

A STUDY OF GASTRIC ULCER

Thesis for the Degree of M.D.

by

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## WITH ILLUSTRATIVE CASES.

The frequency with which ulcer of the stomach has come under my notice in an interesting variety of cases, together with the opportunity in some of them for surgical interference or post-mortem Examination, has led me to take this subject for my thesis.

Gastric ulcer is the most important idiopathic disease of the stomach, not only from its frequency and in many cases the facility of diagnosis during life, but also from its curability and on the other hand its sudden fatal termination in some instances.

By ulceration of the stomach I do not mean the superficial excoriation that often occurs during the course of gastritis; such a lesion is comparatively unimportant and soon undergoes a spontaneous cure. The cases I wish to record are cases of round, perforating or simple ulcers which involve loss of substance of the mucous membrane, unattended by morbid growth.

### ETIOLOGY.

Gastric ulcer is stated by Welch to exist in 5% of the total number of deaths from all causes, scars being/

being found more frequently than the unhealed ulcer. Of the 60 cases which have come under my notice, and which I think worthy of record, 38 were women and 22 were men. The average age at which they occurred among females was 26 years, whilst in men the average was raised to 35 years. None of the cases occurred amongst children.

Among women, the concomitance of anaemia and disorders of menstruation was the rule. Among men, there were no general associations, except that two were cachetic from syphilis and three were habitual drunkards.

As regards occupation, nothing could be gathered to throw light upon the development of the disease, except that the majority of the females were maid-servants and dressmakers, who followed indoor employments and revelled in the luxury of a tea-dinner. In fact two of the female cases confessed to drinking tea, which they kept "brewing" the whole day on the hob, nine and ten times in the 24 hours.

The Mode of Production of a gastric ulcer has been the subject of much discussion. Cruveilhier in 1829 was the first to describe the affection. Many years ago, it was thought to be the result of catarrh of the stomach. Subsequently, it was pointed out by Rokitanski that ulceration was frequently preceded/

preceded by necrosis of the mucous membrane, and this view has been generally accepted, i.e.-that an initial lowering of vitality or actual death of the part has taken place in that portion of the stomach wall which is to be the site of the ulcer.

What is the cause of this Necrosis? The peculiar funnel shaped form of the ulcer which is rarely absent, but which I think is commoner in the more chronic cases, led Virchow and others to believe that the primary necrosis was due to the occlusion of a nutrient artery by embolism or by endarteritis and thrombosis. This view is supported by the fact that when ulceration occurs it generally has its origin where the arterial branches enter the wall of the stomach. Experimental injections of foreign bodies into the crural artery of the dog by Cohnheim and Pavy have led to occasional embolism of one or more of the arteries with subsequent ulceration. At the same time, it is difficult to say that embolism always occurs in the initial stage of gastric ulcer. The gastric arteries are not end-arteries, and cannot be compared to the pulmonary, renal or splenic arteries which have a most imperfect anastomosis. Therefore, it is not surprising that in cases of ulcerative endocarditis it is uncommon to find a detached blood-clot or vegetation obstructing the arteries of the stomach/

stomach. Only on one occasion have I seen this phenomenon post-mortem.

The occlusion of a stomach artery by embolism is of course rendered easy if the foreign particles are injected into a neighbouring blood vessel and not into the general circulation. In a case of aneurism of the coeliac axis, it is recorded that numerous minute ulcers of the stomach have occurred as the result of detachment of portions of the blood-clot. In infective conditions, e.g., pyaemia or anthrax, one can often find evidence of bacterial embolism, and if the stomach be examined after death, numerous minute infarcts and ulcers are to be seen.

Another point which must be considered in a discussion of the pathogenesis of gastric ulcer, though it applies more to the duodenum than stomach, is the occurrence of ulcers in cases of extensive burns of the skin surface. I well remember the case of a big strong man, aet. 45, whom I saw after he had been extensively scalded, especially about the abdomen and thighs, by the bursting of a steam gauge. It was a case of the third degree of burning. After doing fairly well for 7 or 8 weeks he began to complain of pain, soon after the ingestion of food, and this was shortly afterwards followed by vomiting, haemetemesis, collapse and death. I was fortunate enough to obtain permission/

permission to make a post-mortem examination of the abdomen. On opening the stomach and duodenum, acute inflammation of both organs was observed. In the duodenum there were two small ulcers, each measuring about 2 m.m in diameter, deeply punched out with terraced shelving walls. In the stomach, towards the pyloric end on the posterior surface, I found another similar ulcer extending down to the peritoneum, which was inflamed. There were also a few small petechial haemorrhages in the fundus of the stomach.

"Pyæmic embolism" is the theory generally held to explain these duodenal or gastric ulcers following burns. If this is the case, it is difficult to say why the duodenum should be the seat of election rather than the stomach. In ordinary cases of pyaemia the stomach is said to have the preference in the ratio of 5:1. Another theory holds that in these cases of extensive skin burns the glands in the stomach and duodenum, after the destruction of the excretory sweat glands of the skin, take upon themselves more work in order to get rid of the waste materials that would in the ordinary course of events have been thrown off by the skin surface, and in so doing set up a gastro-duodenal catarrh with subsequent ulceration.

In any case, it certainly would seem that whenever/

ever the alimentary canal is called upon to get rid of toxic substances, whether absorbed from the skin or manufactured by microbes, it readily becomes irritated, inflamed and ulcerated.

Inflammation of the nutrient artery and gradual blocking by thrombosis is considered by some to be the cause of the initial necrosis. This may perhaps be possible in some chronic cases, but it does not appear to be a likely cause in the ordinary cases in young female adults. In the cases I have examined post-mortem I never found any evidence of it.

The possibility of the initial necrosis being associated with a small haemorrhagic extravasation must not be lost sight of, especially in view of the frequency of haemorrhagic phenomena in depraved conditions of the blood.

It has been suggested that a condition of venous stagnation may result in a lowered vitality and subsequent ulceration. This probably does occur in some cases of mitral or hepatic disease with portal congestion.

Pressure on any part of the stomach may perhaps be a cause in a few cases. At a post-mortem examination, in a case of aneurism of the abdominal aorta, I saw an ulcer of the posterior wall of the stomach brought about by direct pressure of the aneurismal/

aneurismal tumour.

Van Yzeren holds the view that gastric ulcer may originate in spasm of the pylorus, but in the majority of cases there is certainly no evidence of this. There is probably a confusion of cause and effect in these cases, for hyperacidity may produce pyloric spasm without any ulcer being present.

The possibility of the necrosis being due to microbic invasion from the interior of the viscus is somewhat remote, in view of the antiseptic qualities of the gastric juice, and also the small amount of lymphoid tissue in the stomach wall. This latter fact probably explains the rarity of tubercular ulceration. It is stated that primary ulceration may occur as a local bacterial infection in typhoid fever and anthrax. One must not lose sight of the fact that gastric ulcer generally occurs in the region where the secretion of hydrochloric acid is most active.

So far, I have only considered local conditions in the stomach wall as possible factors in the Etiology. Experimental ulcers have been produced in animals by mechanical and chemical irritation of the mucous membrane and by artificial emboli. But such ulcers have not shown the same life-history, so to speak, as a typical gastric ulcer. Unless the animals/

animals were first rendered anaemic by repeated blood-letting, these ulcers healed rapidly. A general consideration of the cases of gastric ulcer that one comes across in practice, along with a remembrance of the above observation, compel one to believe that whatever may be the cause of the initial necrotic change, there must be some secondary condition of the system or of the gastric secretion which tends to perpetuate the destructive action in the injured mucosa.

Virchow holds that after the necrotic action has been produced the affected area is acted upon by the acid gastric juice. After death, with the stomach empty, there is no change in the stomach wall for some hours; whereas, in the case of a full stomach, digestion of the wall by the acid gastric juice soon occurs. If the wall of the stomach be irritated by mechanical means in the lower animals an ulcer is not necessarily produced; but, if dilute hydrochloric acid be added to the stomach contents and allowed to come in contact with the part whose vitality has been injured ulceration readily occurs. It is estimated that hyperacidity of the gastric juice occurs in 70% of all cases of gastric ulcer. This association of hyperacidity of the stomach contents with an injured stomach wall is,  
I/

I consider, a most important factor in the causation of the disease.

Another all-important secondary or predisposing condition, which one nearly always finds present, is anaemia. The majority of the subjects of gastric ulcer are chlorotic. 30 out of my 60 cases presented anaemia, while 12 of the others developed the condition later, either from repeated haemetemesis and melaena or in consequence of the gastric trouble preventing sufficient ingestion of nourishment.

The examination of the blood, in these cases, always showed diminution of red blood corpuscles and haemoglobin, the latter being most marked, even down to 25% in one case. The red blood corpuscles averaged about 3,000,000 per cm. After a single small haemorrhage there is, in a few hours, a slight increase of the polynucleated white cells, and a few nucleated red cells may appear in a few days. After a large haemorrhage, with haemetemesis, I have noted the  $\frac{\% \text{ of red blood cells}}{\% \text{ of haemoglobin}} = \frac{1}{1}$  with the lymphocytes and polynucleated white cells markedly increased. In a few days, some nucleated red cells appear and then the percentage of haemoglobin and red cells begins to rise, not equally, however, the regeneration of the red cells being faster than that of their contained haemoglobin. It is astonishing what recuperative/

cuperative power the blood possesses in gastric ulcer.

The regeneration of the blood is greatly prolonged if there be repeated small haemorrhages. Therefore, in all cases of gastric ulcer, it is important to examine the stools carefully for evidence of bleeding, even though there be no marked melaena.

Before leaving the consideration of anaemia, reference must be made to the diminished alkalinity of the blood which one finds in chlorosis. The association of this diminished alkalinity with the hyperacidity of the stomach contents is undoubtedly of importance. The question as to whether anaemia causes gastric ulcer, or vice versa, is a vexed one. In the majority of my cases, anaemia was first present and showed itself some time before there were any gastric symptoms. On the other hand, however, some of my cases only developed anaemia after repeated haemorrhages from the stomach. When anaemia is present it certainly greatly retards repair of the stomach wall.

Menstrual disorders are frequently associated with gastric ulcer. Thirty out of my 38 cases exhibited amenorrhoea, whilst the other 8 showed irregularity or scantiness and were generally subject to haemetemesis at the menstrual period. I, for my own part, incline to the view that the menstrual disorder is/

is a result of the chlorosis which is invariably present, and I consider that the amenorrhoea is not a primary factor in the production of the disease.

Tubercle of the Lung may sometimes be present. Two of my patients had phthisis, with haemoptysis, as an accompaniment of the ulcer. In fact, the haemoptysis and haemetæmesis were very confusing in coming to a definite diagnosis, but I shall allude to this later.

One of the men developed phthisis subsequent to gastric ulcer.

From what I could make out the first two patients, who were women, aged 23 & 26 respectively, complained of gastric symptoms before pulmonary ones, and my view of the association of the diseases is that the ulceration and consequent inability to take a proper amount of suitable food lowers the vitality of the system and encourages the attack of the tubercle bacillus, which may have already been lying quiescent in the system.

A syphilitic element was present in two of my cases. In both the disease had been neglected. In one of the cases, in a man aged 45, death occurred, but I was unable to obtain a postmortem examination. I do not believe however that there is any relationship between the two diseases, other than can be explained/

plained by the syphilitic cachexia preventing repair.

Circulatory disorders may co-exist. Mitral valvular disease was present in five of the cases, but there was no evidence of embolism leading to ulceration, nor was I able to satisfy myself that there had been any haemorrhage through rupture of the distended veins, or thrombosis resulting from retarded circulation.

Here, also, I may allude to cirrhosis of the liver causing portal congestion; this occurred in one of my cases in which ulceration was present.

I have already referred to the possibility of aortic aneurism doing injury to the stomach by pressure.

Pyæmia and Septicæmia - especially the former - may show multiple small ulcers in the stomach consequent on septic embolism.

#### MORBID ANATOMY:

I had an opportunity of making an autopsy in what might be called a typical case of gastric ulcer with associated anaemia, and I will now state the facts of the case. It occurred in a young woman, aged 25 years, who died from collapse the result/

result of severe haemetemesis. On opening the stomach, I found a round, deeply punched out ulcer, about the size of a threepenny piece, on the posterior surface near the pylorus. The base of the ulcer, which was smooth, was covered with blood stained mucus and necrosed tissue, and <sup>in</sup> it could be seen a small ruptured blood vessel which was probably a branch of the coronary artery. There was no evidence of any inflammation of the walls of the bloodvessel or of thrombosis.

This is a typical case, but the ulcer instead of being round may be oval or irregular owing to the confluence of two ulcers. They vary in size from that of a pea to even the palm of the hand. The long axis of the ulcer is generally in the direction of the obliterated vessel, and the walls may be terraced, showing steps of mucous membrane, submucosa, and muscle. If this is the appearance it shews chronicity. In a chronic ulcer the edges are often hard and thickened and after healing deformity of the viscus, owing to contraction, generally takes place. As a rule, in an acute case deformity is uncommon.

It will be interesting here to record another case in which the diagnosis was wrong, but the true nature of the condition was shown at operation. The patient/

patient was a man, aged 45 years, whose case was diagnosed as dilatation of the stomach from pyloric cancer. At the operation it was discovered that no cancer was present, and that the cause of the obstruction was due to contraction of the pylorus caused by an ulcer which had cicatrised in that neighbourhood. The viscus was much dilated, the muscular coat of the organ was hypertrophied and the mucous membrane was thrown into heavy folds. The aperture into the duodenum was reduced about two thirds; the narrowing was due to inflammatory thickening of the mucous and submucous coats around the ulcer.

#### SITUATION:

As regards situation, the commonest sites for ulcer of the stomach, according to Brinton, are the posterior surface, the lesser curvature and the pyloric extremity. Of the six cases in which I had the benefit of a post-mortem examination, three occurred on the posterior surface, one on the lesser curvature, one in the pylorus, and the sixth was the case I mentioned before of multiple ulceration in septicaemia, due to septic phlebitis following an injury to varicose veins of the leg, in a young man aged 25 years.

Ulcers, however, are not always single and there may even be five or six present in the same individual.

SYMPTOMS & SIGNS:

The following are the chief symptoms and signs of ulcer of the stomach:--

1. Pain,
2. Localised tenderness,
3. Vomiting,
4. Haemetemesis.

1. The pain in most of the cases was characteristic. It consists of a heavy boring pain, in the centre of the epigastrium, coming on, as a rule, about five to ten minutes after eating and remaining during digestion, after which it ceases. Later, the pain becomes gnawing and burning and after a few weeks a similar dorsal pain shows itself, generally to the left of the 8th to 10th dorsal vertebrae, although in two cases I found it as low as the 1st lumbar vertebra. Both pains are generally increased by pressure on the epigastrium.

2. Localised tenderness: I consider this a most important sign of gastric ulcer. A spot of great local tenderness can nearly always be elicited just below the tip of the ensiform cartilage in the epigastrium.

3. Vomiting generally comes on one or two hours after meals and gives relief to the pain. It consists of food remnants in a watery liquid, and as a rule/

rule contains an excess of hydrochloric acid. It may be absent, the patient simply complaining of nausea or waterbrash and pyrosis with flatulency.

4. Haemorrhage may be the first symptom of ulcer, and it is estimated that it occurs in 40% of cases, although it may show itself only in the stools.

The bleeding may be simply the result of the erosion of some superficial vessel in the mucous or submucous tissue, and may be quite small in amount. On the other hand, a medium sized branch of the coronary artery may be the site of bleeding, and in this case haemorrhage is sudden and profuse. The accident most commonly occurs during the period of digestion, or after physical exercise. The symptoms vary according to the quantity of blood lost. Even before haemetemesis, the patient may become pale and restless, and may exhibit air-hunger. Usually the rupture of the vessel is painless, but sometimes it causes a stabbing pain with the feeling of something having given way. Giddiness and faintness arise; the body and extremities especially become cold and clammy, and the pulse becomes quicker and weaker. Occasionally palpitation, ringing in the ears and dimness of vision manifest themselves; dryness of the mouth and thirst are usually present and syncope may cause death.

If/

If the ulcer is acute, the chief features of the haemetemesis are suddenness, profuseness, and rapid cessation; but if the ulcer is more chronic, then the bleeding is smaller in amount and recurs with brief intervals till death occurs from exhaustion.

A few remarks may here be appropriate as to the differential diagnosis of haemetemesis. Not only is it to be distinguished from haemoptysis, but blood may be vomited as the result of bleeding from the mouth, nose throat and larynx. In haemetemesis, I have generally found the onset preceded by faintness and pain after food, with a previous history of vomiting. In haemoptysis, the onset is generally preceded by a tickling cough, and there is a previous history of loss of flesh and night sweats with coughing. In haemetemesis, if profuse and rapid, the appearance of the blood is usually bright red and clotted, with an alkaline reaction; but if the stomach is full and the bleeding is small in amount and slow, the colour, as a rule, is dark red <sup>like</sup> or, prune juice, with an acid reaction. Then again, melaena is generally a sequel of haemetemesis.

In addition to these distinctions, there are always the physical signs in the chest in the one case and in the epigastrium in the other, but one must/

must not forget that blood from the lungs or elsewhere may be swallowed and brought up as a coffee-ground vomit. Thorough examination of the mouth, nose, throat and lungs should be made to exclude this source of blood.

Several substances besides blood impart a brownish tint to the vomit as, for example, red wines and coffee, cranberries, bile and preparations of iron and bismuth; a similar colouration of the stools results from the administration of calomel and bismuth.

Melaena, if very small in amount, may only be recognised by chemical or microscopical examination. If the quantity of blood evacuated is a little larger, it is generally homogeneously mixed with the contents of the bowel, forming a soft chocolate-coloured mass. If the quantity is still larger, the stool becomes very foul and tarry.

#### FURTHER PROGRESS:

1. Cicatrization, with or without deformity.
2. Progressive ulceration giving:
  - (a) Haemorrhage from erosion of the vessel.
  - (b) Local peritonitis leading to adhesions to other viscera, with or without abscess formation or perforation of stomach wall.
  - (c) Perforation into general peritoneal cavity.
3. Development of malignancy.

1. Cicatrization is very common if the case is properly treated. The fibrous scar generally has a central depression and tends to contract. If the ulcer be near the pylorus, stenosis may result, or excessive fibrous formation may simulate malignant tumour. The latter condition occurred in a case in Professor Fraser's female Ward in the Royal Infirmary, Edinburgh, in the winter of 96-7, the true nature of the case only being discovered at the post-mortem examination. If the ulcer be nearer the centre of the viscus and horse-shoe shaped, hour-glass contraction may result.

2. Progressive Ulceration:

(a) Haemetemesis is generally the result of the erosion of a branch of the coronary artery, but any of the other gastric arteries may be involved. In addition to this, the bleeding may result from the destruction of a large vein, or from involvement of one of the more vascular viscera, if adherent to the stomach.

(b) The commonest adhesions are to the pancreas and the left lobe of the liver. Perforations may occur through these adhesions, with the result that the pancreas may be riddled with fistulae, as it was in one of the cases that came under my notice; or the left lobe/

lobe of the liver may be filled with pus, which discharges by a sinus into the stomach.

Adhesions may also form with the diaphragm, with the result that perforation occurs into the lung and a discharge of pus through a bronchus ensues; or the pericardium and heart may be perforated and death *results*.

Subphrenic abscess though infrequent is also a result. In the great majority of cases, the ulcer which gives rise to it is situated near the lesser curvature in the cardiac region of the stomach. The abscess almost invariably occupies the upper part of the left side of the abdomen; in only a small percentage of cases does it occupy the right side.

On the left side, it is bounded above by the diaphragm; below, by the left lobe of the liver and adhesions between the anterior wall of the stomach and the abdominal wall; on the left, by the spleen, gastro-splenic omentum and by adhesions between the cardiac end of the stomach, spleen and diaphragm; on the right side, by the falciform ligament; and in front, by the abdominal wall and diaphragm.

(c) If the ulcer is on the anterior wall of the stomach, a fistula through the abdominal wall may be formed, but, as a rule, if the ulcer is in that situation/

situation, owing to the mobility of the viscus and the absence of adhesions, perforation occurs into the general peritoneal cavity, followed by death if surgical interference be not adopted. This is the commonest site for perforation, although it is not the commonest situation for ulceration.

The only case of perforated gastric ulcer which I have had the opportunity of fully observing, occurred in a young woman, aged 27 years, who had fortunately only had a small amount of tea and toast two hours previously. Laparotomy was decided upon and performed within one hour. The whole abdomen was distended, and on opening it a good deal of gas escaped. On examining the stomach, the ulcer which had perforated was found on the anterior wall about its centre. Adhesions were absent, but round the rent there was a little localised peritonitis. The opening was about the size of a pea, rounded in shape, with somewhat ragged edges. General peritonitis had not occurred. Treatment was adopted, which will be described under its own heading, and the case recovered.

3. Development of malignancy. Another complication that should be noted, is the formation of cancer on the base of the ulcer or scar. It is estimated that 9 % of all cases of cancer of the stomach/

stomach originate in a simple ulcer. The scirrhus variety is the commonest, but I have not been fortunate enough to see a case.

DIAGNOSIS:

The symptoms and signs of gastric ulcer so closely resemble other diseases that I have annotated the distinctions. The disease has to be differentiated from among other things

1. Cancer of the stomach.
2. Chronic gastritis.
3. Gall stone colic.
4. Nervous gastralgia.
5. Ulcer of the duodenum.

	Gastric ulcer.	Cancer of stomach.	Chronic Gastritis.	Callstone colic	Nervous gastralgia.	Ulcer of Duodenum.
Age	From 20 to 40 years.	Middle and old age, say 40 to 60 years	Middle age.	Generally over 25 years.	18 to 35 years.	Generally middle age.
Sex.	Female 2:1.	Male commonest.	Generally male.	Female $\frac{3}{4}$ cases.	Female.	Male 10:1.
Epigastric pain.	Intense and fixed; soon after food, especially solids; increased by pressure, but ends with digestion.	Less intense; steadier; seldom intermissions even when stomach is empty.	One to two hours after liquid and solid food; moves about.	Irregular and sudden but very intense; often radiates to right shoulder; relieved by pressure.	Relieved by pressure, and alcohol and eating; for days free from pain; no relation to food.	Often 2 to 3 hours after food; generally where right parasternal line crosses right costal margin; continues when stomach empty; not relieved by vomit, but often by food.
Appetite.	Good, but eats little because of pain.	Very poor.	Bad, especially first thing in the morning.	Poor.	Variable; may be increased.	Generally good.
Vomiting.	Soon after food relieves pain; H Cl. in excess; no fermentation.	Not after each meal but once or twice a day or less frequently; diminution of HCl.; lactic, and butyric acids and bacteria present.	In early morning and after food; often contains mucus; no excess of HCl.	Severe retching; no relief to pain.	Rare.	Inconstant; no relation to food and no relief to pain.
Haemetemesis	Clear red or coffee ground; large quantity then long interval; relieves pain.	Coffee ground and small quantity at a time; offensive; short intervals.	Only if cirrhosis of liver is present.	Absent.	Absent.	Very frequent, copious and dangerous bleeding but melaena without haemetemesis is characteristic.

	Gastric ulcer.	Cancer of stomach.	Chronic Gastritis	Gallstone colic.	Nervous Gastralgia.	Ulcer of Duodenum.
Tongue.	Often red and dry, with white central stripe; may be slightly furred.	Thickly furred.	Furred, foul and flabby.	Often furred.	Normal.	Often furred.
Facies and complexion.	Often pale but may be pink and white.	Earthy looking with dry skin.	Auxious, worn, and pinched looking.	Often anxious looking and jaundiced.	Pale.	No anaemia.
Bowels.	Constipation.	Constipation.	Constipation alternating with diarrhoea; piles common.	May be obstruction from stone; often clay coloured.	Confined; often exhaustion after motion.	Variable, may be diarrhoea.
Loss of Flesh.	Only when chronic and then gradual.	Rapid and continuous.	Sometimes gradual loss.	If repeated attacks.	absent	absent.
Physical signs.	Epigastric and dorsal pain; localized tenderness; usually hypersecretion; R.B.C. diminished and Hb. especially; abdominal tumour rare; insidious onset; excess of HCl.	Tumour increasing in size and painful; absence of sudden onset.	Stomach may be dilated; Liver often enlarged and HCl. diminished.	Often rigor; rise of temperature and sweating; enlarged and tender gall-bladder.	Stomach often dilated; no tenderness; HCl. diminished.	Perforation common; jaundice from obstruction of common bile duct; dilatation of stomach from stenosis of duodenum; often associated with gall-stones.

LOCALIZATION OF ULCER:

It is stated that the posture the patient assumes to relieve the pain of gastric ulcer, is a criterion of the position of the lesion: e.g. if the ulcer is in the pylorus, then the easiest position is on the left side, and if it is in the greater curvature, then the pain is increased by standing; but the exact site is only determined rarely.

It is also stated that if pain comes on immediately after the ingestion of food, with vomiting, that is a sign that the ulcer is in the cardiac region or lower part of the oesophagus. Whereas, on the other hand, if pain is not in evidence until two to three hours after a meal, if it is referred to the right of the epigastrium and is associated with melaena it is a sign that the ulcer is in the pylorus or beginning of duodenum.

These modes of localization are, at the best, untrustworthy and although they have been correct in some instances in which I was able to verify my diagnosis, in others they have been misleading.

PROGNOSIS:

I always give a guarded prognosis, because so much depends on the treatment being carried out carefully and in detail, and also on the condition of/  
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of the heart and blood. It is estimated that about one half of the cases recover without leaving digestive impairment. Deaths however may occur from many causes: e.g. perforation, severe haemorrhage, inanition, haemorrhagic anaemia and deformities. The remainder become chronic invalids, and neuralgia of the scar tissue is often met with.

#### TREATMENT:

I will now discuss the treatment I have employed from its dietetic and medicinal aspects, and after that I will record a series of cases which are instructive in illustrating the symptomatology and treatment.

1. Dietetic All food must be avoided that would irritate the surface of the ulcer, mechanically or chemically, and one must also exclude anything that is calculated to stimulate the acid secretions of the stomach. A small quantity of food given frequently is a good rule to follow, for it prevents distension of the stomach, which is inimical to repair, and also prevents any undue excitation of the muscular movements of the viscus. Rest in bed is essential. Absolute physical and physiological rest is obtained by this treatment, along with the administration of nutrient enemata; but, in the majority of simple cases, I consider the rectal feeding too severe an ordeal, and/

and its continuance after a week or ten days is injurious to the system.

Of all the fluid forms of nourishment, milk is the most bland, nutritious and digestible. I generally prescribe it luke-warm, and give it in quantities not exceeding 6 oz. at a time, every hour, to be sipped very slowly. Then I increase the amount, until the patient can take nearly three pints in the 24 hours. If it does not quite agree with the patient, it may be skimmed, mixed with a little lime water or bi-carbonate of soda, or on the other hand sterilised or peptonised.

Very occasionally milk in any of these forms causes discomfort and then I generally fall back upon various meat juices and solutions, such as Wyeth's or Armour's, with perhaps a little chicken jelly.

Additions to the dietary at the end of a week or ten days may be made, but in each case their addition must be watched with care. At the end of three weeks, beef-tea, chicken broth, milk puddings, bread and butter and lightly boiled eggs may be given, and after that tripe, boiled white fish or chicken, and raw scraped red meat may be used. At the end of the second month, ordinary diet may be resumed - excluding pastry, raw acid fruits, potatoes, alcohol and all rich foods and sweets.

If nutrient enemata are required, I often use the following:-

R.

Peptonised milk ..... 2oz.  
 Raw beef juice (steak).....  $\frac{1}{2}$ oz.  
 One egg beaten up.

Sig. To be injected every 4 or 6 hours  
 with the addition of *half an ounce*  
 of brandy if necessary.

Alternately with this I give a meat enule (Burroughs, Wellcome & Co.) every two or three hours. If the enema is not retained, I find that the addition of Tinct. opii. m.15 to 20 to each or every other enema is very effectual.

2. Medicinal: In a simple case of ulceration I have seen the above dietetic treatment work wonders without the exhibition of any drugs. In some cases, however, drugs are necessary, and when the patient is unable to rest in bed I have seen great benefit result from the administration of Argenti nitras in  $\frac{1}{2}$  gr. doses, afterwards increased to 1 gr. I think it helps to heal the ulcer and I feel sure that it relieves the pain and vomiting.

Another valuable drug is Bismuth in the form of subnitrate or carbonate in gr. x-xv. doses. I believe that these salts of Bismuth protect the ulcer, to a certain extent, by forming a coating over it, and at the same time they relieve the pain. They are best given suspended in mucilage or glycerine, and in the case of the carbonate the addition of bicarbonate/

bicarbonate of soda in gr.v. doses is helpful. Again, if the pain is severe the addition of Liquor Morph., Hydrochlor. in m.x. doses, to the bismuth mixture is useful.

Bichromate of Potash, in gr. 1/8 doses, given in pill form has a similar action to Bismuth in promoting the healing and in relieving the pain of gastric ulcer. If there is a good deal of acidity with heartburn and pyrosis, the following powder is useful after meals:-

R.  
 Sod. Bicarb. gr. v.  
 Cretae Praeparat gr. x.  
 Mag. Carb. gr. v.

If given after meals the alkali neutralises the excess of acid and thus hastens digestion. The doses of chalk and magnesia may also be diminished or increased according to the amount of constipation or diarrhoea present, or they may be omitted altogether.

If vomiting is very excessive, the addition of Acid Hydrocyanic dil. in m.ii-iii. doses to the Bismuth mixture is often successful.

Excessive flatulence may be relieved by the use of acid carbohc (mj) resorcin (gr.v.) creasote (mj) or Tinct.Iodi (m.ii) In all cases the bowels require careful regulation. A soap and water, glycerine or castor oil injection per rectum is good, or Carlsbad/

Carlsbad salts ℥ii. to ℥iv., first thing in the morning, in a pint of hot water, at intervals, is successful if there is any constipation.

The treatment of haemorrhage is very important. In all cases the patient must be confined to bed and an icebag applied to the epigastrium. Rectal feeding should be commenced if the case is a severe one. This artificial feeding should not be continued so long as to starve the patient; a week should not be exceeded even in a severe case.

The safest plan in any case of haemetemesis is to give the stomach entire rest for two days, by the administration of nutrient enemata every four hours. After the rest, if the haemorrhage has not recurred, the enemata may be given twice a day, and 2 or 3 ozs. of milk may be allowed by the mouth every 2 or 3 hours.

On the fourth day, the milk diet may be increased in quantity and the enemata discarded.

In the great majority of cases the haemorrhage ceases, and does not recur, so that there is no pressing indication for medicinal treatment. If there is much restlessness a small dose of morphia hypodermically is advantageous. If, however, the haemorrhage is severe and obstinate, a hypodermic of ergotin, gr.v. should be given and the patient should be kept cool/

cool with only very few bedclothes. Should the bleeding be only small in amount and intermittent, Argenti nitras gr.  $\frac{1}{4}$  in a pill with opium quietens the circulation and diminishes peristalsis; the silver also acts as an astringent.

If the haemorrhage is associated with portal congestion the above treatment is assisted by the administration of calomel gr. i., repeated if necessary in a short while.

Should collapse or syncope threaten, ether or strychnine should be administered hypodermically, and diffusible stimulants injected into the bowels; or recourse to transfusion may be necessary. After the gastric symptoms have subsided, the anaemia so often present may require suitable treatment by means of the less irritating preparations of iron and regulation of the bowels.

The following cases illustrate some points in the symptoms, signs and treatment.

1. S.B., female, aged 25 years, complained of vertigo and headache, with shortness of breath and palpitation on the slightest exertion. For some time, she had been suffering from severe gnawing epigastric pain, increased on pressure, which came on about a quarter of an hour after meals, and lasted for fully two hours. After the lapse of a few weeks, she/

she felt a pain in her back to the left of the 10th dorsal vertebra, at the same time as the epigastric pain. Vomiting commenced, generally about half an hour after food, and gave relief to the pain. Her tongue was broad, pale, flabby, and tooth indented; her bowels were constipated and her appetite was good, but she had to "pay for it afterwards" if she ate heartily. Amenorrhoea had been present for the last three months. Being an out-patient at a hospital, she could not be properly looked after. She refused to rest in bed or stop work in the cotton mill, and indulged in quantities of tea. Lately her feet were swelled at nights.

She was put on a light milk diet and nitrate of silver in gr.  $\frac{1}{4}$  doses was given three times a day half an hour before meals; but she neglected her medicine and continued to take unsuitable food. She was running upstairs a few days afterwards when she vomited a quart of blood. She was brought to hospital in a collapsed condition and was put to bed with an ice-bag over her stomach. I injected ergotin hypodermically and later strychnine. The haemorrhage stopped, but she became collapsed. I therefore gave an enema of warm water and brandy per rectum and was preparing to inject sodium chloride solution subcutaneously when the patient expired.

2. The/

2. The next case is one whose pathology I have already described. The diagnosis made was cancer of the pylorus, but at operation it was shewn to be a case of pyloric stricture due to ulceration. The patient was a man, aged 45 years, cachetic and emaciated, with an earthy-looking complexion and an anxious expression. He had suffered for years from "indigestion" but for the last ten months he had been worse and complained of very severe pain about the epigastrium, with vomiting at intervals of large quantities of dark greyish material with a sour smell, containing mucus and the remains of food. Microscopically bacteria and sarcinae were present, but no blood cells could be found. The hydrochloric acid was diminished, but lactic acid was present. Constipation was present in a marked degree.

Inspection shewed peristaltic waves from left to right in the epigastrium, and on palpating the stomach a thickening could be distinctly felt at the pylorus. Splashing sounds were very evident and dilatation of the stomach from cancer of the pylorus was diagnosed.

He benefited considerably from lavage of the stomach; but an operation was decided upon as the only chance of improvement. On opening the stomach, which was dilated, a healed, contracted, annular ulcer/

ulcer causing constriction of the pylorus was discovered. A gastroenterostomy was performed by joining the jejunum to the stomach opening. Convalescence was uninterrupted and the patient is now at work as a gardener, in robust health, able to eat anything, and he has put on about 2 stones in weight since the operation.

3. The following case of haemetemesis is instructive: G.B., aged 26 years, complained of a good deal of flatulence and acid eructations, but had no gastric or lumbar pain and had never vomited. She had suffered for some months from a troublesome cough with expectoration of yellowish green mucus, especially in the morning. Amenorrhoea and constipation were present, and she had lost weight. On examining her chest, the right apex was found to be consolidated and the sputum on examination contained numbers of tubercle bacilli. One day, a profuse haemetemesis occurred without any warning. The patient complained of fullness soon after a meal with nausea; she became restless and uneasy and then blood of a brownish red colour was vomited. Her face became pale and she complained of faintness and cold extremities. She was put to bed, with an ice-bag over her stomach, and a hypodermic injection of morphia was administered.

Nutrient/

Nutrient enemata of milk, eggs and beeftea were given every four hours, the bowel being cleansed night and morning with salt water. On the fourth day she complained of great weakness, noises in her head and giddiness. There was no more haemetemesis but I found the night motion tarry, blown to pieces with gas and very foul. An ice-bag was again applied to the epigastrium and a hypodermic injection of m.v. of ergot was given in the gastric region. Food was given by the stomach on the fifth day after this injection and the patient recovered, but some years later I heard she had succumbed to phthisis.

4. The following facts illustrate a case of perforation, which occurred in a woman aged 27 years, who had been feeling quite well up to the <sup>time of the</sup> perforation, with the exception of a history of amenorrhoea. She only menstruated every three months, and the last time she was unwell was about a week before the perforation, and at this time she vomited a cupful of blood.

When I saw the patient, she complained of severe epigastric pain, which gradually became abdominal. She also told me she had been lifting a table when there was a feeling as if something had given way. Fortunately, she had only had a small amount of tea and toast some two hours previously. The whole abdomen/

abdomen was distended with gas and was very painful on pressure. The flanks were dull on percussion owing to fluid. Her face was pale and anxious; her feet and hands were cold and the body was covered with a clammy sweat. The pulse was very small and 120 per minute, and the temperature was raised to 102° F.

Icebags were placed over the abdomen, and a full dose of morphia was given hypodermically whilst preparations for operation were made. Laparotomy was performed and the perforation found fairly easily owing to the absence of adhesions, although there was a little localized peritonitis round the rent. Perforation was found to have occurred on the anterior surface of the stomach near its centre. The opening was about the size of a threepenny piece, rounded in shape, with somewhat ragged edges. The edges were pared and sutured with Lembert sutures as the stomach was quite empty, the ulcer being inverted and the peritoneal surfaces united together. The peritoneal cavity was then washed out with quantities of warm boracic lotion and carefully dried. The patient was fed, per rectum, for four days and afterwards by the mouth and made a good recovery.

5. The next case is illustrative of the treatment of an ordinary case of ulceration by diet.

G.R./



sucking ice or by sipping cold water.

At the beginning of the third week, Bengers' food, barley and well cooked rice in milk were allowed, every 3 hours, along with a little meat juice or meat jelly.

Towards the end of the week, soft boiled eggs, milk biscuits and a little well scraped raw meat were added to the dietary, and later well boiled chicken or cod was digested without discomfort.

Then ordinary diet was gradually resumed towards the end of the 6th week.

After the beginning of the 3rd week, she was allowed to sit up for an hour in the day and later for the whole day. At the beginning of the 4th week, she was able to walk out of doors and then she resumed her occupation.

If there is any constipation, the addition of a little lactose to the milk may relieve it - otherwise I generally give Carlsbad salts 3ii to iv. in 1 pint of hot water, to be drunk slowly in four parts at intervals of 15 minutes, early in the morning, before breakfast.

This simple treatment by rest and dieting without any medicine was sufficient in this case to effect a cure of the distressing symptoms. After the patient was able to sit up, the anaemic condition was treated/

treated with carbonate of iron in gr.v. doses and she eventually got quite well again.

I have found great benefit in similar cases, where there has been a good deal of pain, from the application over the abdomen of a piece of flannel wrung out of hot water and covered with oiled silk to prevent evaporation. I find it a soothing application and I think it also quietens peristalsis. At night-time, I sometimes substitute a cold flannel compress covered with oiled silk and a flannel binder, and I find it beneficial in inducing sleep.

6. Let me now record a more severe case: viz.,  
that of a man aged 35 years, suffering from portal congestion, and haemorrhoids owing to alcoholism. He was seized with violent pains and haemetemesis after suffering from "indigestion" for a fortnight. An icebag was applied to the epigastrium; perfect rest was enjoined and no conversation or movement of any kind was permitted. A hypodermic injection ~~injection~~ of gr.v. of ergotine was administered.

I have generally found that the thirst in these cases is relieved by sucking ice, but I do not approve of the swallowing of the lukewarm water from the solution of the ice. I think it only hinders the coagulation of the blood from diluting it. There was a good deal of restlessness, so a hypodermic injection of/  
of/

of morphine (gr.  $\frac{1}{2}$ ) was given. The bleeding was very obstinate and collapse was threatening, so I applied ligatures to the arms and thighs, -tight enough to prevent the venous return, but not so tight as to stop the arterial flow, -to withdraw blood temporarily from the circulation. The bleeding was stopped and the patient was fed by the rectum for 5 days. Early each morning, a large enema of one quart of lukewarm water with a teaspoonful of common salt, for cleansing purposes was given. An hour or so after the motion, an enema of half a pint of milk with an egg beaten up in it was given four times a day. Two of these enemata in a few days were replaced by warm beeftea (℥i.) yolk of one egg and ℥ii. of brandy, and the other two were continued as usual. After the fifth day, milk diet was started by the mouth and the man recovered, only to break down again some months later owing to his intemperate habits and the second time he collapsed. On this occasion he was put to bed with his head low and his feet raised. The former treatment was employed and the bleeding stopped, but the collapsed condition got worse. Strychnine was given hypodermically; warm bottles were applied to his feet, and I injected warm brandy and milk per rectum. I saw that the patient was sinking, so I injected one pint of warm saline water (6%)/

(6%) through the rectum. I massaged ~~to get~~ the blood out of the limbs into the trunk, and injected another pint of salt solution into the region about the lower angle of the scapula. Before I had finished the patient expired.

7. One of my cases, a man aged 35 years, could not afford to go to bed. He had typical symptoms of pain and vomiting and had had occasional attacks of mild haematemesis. He was placed on a light diet and was put on:-

R.  
 Argenti Nitras gr. vi.  
 Aq. dest.  $\frac{3}{3}$  vi.

Sig:  $\frac{3}{3}$  in as much water, thrice a day, half an hour after food.

After a week, the dose of silver nitrate was increased to a grain, and this was kept up for a month. The pain left him at the end of the first week, and at the end of the course the man said he had not felt so well for a long time.

8. In another case, I used the subnitrate of Bismuth in gr. ~~x~~ doses, three times a day, in a wine-glassful of water, well shaken up before food. After taking it, I ordered the patient to lie down for 15 minutes on his back, and then for 15 minutes on his right side, so that a protective covering might be formed/

formed over the ulcer. In 2 or 3 weeks, I found all the objectionable symptoms had gone, and in this case as in the majority of cases where I used bismuth, strange to say, there was no constipation.

If perforation occurs into the general peritoneal cavity, perfect rest, with an icebag over the abdomen, and full doses of morphia, exhibited at intervals, are simply of no avail, especially if the stomach be loaded with food, as is often the case.

Laparotomy is the only chance and this operation depends for its success or failure on three factors:

- (1) The time which is permitted to elapse between the occurrence of the perforation and the performance of the operation.
- (2) The treatment of the hole in the stomach.
- (3) The care with which the peritoneum is cleansed.

There are two methods of dealing with the case:

- (1) Closure with suture, with or without paring the edges.
- (2) The making of a gastric fistula externally.

When possible, I advise the first method with pared edges, sometimes at the same time, inverting the ulcer and uniting the peritoneal surfaces together with Lembert sutures.

Cases of subphrenic abscess must also be treated surgically. As soon as the existence of the abscess can/

can be determined, the cavity should be opened and drained; for every day that the operation is postponed increases the risk of secondary inflammation of the thoracic viscera and perforation of the diaphragm.

Incision in the left hypochondrium is as a rule most convenient, but care must be taken not to disturb the adhesions between the stomach and the abdominal wall, which prevent extravasation into the general peritoneal cavity.

The prognosis in cases of subphrenic abscess is not very hopeful. Of the cases on record about 20% only have recovered.