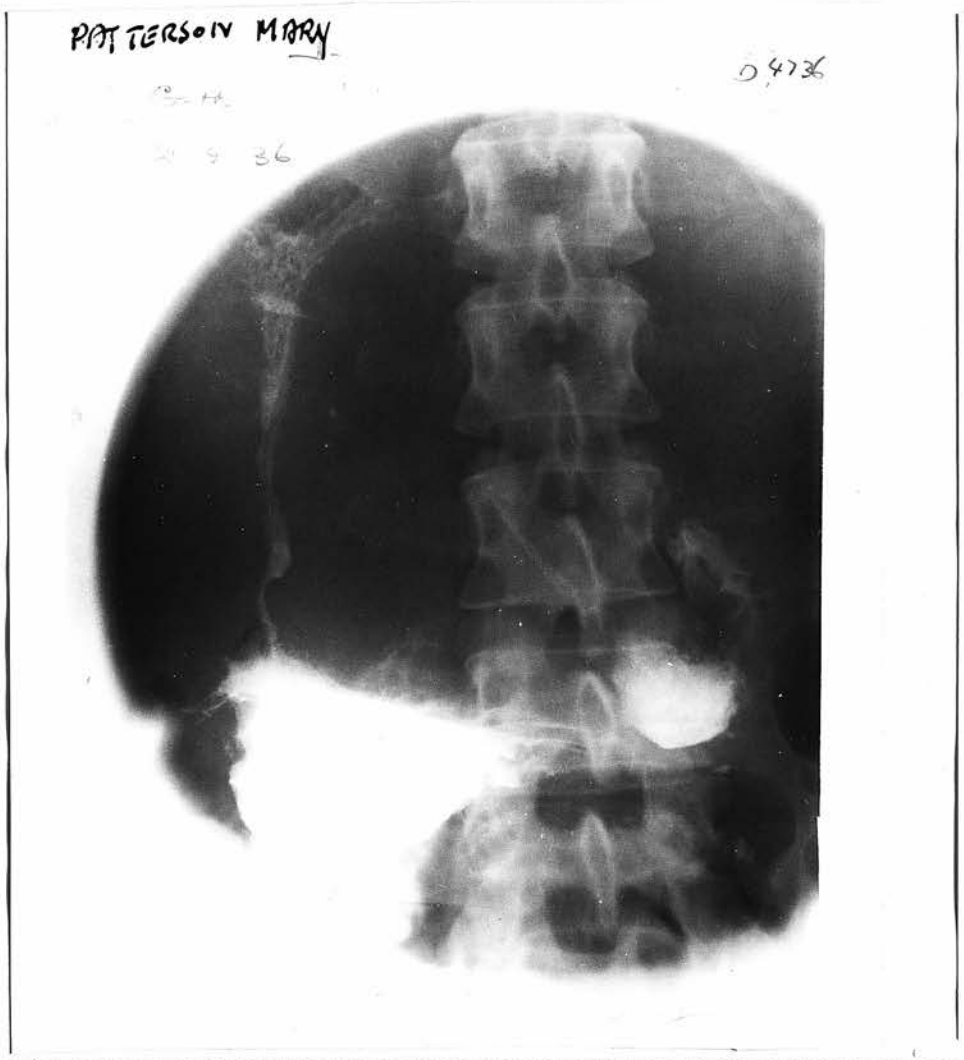
Plate 3.



Patterson Mary. [5 days later]

Same as plate 3. Spasm diminished.

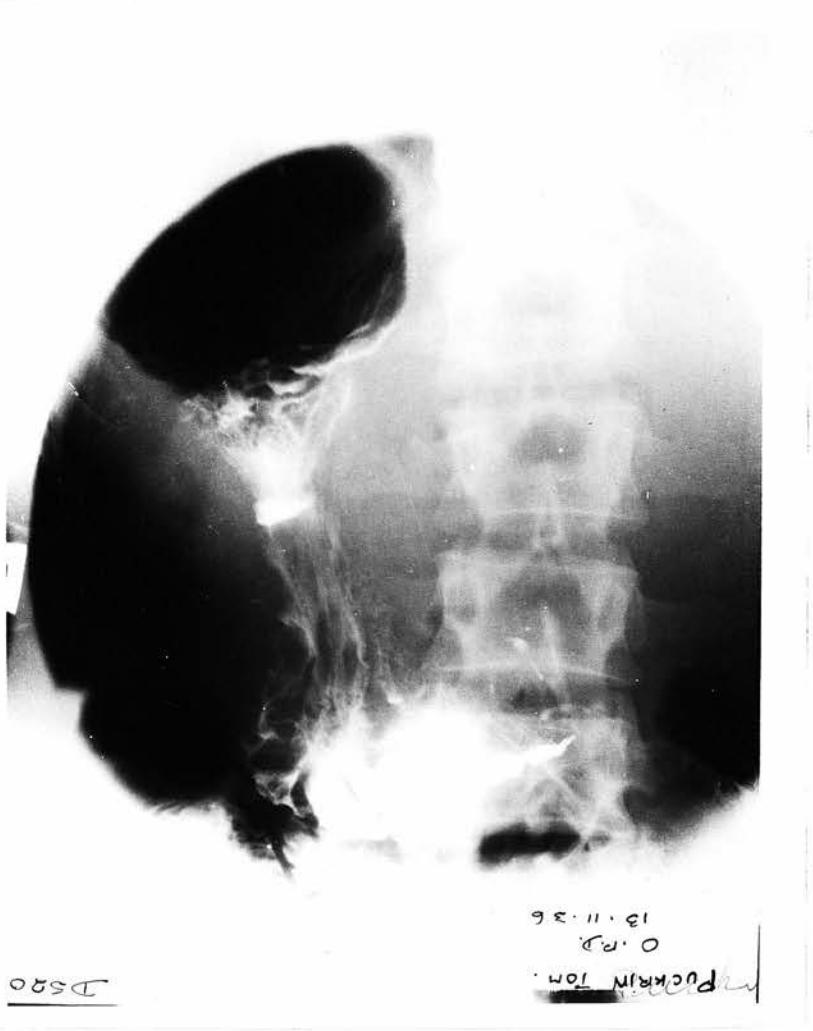
Plate 4.



Patterson Mary. (one month later) Plate reversed

Ulcer much smaller. Spasm greatly diminished.

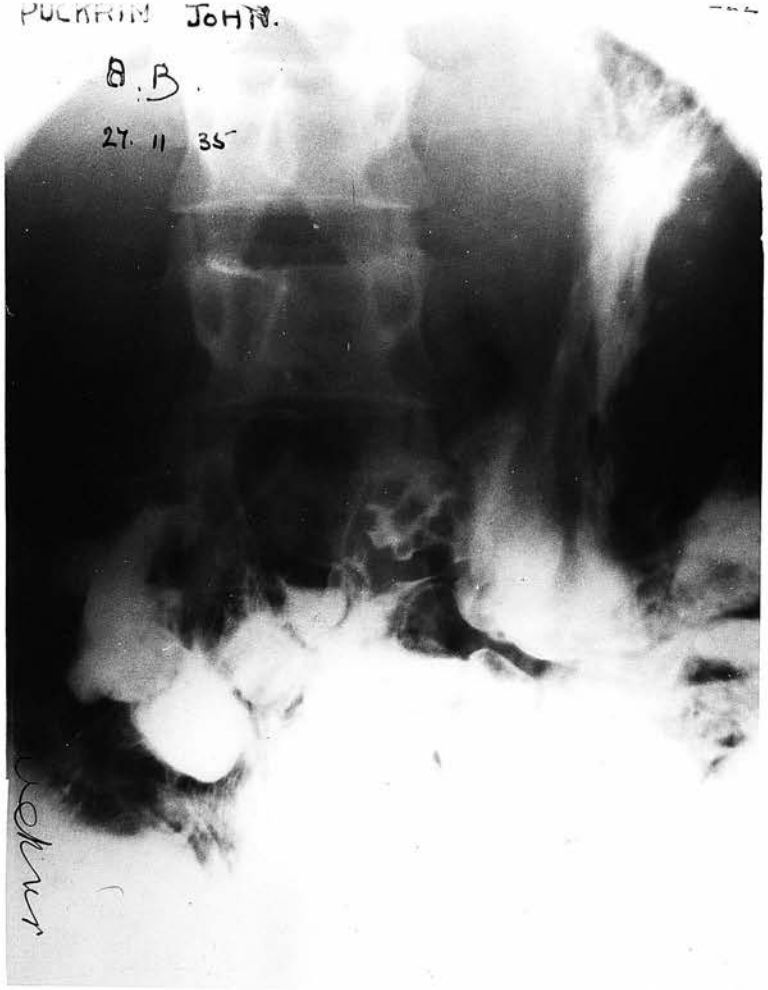
Plate 5.

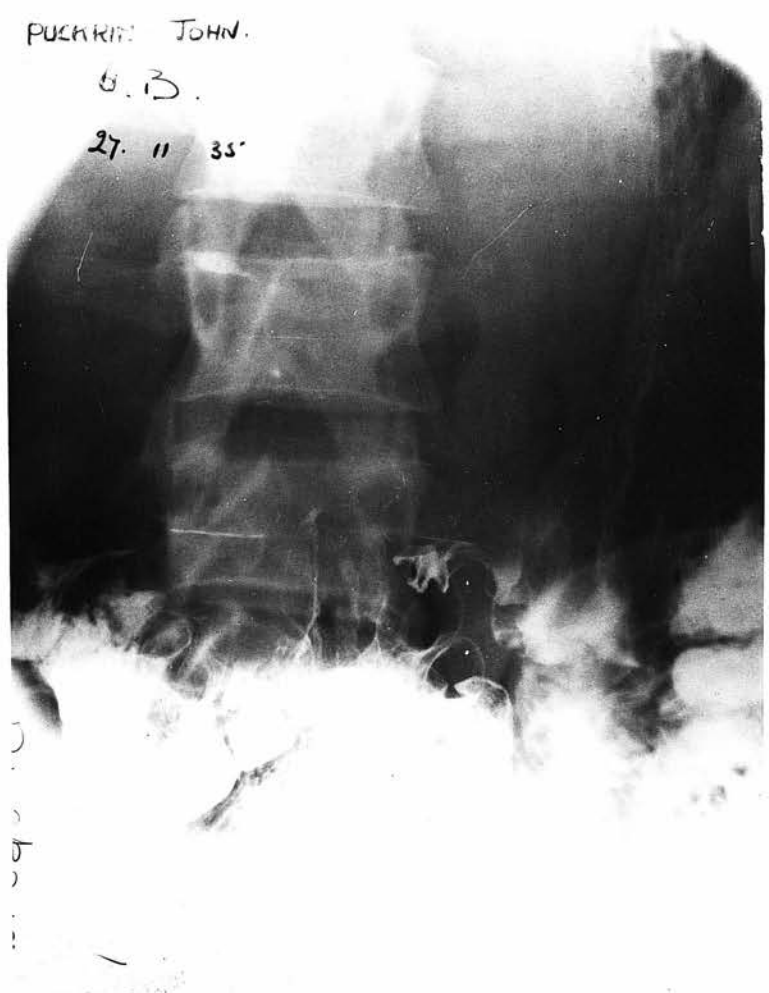


Puckrin. (plate reversed)

Large ulcer at cardiac end of greater curvature.

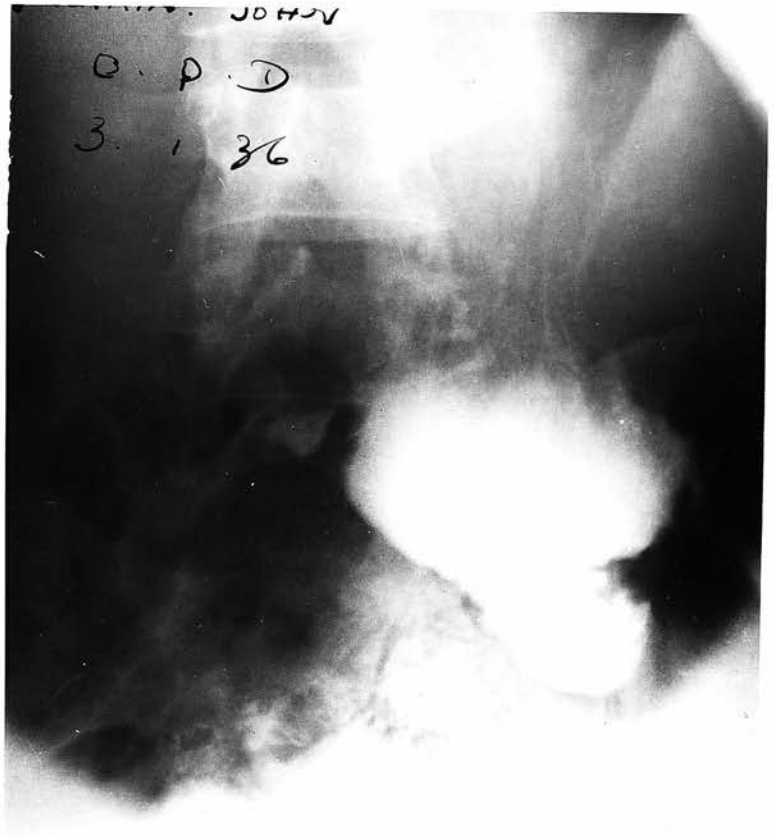
Plate 6.





Puckrin (fortnight later)

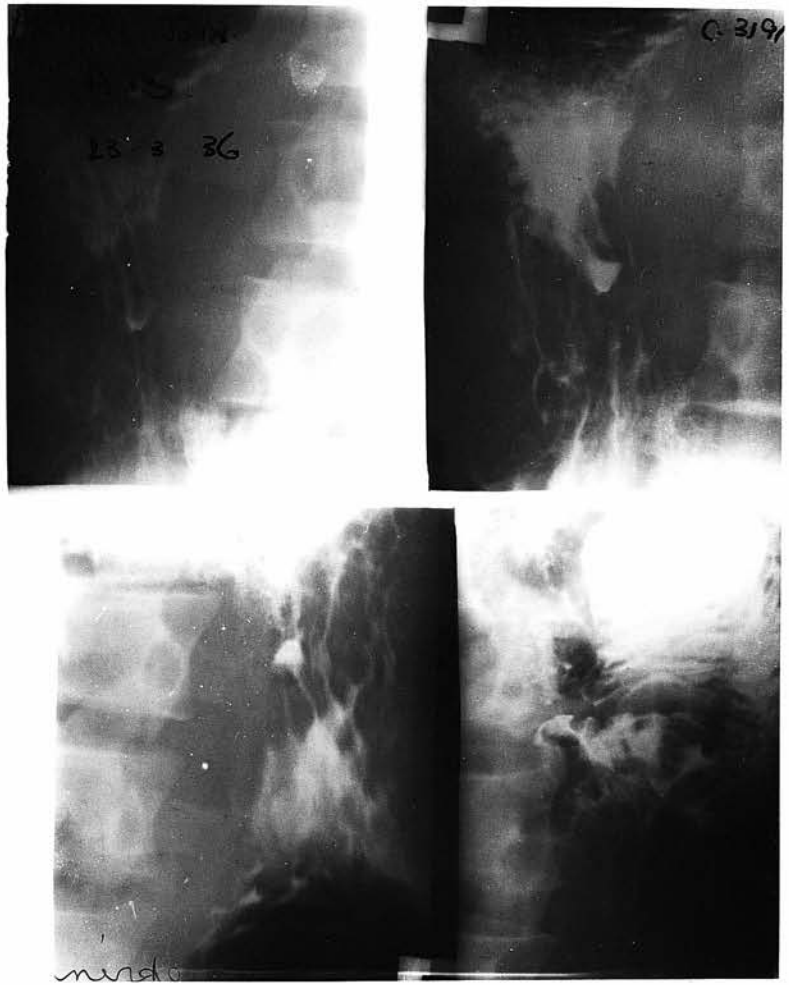
Gastroenterostomy well shown. Large ulcer beyond stoma.
Plates 7 & 8.



Puckrin (two months later)

Meal flows freely through opening. No evidence of ulcer.

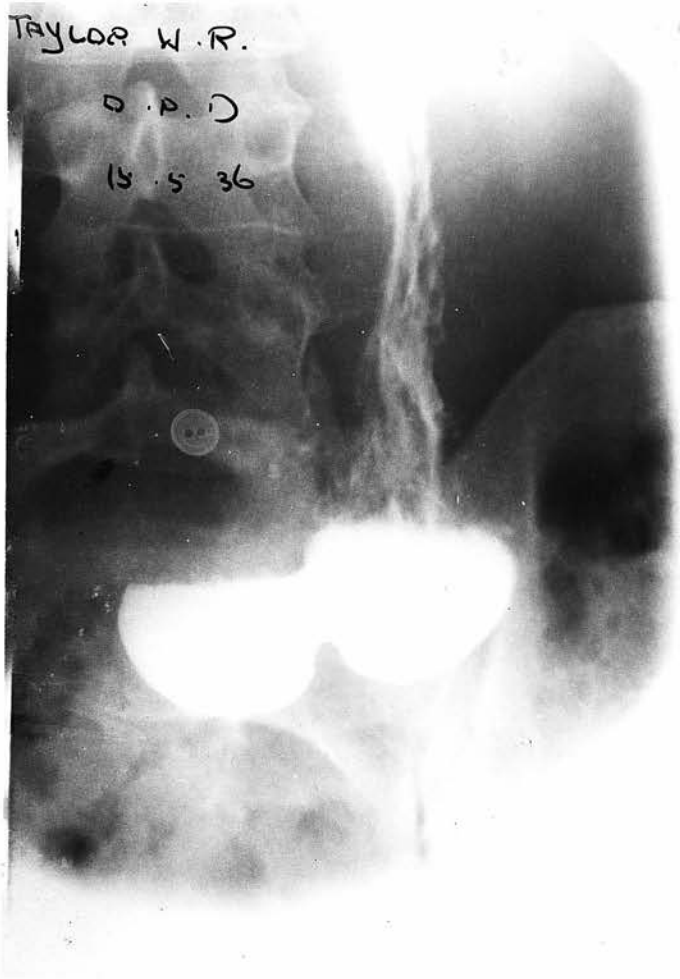
Plate 9.



Puckrin (six months later)

Gastric ulcer revealed again. At the end of nine months no ulcer was shown on screen examination.

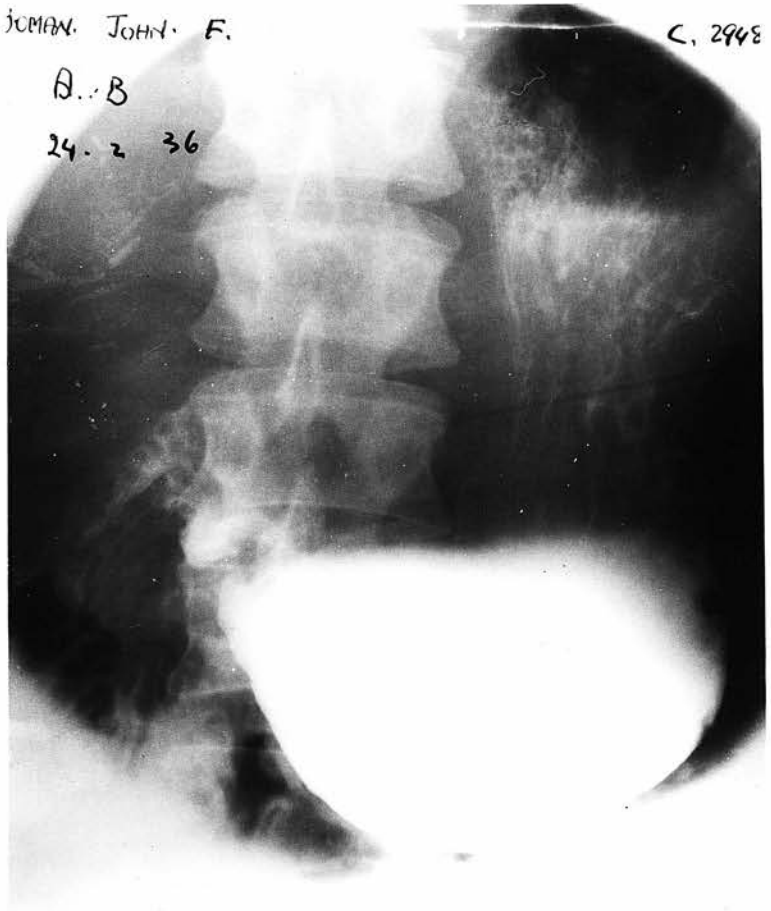
Plate 10.

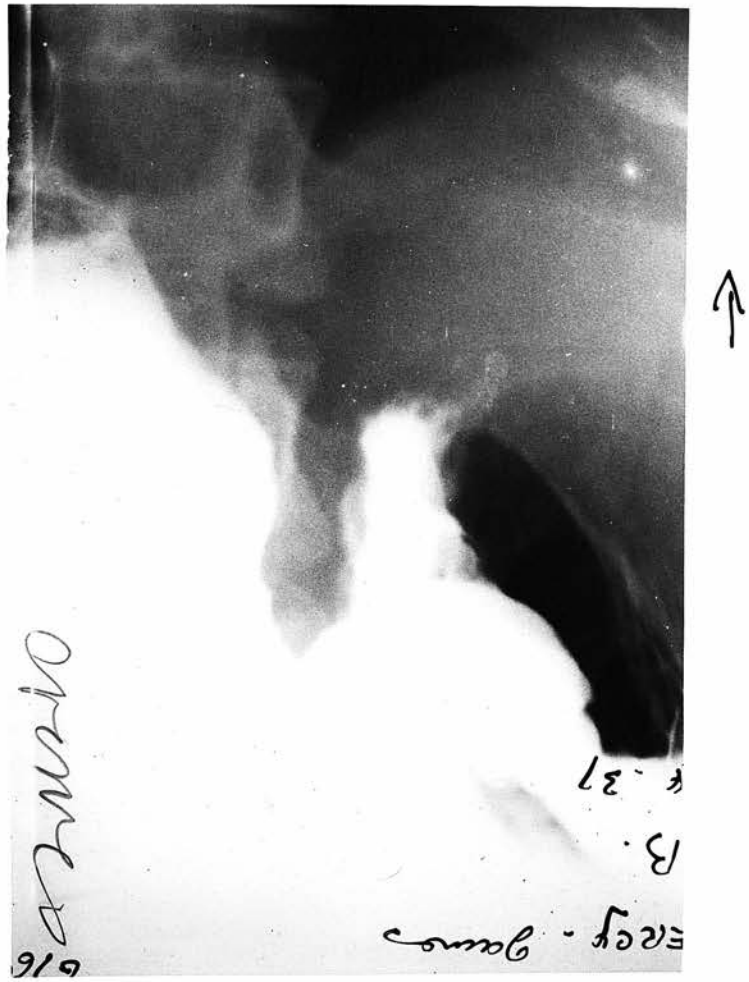


Taylor.

Small ulcer midway between cardiac end and angle of stomach.
(Better shown on screen).

Plate 11.





Bowman.

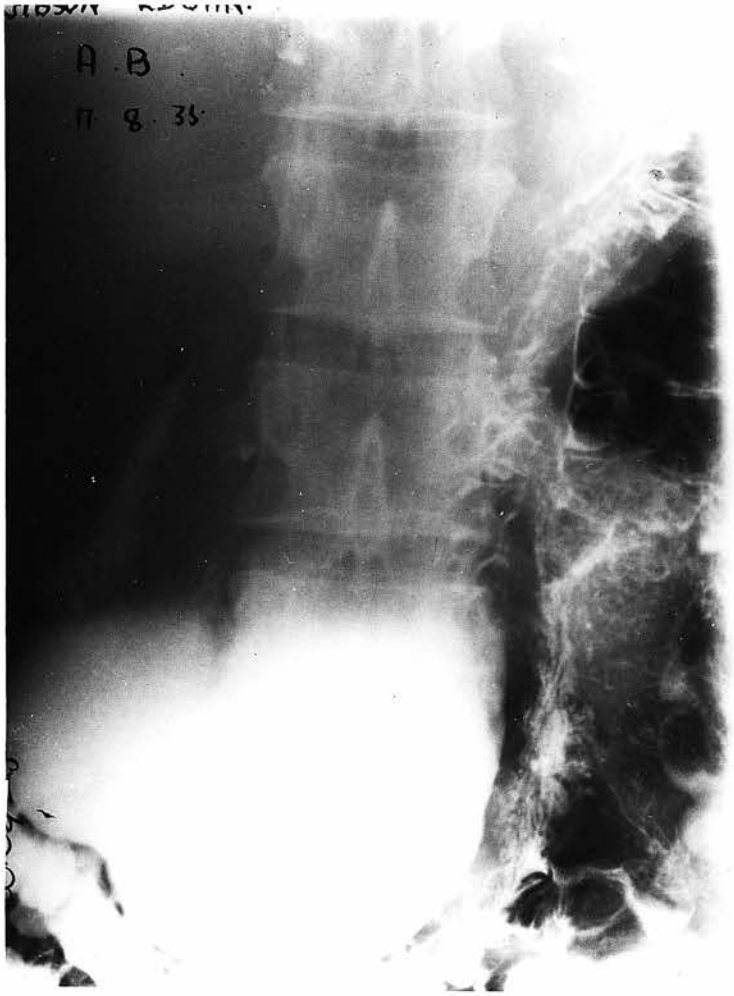
Ulcer at outer side of base of duodenal cap.

Plate 12.

Grimes (reversed plate).

Small irregular cap. Ulcer on outer side just above base. Spasm well marked.

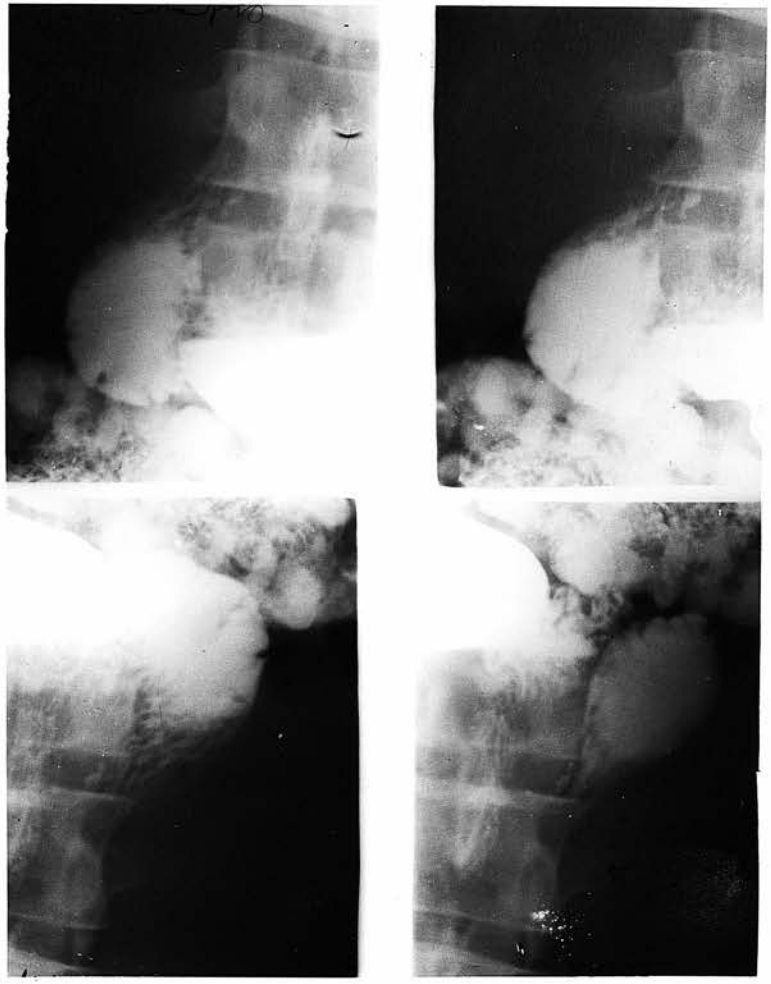
Plate 13.



Gibson, E.

Large ulcer internal aspect. Cap fills with difficulty. Pyloric stenosis developing.

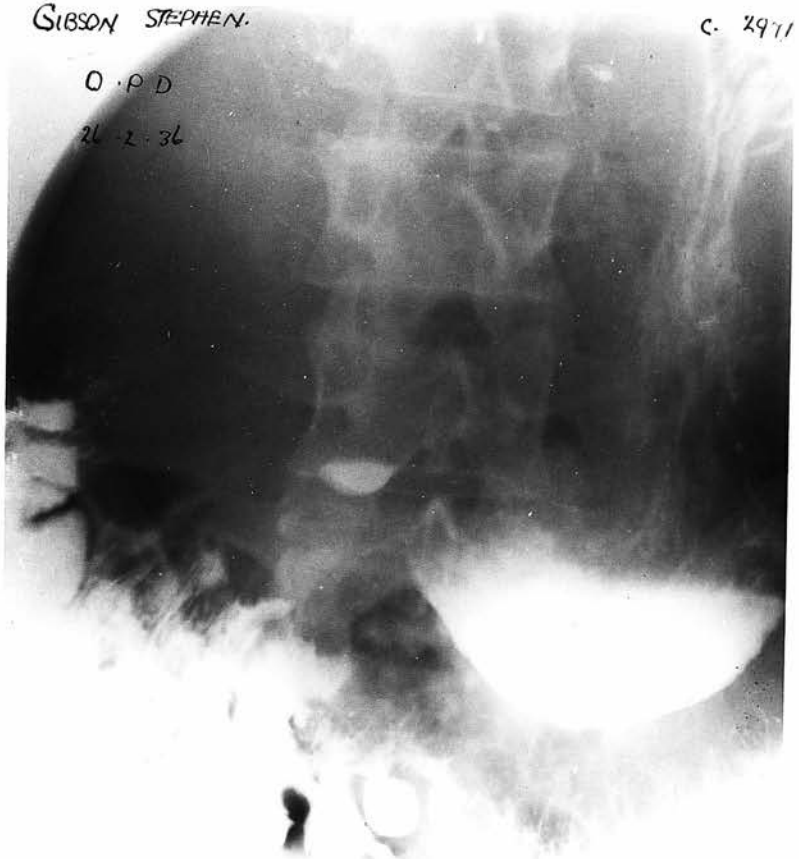
Plate 14.



Gibson, E. (3 weeks later)

Cap normal.

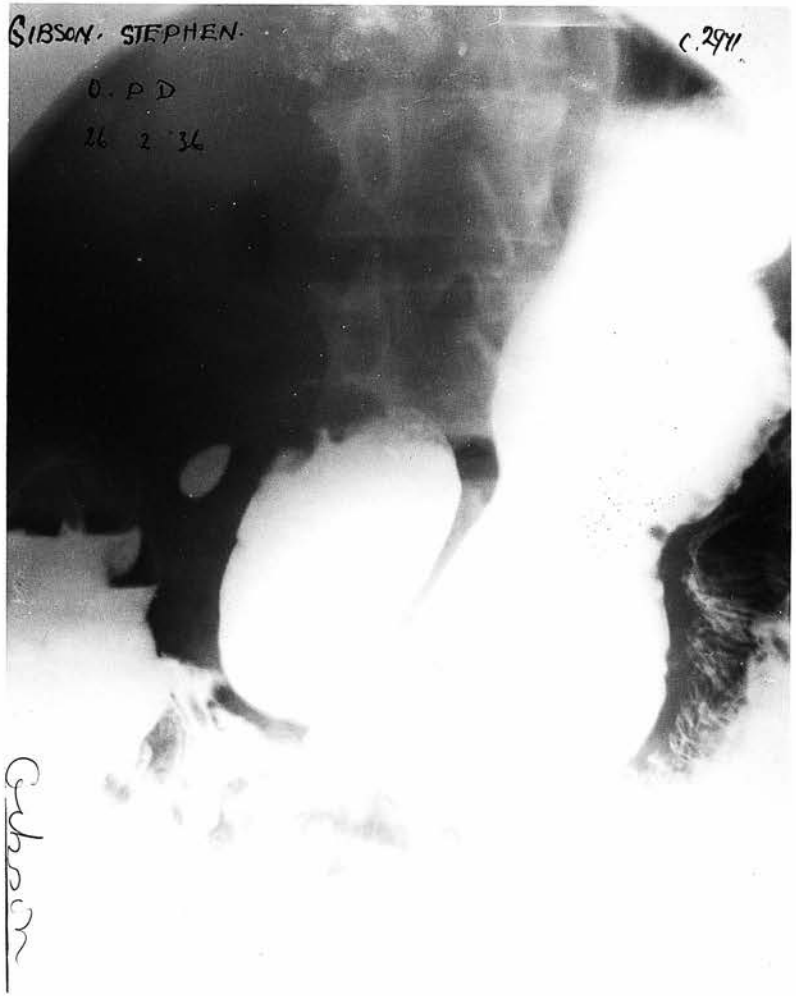
Plate 15.



Gibson, S.

Patch of meal retained in base of cap.

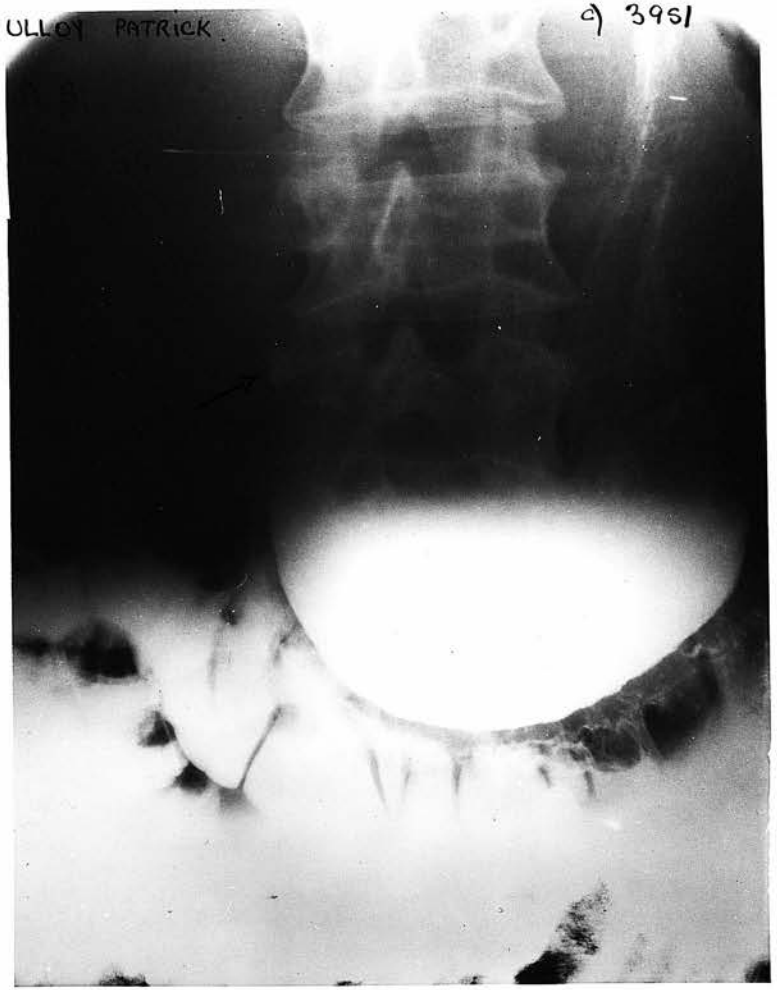
Plate 16.

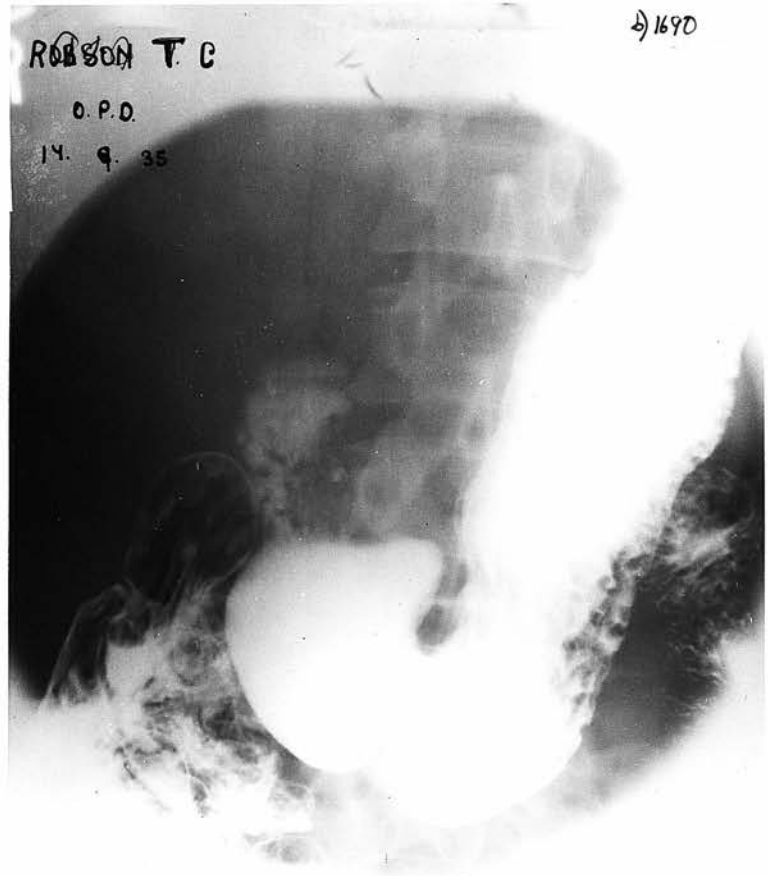


Gibson, S. (same date).

Marked pyloric spasm. Fair sized ulcer at base of cap on outer side.

Plate 17.





Molloy.

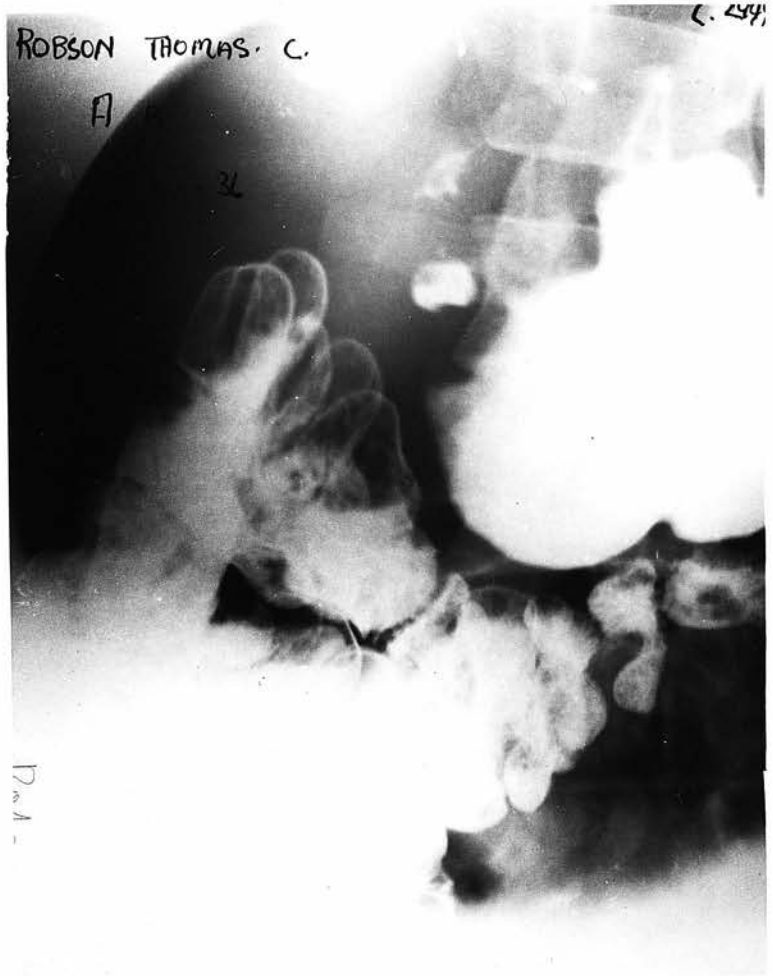
Fair sized ulcer at base of cap. Commencing obstruction.

Plate 18.

Robson, T.C.

Stomach and duodenum normal.

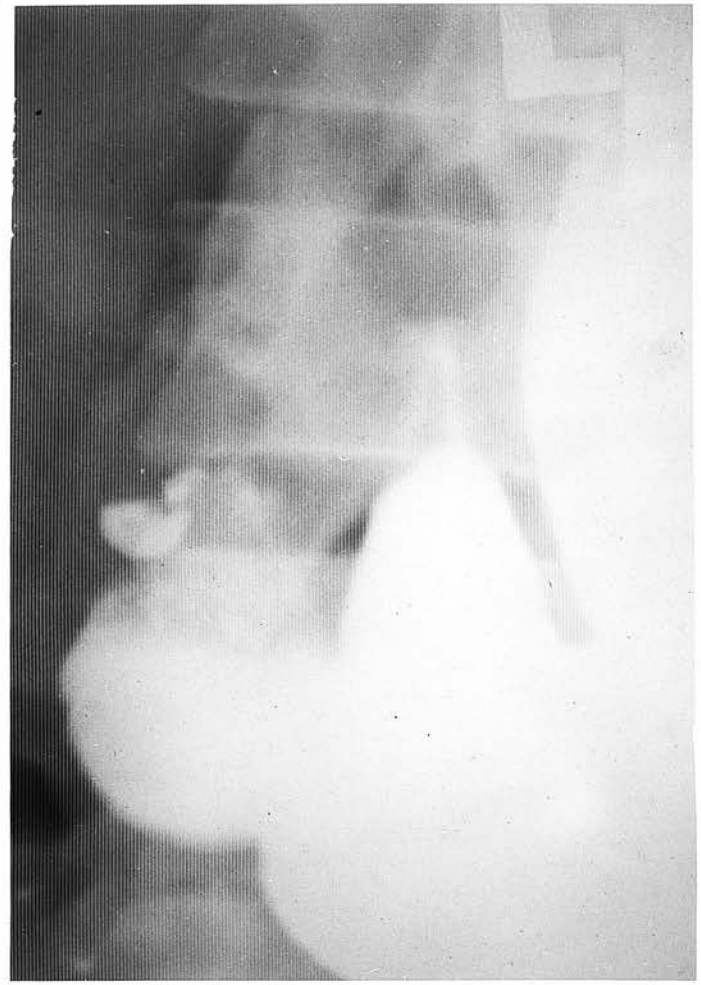
Plate 19.



Robson, T.C. (six months later).

Small residue in cap. After the meal ulcer at base was definite.

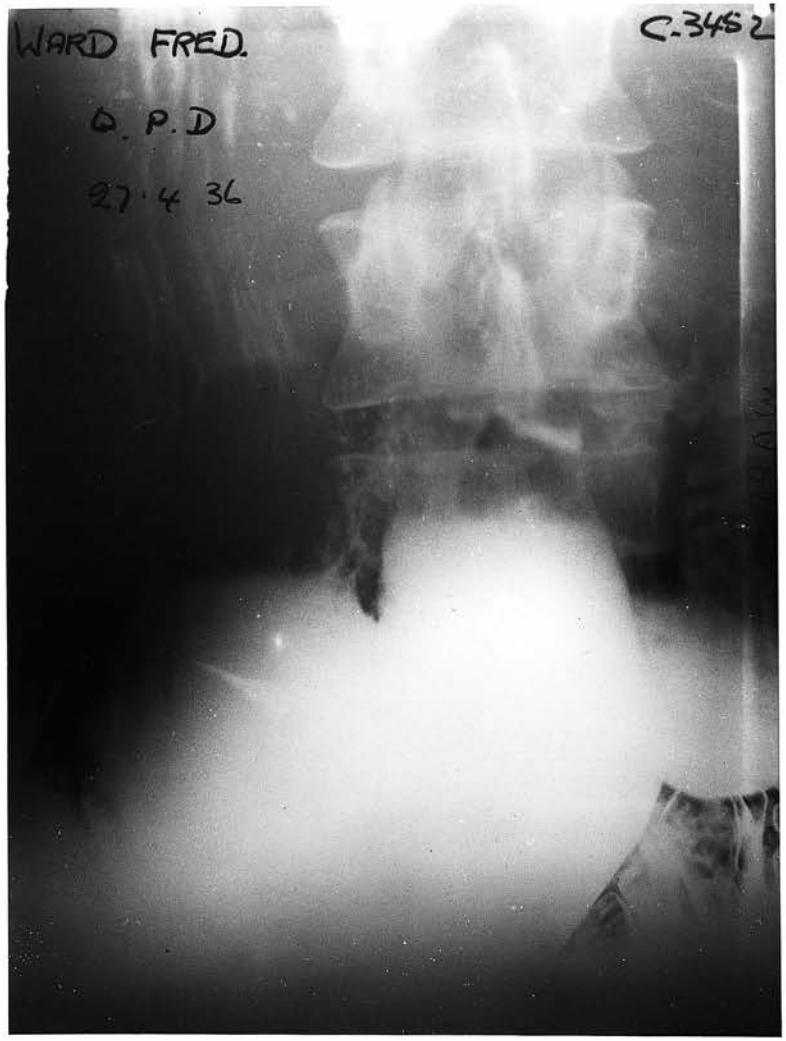
Plate 20.

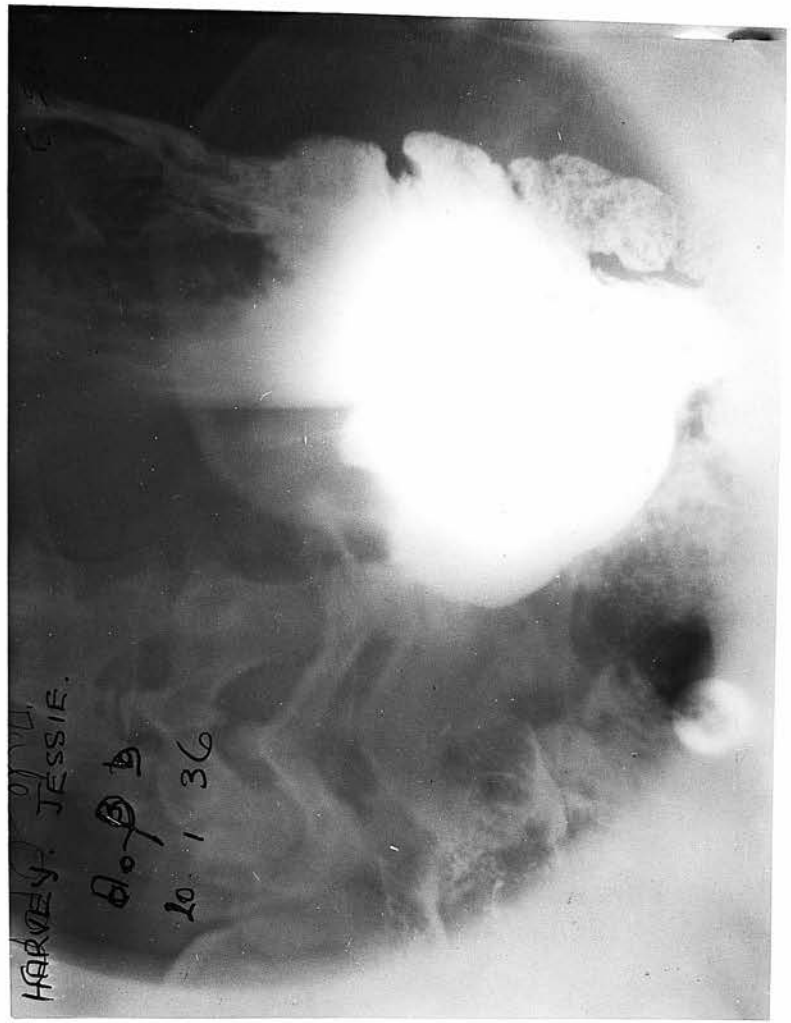


Robson, T.C. (seven months later).

No appreciable change. Spasm in cardiac region of stomach.

Plate 21.





Ward.

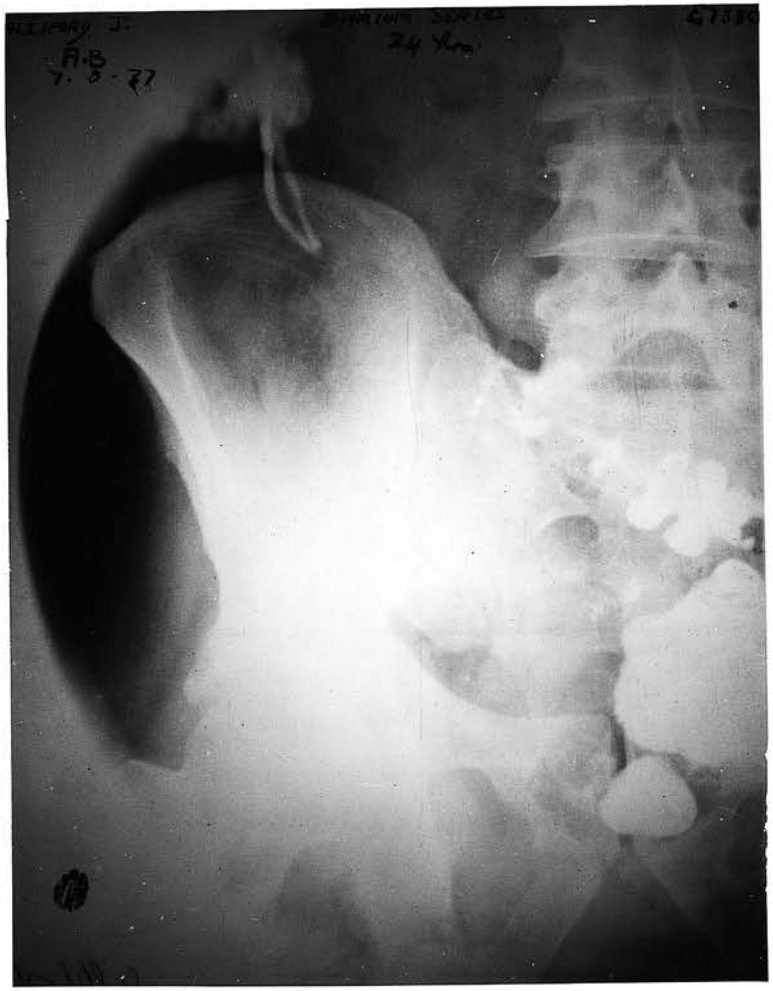
Gastric peristalsis poor. Ulcer at outer side of base of cap. Commencing obstruction.

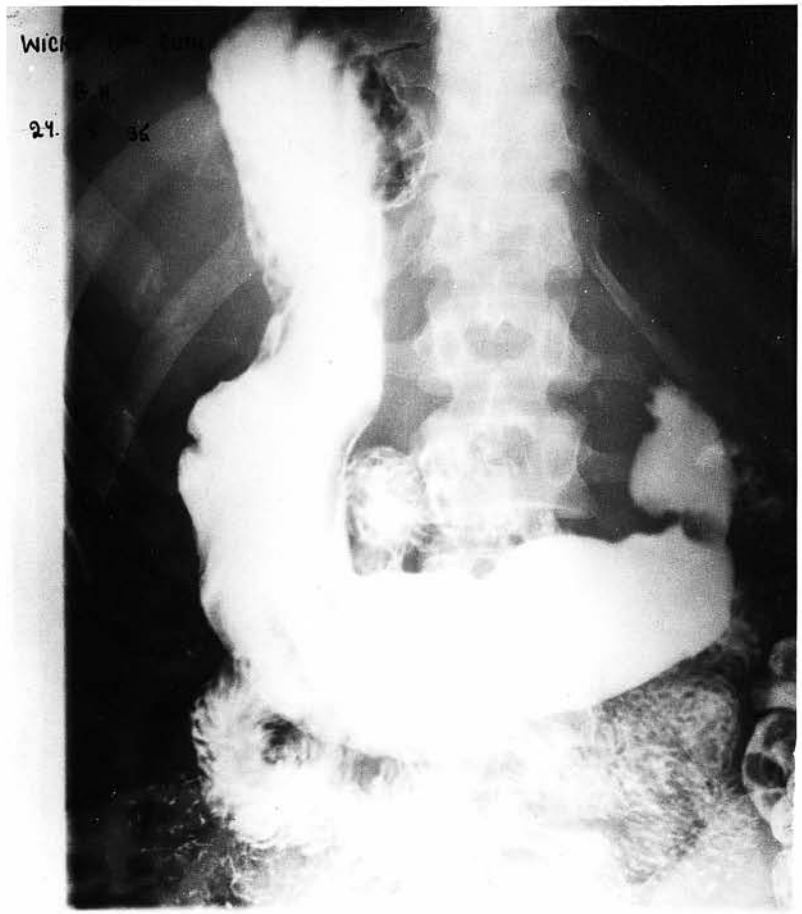
Plate 22.

Harvey.

Gastroenterostomy working well.

Plate 23.





Galliford.

Appendix well shown.

Plate 24.

Wicks.

Marked gastroptosis.

Plate 25.

P E P T I C U L C E R (Cont'd)

T R E A T M E N T

MEDICAL OR SURGICAL:

There is much divergence of opinion as to whether treatment in general should be medical or surgical. It is well, before proceeding further, briefly to elaborate on this difference.

It is as equally true to say that the treatment of ulcer is surgical, unless there is an indication for medical treatment, as it is to say conversely that treatment is medical, unless there is an indication for surgical interference. I know of no surgeon who operates in every case without some probing as to the possibility of medical treatment. The general practitioner has a shrewd idea of the presence of a peptic ulcer and in the main will have already attempted some sort of dietetic regime. It is most interesting to find in one's own records the number of persons suspected of peptic ulceration. I would go so far as to state that more patients are treated for ulcer than actually suffer from ulcer. There will be few who have escaped an attempt at medical treatment. Some of the actual sufferers will have been relieved or even cured. This not inconsiderable quota must of necessity be ignored in any truly

scientific analysis. I cannot support even the suggestion by figures. I am equally sure that I am right.

From an etiological point of view wholesale operative interference is wrong. There can be no justification for any but mechanical faults being treated surgically. Etiological factors, however, are not yet agreed upon. Thus the treatment of perforation which is the result of a mechanical fault is surgical. The treatment of haematemesis, another mechanical fault, is by no means universally looked upon as requiring an operation.

I think it is as well to judge each case on its merits. It is natural that a surgeon will tend to an excess of operations and in all fairness it is difficult for a physician not to over-advise medical treatment. In order to decide on the advice to give any patient some sort of system is desirable. I, myself, have perhaps a little unwittingly followed that suggested by Bennett(54 & 57), who excludes from medical treatment:-

Bennett's contra-indications for Medical Treatment:

1. Those in which perforation has occurred.
2. Those in which there has been severe or recurrent haemorrhage.
3. Those in which cicatrical contraction has caused, or will soon cause, permanent pyloric stenosis, or serious hour-glass constriction of the stomach.

4. Those in which X-ray examination shows deep ulceration with adhesions to neighbouring structures.

5. Those with a short history or other features pointing to the possibility of malignancy.

6. Those which continue to relapse in spite of efficient medical treatment.

7. Those whose economic position makes effective medical treatment impossible.

1. Perforation:

A very definite and urgent surgical condition.

2. Haemorrhage:

The immediate treatment of haemorrhage is medical(13). Joll only advises operation on very selected cases. He does not refer to gastric haemorrhage but only to duodenal haemorrhage(58). He agrees that recurrent haemorrhage usually calls for operation. These opinions were generally held at a meeting of the Medical Society of London(59). Culliman also supports the view that immediate treatment is medical(60).

3. Cicatrical Contraction:

Even Bennett in a later publication(61), does not accept his dictum on this point without some hesitation. There are cases which seem at first to be impossible, cicatrical contractures which respond to treatment by medical means. It is a matter for very shrewd judgment to decide on what the contracture will become. For that reason it may be wise to defer the question of operation until other treatment has been attempted. It is true that from the first one can frequently decide that the mechanical fault can solely be relieved by mechanical means. It is my experience that the most successful results from a surgical point of view are obtained when such a fault is rectified.

4. Deep Ulceration and Adhesions:

This is again a matter of degree. The

decision depends on more than the radiological signs. The whole history of the case must be considered. It is even reasonable to suggest an attempt at conservative treatment. Only by long experience can a decision be made, on a prima facie case for operation from the first.

5. Short History: possible Malignancy:

It is frequently impossible to decide at once whether malignant changes are at the basis of an apparent ulcer. It is well under these circumstances to make certain with little delay. An operation must be advised at once.

6. Persistent Relapses:

Many patients find efficient medical treatment unsatisfactory. They have periodic returns of pain and discomfort. Possibly this may be due to quite comprehensible lapses in diet. They seek operation in the hope that surgery can give more relief than medicine. There are some who do not get relief from medical treatment. They must be few.

7. Economic Difficulties:

In my district this question is one of some moment. More than a fair proportion of patients express themselves in words such as these: "If an operation can do me any good, I am anxious to have one at once, so that I can get the thing cured and get back to work sooner". I have a good deal of sympathy with the point of view. I endeavour to put the matter before them fairly. I tell them that I appreciate their courage, and then discuss the matter in a friendly way. I am certain that it is difficult for a patient to undergo satisfactory dietetic treatment unless he is satisfied that he is doing the right thing. Davies and Wilson[62] indirectly subscribe to this sentiment. They have found an increase in an ulcer shadow when nervous strain is being experienced. Medicine lacks the dramatic effect of the operating theatre.

Further References:

Wilkie[63] has laid down certain dicta from the

surgical aspect. His criteria do not materially differ from those of Bennett. He states that operation is advisable for those ulcers which are judged by X-ray to be more than 1cm. in diameter. He also prefers surgery where haemorrhage has taken place on more than three occasions. He thinks that medical treatment should be given a "fair and prolonged trial" for those patients who have not had six recurrent attacks of symptoms.

Walton[64] has a mortality rate following operation from 2% to 3%. He feels justified in stating that it should be reduced. He himself performed a later series of 180 gastro-enterostomies without a fatality. Broster[65] has a mortality rate of 6.7%.

The St. Bartholemew's Hospital[106] figures for the result of the treatment of gastric and duodenal ulcers may be tabulated as follows:-

		Grade 1		Grade 2		Grade 3		Grade 4		Dead			
										Immediate		Late	
		G	D	G	D	G	D	G	D	G	D	G	D
Number of Cases	Med.	83	48	44	22	19	18	51	33	15	7	10	4
	Surg	52	64	17	20	5	12	13	13	25	6	8	8
Percentage of Cases	Med.	36.0	36.3	19.1	16.6	8.2	13.6	22.1	25.0	6.5	5.3	4.3	3.0
	Surg	43.3	52.0	14.2	16.2	4.2	9.8	10.8	10.6	20.8	4.9	6.7	6.5

The facts to be gleaned from this table are:-

(a) The mortality rate from all surgical methods is much higher in the treatment of gastric ulcer than that from medical treatment - 20.8% to 4.9%.

(b) Fewer patients live in a state of chronic ill-health after operation for gastric ulcer.

(c) Better results are obtained from surgery in the treatment of duodenal ulcer.

Barford[71] found that at the New Lodge Clinic duodenal ulcer responded rather better to medical treatment than did gastric ulcer. At the same time the surgical treatment was also more successful in the former.

Gordon Taylor et alia[66] investigated the late results of 52 patients who had undergone partial gastrectomy for gastric ulcer. The results were uniformly favourable. No evidence was given as to the immediate mortality rate.

David Smith[67] confesses that the late results of medical treatment are not uniformly good. He had investigated 214 cases of peptic ulcer which had been treated at the Glasgow Royal Infirmary. While finding excellent immediate results, the after history was not so favourable. The former indicated 90% of cures while the latter only showed, at the most, 60%. In his paper he includes a table from Mielsen (Act.Med. Scand 1923 LVII, 1-42). It is of interest as it demonstrates that the earlier treatment is begun the better are the chances of success.

Duration of Symptoms	% Cure	% Improved	% Unimproved or operated upon
I. Less than one year	93	7	-
II. One to ten years	63	17	20
III. Over ten years	40	25	35

I was interested to find that Smith was of the opinion that too much attention is paid to the ulcer, and that too little is paid to the patient and to the fundamental cause of the patient's ulcer. This point is also made by D.T. Davies[69]. His Bradshaw lecture ends with a similar phrase, "Should we not concentrate less on the lesion and more on the man and his surroundings ?" Davies is content to feel that the ordinary uncomplicated ulcer can be well managed by medical means. He even thinks that for many cases it is advisable to treat as an out-patient and to allow the patient to carry on with his work.

SURGICAL TREATMENT:

The treatment of perforation and haemorrhage is:-

(a) Perforation:

1. Simple closure with or without drainage.
2. Closure, plus attempt at cure. An interesting note is made by Cellan Jones(79).

(b) Haemorrhage:

On occasion occlusion of the bleeding point is carried out.

Surgical Methods:

1. Excision or occlusion of ulcer.
2. Partial Gastrectomy.
3. Gastrojejunostomy or Gastroduodenostomy with or without (1).
4. Pyloroplasty.
5. Partial Duodenectomy.

Note on After History of Gastro-enterostomy:

The British Medical Association investigated the after results of the operation of gastro-enterostomy, with or without further operative details, in 1928. The operations had been performed between 1920 and 1924. Luff(78) compiled the report. The results in the case of duodenal ulcer were satisfactory in 90% of patients where an after history was obtainable. One third of this number had to make some modification of an ordinary diet. Jejunal ulcer developed in 2.8%, while the mortality rate was 5%.

The figures for gastric ulcer varied considerably with the technique used. An average satisfactory result was 70%. One quarter of this number had to modify their

diet. Jejunal ulcer developed in 0.8%. The average mortality rate was 7.3%. A summary of these statements is added.

	Gastric Ulcer.	Duodenal Ulcer.
Satisfactory Recovery	70%	90%
Immediate Mortality Rate	7.3%	5%
Development of Jejunal Ulcer	0.8%	2.8%

Development of Jejunal or Gastrojejunal Ulcer:

In medical treatment this tragedy cannot come about. It is, however, a not uncommon complication following operation. Walton(107) agrees that Luff's figures following gastro-enterostomy are correct. He mentions that there is a tendency for surgeons to perform a partial gastrectomy to avoid the development of jejunal ulceration. It is now being appreciated that there is still a risk of marginal ulceration occurring after gastrectomy.

The Association of Surgeons has inquired into the risk of jejunal ulcer. Garnet Wright(108) published the results. He states that partial gastrectomy is not immune from a resulting jejunal ulcer; but the risk is less than following a gastro-enterostomy. It may be that if much of the acid bearing area of the stomach were

excised the chances might even be less. High acidity in itself is not a cause of jejunal ulcer but rather is active gastric juice. Another highly interesting fact which emerged from the enquiry is that marginal ulceration is more likely to follow the operation of gastro-enterostomy when used in the treatment of perforation. The cure of jejunal ulcer is best brought about by a partial gastrectomy. The mortality rate is unfortunately as high as 20%.

MEDICAL TREATMENT (70)

1. Von Leube's Regime:

This was the classical treatment for many years. It included a planned diet, rest in bed, a morning drink of Carlsbad water and hot applications to the abdomen. Four periods were devised. The first consisted of two-hourly feeds; only fluids and predigested foods were allowed. Apart from milk there was little of nutritive value in them. After about ten days additions were made. Seven days later meats and potatoes were allowed. In the last period five meals a day were permitted and vegetables were included. The treatment was carried on for about six months.

2. Lenhartz Diet:

Von Leube's regime was responsible for under-nourishment. Lenhartz gave milk and eggs without any preliminary starvation, even in the face of haemorrhage. He encouraged constipation in order to minimise gastro-intestinal peristalsis.

3. Sippy Treatment:

Sippy accepted the suggestion that neutralising or removal of the acidity in the stomach was essential to promote healing of an ulcer. Even in 1915 he had been carrying out these ideas for twelve years. He thus used a clinical reasoning many years before experimental confirmation was available. He did not practise preliminary starvation. His procedure was as follows:-

i. Any excess over 10cc. of night secretion called for nightly gastric lavage. This was repeated until there was no excess.

ii. Rest in bed was necessary for three or four weeks.

iii. Regular feeds commencing with milk and cream were given every hour. He found that sometimes acidity could be controlled by feeding at rather longer intervals, even up to three hours.

iv. Alkalies were administered between the feeds.

There were two powders:-

No.1 Sod.Bicarb. gr.X
Mag.Calc.Pond gr.X

No.2 Sod. Bicarb ... gr.XX
Calc.Carb. ... gr.XX

The powders were alternated, but were varied according to the patient's reactions to them.

v. The feeds were gradually increased to include most soft foods.

The plan outlined by Sippy has been adopted almost universally. There have been modifications of detail. In the main, however, the general system is still employed.

Reduction in Gastric Motility:

Some workers maintain that motility should be controlled rather than the acidity. Amongst these physicians, stand Block & Serby(72). Their conclusions are based on theory, laboratory findings and clinical experience. The patient is put to bed and placed on a bland diet. When the symptoms have disappeared repeated gastric aspiration showed that the acidity was reduced. They admit the legitimacy of the prescribing of alkalies in some cases. They feel that the wholesale use of alkalies is in general due to training and example, and also to a sensation of neglect in its disuse. They do not fear alkalosis as such.

Alkalosis is nevertheless a menace when large quantities of alkali are administered.

Alkalosis:

The symptoms of alkalosis come on insidiously.

They bear no relation to the amount taken. A deterioration in appetite is soon followed by attacks of irritability and melancholy. The mental change may even be so bad as to lead to delusions. The patient in consequence, spreads untrue, scandalous tales. Headache and drowsiness eventually passes to coma.

These extreme symptoms as described by Cope(73) cannot be common. I myself have not been worried by such. I have been impressed by the almost constant complaint of headache and depression experienced by patients during the early days of treatment.

There are definite laboratory findings which assist in the diagnosis of alkalosis.

1. Increase in the blood urea.
2. (a) Increase in the alkali reserve of the plasma as measured in volumes of Co_2
   (b) An increase in the pH of the blood.
3. (a) In spite of any vomiting, the concentration of sodium in the blood is maintained or raised. Severe vomiting in itself would lead to a decrease in this content.
   (b) If the alkaline powder contains calcium, the calcium tends to accumulate. The proportion becomes higher than usual. It simulates parathyroid tumour, in this respect.
   (c) If a magnesium salt is prescribed, there is an increase in the serum magnesium.

Prophyllaxis of Alkalosis:

In order to avoid the disturbances alluded to above, endeavour has been made to find an alternative drug capable of neutralising or buffering the hydrochloric acid of the stomach. Cope himself suggested that disodium phosphate warranted a trial. Following the publications of Mutch in 1936,[74,75,76], I, myself, began to use magnesium trisilicate. My cases did not react well to it. The symptoms were not so quickly or so efficiently relieved as when giving an ordinary alkaline powder. For these reasons I dispensed with it. I cannot remember the particular brand of magnesium trisilicate used. The more recent description of the drug has cast some light on my own (and evidently others) failures. Mutch[77] has now defined the compound in more detail and has given reasons for its past failure. He describes how many marketed brands fail entirely to conform to the standards he has now laid down. I propose to try it again when such a brand is conveniently available.

Tribasic magnesium phosphate is used to some extent at the New Lodge Clinic. The treatment there also includes sodium citrate and magnesia and belladonna.[71].

Atropine and Belladonna:

Atropine and belladonna were used considerably before alkalosis was described. It became part of the armamentarium of the Gastro-Enterologist. It is now used partly in order to reduce the risk of alkalosis.

I have never been satisfied that the drug is absolutely necessary. The unpleasant effects experienced, following a hypodermic injection of atropine in the Pharmacological Department may have had something to do with my opinion. I have been reluctant to employ the alkaloid to any extent. In 1920 Bastedo(81) came to the conclusion that in order to obtain the expected effect large doses were necessary. The dose then given also provoked the less desirable symptoms of extreme dryness of the mouth and throat, tachycardia and dimness of vision. He felt that the less desirable effects were too pronounced to warrant the continuance of its employment.

The effects in the stomach one wishes to obtain from the use of belladonna are:-

(a) Reduction of motility and tone.
(b) Reduction of pylorospasm.
(c) Reduction in secretion.

In 1936 after being subjected to much criticism Bastedo(80) again made his assertions. He compiled a very comprehensive bibliography to support his views. He stated that, were the effects the same in man as in

experimental animals, the use of atropine would be fully justified. He quoted radiologists such as Moore, Barclay, Reizenstein and Frei. These men have almost abandoned the injection of atropine which they had employed for years to reduce spasm. They had come to three conclusions.

(a) Spasm is not overcome in itself by atropine.
(b) Spasm is the result of apprehension on the patient's part.
(c) Spasm is variable and its absence at a second examination is mainly due to the patient's lack of apprehension rather than to the reaction to any antispasmodic drug.

The point (b) would seem to explain the good results obtained by Landau and Hejman(82). They combined the use of atropine with intravenous injection of sodium bromide. They accepted the fact that the soothing of the nervous system by the bromide was an essential factor in obtaining the maximum effect of the atropine. It might well be that the bromide was actually the effective partner.

I conclude this part by quoting Bastedo: "In single maximum doses by hypodermic injection, atropine may have a limited value in reducing secretion and spasm; but in the doses usually employed by mouth, or permissible for any continued treatment, atropine and belladonna are practically without effect on the secretory and motor

functions of the stomach".

Notes on Claims by Hort[83]:

In a letter to the British Medical Journal in 1936, F.A. Hort suggests that a peptic ulcer may be curable by the following means:-

1. Horse serum.
2. Bowel inundation by pure potassium permanganate solution.
3. Chromotherapy.
4. Thyroid and manganese in catchets.
5. Autogenous vaccine.

I can find no scientific confirmation of these suggestions. In spite of such heterodoxy Hort supports the contention that the morale of the patient is a most important item.

DUODENAL FEEDING

Geoffrey Bourne[109] tried this method in 20 cases of peptic ulcer. The tube is passed well into the duodenum. It is closed at the upper end when not in use. The feeds consist alternatively every two hours of egg and milk and Benger's Food. Of the twenty cases, four discharged themselves, a rather high proportion. They did not care for the treatment. Personally I think they were justified. Four weeks with a duodenal tube in situ cannot make life very pleasant. The results Bourne gives do not carry much weight.

TREATMENT BY HISTIDINE

The work of Williamson and Mann referred to in the section devoted to etiology led Aron(quoted99) to investigate the chemical changes involved in their experiments. He found even with the diversion of duodenal contents into the ileum no ulcer followed if tryptophan and histidine or the latter by itself were injected into the animal. The logical result has been the use of histidine by injection and orally in an attempt at what would seem a new and scientific treatment of peptic ulcer.

The method usually employed was a daily subcutaneous or intramuscular injection of 5 cc. of a 4% solution. The number of injections varied, an average being twenty. After Fontes in 1935 had shown that histidine was absorbed when taken by mouth, though to a less extent and rather slowly, tablets of 0.2 gm. were given. Six tablets was the average. They were given along with the injections. More frequently they were prescribed after the intensive twenty injections were finished.

Barry and Florey(100) compiled an imposing list of those who had used the drug with success. At first there seemed to be good reason to believe that a cure had been found by an easy means and unaccompanied by dietetic restriction. There was general agreement

that painful symptoms were relieved.

Barry and Florey, however, did not agree with the basic findings of Weiss and Aron. They thought that scientific justification was not present. They argued in the following way:

1. Aron's theories were based on too small a number of experiments. He failed to keep the animals alive long enough after operation.

2. Drainage of the pancreatic juice alone, which removes trypsim, rarely leads to the formation of ulcer.

3. Aron's theories are only applicable to those ulcers produced by draining the duodenum and thus disturbing normal digestion. The operation of Matthews and Dragstedt, in which a small stomach pouche is drained into a loop of ileum, does not interfere with alkaline digestive ferments; yet a constant, typical peptic ulcer develops in the loop adjacent to the pouch.

These observers performed further experiments on cats and pigs which did not substantiate the claims made.

Sandweiss[102] also approached this method in 1936. His criticisms were the same as above. His clinical findings were not definite. He did not feel that every case should be treated with histidine. He did think that there was a place for it.

The following year Sandweiss published a further address[103]. In his 1936 paper he had mentioned the substitution of distilled water for histidine. In his more recent article this method was tried on a larger

scale. The results of the injection of distilled water compared favourably with those obtained when histidine was injected. In the discussion which followed Dr Sandweiss's address J.H. Fitzgibbon made the following trite and appropriate remark, "The injection of the greatest value..... is injection of commonsense and conservation".

I, myself, was caught in the net of advertisement and used histidine on several patients. It seemed too good to be true. I, personally did not find a rapid relief of symptoms. This led me to lose faith. After a very brief trial I discontinued the adventure.

I feel that:-

1. If the scientific basis which led to recommendation of histidine were correct, all uncomplicated cases should be easily cured.

2. If all such cases are not curable by this means, then there is no adequate reason for its use.

The general opinion held now seems to be the same as mine. Bulmer[101] who was an enthusiast in 1934, was a little doubtful in 1936 and has now, I think, reversed his opinion.

Summary of Methods of Parenteral Treatment:

The reference 102, also contained a list made out by Sandweiss of methods used parenterally in the treatment of peptic ulcer. I have tabulated them:-

No.	Material	Year	Author
1	Vaccineurin	1922	Holler
2	Novoprotein	1922	Pribram
3	Organisms and vaccines ...	1930	Various
4	Aolan	1929	Martin
5	Pepsin	1932	Glaessner
6	Sodium Benzoate	1933	Melocchi
7	Emetine Hydrochloride ...	1931	Pitkin
8	Haemoprotein	1935	Levin
9	Sodium Citrate & Sodium Chloride	1934	Bulman
10	Parathyroid Extract ...	1931	Levy
11	Insulin	1934	Jones

He also gave the following newer methods of non-parenteral treatment:

No.	Material	Year	Author
1	Metaphen	1933	
2	Aluminium Hydroxide ...	1934	
3	Mucin	1931	
4	Okra	1933	
5	Silicon Dioxide	Unpublished	

TREATMENT OF HAEMATEMESIS:

It is still generally held that the treatment of haematemesis is one for the physician. Haemorrhage occurs

not only from gastric and duodenal ulcer but also from anastomotic ulcer. Hurst and Ryle[13] because of their experience do not adopt a pessimistic attitude towards haemorrhages. Ryle found that there were 25 deaths from gastric and duodenal haemorrhage during 20 years. Deaths from other causes sequelae and complications were as much as 128. Though differing in his opinion as to the mortality from haemorrhage Cullinan[84] agrees with expectant treatment. Judgment must find its basis in a just appreciation of all therapeutic possibilities.

Surgical Treatment:

The opinion of most surgeons is conservative. Even so there is growing evidence that early operation is becoming a comparatively safe procedure. The necessity for operation is, however, not easy to assess. The mortality rates from bleeding, treated conservatively, is variously given as 4% by Hurst, 19% by Letherby Tidy[92], and by Hinton in America as 20%. It is difficult to get past the confidence of Hurst & Ryle.

Gordon Taylor[94] is inclined to the view that immediate operation is warranted. His reasons are:-

(a) His own "low" mortality rate of 9%.
(b) Confirmation of the lesion.
(c) The possible cure of the ulcer.

He attributes his good results to the continuous blood drip of Marriott & Kelwick, which he employed during and after operation.

Operation:

The essential feature of an operation is to isolate and stop the bleeding point. Only after this, is further interference warranted. It then depends essentially on the patient's condition.

In 1921 Thomson & Miles made no reference at all to the surgical treatment of duodenal haemorrhage. They did suggest, however, that it was practicable to ligate the vessel in a bleeding gastric ulcer(98).

The surgical references alluded to previously indicate one other important point. The question of the operation depends very much on the inclination and ability of the surgeon. Hurst and Ryle are convinced that surgery often fails because of the inaccessibility of the ulcer. The percentage of haemoglobin they consider is of less importance than the site of haemorrhage.

Blood Transfusion:

The purposes for which transfusion is used are threefold:-

(a) To avert haemorrhage by supplying in particular coagulative elements.
(b) To make up for serious blood loss.
(c) To stimulate haemopoeisis.

The dangers are however:-

1. Haemorrhage is sometimes increased.
2. A serious or even fatal reaction may ensue (97).

Many methods of transfusion have been used and described. In this country the citrated method is used almost exclusively(95,96). The reason is mainly one of convenience. The donor and recipient do not need to be in the same room. An excess of assistants is not required. Clotting does not delay the proceedings.

Indications for transfusion:

(1) Persistent serious haemorrhage.
(2) A haemoglobin percentage of less than 30.
(3) Generally if operation is to be performed.

Medical Treatment:

(Many of these details would a fortiori be also used in Surgical Treatment).

(a) Rest - The patient is kept quiet by an initial injection of morphia gr. ¼. He should not be moved unless conditions at home are very unsatisfactory. Alternatively he may have to be moved when blood-transfusion and/or operations are deemed likely to be required.

(b) Saline - The fluid loss may be made up by either intravenous or rectal saline. I usually give about a pint of normal saline rectally every 3 or 4 hours during the first day.

(c) Diet - Most authorities have held that little or nothing should be given

by mouth for the first few days. The rationale being to avoid damaging the newly formed clot. Theoretically this is correct.

Meulengracht's Method:

Meulengracht[85] has advocated the giving of food from the very first and plenty of it. After reading his article and after considering his results I adopted his method in principle. Like him I was astonished at the change in the general appearance of the patient. His attitude became cheerful, helpful and confident. When using the more accepted, preliminary starvation, the patient was sullen, listless, resentful and often hungry. To my mind the change from this attitude, with its mental outlook, is an important factor in his ultimate cure.

Before changing his methods Meulengracht had a mortality rate from peptic haemorrhage of 8%. Cristiansen had, and I think still has, a mortality rate of 7.9%. The figures are comparable. Now Meulengracht publishes statistics which show that the mortality rate has been reduced to 1%.

My interest in peptic ulcer was not critical previous to 1935. I consider that the treatment is a most important step in the advancement of the conduct of a case of haematemesis.

Details of Treatment (Meulengracht):

1. Sod.Bicarb. Mag.Subcarb. āā 15 gm. Ext. Hyoscyam, 2 gm. - of this one level teaspoonful is given three times a day.
2. Ferri lact. 0.5 gm. is also administered at the same intervals.
3. A puree diet is allowed in as large a quantity as the patient desires. As the dinner consists of food varying from fish balls and mashed potatoes to stewed apricots, one can well understand the patient's good cheer.
4. Meals are given at 6 a.m., 9 a.m., 1 p.m., 3 p.m., and 6 p.m.

I do not use the above diet in detail. For convenience of administration and routine the patient is placed on the same diet as an ordinary case of peptic ulcer (see later). In addition I administer fluids very freely during the first 24 hours. I may withhold food for that time. The patient in such circumstances may have had morphine. He will feel dry for that reason as well as from fluid loss. After the first 48 hours I give Tab. ferrous Sulphate (Glaxo) t.i.d.

The use of Haemostatics and Coagulative Elements:

Gulland and Christie[87] investigated this question with reference to haemophylia. Their experiments were, however, not confined to patients who were unfortunate enough to be suffering from this malady.

They could find no support for the use of:-

(a) Calcium.
(b) Haemostatic serum (Lapenta).
(c) Haemoplastin.
(d) Tissue fibrinogen.

Vitamin "C"

Attention has been called to a deficiency in Vitamin "C" in cases suffering from haematemesis and melaena by Lazarus[91], and by Portnoy and Wilkinson[89]. The former also found three uncomplicated cases of peptic ulcer under treatment to be deficient. Bourne[90] submits confirmatory evidence. He stresses, however, that there does not seem to be any proof that a deficiency of Vitamin C is in anyway a factor in the aetiology of the condition. As has been stated previously Harris[9] suggests otherwise.

The importance of the above investigations lies in the fact that Vitamin C or ascorbic acid should be administered as a routine. Fresh orange juice is the most practical way of doing this. Meulengracht in his paper had come to similar conclusions, though not in such a definite and scientific a manner.

The Alvarez Regime:

I wish to conclude this section of my thesis first with a short description of what I consider to be a practical and simple method of treatment of duodenal ulcer. With easy modification it can be altered to

suit a person suffering from gastric ulcer.

Alvarez[104] rebelled against the Sippy method per se, because he considered that three essentials were required for its proper management:-

(a) Considerable faith, enthusiasm and training on the part of the physician.

(b) An excellent hospital, with excellent "interns".

(c) A patient with plenty of time and money.

He thought that a person suffering from a duodenal ulcer would tend to keep at work. He would only come back for further Sippy treatment under compulsion. He, therefore, felt that a simple method, even though not the best, would be of more practical use.

He drew up a smooth diet list from which are chosen three main meals each day. Between these meals food has to be taken. The suggestion is that each morning such a mixture as the following should be made up:-

Milk - one quart.
Eggs - two.
Cream - ¼ to ½ pint.

The patient takes this to work and drinks a glassful at 10 a.m., and 12, 4, 8 and 10 p.m. Malted milk, milk shake or just plain milk may be used as alternatives - a biscuit or two is not contra-indicated. Breakfast is taken at 7.30, lunch at noon and dinner at 6 p.m. Alvarez rarely finds any alkalies necessary. The times

for the between feeds may have to be reduced if pain comes on earlier than two hours after food.

The patient is asked to rest as much as possible, particularly on Saturday and Sunday. No other exercise than walking is permissable. The treatment is continued for at least six months. It may be necessary for food to be taken between breakfast and lunch and lunch and dinner for some months further.

In a cursory investigation which I carried out a few years ago to assist me in my work at the North Riding Infirmary, I came across a reference to the Alvarez diet. I was at once impressed by its logic and ease of application. Having studied his smooth diet list(105), I made up a general dietary which with modifications can easily be made to suit the individual.

My Routine Treatment of Peptic Ulcer:

I do not make any claim for originality for my routine treatment of peptic ulcer. I drew up the dietary so as to have an easily applied scheme. I determined to make a simple diet sheet to cover three weeks, so that without great alteration in outline individual differences could be inserted or withdrawn.

In the first instance it was drafted to make the work of the supervising nurse easier for her.

Secondly, it kept the treatment constant. House Physicians were not tempted to alter the method. The nursing and medical staff soon found the chart extremely helpful. It has become the custom to refer to the stage of treatment as being that of a certain week. That week may have been employed for a fortnight. It is still called by the week on the chart.

Even now I cannot decide why three weeks were chosen. I think I was influenced by a chapter in "Nutrition & Diet"[70]. The diet has been in use for several years and has given fair satisfaction. I have noticed that other practitioners use it. I have never published it or spoken of it in public. The patient is frequently given a copy and hence it has crept into the armamentarium of fellow medical men. Frankly, I think, the main merits are that it is simple, reasonably cheap, easy to alter. Perhaps of equal importance it is to others "ready-made".

As it stands the scheme is more particularly applicable to the treatment of duodenal ulcer. The speed of increase is in many instances too rapid for the treatment of gastric ulcer. Yet, I have had a case under my care recently, who had a large lesser curvature ulcer. At first it seemed too large for me to deal with. It resembled an accessory pocket. My fears were apparently justified. The alternation of milk and powder did not

relieve the symptoms. She pleaded for operation to relieve her pain. In desperation I altered treatment so that she took semi-solids. As soon as she had the second week diet with custard and then gruels instead of milk she quickly responded. The X-ray shadow diminished regularly and rapidly. I saw her in out-patients lately and after X-ray found that the ulcer had disappeared. I do not attempt to explain this peculiar reaction.

In spite of the diet on which this patient was placed, I lengthen the three days of milk and powder to six days. Sometimes I double the time during which the second weeks' diet is taken. I am inclined to pay more detailed attention to the patients suffering from gastric ulcer.

While under treatment the powder I use is given in milk or in water. At first I used the following prescription which was given by the late Professor McLean at a local medical meeting.

R.

Mag. Carb. Pond
Cret. prep.
Sod. Bicarb. aa ℥i } ℥i level = dose.

From experience the proportions were gradually altered and the ingredients changed.

R.

Pulv. Cret. ℥iv
Mag. Calc. Lev. ℥vi } ℥i Level = dose
Ol. M. Pep. ♏vi

I have found no defect in the omission of sodium Bicarbonate. The patients complained of the taste. The addition of the small amount of peppermint oil has been much appreciated. The incredients are alkaline and a little absorptive. In the average person the powder has a mild laxative action. There is no difficulty in altering the proportions to remedy diarrhœa or constipation.

During the first three days or while on milk it is advisable to give a daily saline enema. The rest in bed combined with the milk practically always results in uneasy constipation.

I usually allow a duodenal case to get up for a little while after the middle of the second week. This acts in two ways.

1. The patient feels he is rewarded for his good behaviour in keeping to a monotonous routine.
2. He becomes accustomed to standing, so that an X-ray is more conveniently taken before he leaves hospital.

Rest is essential for the initial period. Not only is this so from the point of view of treatment. It is also more convenient to ascertain the condition of the stools with regard to haemorrhage.

In addition some patients have to be watched closely at first to ensure that no addition is made to the prescribed diet.

An example of the need of such watchfulness occurred recently. A man was admitted following a copious haematemesis. His red count was down to two millions. I went to his bed one day. He exhibited an unusual modesty when I began to examine him. I found a large packet of sweet biscuits lying next to the skin of his abdomen. Patients suffering from gastric ulcer are kept in bed for three weeks. I usually advise a minimum stay in hospital for four weeks.

From the very start I impress on the patient that adequate treatment will take a considerable time. I explain that the stay in hospital is only a preliminary. The routine of treatment with the resulting relief of pain is usually a sufficient proof to the patient that he is on the right track.

On ceasing to be an in-patient, a sheet is given which indicates the main foods which are permissable. Elasticity is obtained by questioning him as to what has been found to disagree with him. Extra types of food are added as found suitable.

I try to get my old patients to attend at the out-patients' department periodically for about six months. It is extremely difficult for many of them to do this. Until recently the case recording methods at the North Riding Infirmary have been quite hopeless.

The filing systems were old fashioned. Case notes have been "put away" rather than filed. Important follow-up notes and clinical findings have thus been unwittingly wasted.

Note on Belladonna:

I do not regularly resort either to belladonna or atropine. Occasionally a single large dose has been given by subcutaneous injection. I have not found a necessity for routine administration.

DIETARY NO. 1.

FIRST WEEK

Days 1, 2, & 3.

On awakening - powder.
One hour later - tumbler of milk (hot or cold).
And then every hour alternately powder and milk.

Days 4, 5, & 6.

As days 1,2,3, but at dinner-time add one egg and a teaspoonful of sugar to milk, and similarly at 6 or 7 p.m.

Day 7.

As days 1,2,3, but at breakfast-time take one egg and milk as above; at dinner-time have one poached egg covered with bread crumbs, and a glass of milk. At supper-time have one egg and milk.

SECOND WEEK.

As days 1,2,3, but add:

Breakfast	-	Two half slices of well-done toast buttered when cold, and one lightly-boiled egg. Glass of milk.
Dinner	-	Tablespoonful of mashed potato with butter. Small helping of Tapioca or ground rice, or custard pudding.
Tea	-	Two half slices of well-done toast with plain jam, (having no pips or stones). Glass of milk.
Supper	-	One boiled egg (light), mashed up with baked bread crumbs. Glass of milk.

THIRD WEEK.

POWDER NOW TO BE TAKEN EVERY TWO HOURS BETWEEN MEALS AND ON AWAKENING AND GOING TO BED.

Breakfast.	Small helping of porridge with sugar and milk. One poached egg on toast.
10 or 11	Glass of milk.
12 or 1	Small helping of minced meat, two tablespoons of mashed potato. Pudding as in second week.
3 p.m.	Glass of milk.
5 p.m.	Two slices of toast with jam or jelly. Glass of milk.
6.30 p.m.	Soft boiled egg, one slice of thin white bread and butter. Glass of milk.
9 p.m.	Well baked apple (without skin or core). Glass of milk.

IF AT ANY TIME PAIN RECURS GO AT ONCE TO DAYS 1,2,3. In addition the juice of one orange should be taken daily.

Follow-Up Diet

CAN BE EATEN:	CANNOT BE EATEN.
Orange juice.	MELON.
Coffee in moderation.	Shredded Wheat.
Chocolate, tea, cocoa.	Smoked or tinned fish.
Eggs, bacon, ham.	Pork.
White bread and butter.	Veal.
Toast.	Crab.
Smooth thin gruel.	Lobster.
Cornflakes.	Bran biscuits.
Broths.	Salad.
Small portion of meat, fish, chicken.	Spinach.
Potatoes, baked or mashed.	Nuts.
Stewed tomatoes: NOT RAW.	Raisons.
Cauliflowers.	Cheese.
Brussell sprouts.	Most green vegetables.
Macaroni.	Raw fruit.
Spaghetti, soft with or without cheese.	Strong sugary things.
Banana - fried or baked: NOT RAW.	Sweets between meals.
Trifle Pudding.	
Custard.	
Ice Cream.	
Plain Cake.	
Stewed fruits.	
Tinned pears or peaches.	

A Glass of milk, Ovaltine, or Horlicks Milk between meals, and last thing at night.

McLeans' Powder, - one teaspoonful after meals.

One tablespoonful of Medicated Paraffin, night and morning.

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P E P T I C U L C E R (Cont'd).

PROGNOSIS.

Prognosis may conveniently be dealt with under three headings. They are:-

(1) As to life
(2) As to recurrence
(3) As to future health.

1. As to Life:

When discussing the value of treatment I have mentioned some of the mortality rates. Naturally deaths from surgery are in some instances higher than deaths when under medical treatment. As all perforations are dealt with by surgery the explanation of this higher rate is adequate. In the St. Bartholemew's report (106) quoted before the immediate mortality rates are given along with the late mortality rates.

	Immediate %		Late %	
	Gastric	Duodenal	Gastric	Duodenal
Medical	6.5	5.3	4.3	3.0
Surgical	20.8	4.9	6.7	6.5

It is evident from the figures given that the treatment of perforation of gastric ulcer is one of much more gravity than that of duodenal ulcer. Hamilton Bailey[111] has a death rate from all perforations of over 25%. As perforations occur in say 20% of all cases (though Stewart's post-mortem figures are 50%) then the percentage for immediate deaths from surgical treatment should be reduced by 0.2% for fair comparison. The severe procedure adopted for the surgical treatment of gastric ulcer must be the main reason for the high mortality rate. The death rate and incidence of haemorrhage does not warrant any deduction from the medical figures.

(2) Prognosis as to Recurrence:

(a) Medical Treatment:

There is no great divergence between medical and surgical claims. It would seem that the earlier treatment is begun the better is the chance of cure from medical measures. On an average, recurrence may be taken as about 40%

(b) Surgical Treatment:

There is an added factor here. There is the chance of marginal, jejunal or gastrojejunal ulcer developing.

I do not include any of my cases as I regard at least 10 years as being necessary before cure should be claimed.

(3) Prognosis as to future health:

Basford and Scott[112] have investigated working capacity after duodenal and gastric ulcer. Their conclusions are based entirely on the after history and do not include the original disease. Irrespective of the class of work performed, irrespective of the type of lesion and the type of treatment, the expectation of working incapacity is high.

These results would indicate further, that the future health of a person who has had a peptic ulcer is below that of the average person. The investigation was made in the personnel of the London General Post Office and this represents the effect on a very large and varied assortment of persons.

P E P T I C U L C E R (Cont'd)

CONCLUSION

The probable explanation of the development of a chronic peptic ulcer is that there is a lowering of local vitality. Important amongst numerous predisposing causes for this are faulty diet and nervous strain. Described types of organisms either attack the stomach and duodenum before such a devitilisation or more likely after it. The acid of the stomach tends to keep the ulcer open and to counteract a tendency for spontaneous cure. As Judd(41) has truly remarked, "The final word concerning (the pathogenesis of) gastric and duodenal ulcer has not been spoken".

Diagnosis rests particularly on two considerations. The first consideration is that following a careful clinical examination where especial note is made of the history. The second is a consideration of an X-ray examination. When operation has been advised or a post-mortem has been made, the excellence of modern radiology is proved. I have no regrets for basing my diagnosis so essentially on this ancillary science.

Claims that a proper etiological basis had been found, led to the administration of histidine. Its use has been very doubtful, if not useless.

A recent successful treatment of haematemesis has been described.

The mortality rate is not high. The future health and working capacity of the individual is frequently impaired.

Gastric acidity must be neutralised or buffered. Thus diet, alkalies and buffering agents are necessary. A reasonable proportion of cases does well. Relapses are likely in the present state of our knowledge.

Routine treatments are useful. Physicians and Surgeons are co-operating to obtain a higher proportion of cures. There must, however, be a decided change from general treatment of the disease, to the individual study of the patient, and his circumstances.

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T H E S I S

- on -

P E P T I C U L C E R

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OCTOBER 1938.