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PEPTIC ULCER OF THE STOMACH

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by

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PEPTIC ULCER OF THE STOMACH.

HISTORICAL.

Peptic Ulcer of the Stomach was first described by Cruveilhier about 1830. In 1856 ("Archives Generales de Medecine, February and March") he published a further account. He describes it as being characterised by dis-comfort in the epigastrium after food, the patient only being at ease when the stomach is empty, and by loss of appetite. Later the dis-comfort becomes an actual pain; there is in addition Haematemesis. The pain is about the ensiform cartilage, is increased by pressure, and directly caused by food, coming on shortly after a meal and lasting till the stomach is empty. In some cases it radiates upwards behind the Sternum or into the lower intercostal spaces. He further points out that when severe there is a corresponding pain in the back to the left of the 10th, 11th and 12th Dorsal Vertebra; he admits that it is difficult to distinguish the pain from that occurring in some conditions without ulcer. Brinton, "Ulcer of the Stomach", 1837, is still regarded as a classic. Rokitansky (Oestr. Med. Jahrb 1839) gives an account of the condition which he regarded as a form of necrosis caused by venous hyperaemia, Trousseau, "Clinical Lectures, Vol.IV, pages 64 to 92" gives



a most interesting description of the condition. Later, Virchow and Pavy worked at the subject from a pathological standpoint. Leube and Kussmaul introduced the stomach tube and thus demonstrated various alterations in the gastric juice.

ETIOLOGY.Incidence:

The postmortem percentages given by different authorities vary, thus, Dietrich in 10,103 examinations found open ulcers in 126 and cicatrised in 224 i.e. 3.04%. Brinton gives 5%. W. H. Welch 5%. Others give 10 to 13%. Clinically, it is found to vary in different countries, thus, it is less frequent in America than Europe (Da Costa); and is more frequent in Canada and Massachusetts than in Baltimore (Osler).

Sex and Age:

Welch, in 1699 cases, found 40% in men, 60% in women: Fenwick, in 2031 cases, found 40% in men, 60% in women. In 89 cases specially examined Fenwick found 30 acute and 59 chronic, 10% of the acute were in men, and 73% of the chronic were in men. The general view seems to be that the proportion in women is about 2.5 or three times that in men. In women 75% of the cases occurred under 30 years. In men 25% under 30 years. Though the majority of cases occur in the 3rd and 4th decade, it is not limited to this period. Goodhart has reported a case in an infant 30 hours old; among 390 autopsies at the Babies Hospital, New York, five cases were found. In general practice, I have had

a fatal case in an infant six months old, and a typical case in a woman of 58.

Heredity:

Dreschfield notes six cases. In two, mother and daughter suffered. In one, father and daughter; in two, two sisters; and in one, brother and sister. In my practice, a mother and son have both suffered, the rest of the family being healthy; and in another family, a brother and sister; in the latter case the sister was a dressmaker, the brother a clerk, and it would seem most probable that the mode of life might be of more importance than any hereditary influence.

Occupation:

Different observers state that different occupations present most cases, such as cooks, housemaids or metal turners.

Trauma:

A blow in the stomach region and tight-lacing have both been accredited as causes, but I think it will be conceded that the number of cases of either in which an ulcer occurs, is infinitely smaller than the number in which it does not occur.

Associated Diseases:

Chlorosis has a universally accepted association

with gastric ulcer: other conditions such as tubercle and syphilis may cause specific ulceration of the stomach in their course. It is very possible that syphilitic endarteritis might be a factor in causing peptic ulcer.

PATHOLOGICAL ANATOMY.

On examination, either on operating or post-mortem, of cases diagnosed as gastric ulcer, various conditions may be found. They may be classified as follows:-

A. Erosions:

- (a) Simple erosions: these appear to be mere abrasions, they are very small and may easily be overlooked post mortem. If seen when bleeding, the mucous membrane seems to be studded with numerous bleeding points. It may be noted here that in some cases in which most severe haemorrhage has taken place no erosion may be found on the most careful examination.
- (b) Exulceratio Simplex (Dieulafoy). The surface layers of mucous membrane are removed exposing the arterioles and giving rise to terrible haemorrhage. I have not found any cases of this condition described.

B. Ulcer:

- (a) Acute round ulcer.
- (b) Chronic ulcer.

The ulcer has a characteristic appearance and is found in the stomach, first part duodenum, and lower end of the oesophagus. It is single in five cases out of six and is most commonly found at the pyloric end towards the lesser curvature; the following table gives the statistics of its situation:-

| | | | % | % | Chronic | Acute |
|-------------------|----------------|--------------------|---|--------------------------------------|---------|--|
| Posterior surface | 43 | 235 | | 254 | 25 | |
| Anterior " | 4 | 96 | | 82 | 8 | |
| Both surfaces | 6 | - | - | - | - | |
| Lesser Curve | 27 | 288 | | 366 | 36 | |
| Pylorus | 16 | 95 | | 158 | 15.6 | 53 75% |
| Cardiac End | 2 | 50 | | 80 | 7.9 | 10 |
| Greater curve | 2 | 27 | | 42 | 4.14 | |
| Fundus | | 24 | | 33 | 3.3 | |
| Middle Zone | | | | | 7 | |
| | 100 Brinton | 793 cases Welch | | 1015 Fenwick col- lected cases | | Fenwick's own cases 70 chronic 39 acute. |

Fairly equally distributed.

The more recent or Acute Ulcer is circular; $\frac{1}{2}$ to 1 inch in diameter and of varying depth. It is clean cut, or punched out, and is classically described as being funnel-shaped or stepped. The latter appearance being caused by successively less of the succeeding layers of the stomach wall being involved. There is no thickening, but sometimes the surrounding mucous membrane may show small haemorrhages or areas of congestion. The depth varies, one or all of the coats may be perforated. The floor is usually smooth, and rarely has been necrotic in part.

The Chronic Ulcer is more irregular in outline and of greater size. The edges and floor show thickening due to infiltration. If near a solid fixed organ the ulcer may have become adherent to it and may even have penetrated it. The pancreas, liver or spleen, may be thus involved, in other cases adhesions take place to the diaphragm or mesentery. These adhesions are protective preventing perforation into the peritoneal cavity. The anterior surface of the stomach is in relation to the moving abdominal wall so that adhesions do not readily take place, and the perforation into the peritoneal cavity of ulcers in this situation is thus more frequent. Sometimes perforation occurs into the transverse colon, the duodenum, or small intestine (in order of frequency) causing a bimuscular fistula. Rarely such a fistula is found into the pericardium, the pleura, the gall bladder or a bronchus. Subphrenic abscess may also be caused either by a slight leakage or from the spread of inflammation. In cases of fatal haemorrhage the open end of the artery is sometimes found in the floor of the ulcer.

Healing of the ulcer takes place with varying results. Slight ulcers or erosions involving only the mucous membrane, heal without leaving any trace. Deeper ulcers leave a dimpling or thinning of the stomach wall. While if the process has extended to,

or nearly to, the peritoneal coat there is marked cicatrisation. This by contracting may cause stenosis of the pylorus or hour-glass stomach.

Microscopically various changes have been found.

In Acute Ulcer these are not seen but in chronic ulcer there is marked alteration in the glands which are lined and filled with cubical cells, the interglandular tissue, submucous, and muscular layers, show cell infiltration with formation of fibrous tissue which is also found replacing the muscle in the floor of the ulcer, the density varying with the degree of chronicity. The arteries sometimes show periarteritis and endarteritis; sometimes appear to be changed into embryonic tissue thus rendering haemorrhage easy of occurrence.

Jaworski and Korezenski state that they have found cell infiltration etc. in the mucous membrane in all cases of gastric ulcer, even at some distance from the ulcer, and marked inflammatory change in the artery walls. They regard this as the cause of the ulcer.

It would seem most probable that these changes are due to an associated gastritis.

THEORITICAL AND EXPERIMENTAL.

Numerous theories have been put forward and a considerable amount of experimental work done to account for the peptic ulcer. Primarily we must consider the stomach as a tidal organ which may be influenced in different ways. Thus stomach symptoms may be caused by derangements of the nervous system, by defects in the circulation, by alterations in the blood, by alterations in the gastric juice, by unsuitable food, and by external influences.

The Nervous System controls the secretory function, the vascular tone, and the motility of the organ. Thus in nervous dyspepsia there is an increase in the acidity of the gastric juice; in some diseases of the nervous system small haemorrhages occur in the mucous membranes; in other conditions muscular spasm may be caused. These facts have each been given as a cause of gastric ulcer. The increase of acidity will be considered later; punctiform haemorrhages due to nerve disease are too rare to be of signification; muscular spasm whether due to nervous disease or not, may have an influence. Thus, Dr Van Ijzeren resected the vagi below the diaphragm in rabbits. The result was slight paralysis followed by permanent spasm of the muscular

coat, in two or three weeks he as a rule found an ulcer in the pyloric region, near the lesser curvature. This was generally single and did not heal. The gastric juice was not hyperacid. He further found that by dividing the pylorus parallel to its axis, down to the muscularis mucosae, the ulcer was prevented eleven times out of twelve. Gastroenterostomy also prevented its formation. He considered that as gastroenterostomy gave good results the spasm was reflex from the passage of food through the duodenum. His deduction is that the spasm nips the blood vessels and thus causes necrosis of the mucous membrane. There can be no doubt that the presence of an ulcer or any irritation of the stomach may cause spasm.

Dr Dalla Vedova has by dividing the vagi and destroying the coeliac plexus obtained in about half the cases distinct changes. These were found chiefly in the antrum pylori, and were ulcers, haemorrhages, spots of necrosis, and cicatrices of ulcers. He regards the site as being determined by the more highly acid juice there and by the constant irritation of the weakened wall by the passage of the more solid parts of the chyme.

Circulatory defects greatly affect the stomach as in the backward congestion of heart disease.

These cases are, however, not prone to peptic ulcer. Rokitansky considered that venous hyperaemia was the cause of the condition. Virchow attributed the condition to embolism and this view is held by many. The shape of the ulcer is considered to support it, but Fenwick has shown, that, if a cautory be applied to the mucous membrane destroying irregular portions, the resulting ulcer always takes the round funnel shape. Further, to find the embolus is exceedingly rare; and, if solid particles be injected into the circulation, the stomach is one of the last organs in which emboli are found. Dreschfield points out that ulcer usually occurs in those with healthy vessels so that thrombosis as a cause may also be regarded as of little importance. It seems feasible that the stomach being an elastic organ, the circulation to some parts may at times be mechanically impeded.

Alterations in the blood would appear to have an important influence as evidenced by the frequency of the occurrences of ulcer in chlorosis. Quincke and Daettwyler (Correspondenz - Blatt fur Schwertzer Aerzte 1875, page 101), injured the mucous membrane in animals rendered anaemic by bleedings. The abrasions did not heal but continued to form distinct ulcers which in some instances perforated.

Silberman (Deutsche Med. Woch. 1886, No.29) produced haemoglobinuria, injured the mucous membrane, and found ulcers result.

This may be compared with the ulcer following burns. Considering that it has been shown that the latter appears to rise from irritation of a solitary gland which has probably been rendered susceptible by the spread upwards of duodenitis caused by toxins excreted in the bile, it seems fair to conclude that some circulating toxins may, in the presence of HCl and given diminished vitality, cause necrosis of the tissues. That the blood may be definitely affected in constipation is shown by the occurrence of enterogenous cyanosis (West and Clarke, Lancet, February 2nd, 1907).

Alterations in the gastric juice have been credited with great causative importance. Excessive HCl is found clinically in the great majority of cases. There are, however, a few cases in which the HCl is diminished or even absent, and there are other conditions in which there is great hyperacidity but no ulcer. As, however, peptic ulcer is only found where HCl is also found, it would appear that the presence of the latter is essential to the production of the condition.

Mathes injured the mucous membrane of a dog's stomach, and, by administering HCl daily, succeeded in producing an ulcer.

Dr Pavy suggested that the stomach was digested by the gastric juice if the protecting layer of mucous membrane was destroyed, or weakened by diminished blood supply. Against this must be set the fact that superficial injuries of the mucous membrane heal very rapidly. I have myself, in washing out the stomach, brought away several pieces of what, on microscopical examination, was proved to be gastric mucous membrane; yet on post mortem some six weeks later, there was no sign of ulcer.

Unsuitable food has been credited with much. Thus, it has been suggested that cooks suffer very frequently owing to their habit of tasting very hot food, thus causing a spot of necrosis. Haemorrhagic erosions, and in one case a typical acute ulcer, have been produced in this way in dogs (Decker) Unsuitable and indigestible food is a cause of gastritis, and it is more probable that it may predispose to ulcer in this way. Thus shop-girls and house-maids who frequently live to a large extent on tea and bread, or buns, are very frequent sufferers from dyspepsia in all its forms.

Under this heading one might also consider the possibilities of sepsis. There are numerous organisms in the food and in the mouth, etc. If the stomach is secreting at the time the organisms are swallowed, they are killed by the acid juice; if it is not, they may manage to find a lodgment. It may be noted that the glands at the pyloric end do not secrete HCl so that the mucous membrane in this region is more susceptible of microbic invasion than at the cardiac end. Mayo Robinson has suggested that pyorrhoea alveolaris may in this way have an influence.

External influences such as tight-lacing, constant pressure, as in shoemakers and weavers, and blows on the epigastrium have all been credited as causes, at the best they would appear to be remote though Ritter and Vanini have produced various stomach injuries in animals by repeated blows on the epigastrium.

From a consideration of the foregoing the following conclusions may be drawn:-

- (1) The peptic ulcer can only occur in the presence of acid.
- (2) That the acidity of itself is not sufficient to cause necrosis in a reasonably healthy stomach, even if the mucous membrane be injured.

- (3) That the portion of stomach that does not secrete HCl is most susceptible.
- (4) That no one definite cause can be given, but that diminished vitality of the tissues as in chloris, or as a result of prolonged and unsuitable food, pre-disposes. If to this be added some gastritis, then the special weakening and possibly the excess of HCl with some slight further irritation either in the food, or due to organisms, or to interference with the blood supply, may cause the necrosis.
- (5) Possibly circulating toxins if the tissues are weakened may be the cause.

The fact that the pyloric end towards the lesser curve is the commonest site of the ulcer may possibly be explained by the following:- (1) The non bactericidal nature of the secretion. (2) The blood supply of the part if derived principally from the pyloric branch of the hepatic artery. When the stomach is distended, the pyloric portion becomes bent on itself, and consequently there is some kinking of the artery interfering with the blood supply. Also, if, in the cadaver, ligatures be placed on the termination of the oesophagus and beyond the pylorus and the stomach be then distended through a small incision, it will be found that it presses distinctly on the origin of this artery, thus further diminishing the blood supply. I have only had one opportunity of doing this, the body was not specially prepared.

SYMPTOMS.

There are three cardinal symptoms, pain coming on about twenty minutes after food, and lasting till the stomach is empty, vomiting, and haematemesis. In a typical case the patient will state that for some time pain has been felt after food, that it is made worse by pressure, and occasionally she has vomited, after which the pain has been eased, and that she has noticed that the vomited matter has been dark brown at times. There are all possible variations on the above. Thus taking the symptoms in series.

Pain: This is most commonly in the epigastrium at the pit of the stomach; it is dull or boring in character, and sometimes is felt to extend through to the back, to the left of the 10th, 11th and 12th dorsal vertebrae. It is usually increased by pressure. Robson and Moynihan state that pressure on the above spot on the back elicits tenderness until the ulcer is healed. I have not found this to be so; in two convalescing cases in which I found no tenderness, there was a marked return of the symptoms in a couple of days.

A sympathetic neuralgia occurs in some cases, pain being complained of about the 5th and 6th dorsal vertebrae, or at the tip of the shoulder or even radiating down the arm. An attempt has been made

to divine the precise site of the ulcer from the situation of this pain, but as it is sympathetic this would not seem to be feasible.

Its occurrence varies in different cases: commonly it comes on fifteen to twenty minutes after taking food, being then caused by the acidity of the contents during digestion irritating the ulcer. It then lasts until gastric digestion is complete, or until the stomach is emptied by vomiting. In other cases it comes on at a longer interval after food, sometimes as much as two or three hours. This may be because the ulcer is at the pylorus, the passage of the chyme causing it; or in other cases, in which it is immediately relieved by taking a little food, as a glass of milk and a biscuit, or by a dose of bicarbonate of soda, it may be that there is an inopportune secretion of acid when the stomach is empty. Duodenal ulcer might give rise to a delayed pain, but the immediate relief afforded by a little bland food in the majority of these cases, would seem to show that the cause is in the stomach.

More rarely it immediately follows the taking of food; it must then be due either to the ulcer being at the cardiac end or to very great irritability of the stomach.

Sometimes when hyperacidity is marked, pain comes on worst during the early morning, and this

may be the only time when it is felt; more commonly, with this early morning pain, there is a delayed pain after food.

The quality of the pain varies also, most typically it is dull and boring and is then sometimes described by the patient as being like a gimlet being bored through. In other cases it is more dull in character and is then compared to a dead weight like lead. In others it is at times most severe, the patient writhing in agony. As a rule it is found that pressure on the epigastrium increases pain, though in some cases relief is obtained by lying flat on the face with a hard pillow under the stomach; it has been suggested that the ulcer is then on the posterior aspect of the stomach.

In the majority of cases careful palpitation of the epigastrium will reveal a distinctly tender area or at any rate a small area of greatest tenderness. This is of great importance diagnostically.

There are certain cases of ulcer in which a dull aching pain between meals is the only symptom (Osler). I have myself had one case in which there was only this dull ache. Two years careful dieting (milk and milk foods) were required to effect a cure of the pain which I am inclined to attribute to an ulcer.

There are other cases in which pain is entirely absent. Personally I have always found it to be present at some time though not always the symptom which draws attention to the case. Thus a girl of 22 complained of belching up great quantities of wind, there was no pain, but two days later haematemesis came on, as she recovered and food by the mouth was given she complained of pain which proved troublesome and continued for some months.

It is to be noted that during menstruation pain is much aggravated. In one case of mine it was so intense and continuous at this period as to cause the greatest anxiety to the friends, the pulse, however, remained of good volume and was not quickened.

Sometimes anaemic girls suffer from pain with most of the characters of that caused by ulcer; thus it comes on fifteen to twenty minutes after food, is situated in the epigastrium and is boring in character. Some of the cases are undoubtedly due to ulcer; if so, there is a definite spot of tenderness in the epigastrium.

Haematemesis: The vomiting of blood has been accepted as the great characteristic of peptic ulcer.

There are cases in which it is the only symptom, but also there are cases in which it is absent, and others in which it is most severe and sometimes fatal and yet no gross lesion of the stomach is to be found.

This was perfectly recognised by Trousseau; in commenting on it (Clinical Lectures, Vol.IV., p.71) he says:- "In fact though gastrorrhagia is observed in the majority of cases of simple ulcer, it is a symptom that is sometimes wanting; moreover, it is a phenomenon also belonging to cancer of the stomach, which sometimes shows itself in non-ulcerous chronic gastritis, and is likewise met with in a considerable number of cases presenting no apparent lesion of the organ which is the seat of the haemorrhage." He then gives illustrative cases.... p.72. One case of perforative peritonitis without previous symptoms; p.73, one case with slight pain and slight tenderness, but no gastrorrhagia in which a partly healed ulcer was found post mortem; p.79, one case of gastrorrhagia without any previous symptoms and without pain, in a woman, aet 65. This passed off in three days under iron and rhatany; three days later no trace of blood was to be found in the stools: a month later she was well: five years later on enquiry her health was excellent; p.80, a man aet 33 died

suddenly vomiting blood, post-mortem, there was no trace of ulceration of the stomach or duodenum, the lungs, heart and brain were healthy.

Hale White (Lancet, November 3rd, 1906, p.1189 et seq.) has a paper on haematemesis without obvious gastric lesion. He suggests gastrostaxis as a name for the condition, which he regards as most probably due to a blood state. He gives a bibliography and notes 29 properly authenticated cases but does not mention Trousseau nor include the last case quoted above.

In ulcer the haematemesis varies in quantity and appearance. There may only be a slight oozing, the blood being then usually vomited mixed with the food, giving it a brown or "coffee-ground" colour, due to its alteration by the gastric juice. If rather more blood has been lost, as in slight haemorrhage from an arteriole, the blood may remain for some time in the stomach and is then vomited as clots or lumps of a dark colour. If the haemorrhage is very severe, the patient feels faint, turns pale, and breaks out in a clammy sweat, there is a feeling of abdominal discomfort, and then, suddenly, almost without effort, the blood is ejected.

An unpleasant salt taste in the mouth is usually noted. Under treatment the haemorrhage may not recur, but as a rule, it is repeated in a day or two.

Thus, a girl aged 22 already alluded to, felt a sudden desire to go to stool, she got up and immediately fell forward in a semi-fainting state, and vomited up about two pints of bright liquid blood, two days later the haemorrhage was repeated, this time the blood was in dark lumps, and one day later there was a slight vomiting of blood-stained fluid.

Another, a woman aged 54, vomited about a pint and a half of blood and blood stained fluid at 7 p.m. two days later at 2-50 p.m. she vomited about two pints of blood clot and blood stained fluid. The hand laid on the epigastrium felt a peristaltic wave, every five minutes or so, in the pyloric region, accompanied by a thrill and a bubbling sound. She became very faint and blanched. Next morning, the bowels acted copiously twice, the motions consisting of black tarry liquid. She eventually recovered.

When gastric haemorrhage takes place, the blood is not all vomited. A varying portion escapes into the intestine and causes melaena. When only a very slight oozing is going on and no vomiting, the blood may still be detected in the motions. Sometimes the guiacum and ozonic ether test is required to detect it. Until this gives a negative result it cannot be considered that the haemorrhage has ceased.

In other cases, all, or the greater portion of the blood may pass into the intestine. Thus, I saw a lad of twenty, one night, vomiting blood. He was very collapsed and appeared to have lost a considerable quantity. Next day there was no recurrence of the vomiting, and his condition had much improved by the evening, but during the night he became very restless and faint and suddenly sat up in bed and fell back dead.

In another case which I was called to see in consultation, the patient, a woman of 25, had a week previously had a severe haematemesis, this had been repeated two days later. When I saw her there had been no further vomiting, but she was blanched and gasping and died in a few minutes.

As a general rule, even in severe cases the patients recover.

Haemorrhage from the stomach also occurs in cases of cancer, cirrhosis of the liver, and backward congestion from heart disease. In these conditions, however, it is generally easy to differentiate.

Vomiting of food occurs frequently. The food may be vomited immediately after being taken if the stomach is very irritable, or an hour or more after when the cause is the irritation of the acid juice. The vomited matter in this case will be found to

contain an excess of HCl. According to Dreschfield, in about a fourth of all cases of ulcer the acid is normal, while in some chronic cases with anaemia or dilatation of the stomach it is diminished. In old standing cases with dilatation of the stomach, vomiting, characteristic of that condition, occurs.

Clinically four main types may be distinguished:

- (1) The fulminant; the first symptom being perforation or severe haemorrhage.
- (2) The anaemic type.
- (3) The gastric type.
- (4) Chronic cases.

The anaemic type includes a great number of cases. The patients complain of anaemic dyspepsia, they vomit food at times and haematemesis is not very frequent. The tongue is clean but pale and flabby. Under treatment for the anaemia they improve but generally relapse once or twice. Thus one girl of 20 has been under my care during the past fourteen months she has never had haematemesis, but there was a definite spot of tenderness near the ensiform cartilage. She suffers from chlorosis and at intervals comes under observation, when this becomes worse. She then has pain after food and occasionally vomiting about one hour after food, these attacks are, however, becoming less frequent and less severe.

In the gastric type the pain and vomiting are more severe and haematemesis is commoner. In these cases if they carry out orders the prospect of a quicker permanent cure seems better.

The woman aet 54, already alluded to, had had very severe pain, totally incapacitating her for four years previous to coming under observation. On one occasion during this time she vomited a little blood. After three months treatment she had no recurrence of the pain, was able to take a fairly mixed diet, and to do housework. Chronic cases result when the patient is neglectful or when the environment is bad.

The haemorrhagic type mentioned by Dreschfield (Allbuts, System of Medicine, Vol.III., p.539) must be accepted with reserve in view of the possibilities of gaströs taxis.

Complications: Perforation is the most serious of these, the signs are: collapse followed by general peritonitis. If operation is not resorted^{to} within twelve hours, the case may be regarded as hopeless. Some few cases in which the perforation is minute or the leakage limited by adhesions are said to sometimes recover.

Subphrenic or localised abscess may occur if adhesions have previously formed about the site of

the perforation. The signs are those of a localised collection of pus following the symptoms of perforation, there being special symptoms in the stomach region. Various other results of perforation as given by Dreschfield have been noted under pathological anatomy.

Cicatrisation of an ulcer may cause stenosis of the pylorus leading to dilatation of the stomach, or if it be situated differently, to hour glass contraction. I have seen one case of the condition the result of oxalic acid poisoning. There was no dilatation of the stomach to be made out but vomiting was persistent and as a rule "bilious", and emaciation was extreme. Gastroenterostomy afforded some relief. Adhesions to surrounding organs after the ulcer has healed may cause severe symptoms. Robson and Moynihan report several cases on which they have operated.

Various alterations in the urine occur in some cases. Albumin is sometimes found; in others diacetic acid and acetone.

Nervous symptoms are said to occur in some cases. Thus in chronic cases with dilatation of the stomach, tetany may arise. Robson and Moynihan report several cases.

DIAGNOSIS.

The points on which the diagnosis rests are, pain following food, vomiting followed food, haematemesis, and a definite point of greatest tenderness in the epigastrium. The following conditions present the greatest difficulty in differentiation.

Gastrostaxis: Hale White says: "It is often very difficult or impossible to separate these cases from gastric ulcer unless the gastric ulcer has led to hour glass stomach, gastric dilatation, tetany, a mass of thickening that can be felt, or a subphrenic abscess, but points that will help are that these cases of gastric oozing of blood are almost confined to women, while a considerable proportion of gastric ulcers are present in men. If the patient has not been starved as a result of treatment, I think that a sufferer from gastric ulcer is more likely to be wasted than one afflicted with gastric oozing, and although some of these give a history of chronic dyspepsia, pain after food, and tenderness over the stomach, yet the occurrence of considerable intervals during which there is no complaint of gastric symptoms is, perhaps on the whole in favour of gastric oozing."

"In the present state of our knowledge, the diagnosis must sometimes be a matter of consider-

able difficulty."

It would seem feasible that the presence of one definite point of tenderness at the same spot on several examinations, should be accepted as in favour of ulcer.

Anaemic Dyspepsia: This is characterised by pain after food and occasional vomiting. The vomiting, however, has not the definite relation to food that it has in ulcer, occurring irregularly and sometimes at night without much relief from the pain. There is not one fixed tender spot. If this latter point be carefully looked to, it should rarely be necessary to decide from the effects of treatment as recommended by Dreschfield.

Other forms of dyspepsia: In these the vomiting is irregular and the pain not so localised and not increased by pressure.

Cancer of the stomach: If the tumour cannot be felt, may present some difficulty. Regard must be paid to the age of the patient. An examination of vomited matter will reveal diminution of HCl. Cachexia is not to be relied on. The effects of treatment might be watched for a short time. It must be remembered that a palpable mass of adhesions may be due to ulcer.

Duodenal ulcer is as a rule not to be distinguished clinically. From the pain coming on some hours after food and being situated more to the right, a diagnosis has been made once or twice. (Osler. System of Medicine). Dreschfield quotes Boas as stating that the vomit has an alkaline reaction, contains bile, and digests fibrine. This seems rather far fetched.

Gall stones, if jaundice is not present regard must be paid to the sudden onset of the pain without definite relation to the food; to the time which it lasts, usually for several hours; and to the fact that it is situated more to the right than that in ulcer. The temperature is also raised.

Movable kidney is found on examination.

Finally, Trousseau's remark in commenting on the subject, might be quoted. "I cannot help thinking he has somewhat exaggerated the frequency of the cases in exaggerating the significance of the symptoms."

PROGNOSIS.

Robson and Moynihan (Diseases of the Stomach, p.169) give the following statistics.

Lebert quoted by Dreschfield states that 10% of all ulcers are fatal, perforation accounting for $6\frac{1}{2}\%$, haemorrhage for $3\frac{1}{2}\%$.

Habershon 18% of all cases perforate.

Brinton 15% " " " "

Muller 11% die of haemorrhage.

Average of all authorities gives 5% die of haemorrhage.

They (R. & M.) consider that at least 20% are fatal, medically treated, and add that statistics do not take account of deaths from complications.

Debove and Rinauld (Ulcers de l'estomac) give the following:-

| | |
|---------------------------|----|
| Perfect cure | 50 |
| Foudroyant Haematemesis . | 5 |
| Perforation & Peritonitis | 13 |
| Pulmonary tuberculosis .. | 20 |
| Inanition | 5 |
| Different complications . | 7 |

Mansell Moullin (B. M. J. Ap.25, 1903) quotes Bulstrode's statistics for the London Hospital from 1897 to 1902. During this time there were 500 cases; 98 in men, 402 in women and 89 deaths.

| | | | |
|--------------|----|---|--------------|
| Peritonitis | 48 | = | nearly 10%) |
| Haematemesis | 13 | = | 2.5% (18% |
| Other causes | 28 | = | 5.5%) |

82% were discharged as relieved or cured.
 211 had suffered previously = 42%.
 116 had had one previous attack.
 41 " " two " attacks.
 15 " " three " "
 39 " " four or more " "

i.e. recurrence or relapse occurs in 40% of cases. Robson and Moynihan continuing, state that to the above must be added the cases admitted for different serious complications.

Pyloric stenosis (Gerhardt 10%, Warren 10%)

Hour glass contraction.

Gastric dilatation.

Chronic dyspepsia.

Constant vomiting.

Perigastric adhesions.

Cardiac stenosis.

Cancerous development (Hauser 6%).

It must be remembered that the Hospital patients who form the basis of the above statistics are, as a class, most careless; their stay in hospital is generally six weeks or two months at most; to cure a gastric ulcer two years attention and care in diet is required.

Consequently one is not surprised to find that in general practice the results are much better, a cure generally resulting, though relapses may occur once or twice.

TREATMENT.

The ideal in treatment is to put the stomach at rest, prevent irritation of the ulcer and thus allow it to heal. The usual way in which this is attempted is by perfect rest in bed, the administration of a teaspoonful of milk and lime water by the mouth and the use of nutrient enemata every four hours. Should haemorrhage be present, ice is given to suck, and ice bags laid on the epigastrium. Various drugs are also given.

The nutrient value of enemata even where previously digested, has been called in question, but Sharkey (Lancet, November 10, 1906, p.1263), has shown that sugar, peptones and pulverised casein have a good calorific value and are well absorbed.

W. Pasteur (Lancet May 21st, 1904, p.1418) got good results by injections of plain water at the body heat, some cases having no nourishment for ten days or a fortnight, others for as long as three weeks.

Whether this method really gives rest and freedom from irritation has been questioned, especially if there be excessive secretion of HCl. Thus Ewald (Folia Therapeutica, Jan. 1907, p.8) points out that the ulcer is commonly grafted on some other condition as chlorosis or anaemia, and is associated

with hyperchlorhydria, and states that a Dr Lennhartz of Hamburg treats these by oral feeding from the first, even when haemorrhage has occurred. The diet consists of two eggs beaten up per day and about five drachms of fresh milk every two hours. This is increased by one egg and three ounces of milk per diem till a total of eight eggs and one litre of milk is reached. From the third day sugar and from the sixth day scraped meat are added; from the eighth day rusks, rice pudding, raw ham and butter. Rest in bed is enforced for from two to four weeks. In 60 cases, one died, seven had a recurrence of haemorrhage during treatment; pain ceased directly the albuminous diet was begun.

Ewald, after haemorrhage, gives nutrient enemata for three or four days; then, when the stools give a negative result with guiacum, he feeds by the mouth, rapidly increasing the amount. He states that the weight rapidly increases unless some complication be present.

In "older non bleeding ulcers" he considers that much depends on whether the ulcer is healing, or the symptoms are due to adhesions. If the ulcer is healing he gives nutrient enemata.

As the object of treatment is to encourage healing this would seem rather anomalous, unless one is to regard his cases of haemorrhage as gastrostaxis.

It may be concluded, therefore, that the old routine treatment is correct; but it would seem that eggs beaten up with milk might be given within the first week provided there was no haemorrhage and that they did not cause pain.

The drugs recommended as specifics are numerous. As regards most of them it is said that they act, if at all, by influencing the associated dyspepsia; even if this is so, it must be beneficial.

Trousseau recommended Nitrate of Silver, gr. $\frac{1}{4}$ in pill. He continued his treatment for a long period.

Professor Fraser states that he has never known a failure with this drug.

Iron in some form would, of course, be of great use in anaemic cases. I have found that if there be no bleeding the Citrate of Iron ^{and} Ammonia, or the perchloride, may be given from the first; if bleeding has occurred after the lapse of a week.

Bismuth in powder is said to form a protecting coat to the ulcer. It can have no good effect in this way, as if there be food passing over, it would scrape it off, if there were no solid food present the liquid would soak through it. It is, however, probably of use given as the carbonate if there be excess of acid present.

With it the carbonate of soda and magnesium may be combined.

Haematemesis has a number of remedies. Ergot or its preparations raise the general blood pressure and thus might keep up the bleeding. Adrenalin 5 minim doses of 1:1000 solution (Parke Davis & Co) by the mouth, has a local action on the mucous membrane. To ensure an effect this dose should be repeated every fifteen minutes for four doses, then every half hour or one hour for twenty four hours. Thus given, I have always found the haemorrhage ceases. If it recurs in a day or two the process is repeated. In my fatal case of haematemesis I had no adrenalin. Morphia $\frac{1}{2}$ a grain, or even one grain by hyperdermic should be administered at once; it calms the patient, steadies the pulse and puts the stomach at rest.

Pain: When on proper dietetic treatment pain usually disappears at once. If it should not do so, a rubber hot water bottle applied to the epigastrium is usually sufficient. If it persists in spite of rectal feeding, it is practically certain that it is hysterical.

In some cases after about six weeks treatment when the diet is becoming a little varied, pain after food comes on and is troublesome, persisting in spite of limiting the diet to milk.

In these cases I have always found that pancreaticized milk, if ordered to be kept up for a

month, is curative. In cases with adhesions this would of course not be so.

Vomiting: The proper diet is again curative. A mixture of bicarbonate of soda with two minim doses of acid Hydrocyan. Dil. may be given. Opium is rarely required.

The question of operative treatment may be considered as unsettled. It would seem that unless the haemorrhage is due to the erosion of a large artery, the case will recover without operation. If it is due to this cause the patient will die before an operation can be done. Perforation requires immediate operation. Adhesions and thickening about the pylorus might be treated in the first place by hypodermic injections of fibrolysin as mentioned by Ewald in the paper already quoted. If this fails operation has given fair results.

The whole question of the operative treatment of gastric ulcer was freely ventilated at the Royal Medical and Chirurgical Society, London. (Lancet, November 24th, December 8th and December 15th, 1906). The opinions of most was that operative interference had not justified the expectations which were had of it.