ON PEPTIC ULCER

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

Though it cannot be said that gastric or duodenal ulcer is a very common condition, still it may be truly stated that it is by no means rare at the present day.

During my tenure of office as Resident Medical Officer to the Bradford Royal Infirmary it has been my experience that almost at no time has there been total absence of such cases in the wards and usually there are at least two or three in hospital at one time out of a total of 210 beds.

Now when we consider that as recently as fifteen or twenty years ago the condition was considered a rare one - so rare indeed that Perry and Shaw were able to collect all the cases previously reported and diagnosed as duodenal ulcer, we are at once prompted to ask for an explanation of this great increase in the number of cases reported.

Before attempting to answer such a question we must first assure ourselves that there has really been an increase in the number of cases of gastro-duodenal ulcer. And that, at once brings us to the most important consideration of the question. Is the increased number of reported cases in recent years to be accounted for by a true increase in the number of cases or is it that cases now-a-days are being recognised and diagnosed as gastric

and duodenal ulcers which formerly would have been missed or at most have been considered as cases of mere gastritis?

If we believe that the number of cases of gastro-duodenal ulcer has actually increased, then how are we to explain that increase? Is it that in recent years the manner of living has materially changed? Does the food taken now differ so greatly from that indulged in fifteen or twenty years ago as to cause such an increase in the production of gastric ulceration?

Or must we look to the state of the teeth and to oral sepsis as the chief factors in the production of the condition?

If so, have we any evidence that in later years, the teeth have not been attended to as well as fifteen or twenty years ago, and though we must admit that with a certain class of patient, oral sepsis is extremely common, yet here again we have no evidence that there is more oral sepsis to-day than formerly.

Many other factors - important certainly in the causation of the condition under consideration - could be mentioned, but no-where can we find or are we justified in saying that these factors are commoner now than before.

Surely the true answer to the question must be as I suggested before, viz: that we are to-day diagnosing and classifying as cases of gastro-duodenal ulcer, conditions which formerly were not recognised as such.

That medical science and art are always progressing and that every day we are getting a clearer knowledge of medicine and are more and more able as time goes on to appreciate the significance of symptoms and to interpret them aright is admitted by all.

It must also be granted by all that, owing to the great progress that has been made in abdominal surgery during the past twenty years, our experience in gastro-duodenal ulcer, as in innumerable other conditions, has been enormously enriched.

And so we are led to the conclusion that the greater number of gastro-duodenal ulcers to-day does not in any way indicate a greater prevalence of the disease. And when we also consider that 60% of the cases collected by Sir C. Perry and Dr. Shaw are reported to have presented no diagnostic symptoms prior to fatal perforation or haemorrhage, our view that many cases of true gastro-duodenal ulcer in former times were not recognised seems to be strengthened.

But this now brings us to a very important question, namely: How are we to diagnose cases of gastro-duodenal ulcer and are all cases diagnosed as such (I exclude, of course, cases which have been operated upon) truly cases of ulceration?

Firstly - must we wait for the appearance of haematemesis or melaena before we are justified in diagnosing

gastric or duodenal ulcer? If so, what are we to say about cases in which these two symptoms are never present?

Haematemesis - it is stated by Dreschfield, occurs only in about one third of all cases of gastric or duodenal ulcer, while about the same proportion of cases have melaena. Perforation has occurred in many cases in which a history of no other symptom apparently than dyspepsia, could be elicited.

Are there any other symptoms, therefore, less pronounced and less alarming which may permit us to clearly say that we are dealing with a case of gastric or duodenal ulcer?

Sir Berkeley Moynihan, who has done a great deal of work on duodenal ulcer lately, lays great stress on the significance of "hunger pain" in the early diagnosis of that condition. In fact he holds that "hunger pain" is pathognomonic of the disease.

Mayo Robson also, in an article which he published in the Medical Annual of 1906, says that in his opinion duodenal ulcer is a much more common disease than is generally supposed, as patients very often are apparently healthy except for a burning sensation three or four hours after food which is relieved by taking more food - "hunger pain."

On the other hand, we find another school, chief amongst whom is Hertz, who place no diagnostic value in

hunger pain and believes that one is not justified in diagnosing duodenal as gastric ulcer without the classical symptoms - haematemesis and melaena.

It will, therefore, be my object in this Thesis to present the views of the various authorities on this subject - to weigh the "pros" and "cons" of each and to give an account of personal observations which I have made on a number of cases.

HISTORICAL SECTION

As I have already stated, gastric and duodenal ulcer or peptic ulcer as I shall refer to them later are comparatively newly recognised conditions.

Regarding the history of gastric ulcer, little was known of the condition prior to 1829. In that year Couvielhier in his Anatomie Pathologique describes gastric ulcer and recognises it as a typical well characterised disease form. He described not only its anatomical characters but also gave a careful description of its clinical symptoms and of many of its therapeutic measures.

Following the investigations of this author, many other classical investigations were published, chief amongst which was that of Rokitansky - based on a large number of cases.

After the fourth decade of last century new contributions to the subject were furnished every year and more recently still, great progress has been made and our knowledge of gastric ulcer has been vastly enriched.

Duodenal ulcer has very recently become recognised and we have ~~only~~ to look back less than a hundred years to find the first reported case of it.

In the year 1817 Mr. Travers reported two cases of duodenal ulcer in the London "Medico Chirurgical Transactions." Both cases died and in both post mortem

examinations were made and definite ulcers of the duodenum were demonstrated.

In both cases the symptom which we now know as "hunger pain" was present, and it is interesting to note that this sign was apparently not taken notice of as we have to wait until the year 1896 before Mayo Robson draws attention to its significance in the diagnosis of duodenal ulcer.

About thirteen years later, i.e. 1830, Dr. John Abercrombie, in the second edition of Pathological and Practical Researches on diseases of the stomach, records five cases of duodenal ulcer collected from literature. One was reported by Irvine of Philadelphia (1824) two were reported by French physicians and the fifth was related in the Midland Medical and Surgical Reporter (1829). In dealing with these cases, Abercrombie points out that the leading clinical feature in them was that food was taken with relish and the first stages of digestion were not interfered with but about the time when the food should be passing out of the stomach, the pain came on.

This observation also remained unattended to.

Later, in 1864, we find eighty cases reported by Trier of Copenhagen, ~~and between that date and the year 18__ many additional cases were added to the literature on the subject~~.

Between 1864 and 1882 a few Parisian theses appeared otherwise no advances were made on the subject.

In 1883, Chrostie reported eight of his own cases (Adq. Wien Med. Zeit) also 136 from other sources.

Bourquoy in 1887 recorded five cases of duodenal ulcer diagnosed entirely from symptoms alone.

Prior to this time it had been considered impossible to diagnose the existence of the ulcer of the duodenum during life.

The next contribution to the subject was furnished by Oppenheimer who produced in 1891, a work "Das ulcus pepticum duodenale "Warzburg" which contained a summary and tables of most of the cases recorded up to that time.

In 1894, Collins of Paris wrote up 257 cases recorded up to that date with five of his own cases.

The next important work on the subject was by Drs. Perry and Shaw which appeared in Guy's Hospital Report. The subject was treated from the Pathological standpoint, the basis being the records of 17,652 post-mortems at Guy's Hospital between 1826 and 1892.

In 1894 the first successful case of duodenal ulcer treated by operation is recorded by Mr. H. P. Dean.

Another similar case is reported by Mr. Dunn immediately afterwards.

The first operation for chronic ulcer was performed by A. J. Codiville of Firenza in 1893.

In January 1900 Moynihan of Leeds, operated on his first case of duodenal ulcer and in 1901 he published his first paper dealing with the various forms from the surgeon's point of view.

Since then, valuable contributions have been made by:

W. T. Mayo in 1906 and 1908.

Dr. David Drummond and Mr. Rutherford Morrison (B.M.J.) in 1909.

Mr. Mayo Robson in an address before the Norfolk and Norwich Medico-Chirurgical Society in 1909.

Manson Moullin in an article in 1910 on the essential cause of gastric and duodenal ulcer.

And finally by Dr. E. C. Hort in a lecture on the treatment of gastric and duodenal ulcer at the Medical Graduates' College in October 1910.

The apparent increase in the proportion of duodenal ulcer to gastric ulcer has been explained by the fact that a more accurate anatomical classification is now adopted.

Formerly all ulcers about the pylorus were classified as pyloric and, therefore, gastric, and 95% of all duodenal ulcers extended up to the pylorus or within $\frac{5}{4}$ inch of it and adhesions more or less obscured the field.

The facts were but slowly brought forward.

S Y M P T O M A T O L O G Y

Let us now consider the symptoms which present themselves in well marked and typical cases of peptic ulcer and also those which, though less constant in ~~this~~ their occurrence, are still of great diagnostic value when present.

I do not intend here to discuss the cause of peptic ulcer or its morbid anatomy, nor do I intend to consider the etiological factors concerned in the condition. What I want to do in this section is merely to take up in order, the generally recognised symptoms in peptic ulcer and study them individually in order to arrive at some conclusion as to their clinical diagnostic importance.

The symptoms which one generally expects to find in a case of either gastric or duodenal ulcer are as follows: Pain and tenderness, ~~vanishing~~ vomiting, haematemesis or melaena or both, and dyspeptic symptoms. Added to these we may find a certain amount of hyperaethesia in certain areas, usually over the supposed seat of the ulcer and on either side of the vertebrae behind, and also in some cases neurotic symptoms are met with.

I. Pain and Tenderness. Pain is the most frequent symptom in peptic ulcer and one on which we should endeavour in all cases to make our diagnosis. It is said to occur in about 80 to 90 per cent of all cases. Of the fifty consecutive cases on which this Thesis is based, the

percentage is a hundred. In no case was pain absent.

The pain of which the patient complains most is, as a rule, situated in the epigastrium just below the ensiform cartilage. If the ulcer is in the duodenum or at the pyloric end of the stomach, the pain complained of was to the right of the umbilicus and slightly above its level. The pain differed in character and severity in different cases. In some cases it was described as a burning pain shooting through to the back. In a smaller number of cases the pain was colicky in nature, while in others, the epigastric pain was not acute but was more a feeling of fulness and uneasiness associated with tenderness on pressure.

In some cases - in my series of cases nearly all of them - a definite pain in either the right or left shoulder was present and I consider it a most important diagnostic sign in the condition. In the considerable number of perforated gastric or duodenal ulcers which I have seen, a prominent symptom has always been pain either in the right or the left shoulder.

The patient has as a rule mentioned this symptom without being asked about it; at other times he admitted its presence when one enquired. In no case of gastric or duodenal perforation have I known this symptom to be absent. My experience is based on two and a half years

as resident in a general infirmary as well as a few cases seen in general practice.

Now, though it cannot be said that this symptom occurs as frequently in unperforated ulcers of the stomach as it does in perforated ones, still in a great number of cases - in fact we may go further and say that in the great majority of cases we find it.

What may be the exact cause of the pain we are not prepared to say, but some reflex irritation of the intercostal nerves suggests itself as a possible explanation or more probably a communication of the branches of the sympathetic nervous system with branches of the axillary plexus. In either case, this sign will only manifest itself with lesions connected with the higher segments of the cord. I consider this symptom, therefore, a most important one not "per se" but as one which helps to differentiate gastric and duodenal lesions from other lesions lower down in the abdomen which are left to simulate the former conditions in other respects.

This sign is also helpful in roughly determining the site of the peptic ulcer.

In cases where the ulcer is in the pyloric end of the stomach or in the duodenum, we find the pain situated in the right shoulder, whereas ulcers of the cardiac end give rise to a painful left shoulder.

Pain is also felt in the back in most cases. In practically all my cases this was noticed - the pain

being situated usually over the region of the spine between the sixth and tenth dorsal vertebrae. In a few cases, the pain was slightly further down.

As regards the time of occurrence of pain, much has been written. It is generally stated that the pain of gastric ulcer comes on immediately after the injection of food, while in the duodenal variety, two or three hours usually elapse before the onset of pain.

In duodenal ulcer, Moynihan states that the pain is usually felt from two to two and a half hours after taking food. It is increased by pressure according to Dreschfield (Allbutt and Rolleston, Vol. III p. 561). It is often relieved by pressure according to Moynihan (Moynihan's book on Duodenal Ulcer p. 104).

In my experience those cases in which the pain has been relieved by pressure, the patient has been of a neurotic type and I have been led to the conclusion that in these the pain has been of a purely ~~hem~~neurasthenic type - the ~~hem~~neurasthenic symptoms masking the symptoms and signs of the organic disease.

In two cases of duodenal or gastric ulcer, it has been my experience to find that the pain is increased by pressure.

It is a well known fact that the pain in duodenal ulcer is usually relieved by taking food. What the cause of this is has never been satisfactorily explained.

It has been suggested that the food causes a reflex closure of the pyloric orifice preventing the passage of food into the duodenum. That this explanation is not the true one is proved by the fact that if a meal be given in which bismuth is mixed, the X-ray ~~serum~~ *screen* shows food passing from the stomach into the duodenum in the first few minutes.

The character of the pain in duodenal ulcer varies from a dull, aching pain, often just a feeling of uneasiness and oppression, to an acute burning pain. According to Dreschfield, the pain is rarely localised over a circumscribed area, but may radiate to the epigastrium, to the umbilicus or to the right side.

Charles F. Martin, of Montreal, on the other hand, holds that the pain is usually localised though it may radiate to the breasts, shoulders or back. I have already pointed out that in my experience, radiation of pain is the rule and the shoulders are usually the chief seat of this referred pain.

The pain in duodenal ulcer is not relieved by vomiting according to most authorities. With this general statement I agree, but I have found that in the majority of cases, the eructation of wind or sour material is usually followed by relief.

In the majority of the cases collected by the Fenwicks (Fenwick, Samuel and Soltan: Ulcer of the

Stomach and Duodenum, London 1900) the occurrence of pain was entirely independent of the injection of food being often most severe when the stomach was empty.

In my experience the ~~lesion~~ time incident is not a very important point in the diagnosis of the situation of the peptic ulcer as there are certainly exceptions to the general statement that "in gastric ulcer the pain comes on immediately after food while in duodenal ulcer it occurs two or three hours later."

Tenderness as a sign is of extreme value. This in all cases, in my experience, is definite and markedly localised. Palpation in many cases may not be sufficient to elicit any acute pain, but the application of a weak galvanic current picks out a small area of tenderness, as a rule not larger than about an inch square.

This tenderness, therefore, localised in character is a most important diagnostic sign, inasmuch as it differentiates cases of peptic ulcer from cases of hyperacidity or mere gastritis in which later two conditions, no localisation of tenderness can be got.

It is held by many that the pain in gastric ulcer is localised. This is not my experience. The patient, as a rule, complains of an indefinite epigastric pain and this is easy to understand as in most cases the ulcer is associated with dyspeptic symptoms, and the latter invariably mask the former. Tenderness is localised when

the pain is indefinite and can even be elicited when no pain is complained of.

Before passing from the consideration of pain as a symptom of peptic ulcer, let me mention a few statements made by Sir Berkeley Moynihan regarding the significance of the various degrees and characters of pain in ulcer of the duodenum. He states that when the pain occurs four hours or more after food, the ulcer is of the "tucked back" variety and in operating presents considerable difficulty in bringing it out of the abdominal wound. Pain two hours after food according to the same authority, indicates that the ulcer has contracted adhesion to the liver or anterior abdominal wall.

The following statements are also taken from Moynihan's book on Duodenal Ulcer: "The pain in duodenal ulcer comes "on sooner if liquid diet be taken.....The pain comes on "when patient begins to feel hungry....Food relieves the "pain. The pain is nearly always preceded by a sensation "of weight and feeling of fulness. Eructation of gas re- "lieves the pain.....Regurgitation of food often occurs "and the taste is acid and produces a scalding feeling in "the throat; a free gush of saliva often occurs..... "Pain is often relieved by pressure."

Hunger Pain will be discussed later.

II. Vomiting. This is considered by many authorities to be an important symptom in the diagnosing of peptic ulcer. In the majority of the fifty cases recorded below, vomiting did not occur though in nearly all of the cases a history of vomiting was given occurring at the onset of the illness. Vomiting - as I have stated - did not occur in the majority of my cases but in those in which it was a feature, it made its appearance later in the illness and coincident with it, one was able to demonstrate a dilated stomach. We may, therefore, distinguish two varieties of vomiting in peptic ulcer, viz: (1) Early vomiting, occurring before a definite ulcer has formed in the stomach. This vomiting is often merely a regurgitation of food or eructation of sour material, and (2) Later vomiting occurring when the stomach has become dilated.

In nearly all cases, early vomiting is present, whereas late vomiting only occurs in those cases where the pylorus has become constricted and dilatation of the stomach has supervened.

As regards the true cause of vomiting in these cases, no satisfactory theory has, as far as I know, been advanced.

In all cases of gastric and duodenal ulcer which have gone on for some time, I have noticed that the blood pressure is low, but in cases where early signs of gastric ulceration are beginning to manifest themselves

the blood pressure - if taken after a meal - is high. Is it not possible, therefore, that in the early stages of gastric ulceration we get a spasm of the pylorus which causes regurgitation and vomiting, and at the same time sends up the blood pressure.

As regards the character of the vomited material, the question of hyper and hypo-chlorhydria will be discussed later. Suffice it here to say that the vomiting which occurs before we suspect that a true gastric ulcer has formed, consists as a rule chiefly of un - altered food, whereas the vomiting which we get when dilatation of the stomach is present, comes on some time after the injestion of food and is pale and frothy in appearance.

III. Haematemesis and Melaena. We may take these two symptoms together as they differ only as regards their origin. It is now a recognised fact that haematemesis is a more marked feature in gastric than duodenal ulcer and melaena is more often present in the latter than in the former.

Haematemesis is said to occur in one third of all cases of peptic ulcer. This, as will be seen, is about the percentage in my cases.

Haematemesis in itself cannot be regarded as a diagnostic symptom of ulcer of the stomach or duodenum.

According to Moynihan, this is rather a conflicting

than a typical sign of the disease. The haemorrhage in duodenal ulcer may be very slight or it may be extremely profuse.

In some cases, the signs of internal haemorrhage are evident while melaena and haematemesis occur: at other times the haemorrhage occurs most insidiously. Moynihan (p. 115 Duodenal Ulcer) quotes the case of a man with a blanched appearance who was sent to him because of a right inguinal hernia. The patient had noticed no loss of blood but had suffered from indigestion for years and it had been recently severe. It was soon discovered that he had melaena and subsequent operation demonstrated the presence of a duodenal ulcer.

Haematemesis occurs more frequently in gastric than in duodenal ulcer.

Melaena is often a difficult symptom to detect from the patient's history as in many cases he gives the history of having tarry stools but admits to having taken bismuth freely as treatment. Bismuth, therefore, masks the melaena.

A more important sign than melaena is the detection of blood in the faeces as blood may be present in the stools in fairly large quantities without being detected by the naked eye appearance of the stools.

The tests for occult blood are:

1. Microscopic.
2. Chemical.
3. Spectoroscopic.
4. Microchemical (Teichmann's test)

The chemical tests are the most important.

IV. Dyspeptic symptoms. These symptoms are more common in duodenal than in gastric ulcer.

There is often a feeling of heaviness and distension coming on some hours after taking food. Flatulence, acidity and pyrosis are often complained of. Vomiting is not a frequent symptom and is often absent.

HCl varies in amount - normal, diminished or absent or increased.

Constipation is more frequent than diarrhoea. At first the patient seems to suffer but little in general health, later there is emaciation.

SPECIAL SYMPTOMS

In peptic ulcer and more particularly in ulcer of the duodenum, there are certain symptoms which are generally associated with the condition and even regarded by some authorities as pathognomonic of the disease.

The three chief special symptoms which I wish to consider in this Thesis are:

I. Hunger pain.
II. Hyperchlorhydria, and
III. Occult blood.

I. Hunger Pain.

First of all it is necessary that we should be clear as to what exactly we mean when we talk of "Hunger Pain." Secondly, how it is produced and finally and most important of all, what diagnostic value can we put on it.

Sir Berkeley Moynihan was the first to give the name of hunger pain to a group of symptoms occurring in the course of duodenal ulcer. In his book on Duodenal Ulcer (p. 101 et seq) Sir B. Moynihan describes this group of symptoms in the following manner: "A patient may have suffered for a considerable por-"tion of his life and when asked how long he has had

"the complaint, he will answer "All my life." If the "patient remembers his earlier history well, he will say "that the symptoms of weight, oppression or distension "in the epigastrium after meals came on insidiously or "almost imperceptibly. At first the discomfort may be "capricious but the patient is not long before he can "assign a definite time to its appearance, viz: about "two hours or a little more after meals. He also finds "out that the pain or discomfort is relieved by the next meal."

Sir B. Moynihan goes on to point out that many patients say that the pain comes on just about the time they are beginning to get hungry, and that on this account, he suggested the term "Hunger Pain" in one of his earlier papers on the subject.

We must, therefore, understand by the term "Hunger Pain" a group of symptoms occurring in the way in which Moynihan has described.

How is hunger pain produced? In this Thesis I do not intend to discuss the various theories regarding the actual causation of "hunger pain." I wish rather to consider its clinical significance when present.

It has been suggested that the "pain" is caused by the food passing over the surface of the ulcer, but I have already pointed out that food passes into the duodenum during the first five minutes after taking it. The pain comes on, therefore, when more than half the

food has left the stomach. To my mind, no satisfactory theory of the production of hunger pain has ever been put forward.

Let me now pass to the third point in consideration of "hunger pain" namely, what diagnostic value can be put in this symptom.

In this section I intend merely to put forward the views expressed by different authorities on the subject. My own opinion, based on my series of cases, recorded later, I will give in the "conclusions."

As regards the importance of this symptom we have, as I stated in my introduction, two schools entirely opposed to each other. The one represented by Moynihan and others who regard recurring attacks of hunger pain as almost pathognomonic of duodenal ulcer. The other, headed by Dr. Herts, who do not place much importance in hunger pain in the diagnosis of the condition, and would not make a diagnosis of duodenal ulcer without the classical symptoms being present, viz: haematemesis and malaena.

Moynihan goes as far as to assert that recurring attacks of "hunger pain" are quite sufficient on which to make a diagnosis of duodenal ulcer and that physical examination is unnecessary. Physical examination, he contends, is useless at the stage of the disease when a

diagnosis should be made as no physical signs of organic disease are present. Melaena and haematemesis, he regards as complications, not signs.

The opposite school, on the other hand, hold that far too much importance is laid on "hunger pain" as a sign in duodenal ulcer.

Dr. R. Hutchison, in a paper in the B.M.J. of Jan. 22 1910 (p. 203, 204) contends that there is a condition in which the mucous membrane of the stomach and duodenum are so hypersensitive as to permit gastric juice of normal acidity to produce symptoms of distension, feeling of oppression and a certain amount of pain two or three hours after food and relieved by injestion of more food - "hunger pain" without there being any organic lesion present. Dr. Herts is also of the opinion that "hunger pain" cannot be regarded as pathognomonic of duodenal ulcer. He holds that the pain is not due to the action of hydrochloric acid on the raw surface of the ulcer and also quotes a case operated on by Mr. Sherron proving that "hunger pain" even of so long duration as nine months does not necessarily mean duodenal ulcer.

Mr. H. J. Paterson says that in his opinion there is very little uniformity in the symptoms of duodenal ulcer and also adds that only a very few of his 41 cases of duodenal ulcer on which he had operated during the past three years gives the history of "hunger pain."

Dr. Craven Moore says that in cases of gastric and duodenal ulcer, there is an acid dyspepsia due to excessive secretion of normal gastric juice and this condition can give rise to "hunger pain" and other symptoms. In his opinion, therefore, "hunger pain" is not exclusively significant of duodenal ulcer.

In a report of a meeting of the Edinburgh Medico Chirurgical Society (B.M.J. 1911, 1. 625) Prof. Caird considers Mr. Moynihan's definite symptom group for duodenal ulcer a valuable one but he does not consider it pathognomonic of duodenal ulcer as he asserts that the symptoms may be present in other conditions such as cancer in the neighbourhood of the duodenum.

Mr. Stiles also stated that malignant tumour of the hepatic plexure of the colon and chronic adhesive peritonitis round the gall bladder may give rise to symptoms akin to those of duodenal ulcer.

Dr. Russell, at the same meeting, expressed as his opinion that gastric and duodenal ulcer are both able to produce the same clinical picture and, therefore, hunger pain is not diagnostic of the duodenal variety of ulcer.

Hyperchlorhydria.

That hyperchlorhydria or excessive formation of free HCl is present in many cases of gastric and duodenal ulcer is admitted by all but we have to admit at the same time that there are cases in which the HCl may be normal in

amount and in some cases even diminished. According to Mansell Mullin (Lancet Oct. 1. 1910, p. 997) hyperchlorhydria is a secondary consequence of spasmodic contraction of the pylorus or of hyper-secretion of gastric juice or of both together. These are the result of a morbid condition of the mucous membrane of the stomach brought on by irritation from an ulcer or from septic poisoning. This authority holds that there is not an excess of free HCl present, but rather an accumulation of gastric juice producing the symptoms which we term Hyperchlorhydria. This theory I think a feasable one and is borne out in my experience of certain cases of my own which at operation have proved to be duodenal and gastric ulcer.

In two or three of my cases, reported in detail further on, I tested the gastric contents and found a diminution both in the total acidity and in the free HCl. In these cases, symptoms of so-called hyperchlorhydria were marked. No doubt in these cases there was a hyper-secretion of gastric juice, though free HCl was actually below normal in quantity.

Dr. Charles F. Martin in (Osler & Macrae, System of Medicine 181) makes the following statements with regard to the relation between hyper-acidity and gastric ulcer.

(1) Gastric ulcer only occurs when HCl is normally present. Hyper-acidity is common in gastric

ulcer but there are cases when the HCl is normal or subnormal in amount.

(2) Hyper-acidity occurs frequently in cases other than gastric ulcer. This statement accords with my experience. In one of my cases, which at operation proved to be a malignant glandular mass behind the stomach and duodenum, hyperchlorhydria was the most distressing symptom to the patient and gastric analysis demonstrated an increase in the total acidity or free hydrochloric acid.

(3) An ulcer of the stomach cannot be produced by hyper-acidity alone.

(4) As hyper-acidity occurs frequently in chlorosis, it may be an additional factor in the etiology.

(5) Hyper-acidity may produce gastric ulcer if the mucous membrane of the stomach has first been infused - the acid in this case acting on the exposed ends of the blood vessels constricting them, thus producing a condition of local anaemia and necrosis.

(6) Gastric ulcer is prevented from healing by the presence of excess of free HCl.

These, briefly, are the conclusions arrived at by Dr. Martin with regard to the relationship of hyper-acidity and gastric ulcer.

The symptoms produced by hyperchlorhydria are regarded by Moynihan as pathognomonic of duodenal ulcer.

but, on the other hand, Dr. R. Hutchison shows that from post mortem evidence, proof has been obtained of the existence of a condition of the gastric and duodenal mucous membrane which permitted normal gastric juice to produce symptoms like those described as hyperchlorhydria without any organic lesion being present.

In a paper in the B.M.J. Jan. 22. 1910 (p. 204) Mr. Paterson states that in his opinion the presence of true hyperchlorhydria denotes organic lesions.

Dr. Craven Moore denies the existence of hyperchlorhydria and says that the condition of "acid dyspepsia" due to excessive secretion of normal gastric juice produces the symptoms.

Occult Blood.

The detecting of occult blood in the faeces must be regarded as a most valuable sign in the diagnosis of gastric and, to a greater extent, of duodenal ulcer. Occult blood of course does not necessarily mean gastric or duodenal ulcer but its presence at once limits our diagnosis to some organic lesion and thus puts out of court all functional disturbances such as gastric neurosis or anomalies in the gastric secretion.

It is necessary first of all, however, to make certain that the occult blood got is from the gastro intestinal tract and not due to such fallacies as the

injection of large quantities of raw or ill-cooked beef or blood sausages. We must also exclude bleeding from the gums or nose or any injuries to the oesophagal or gastric mucous membrane caused by passing a stomach tube. Fissures in ano and haemorrhoids must also be thought of.

General diseases such as profuse scurvy, haemophilia, arteriosclerosis and enteric fever may also give rise to the presence of blood in the faeces and must, therefore, be excluded.

Provided then that we have excluded all those fallacies, how are we to regard the importance of occult haemorrhage?

It is well known that persistent blood in the faeces is a sign of malignant disease.

Dreschfield (Allbutt & Rolleston, vol iii, p. 481) points out that occult haemorrhage is more constant and characteristic of cancer than of ulcer. Ruthmeyer's statistics support this contention (Osler & Macrae Vol.V. 195).

I have found repeatedly in my own cases that in cases of gastric and duodenal ulcer, occult blood has not been detected until two or three examinations of the faeces have been made. This would suggest, therefore, that in gastric and duodenal ulcer, occult blood is not constant, but occurs only at intervals. This, therefore, is a point of difference from cancer. If an ulcer is suspected, it is of great importance to examine the faeces after pain.

Dr. Craven Moore holds that ~~there~~ it is of very great value in diagnosis if occult blood be detected in the stools after a few days of diet free from haemoglobin; he himself has found it in every case of ulcer he has investigated.

The persistence of occult blood in the stools naturally suggests an unhealed ulcer, therefore our prognosis and mode of treatment are guided by this sign.

DIFFERENTIAL DIAGNOSIS

An accurate examination of the patient and careful consideration, not only of the symptoms of which the patient complains at the time, but also of the past history should, in the majority of cases, enable us to say with more than a fair chance of being right, that we are dealing with a case of peptic ulcer. The position of the ulcer in the stomach or whether it is situated in the stomach or duodenum is more difficult to recognise.

I shall, therefore, in this portion of my Thesis, begin by comparing gastric with duodenal ulcer and discuss the differential diagnosis of the two conditions.

Moynihan in his book (p. 122) on Duodenal Ulcer, asserts that though in his earlier cases he experienced some difficulty in distinguishing gastric from duodenal ulcer, now he holds that the conditions are quite distinct and should present no difficulty.

According to this authority, the time at which pain comes on after taking food and also the striking recurrence of the attacks at various seasons of the year - especially when it is cold and wet, are most valuable points in the diagnosing of duodenal ulcer. While agreeing with Moynihan as to the importance of the former point in differential diagnosis, still in observations made from 50 cases of peptic ulcer (gastric and duodenal) my

experience has been that in a few cases where pain came on 3 to 4 hours after food - suggesting duodenal ulcer - on operation an ulcer situated to the gastric side of the pyloric vein was discovered. I can not, therefore, agree with the statement that pain occurring two to three hours after the injection of food necessarily means duodenal ulcer.

As regards the recurrence of attacks at special times of the year - in cold and wet weather - I have looked in vain for this ~~sign~~ incident in cases of duodenal ulcer while in many cases of undoubted gastric ulcer, the patient has stated that the attacks have come on each year about the same time - usually the late winter.

Stengel (Osler & Macrae, vol. V, 292) is of the opinion that in the majority of cases it is impossible to make a definite diagnosis between gastric and duodenal ulcer. In his opinion, long continued or recurrent burning oraching pain coming on at a definite time, usually from two and a half to four hours after food accompanied by evidences of chronic gastric disease hyper-acidity and stagnation of stomach contents, haematemesis or melaena or occult blood suggest ulceration at the pyloric vein but one cannot go farther.

In straight forward cases, however, one should have no difficulty in diagnosing gastric from duodenal ulcer. That is to say when we have a definite history

and typical signs and symptoms present. In my opinion no one symptom is diagnostic of gastric or duodenal ulcer.

The following are the chief points of difference between gastric and duodenal ulcer:

1. In gastric ulcer the pain comes on as a rule immediately or shortly after food. In duodenal ulcer two or three hours after food.

2. When vomiting occurs in gastric ulcer, pain is relieved, not so in duodenal ulcer.

3. In gastric ulcer the appetite is often good but the patient is afraid to eat on account of the consequent pain. In duodenal ulcer the appetite is usually poor.

4. The point of tenderness differs according to whether the ulcer is situated in the stomach or duodenum. In gastric ulcer I have found the point of maximum tenderness above the umbilicus in the middle line of the epigastrium or slightly to the left.
In duodenal ulcer the tender spot is situated to the right, i.e. in the position of the duodenum.

5. If haemorrhage occurs in gastric ulcer, it usually takes the form of haematemesis while in duodenal ulcer, melaena is the more common. If both haematemesis and melaena occur, in gastric ulcer the former usually appears first while in duodenal ulcer the opposite is the case.

6. Shoulder pain - a sign which I have already referred to, is felt usually in the right shoulder in cases of ulcer of the pylorus and duodenum, while left shoulder pain is found when the ulcer is situated near the cardiac end of the stomach.

Having discussed the differential diagnosis between gastric and duodenal ulcer, we may now pass on to other conditions which must be differentiated from peptic ulcer.

The following conditions I consider the most important in a differential diagnosis from peptic ulcer:

1. Carcinoma of Stomach: Here a consideration of the following points should make the diagnosis easy. First of all the age. Carcinoma of the stomach is rare under 40 years of age. Gastric ulcer is usually met with in young adults.

Pain is continuous in carcinoma and vomiting does not give relief. Food has little influence on the pain in carcinoma. Anorexia is marked in carcinoma, whereas in gastric ulcer the appetite is often good but the patient is afraid to eat.

An analysis of the gastric contents reveals in carcinoma a diminution and often a complete absence of free HCl and in a microscopical examination of the residue, we can usually detect sarcina ventriculi, torula and less frequently cancerous fragments.

It is said that loss of flesh is a distinguishing feature of carcinoma in contra-distinction of simple ulcer of the stomach, but it often happens that after a long attack of gastric ulcer, loss of flesh is marked and even a cachectic appearance may develop. Cachexia nevertheless is more marked in carcinoma than in the

worst cases of gastric or duodenal ulcer.

Finally, in cases of carcinoma of the stomach we can usually detect a tumour in the epigastric region. No epigastric tumour can be felt in simple ulcer of the stomach or duodenum.

2. Neurasthenic Gastralgia. I have already stated that we must regard neurasthenic gastralgia with scepticism. In my opinion, based on a considerable number of cases, on all of which unfortunately I have not managed to procure notes, gastric neurosis is oftener a secondary symptom than a disease in itself.

The neurasthenic symptoms are often of so pronounced a character that they entirely mask the primary cause and one is frequently too apt to consider the case merely as a neurosis, whereas the neurosis is merely the ~~absence~~ result of long protracted organic trouble.

In true neurasthenic gastralgia the pain comes on suddenly at any time and is not, as a rule, influenced by food. Light pressure usually causes pain - the patient often complains that the bed-clothes aggravate the pain - firm pressure, as a rule, relieves it.

Haematemesis and melaena never occur.

The points, therefore, of most importance in making a diagnosis of this condition from gastric or duodenal ulcer are: The absence of haemorrhage and the relief

of pain on deep pressure. The other points I consider may perfectly well exist in a case of organic disease with a secondary neurosis.

3. Gall-stone colic: This condition may simulate duodenal ulcer but in the former the pain comes on more suddenly, is more severe and nausea and vomiting always accompany the pain and jaundice may supervene. The liver may be enlarged and pressure over the region of the gall bladder causes pain. Cases of duodenal ulcer, however, have been mistaken for gall stone colic. Dr.Rankin says (B.M.J. 1910 11. 181): "Cases have been recorded of "duodenal ulcer, confirmed by operation, in which the at- "tacks of pain so closely resembled biliary colic that they "necessitated the patient being put to bed with hot bottles "and the administration of morphine."

4. The gastric crises of locomotor ataxia. One should always be on the look-out for this condition but having it in mind as a possibility there should be no difficulty in making the diagnosis.

The history of the disease - lightning pains, absence of knee jerks - ocular symptoms and the ataxic gait makes this condition easily distinguished.

5. Cirrhosis of the liver is another condition which in some cases may present difficulties. In one of my cases which at operation proved to be gastric ulcer, the diagnosis of cirrhosis of the liver was first made.

The history of the case, however, - usually an alcoholic history - the tenderness in the region of the liver with the resulting symptoms of portal congestion and the absence of the true classical signs of peptic ulcer should in most instances enable the examiner to differentiate the two diseases.

I shall not deal with the various conditions which may simulate gastric or duodenal ulcer after perforation has taken place. This Thesis is based on the observation made on unperforated ulcers of the stomach and duodenum. The three or (four) cases of perforated ulcer are inserted merely to illustrate the organism theory in the causation of the disease.

The following fifty cases are those which came under my care in the Royal Infirmary, Bradford, from November 1912 until the end of 1913. They are arranged in the order in which they were admitted to hospital.

I have briefly described the symptoms complained of and the signs which were present on admission and during the course of their stay in hospital. In this section of my Thesis I have not attempted to give in detail the medical treatment adopted in those cases which were not operated on. The medical treatment which I employed I shall describe in my next section.

In reviewing the cases, the following points are worthy of mention.

Firstly, as regards sex, it will be noted that of the fifty cases of peptic ulcer, 26 were in females while 24 were in males.

Secondly, it will be noted that out of the total number of my cases, 15 were in or at least suggested a duodenal site while the remaining 35 were gastric in origin.

It is also to be noted that of the 15 cases of duodenal ulcer, only two were in females while the remaining 13 were in males.

Looking at these results, therefore, it has been my experience that of cases of peptic ulcer, the duodenal variety is the more common in the male, while in the female it is uncommon, the ulcer usually occurring in the stomach proper.

C A S E S

Case I. W.W. age 40. Tramway repairer.

Vomiting and pain 3 or 4 hours after food for ten years, worse during last 4 years. Trouble with indigestion "all his life."

No actual pain but feeling of fulness and distension. Hunger pain. No haematemesis or melaena. Lost weight. Apparently healthy. Teeth bad. Not influenced by weather.

Examination: Dilated stomach. No hyperchlorhydria. No mass to be felt. No tenderness. No hyperasthenia. Pain felt in right shoulder.

Operation: Duodenal ulcer - posterior wall.

Recovery: Well before operation performed.

Case II. C.C. 34, Carter.

Pain in epigastrium. Vomiting immediately after food. Duration 3 years. Lost weight. Had haematemesis. No melaena. Hunger pain present. Pain in left shoulder and back. Hyperasthesia in front and behind. Not influenced by weather. Slight excess of free HCl. Teeth bad.

Operation: Gastric ulcer (not duodenal)

Well.

Ulcer is in stomach.

Case III. M.S. 33. Housewife.

Pain in epigastrium. Vomiting after food. Duration, 10 years. Numerous attacks. Pain came on a few hours after food. Hunger pain. Pain in right shoulder. No haematemesis but melaena on several occasions - apart from taking medicine. Pain is not influenced by any special food. Tenderness in epigastrium - tenderness localised - hyperasthenia behind and in front on left side. No hyperchlorhydria. Bad teeth.

Diagnosis: Pyloric ulcer.

Case IV. J.C. 24. Warehouseman.

Admitted on account of severe malaena occurring on 3 occasions during last 8 days. Indigestion for years. Pain in right hypochondria and epigastrium for 8 days. Pain in right shoulder and back. No haematemesis. Tenderness very localised in duodenal ulcer. Only got by faradic current. Hunger pain not present.

Diagnosis: Duodenal ulcer. Cleared up by medical treatment.

(Hunger pain not present yet typically duodenal)

Case V. B.N. 48. Motor Inspector.

Admitted with pain in right hypochondrium. Has suffered for 7 years.. Worse in cold weather. Pain relieved by taking more food. Hunger pain. Pain one hour after food. Food: No special food influences pain. No haematemesis. Melaena.

Examination: Tenderness localised just under right costal margin. Pain right shoulder.

Free HCl diminished.

Operation: Duodenal ulcer found. Well before operation.

Case VI. H.K. 31. Labourer.

Admitted with complaint of feeling of fulness in epigastrium. No pain. Dyspepsia for two years. Not influenced by weather. Vomiting has been a symptom - 2 hours after food.

Examination: Tenderness localised just behind right costal margin. Hyperasthenia in front and behind. Neurotic temperament. Pain in right shoulder. No hunger pain. Teeth bad.

Operation: Hypochlorhydria. Duodenal ulcer (1st part of duodenum)

Case VII. M.B. 24. Spinner.

Pain in epigastrium. Vomiting - immediately after food. Haematemesis on 3 occasions. One bad tooth. Anaemia.

No melaena. No hunger pain. Tenderness to left of umbilicus. Got with faradic current and distinctly localised. Slight pain in left shoulder and back. Gastric contents normal.

Diagnosis: Gastric ulcer. Relieved by medical treatment.

Case VIII. H.G. 47. Tailor.

Admitted with feeling of fulness in epigastrium, wind and flatulence. Chiefly after food. No haematemesis or melaena. No hunger pain. No tenderness or pain can be made out. No increase in free HCl, diminished on one examination.

Operation: Ulcer at pylorus - malignant. Not followed.

Case IX. A.B. 49. Mill worker, male.

Admitted with pain in epigastrium. 2 years. Haematemesis - melaena. Pain in left shoulder. Pain after food. No hunger pain. Tenderness to left of umbilicus. An excess of free HCl.

Case X. P.B. 46. Labourer.

Admitted on account of severe haematemesis occurring a few hours before admission to hospital. Symptoms of 6 months' duration and consist of pain in epigastrium. Made worse by firm pressure. Feeling of fulness in epigastrium and wind. No melaena. Pain comes on 3 hours after food. No hunger pain. Marked alcoholic history. Marked pain in right shoulder and in back.

Tenderness well marked and localised just to right of umbilicus. Gastric contents not examined.

Operation: Gastric ulcer found and excised.

NOTE.- Pain came on 3 hours after food.
Tenderness to right side of umbilicus - pain in right shoulder.

Case XI. Sister F. 28.

Pain in epigastrium. Feeling of fulness about ½ hour after food. Vomiting - relieved pain. Haematemesis. No melaena. Duration, many years.

Examination: Acute tenderness 1½ inches above and to left of umbilicus. Localised with faradic current. Pain in both shoulders.

Operation: Gastric ulcer.

Case XII. M.S. 56. Housewife.

Admitted complaining of pain in the region of the stomach. Duration 10 years. Not influenced by weather. Pain in epigastrium and back and left shoulder. Haematemesis on one occasion. Vomiting frequently. Hunger pain present. No hyperchlorhydria.

Relieved by medical treatment. Went home well.

Diagnosis: Gastric ulcer. Yet hunger pain present. (Pain ½ hour after food)

Case XIII. J.B. 32. Wool comber.

Admitted with pain ¼ hour after food. Attacks of epigastric pain during the night. Duration 8 months.

Pain came on ½ hour after food, lasted for 3 or 4 hours and was not relieved by taking more food. No haematemesis or melaena. Lost weight. Pain in right shoulder. No alcoholic or tobacco history. All teeth bad.

On examination: There is a point of extreme tenderness with the cathode 1¾ inches to the right of the umbilicus and at a level with it.

Operation: Duodenal ulcer.

No melaena or haematemesis.

Case XIV. T.H. 31. French Polisher.

Admitted with pain in upper part of abdomen which shoots through to back and left shoulder. Duration - 2 months. Attacks for 4 years. Pain in epigastrium immediately after meals and vomiting. Vomiting relieves the pain. Melaena and haematemesis have both been present. The pain is felt a little to the right of the middle line. Teeth very bad. (Streptococci short chained got) Point of maximum tenderness just to right of middle of umbilicus. Stomach dilated.

Diagnosis: Gastric ulcer. Relieved by medical treatment.

Case XV. J.B. 64. Dyer. M.

Admitted with pain in epigastrium. Hunger pain. Duration - 20 years. Alcoholic. Melaena but no haematemesis. Pain in back and right shoulder. Many previous attacks, occurring usually in the early autumn. Teeth bad. Tenderness not marked but got with faradic current. Localised just below right costal margin. Nil palpable.

Examination of gastric contents. Combined HCl but no free HCl.

Diagnosis: Duodenal ulcer.

Treatment: Relieved by medical treatment.

Case XVI. E.F. 21. Mender. F.

Admitted on account of vomiting. No pain but vomiting after food (immediately). Feeling of weight in epigastrium. Duration - 3 years.

Haematemesis frequently. Pain after food came on later in the illness. Lost weight. Teeth good. Tenderness localised to right of umbilicus. Pain in both shoulder and back. Hyperasthenia.

Diagnosis. Gastric ulcer.

Relieved by medical treatment.

Case XVII. A.C. 24. Mantle Maker. M.

Admitted with pain in stomach region. Occurs immediately after food - vomiting. Duration - 3 weeks. Vomiting relieves pain. Pain in left shoulder. Teeth good. Tenderness to left of umbilicus. Got with faradic current. Relieved by medical treatment. Blood in stools.

Diagnosis: Gastric ulcer.

Case XVIII. T.H. 30. Labourer. M.

Pain in pit of stomach. 16 months. Pain comes on 2 hours p.e. No hunger pain. Vomiting relieves the pain. Haematemesis on one occasion. Not alcoholic. Teeth bad. Tenderness to left of umbilicus. Got with faradic current. Relieved by medical treatment.

Case XIX. W.B. 25. Wool comber. M.

Admitted on account of melaena. Duration - 1 week. No history of indigestion or past illnesses. No hunger pain. Teeth bad. Pain in right shoulder. No other pain. Acute localised tenderness $\frac{5}{4}$ inches to right of umbilicus.

Diagnosis: Duodenal ulcer.

Cured by medical treatment.

Case XX. C.I. 29. Charwoman. F.

Pain in epigastrium $\frac{1}{2}$ hour after food. 2 months. Suffered from indigestion for many years. No haematemesis or melaena. Pain in left shoulder and back. Tenderness to left of umbilicus. Localised. Teeth bad. Blood in stools, H.C.L. negative. Blood corpuscles found microscopically

Diagnosis: Gastric ulcer. Relieved by medical treatment.

Case XXI. H.F. 38. Weaver. F.

Admitted on account of pain in left shoulder. Duration - 10 weeks. Vomiting and pain in epigastrium came on later and occurred immediately p.e. No haematemesis or melaena. No hunger pain. Considerable tenderness to left of umbilicus.

Diagnosis: Gastric ulcer. Relieved by medical treatment.

Case XXII. J.S. 35. Housewife. F.

Pain immediately after food - haematemesis - 12 months. Vomiting relieves pain. Pain in left shoulder and in back. Anaemia. Cough and slight haemoptysis. Considerable tenderness to left of umbilicus. no T.B. found in Sputum.

Diagnosis: Gastric ulcer. Relieved by medical treatment.

Case XXIII. W.G. 27. Dyer's labourer. M.

Pain and sickness after food. Duration 2 years. Pain occurred 2 hours after food. Vomiting gave relief. Pain goes through to back - right shoulder. No haematemesis or melaena. Marked tenderness below right costal margin.

Diagnosis: Duodenal ulcer. Relieved by medical treatment.

Case XXIV. A.G. 24. Spinner. F.

Pain and vomiting 10 minutes after food. 4 years. No hunger pain. Haematemesis once. No melaena. Pain in left shoulder. Tenderness to left of umbilicus.

Diagnosis: Gastric ulcer. Relieved by medical treatment.

Case XXV. M.B. 24. Drawer. F.

Pain after food - vomiting - relieved pain. Haematemesis. No melaena. Pain in left shoulder and back. Tenderness to left of umbilicus.

Diagnosis: Gastric ulcer. Relieved by medical treatment.

Case XXVI. H.F. 53. Housewife. F.

Pain in epigastrium, back and left shoulder. 8 weeks. Occurrence of pain has no relation to food. Relieved by taking food. Later hunger pain. Haematemesis. No melaena. Attack 20 years ago. Teeth bad. Marked tenderness localised just above and to right of umbilicus. No increase of free HCl.

Diagnosis: Gastric ulcer. Relieved by medical treatment.

Case XXVII. A.T. 30. Dyer's labourer. M.

Pain in epigastrium. Comes on 1 hour after food, lasts for an hour - relieved by vomiting - ~~by mouth~~. Hunger pain. Haematemesis. No melaena. Teeth bad. Marked tenderness in epigastrium 3 inches below the ensiform cartilage and 1 inch to right of the middle line. Pain in right shoulder.

Patient died. Duodenal ulcer found at P.M.

Case XXVIII. M.B. 27. Cook. F.

Pain in epigastrium. Vomiting and haematemesis 4 days. 18 months.

Had been troubled with indigestion for many years. Haematemesis. Pain relieved by eructation of gas not vomiting. Melaena has been present. No hunger pain. Pain and great tenderness to left of umbilicus and also in back and left shoulder.

Hyperasthenia in epigastrium and on either side of spinal column from 2nd dorsal down to lumbar region. Teeth bad.

Diagnosis: Gastric ulcer. Relieved by medical treatment.

Case XXIX. H.B. 24. Domestic. F.

Severe pain in abdomen and sickness. 2 months. No haematemesis or melaena. Pain in left shoulder, and back. Pain worse when patient moves. No hunger pain. Blows in Stock.

Marked localised tenderness to left of umbilicus. Teeth bad.

Diagnosis: Gastric ulcer. Relieved by medical treatment.

Case XXX. T.W. 61. Wool Comber.

Admitted with severe haematemesis in a collapsed condition. Gastric trouble for many years. There is great tenderness in epigastrium to left of middle line. Pain in left shoulder and back is also complained of. No history of melaena or hunger pain.

Diagnosis: Gastric ulcer. Died. Not perforation. No P.M.

Case XXXI. S.M. 42. Barber and Mender.

Admitted with acute abdominal pain and vomiting. History of gastric trouble for 20 years. Haematemesis and melaena both been present. No hunger pain. Teeth bad. Pain in right shoulder - severe.

Operation: Perforation near pylorus on anterior wall. Scraping from ulcer and fluid in peritoneal cavity shewed short chained streptococci. Vaccine prepared and patient made a quick recovery.

Case XXXII. Nurse B. 28. Nurse.

Admitted with pain in abdomen and vomiting. Haematemesis once. No melaena. No hunger pain. Pain between shoulders and in left shoulder. Tenderness in left hypochondria - localised and got with faradic current. Duration - many attacks during 2 years. No increase in free HCl.

Diagnosis: Gastric ulcer. Relieved by medical treatment.

Case XXXIII. Nellie Mason. 31. Servant.

Admitted with pain in abdomen and vomiting. Pain immediately after food. Vomiting relieved pain. Melaena and haematemesis. Melaena came on first. Very definite hunger pain. Pain in right shoulder. Tenderness marked in right hypochondria.

Operation: Patient was operated on a year before admission to this hospital when she suffered from exactly similar symptoms. No ulcer was found. Relieved by medical treatment.

N.B. Very definite history suggesting duodenal ulcer.

Case XXXIV. K.R. 23. Drawer.

Admitted with pain in epigastrium and back, left shoulder. Vomiting and pain immediately after food. No hunger pain.

Definite tenderness to left of umbilicus.

Haematemesis but no melaena.

Sweats at night and cough - no T.B. found in sputum. Teeth bad.

Diagnosis: Gastric ulcer. Relieved by medical treatment.

Case XXXV. M.M. 23. Weaver.

Pain in epigastrium and sickness and vomiting 5 weeks. Had suffered from chlorosis. Melaena but no haematemesis. Pain in left shoulder and back. Teeth bad. Marked tenderness very localised just above and slightly to left of the umbilicus.

Diagnosis: Gastric ulcer. Relieved by medical treatment.

Case XXXVI. B.M. 36. Cardroom worker.

Admitted with pain in epigastrium and vomiting and haematemesis - 8 months.

Pain immediately after food. Haematemesis on one occasion. No melaena. No hunger pain. Pain in left shoulder and in back. Tenderness marked and localised to left of umbilicus and slightly above. Hyperæsthesia in epigastrium and back.

Diagnosis: Gastric ulcer. Relieved by medical treatment.

Case XXXVII. H.I. 45. Agent.

Pain in epigastrium and flatulence about 3 hours after food. Vomiting. Melaena. Admitted in collapsed state. Definite hunger pain. Pain in back - burning pain - pain in right shoulder. Teeth bad. Tenderness very marked below right costal margin. Improved greatly by medical treatment.

Operation: Duodenal ulcer found on posterior wall.

Case XXXVIII. M.S. 35. Housewife.

Admitted with continual pain in epigastrium - worse at nights. Pain not made worse by food. Haematemesis once. Melaena every day for a week. Pain in left shoulder and back. No hunger pain. Teeth bad. Tenderness to left of umbilicus.

Diagnosis: Gastric ulcer. Relieved by medical treatment.

Case XXXIX. J.J. 26. Plasterer.

Admitted with pain in epigastrium, sickness and occasional vomiting. Pain comes on about 2 hours after food. Hunger pain. Melaena. No haematemesis Right shoulder pain. Also pain and hyperæsthesia in back. Teeth bad. Tenderness marked to right of umbilicus.

Diagnosis: Duodenal ulcer. Relieved by medical treatment.

Case XL. G.S. 34. Labourer. M.

Admitted on account of severe haematemesis and pain in abdomen. Haematemesis but no melaena. Alcoholic history. Pain comes on 3 or 4 hours after food. Hunger pain very definite. Tenderness marked and definitely localised just below right costal margin. Pain in right shoulder severe. Mouth very septic. No increase in HCl.

Operation: Chronic ulcer found, posterior wall of stomach in pyloric portion of the stomach with some omental adhesions.

Case XLI E.C. 20. Maid. F.

Admitted with feeling of discomfort in the epigastric region and vomiting. Haematemesis. Pain very constant but most severe just after meals. No hunger pain. Melaena has been present.

Very marked tenderness in the epigastrium and left hypochondria. Pain im back and shoulders. Teeth bad.

Three days after admission signs of phthisis manifested themselves. Left apex - haemoptysis.

Stomach symptoms got worse.

Treated as phthisis and improved - sanatorium treatment later. Tenderness in epigastrium still present.

Diagnosis: Gastric ulcer with phthisis.

N.B. Apart from the haemoptysis there was definite haematemesis. No question of haematemesis being mistaken for haemoptysis.

Case XLII. E.P. 21. Housemaid F.

Admitted with pain in epigastrium immediately after food - 7 weeks. Indigestion for many years. No hunger pain. Pain in shoulders - chiefly left. Tenderness in left hypochondrium just below left costal margin. No haematemesis or melaena.

Diagnosis: Gastric ulcer.

Case XLIII. E.R. 30. Housewife F.

Pain in epigastrium 2 hours p.e. No hunger pain. Vomiting relieves pain.

Tenderness above and to right of umbilicus but no definite tender spot to be made out. No haematemesis and no melaena present. No pain in shoulders. Free HCl present .05%

Diagnosis: Gastric ulcer ? Not a case of hyperchlorhydria. Probably one of those cases presenting ~~no food~~ symptoms and one which would be likely to go on to perforation.

Case XLIV. M.N. 26. Drawer. F.

Pain immediately after food and vomiting - 3 months. Severe pain in left scapular region. Pain was relieved by vomiting. No hunger pain. No haematemesis and no melaena. Anaemia. Teeth mostly artificial, some stumps. Marked tenderness to left of umbilicus localised. No increase in free HCl.

Diagnosis: Gastric ulcer. Relieved by medical treatment.

Case XLV. N.M. 18. Mill-hand. F.

Pain ½ hour p.e. 4 years. Vomiting usually relieves the pain. Haematemesis. No melaena. Teeth are very good. A tender spot is made out 1½" above and to right of umbilicus and behind, also a tender point

1" to right of 10th spine D.V.

Pain in both shoulders. No hunger pain.

Diagnosis: Gastric ulcer. Relieved by medical treatment.

Case XLVI. C.M. 36. Maid.

Pain immediately after food. Vomiting relieves the pain. Hunger pain. Food relieves the pain. Haematemesis and melaena both present. Definite tender spot to right and above umbilicus. Stomach is dilated. Pain in right shoulder.

Diagnosis: Duodenal ulcer. Relieved by medical treatment.

Case XLVII. W.L. 25. Grinder. M.

Pain in left side of epigastrium. 4 years. No haematemesis, no melaena. Motions dark but patient was taking bismuth. No hunger pain. Very tender spot, got with weak faradic current, midway between umbilicus and ensiform cartilage, 1½" to right of middle line. On the back the painful point on application of the cathode is very marked and is at the level of the 9 D.V. and 2" to right of middle line.

Diagnosis: Duodenal ulcer or ulcer at pylorus.

Case XLVIII. M.C. 43. Traveller. M.

Admitted with pain in abdomen chiefly on right side. Distension. Diminution of liver dulness free gas in peritoneal cavity.

Pain felt to right of umbilicus and above. Felt in back and right shoulder (severe) No history of gastric trouble before a week ago when he had slight discomfort and feeling of fulness in epigastrium. No actual pain. No haematemesis or melaena at any time.

Diagnosis: Ulcer at pyloric end of stomach perforated.

Operation: Perforation in anterior wall of stomach at pyloric end of stomach.

Fluid from peritoneal cavity examined and found to contain a short chained streptococcus. Vaccine made. Patient recovered.

Case XLIX. G.M. 36. Hawker. M.

Admitted with pain 3 hours p.e. relieved by taking food. Pain in back and right shoulder. Duration nine years - pain never severe. Attacks usually come on in Autumn. Never been any vomiting. No haematemesis but melaena has been present at times. Slight pain and localised tenderness got by electric current above and to right of umbilicus. No hyperanthesia present.

Diagnosis: Ducdenal ulcer. Relieved by medical treatment.

Case L. A.J. 28. Housemaid. F.

Admitted with frequent attacks of pain in epigastric region, sickness and vomiting. Present attack is of 3 weeks' duration. Has been troubled with gastric symptoms for past five years.

Pain comes on immediately after food. Made worse by ingestion of more food. No hunger pain. Pain in left shoulder. Attacks do not occur at any special time of the year. Haematemesis and melaena.

On admission: Definite tenderness got in epigastrium above and to left of umbilicus. Pain in left shoulder and back. Hyperasthenia is present in epigastrium and left hypochondrium. Hyperasthenia on either side of spinal column from level of 6th dorsal vertebrae downwards to lumbar region.

Patient has swinging temperature - cough, spits and sweats at nights. T.B. found in sputum. Haemoptysis. Case still not relieved.

Diagnosis: Gastric ulcer with pulmonary phthisis.

C O N C L U S I O N S.

Having discussed the etiology, symptomatology and differential diagnosis of peptic ulcer I have endeavoured to put forward the views of the leading authorities regarding the various peculiarities in the condition, and in my own series of 50 cases I have merely stated the facts of each individual case as I found it. I now, in this section of my thesis, wish to draw some conclusions from my own experience in the diagnosis and treatment of peptic ulcer.

And firstly I shall discuss the early diagnosis of cases of ulcer of the stomach or duodenum.

By many it is believed that one is not justified in diagnosing peptic ulcer before the occurrence of such symptoms as haematemesis or melaena. Now in a great number of my cases neither of these symptoms presented themselves and yet one was bound to admit that the patient was the subject of peptic ulceration. In my opinion haematemesis and melaena are symptoms which though of extreme value when present are still indispensable in diagnosing the existence of an ulcer of the stomach or duodenum. They are to be regarded either as late manifestations of the condition or mere accidents in the progress of the ulceration. An ulcer,

though extremely small, may give rise to symptoms of considerable severity, though it may not erode any vessel in the stomach or duodenal wall. It is, therefore, quite reasonable to believe that in many cases bleeding may never occur, or, if it does occur, it may be in such small quantities as to be unrecognisable as "haematemesis" or melaena. Erosion of a vessel of fair size is required in order that haematemesis or melaena may occur.

In my opinion, therefore, one is not justified in withholding a diagnosis of peptic ulcer simply on the grounds of absence of haematemesis and melaena. To wait for these symptoms is equivalent to waiting till an appendicitis goes on to abscess before diagnosing the condition.

Of far greater importance in the diagnosis of peptic ulcer than haematemesis and melaena is the presence of occult blood or the detection of blood corpuscles by microscopical examination of the gastric contents. In my cases it will be noticed that in ~~quite~~ a ~~number~~ *few* occult blood was present in the stools and blood corpuscles detected *in one case* in the gastric contents, though the symptoms of haematemesis and melaena had never occurred.

Since the early adoption of treatment in these cases is extremely desirable since many early ulcers may be completely cured by rest and suitable diet, it is of

the utmost importance to commence a rigorous mode of treatment before such symptoms as haematemesis or melaena occur, when too often nothing short of surgical interference can offer the patient any hope of recovery.

This now brings me to the consideration of the symptoms short of haematemesis and melaena which should enable us to say when we are dealing with a case of peptic ulcer.

In duodenal ulcer Moyniham considers that hunger pain is pathognomonic. He is bold enough to state that this symptom alone is sufficient on which to enable one to make the diagnosis of duodenal ulcer.

Now, though I agree with Moyniham to the extent that a history of hunger pain is extremely suggestive of duodenal ulcer and that the presence of this symptom, when existing with other symptoms, is of the utmost help in the diagnosis. Still it will be seen from my series of cases that in a number of them hunger pain was not present though operation finally proved them to be cases of duodenal ulcer. And again, a few patients gave a typical history of hunger pain who were the subjects of gastric ulcer and not duodenal ulcer.

My experience, therefore, leads ~~made~~ me to say that one cannot regard hunger pain as absolutely pathognomonic of duodenal ulcer, though in combination with other symptoms it is suggestive of the ulceration being of the

duodenal variety.

A sign of far greater importance, to my mind, in the diagnosing of peptic ulceration is the presence of tenderness in the epigastric or hypochondric regions. The tenderness can usually be elicited by palpation but in some cases it may be so slight as to be missed by this manner of examination, and we have to resort to some other way of eliciting its presence.

Now the way which I have found of most value in my cases is the application of the weak galvanic battery to the epigastrium. It is my belief that in no case of peptic ulcer will this method fail to pick out a small area of tenderness.

During my tenure of office as Resident Medical Officer in Bradford Royal Infirmary, I made a special point of applying the galvanic current to all gastric cases in which there was any suggestion of ulcer and in all cases where no localised tenderness could be detected we were able later to satisfy ourselves that we were not dealing with peptic ulcer - whereas in those cases in which the diagnosis became evident later or in which an ulcer was demonstrated at operation the galvanic current test was invariably positive. The tenderness elicited was seldom greater in size than about an inch square, but repeated applications of the battery resulted always in picking out exactly the

same area.

The importance of localised tenderness, whether elicited by palpation or by the galvanic battery is, to my mind, very great as it is not only present in conjunction with other symptoms and signs but persists after the other symptoms have disappeared. I often found that a patient would state that he or she felt "quite well again" but on examination the localised area of tenderness was still present.

This persistence of tenderness in spite of the alleviation of the more subjective symptoms, is, I consider, an important, if not pathognomonic sign in gastric and duodenal ulcer.

The site of tenderness naturally varies according to the position of the ulcer - usually being to the right of the epigastrium in duodenal ulcer and more to the left in the gastric variety.

I have already mentioned the pain in the shoulder which one gets in these cases. I am bound to say that this, in my experience, is an ~~external~~ namely useful additional sign as I know of no other condition in which precisely the same symptom is got.

To sum up, therefore, I would say that given a patient complaining of pain in the epigastrium worst after food, perhaps relieved by vomiting or giving a typical history of hunger pain (in the case of duodenal ulcer) and in whom a definite small area of tenderness

could be elicited with the addition of the shoulder pain I have described the diagnosis of peptic ulcer can be made. If in addition occult blood was detected in the stools (the best time to find it is immediately after pain) then the diagnosis is still more certain.

Haematemesis or melaena need not be waited for. The question of hyperchlorhydria in these cases I do not consider of great importance except in differentiating them from a malignant condition of the stomach.

In my opinion we must distinguish two kinds of peptic ulcers - viz., those which form and develop in stomachs in which hyperacidity is present and secondly those which occur in cases of sub-acidity. It has been generally accepted that hyperacidity is present in nearly all cases and also that that state was necessary for the production and progress of an ulcer. Reigiel states that there is needed in the production of a gastric ulcer an excess of pure HCl in the gastric contents and maintains that hyperacidity is a feature of all cases of gastric ulcer and is primary to that lesion. However hyperacidity is not constant in all cases of gastric ulcer. In fifty four cases reported by Howard in 17.6% only was there hyperacidity while there was actually subacidity in 26.4% of the cases; the remaining 56% having a normal acidity.

This, therefore, forces us to believe that we have

two separate causes of peptic ulcer or that there are two distinct kinds of ulcers. In the first we have cases in which an excess of HCl in the gastric contents is the chief factor. In the second, cases in which the condition is microbic and the organisms owe their virulence and vitality to the diminished quantity of free HCl present.

The second class I consider the most serious from a clinical point of view and are the cases which are most likely to go on to perforation. I make this assertion from experience gained from a series of cases of perforated peptic ulcers, from which I took swabs and scrapings and in which I was able to demonstrate each time the presence of the "short chained" streptococci.

The short chained streptococci are in manycases to be found from scrapings, taken from the roots of the teeth and gums. That the primary infection is to be found in the mouth has suggested to me the possibility of treatment of peptic ulcers by autogenous vaccines. I have tried this but have never been able to satisfy myself that any marked improvement resulted, though the administration of polyvalent streptococcus serum in perforated cases after operation seemed to make a more hasty recovery than other cases where no serum was given.

It has been stated, and is, in fact, generally held that ulcers of the stomach and duodenum are more common on the posterior wall than on the anterior wall. In perforated cases the reverse is the case.

I have also noticed that many cases of ulcer which have perforated have presented practically no symptoms before the perforation.

Is it not possible, therefore, that the painful symptoms of gastric and duodenal ulcer are due to adhesions with the pancreas and consequent connection with the sympathetic nervous system?

Such cases do not perforate or rather if the ulcer does erode the wall of the stomach the pancreas forms a base to the ulcer.

The importance of early diagnosis in these cases is on account of applying the proper treatment at once.

The question which naturally arises is should one operate or is medical treatment sufficient to produce permanent cure?

My experience makes me say that in the majority of cases of gastric ulcer medical treatment, if rigorously carried out, is sufficient to affect a great improvement if not a complete cure. With the duodenal variety medical means are not so effective.

I consider that in view of our present knowledge a diagnosis of duodenal ulcer makes it imperative to

carefully consider the question of surgical interference, and certainly, if the condition does not yield at first to medical treatment, operation is imperative.

The occurrence of haematemesis or melaena does not, in my opinion, make operation imperative.

If, therefore, one decides to submit a patient, suffering from peptic ulcer, to medical treatment what will be the lines on which we should go?

The following detailed treatment is the one which I have adopted in the majority of my cases and which I have found the best.

In only one case of my series was the total withdrawal of food adopted at the commencement. In all the other cases the treatment was as follows:-

Firstly. The teeth, gums, mouth, tonsils and pharynx, if in bad condition, were properly attended to. Then the patient was given a cathartic, such as 3 grains of calomel followed by salts, or the following prescription.

Hydrarg. Chloride ~~water~~ mitis gr. ii
Pulv. Rhei. gr. iv.
Sod. bicarb. gr. vi

Followed by salts in the morning.

Absolute rest in bed for at least three weeks was enjoined.

For the first 48 hours no food of any kind was allowed but a glass of hot water with a quarter of a

teaspoonful of sodium bicarbonate was given every four hours for four times. The object in this was to clear off thoroughly the mucous from the inflamed stomach or duodenum. After this no water, except sips for thirst, was allowed in order to give the stomach and duodenum a complete rest.

After 48 hours, i.e., on the third day a small amount of nourishment was allowed.

In my cases this took the form of two ounces of peptonised milk with an ounce of Vichy water given every three hours.

On the fourth day another ounce of peptonised milk was added at each feed to the same quantity of Vichy water.

Between each feed a glass of hot water was given in order to wash out the stomach and duodenum.

On the fourth day the bowels were moved by means of an enema and this was repeated each day for ten days.

On the fifth day 5 ounces of peptonised milk with 2 ounces of Vichy water were allowed, and as the patient was getting no iron in the diet a pill containing iron and aloes was given.

On the sixth day a gruel with one raw egg was introduced into the diet which consisted of small quantities of ordinary milk dilated with Vichy water, given at frequent intervals.

Hot water between feeds was still continued. After ten days we made a gradual increase in the quantity of food given, introducing pounded meat, pounded fish and lightly boiled eggs to the diet.

This diet was continued until the end of three weeks when the patient was allowed to sit up in bed.

During the fourth week an ordinary diet was commenced and gradually worked up until the patient was taking ordinary food.

At the end of the fourth week as a rule the patient was dismissed and sent to a convalescent home where he or she remained for a fortnight.

This treatment was carried out in nearly all my cases with extremely satisfactory results. In a few a bismuth mixture was given in the third week, the doses being taken at night after the stomach was quite empty.

In a few of my early cases (not in my series of 50 reported in this thesis) horse serum was given but with no good results.

To sum up it may be said that most gastric ulcers will undergo great improvement if not be completely cured by medical treatment carried out on proper lines.

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