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THESIS

ON

DUODENAL ULCER.

(including an examination of the cases
published during 1902 to 1906, inclusive).

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CONTENTS

	page		
Introduction	4	Clinical features—	31
Anatomy	5	Pain	31
Histology	7	Pyloric spasm.	33
Embryology	10	Appetite.	34
Physiology	10	Vomiting.	34
Situation.	11	Bleeding.	35
Characters.	12.	Flatulence	37
Causation (aetiology) 15.		Constipation	37
<u>A.</u> Experimental 16.		Stomach contents	37
<u>B.</u> Bacterial 17.		Tenderness	38
<u>C.</u> Non-bacterial. 17.		Rigidity	38
Thrombosis &c 17.		Swelling	38
Nerve theory. 18.		Loss of weight:	39
Injury 18		Urine	39
Anaemia 19.		Intermittency	39
Sepsis 20		Complications—	40
Burns 21		1. Haemorrhage	40
Bright's disease 22		2. Perforation.	41.
Cardiac. 24		3. Abscess	47.
Parasites 24		4. Cicatrization.	48.
Hyperchlorhydria 24		5. Adhesions	48.
Acute & chronic ulceration 28		6. Cancer.	48.
Age	30	7. Biliary & Pancreatic	49.
Sex	30	diseases.	

Association of Duodenal with gastric ulcer	50.
Diagnosis.	50.
Treatment.	53.
Author's case	54.
Literature.	64.

Introduction.

Ulceration of the duodenum has been known & described for at least eighty years; more than a thousand cases have been recorded; but it is only within the last decade that the subject has received the attention which it deserves. It may be, owing to our diet & mode of life, that the condition has become more common. It is only latterly, however, that its dangers—especially those of haemorrhage & of perforation—have been emphasized.

I feel that the general practitioner, who is quite au fait with gastric ulcer in anaemic young women, & with its complications, has not yet been taught to recognize the similar condition met with in the duodenum, which is more frequently found in the other sex. It must be allowed, however, that many patients, the subjects of duodenal ulceration, do not consult a doctor until the occurrence of perforation, & that some of them have suffered from no previous indigestion. In such cases, also, the medical man must be able to what has happened, as it is only then by

prompt operative interference that life can be saved.

Anatomy.

The duodenum is about ten inches long, & considered as part of the intestine, is remarkable in the following particulars.

- 1. It is more fixed to the posterior abdominal wall than any other portion of the gut.
- 2. It has no mesentery.
- 3. It surrounds the head of the pancreas.
- 4. It has a larger diameter ($1\frac{1}{2}$ to 2 inches) than the rest of the small intestine.
- 5. It is remarkable in that its commencement & its termination are at almost the same level. If contractility were denied to the duodenum we would compare it merely to an S-shaped trap, in which water stagnates & prevents the return of obnoxious gases. But this shape is not without significance; it offers a mechanical obstruction to the passage of the chyme, & thereby helps to secure a thorough mixing of the acid chyme with the alkaline juices which flow into the second part of the duodenum at the biliary papilla.

With regard to ulceration, it is the first part that concerns us most; this is two inches in length & extends from the pylorus to the neck of the gall bladder. This part is usually described as possessing an anterior & a posterior surface; it is better described as having a right lateral & a left lateral surface. The right lateral surface is entirely covered by peritoneum; the left lateral surface is only covered by peritoneum at the pyloric end.

Above lie the quadrata lobe of the liver, the gall bladder, & foramen of Winslow. Below lies the pancreas. Crossing the left lateral surface are found the gastro-duodenal artery, the portal vein, & common bile duct. It is well-protected by the liver in front.

Special mention should be made of the numerous named vessels lying in relation to the duodenum. This will best be recognized by quoting (mostly from "Coynihan") the vessels which have been found perforated in the course of duodenal ulceration—

Aorta (2 cases)

Hepatic

Gastro-duodenal.

Sup. pancreatico-duodenal (9 cases).

Pyloric

Pancreatica magna.

Right gastro-epiploic.

Portal vein.

Sup. mesenteric vein.

The second part of the duodenum descends vertically for three inches. On its left side is the head of the pancreas; on the right, the hepatic flexure of the colon. It is crossed in front by the transverse meso-colon, & transverse colon. Behind lies the hilum of the right kidney. Into this part, a little below the middle, enter the bile duct, & the duct of the pancreas; these ducts usually join each other before opening into the duodenum.

It will be observed, therefore, that the duodenum lies far back - on the posterior abdominal wall - & is in close relation to other important structures, such as the gall bladder, pancreas, & right kidney.

Histology

The duodenum has the following

characteristics,—

1. The valvulae conniventes make their appearance here, one or two inches beyond the pyloric ring, & extend thence down the jejunum.
2. Brunner's glands. These are most abundant at the commencement of the duodenum, gradually diminishing, as the tube advances. They are small, ^{compound} racemose glands, very similar in structure to the pyloric glands, & are situated beneath the muscularis mucosae, in the submucous layer. The duct of each gland pierces the muscularis mucosae, opens on the surface of the mucous membrane, & discharges a serous secretion.
3. Biliary papilla. This consists of a small projection of the mucous membrane, situated in the second part of the duodenum, three or four inches from the pylorus. The bile & pancreatic juice enter here.
4. The sphincter of Ochsner². While operating for gall stones & on the stomach, Dr Ochsner, of Chicago, frequently found the duodenum distended with gas to a point just below the entrance of the common bile duct, & contracted below this point. (This fact has been observed by numerous

surgeons + anatomists.) He accordingly made a considerable number of dissections, + found in every case a more or less marked thickening of the gut at a point two to four cm. below the entrance of the common bile duct. A careful study of this thickening demonstrated a marked increase of the circular muscle fibres. The arrangement of the fibres was more diffuse than that found at the pylorus, thus constituting a broader sphincter. D. Osborn suggests that these facts point towards the presence of a sphincter at this point, + that its physiological function consists in providing a means of retaining the chyme in the upper part of the duodenum sufficiently long to provide for a thorough mixing with bile + pancreatic juice. He points out how obstruction - physiological + pathological - may occur at the pylorus + at the ileo-caecal valve; in the latter situation, the obstruction is due to inflammation in its vicinity, causing a stoppage of peristalsis, + is most frequently met with in cases of appendicitis. He considers, therefore, that in certain forms of irritation or inflammation

of the gall bladder, or its ducts, (& we may surely add the Duodenum itself) this sphincter may play no unimportant part. It is normally, however, a physiological sphincter.

Embryology.

The foregut forms the tongue, the posterior wall of the pharynx, oesophagus, stomach & duodenum to a point just below the entrance of the common bile duct, probably the sphincter of Oddi. The pancreas, liver, & their ducts are offshoots from the upper part of the duodenum.

Therefore, from an embryological point of view, the upper part of the duodenum is not part of the small intestine, & we shall see later what a significant bearing this has on the functions of the part.

Physiology.

The acid chyme which emerges from the stomach through the pylorus is the normal stimulation³ to the flow of the pancreatic juice. The food accumulates in the duodenum & there undergoes a process

of rhythmic segmentation⁴ which serves to mix it thoroughly with the bile & with the pancreatic juice. The great physiological importance of the latter secretion is, of course, the digestion of all three classes of food-stuffs — carbohydrates, proteids, & fats. Thus the duodenum, like the stomach, serves to retard the progress of the food, giving time for its conversion into absorbable products. No absorption, with the possible exception of certain stimulants, takes place before the jejunum is reached.

Blue litmus paper is turned red when it is placed on the surface of a perforating ulcer, but the mucus exuded at that ulcer gives a strong alkaline reaction (Caird).⁵ Below the biliary papilla an alkaline reaction is obtained.

Situation of the ulcer.

The situation of duodenal ulcers is one of the most remarkable facts in regard to this lesion. In the vast majority the ulcer is found in the first part, & most of these are on the right

lateral wall. It will be observed, therefore, that the ulceration occurs when the contents of the tube are acid, & this is forcibly brought out by reference to the following statistics.

	1 st part	2 nd part	3 rd part	4 th part	Total
Collin ⁶	242	14	3	3	262
Perry & Shaw ⁷	123	16	2	—	141
Author	107	7	1	—	115

The first & second parts were affected together in 3 cases; the situation was not stated in the other cases.

It may here be stated that this is the situation, too, of post-mortem digestion. This condition is more common in the stomach than in the duodenum, but is never found below the biliary papilla.

Characters of the ulcer.

Two forms of ulceration may be described according as induration is present or not. There are many other ways, of course, in which a basis of classification could be formed, but we find this the most useful in our present contention.

The great majority of the ulcers are indurated; they are obviously chronic. In most of them all the coats of the tube are affected, & there are signs of a chronic peritonitis around the ulcer, which may be found adherent to any of the structures surrounding it. The scarring on the outside of the duodenum may extend over a distance of two or more inches. These are the cases which lead to stenosis of the gut, with secondary dilatation of the stomach, but, in spite of this tendency to healing, they often give way & perforation occurs. Looking at these ulcers from the interior, they are sometimes terraced, sometimes deeply punched out; they are practically devoid of granulations, & a vessel, e.g., the gastro-duodenal artery may be found exposed on the surface. The margins of a perforated ulcer are friable & often oedematous; they do not therefore hold sutures well.

In the other class of ulcers there is no sign of induration, no hint at repair. They have more the appearance of having been freshly produced. They may be

bleeding mucous ulcers, or deeply punched out, or perforated.

CAUSATION

It must be understood at the outset of this inquiry that we consider the causes at work in the production of duodenal ulcer are similar to those which account for ulcer of the stomach; in fact, many cases have been recorded where both lesions existed in the same patient.

A. Experimental.

B. Bacterial.

C. Non-bacterial:-

- (1) Embolism; thrombosis theory (Virchow).
- (2) Nerve theory.
- (3) Mechanical injury; (+ foreign bodies).
- (4) Anaemia.
- (5) Sepsis.
- (6) Burns.
- (7) Bright's disease.
- (8) Cardiac disease.
- (9) Parasites.
- (10) Hyperchlorhydria.

A. Experimental causation of duodenal ulcer.

Ulcer of the duodenum has been experimentally produced in the following ways:-

- ① By giving animals cultures of colon bacilli in their food. (Turck)
- ② By the introduction of HCl into the stomach (Ewald).
- ③ By giving animals subcutaneous injections of toluendiaurene. (Hunter) ^{8.}

The first of these experiments makes us wonder if there is not sometimes a connection between oral sepsis & duodenal ulcer, though it must be confessed the former is notoriously common while the latter is not. Another point in favour of the infective origin of gastric & duodenal ulcer is the fact that it is not uncommon to find two ulcers—the one exactly opposite the other.

Our views are entirely in accordance with the results of the second experiment.

With the third experiment we have little to do, though Dr Hunter thought he had found an analogy between ulcer occurring in cases of burns & those produced by his injections. His contention is partly correct.

In his experiments it is to be noted that the inflammation, set up by some toxic substance excreted with the bile, extended from the biliary papilla to a variable distance down the intestine. It will be shown later that the ulceration found in cases of burns is mostly above the entrance of bile, & that there is no question of it being produced by anything excreted at Vater's ampulla.

B. There are some ulcers of the duodenum which can clearly be ascribed to the action of bacteria. They are:

- 1. Tubercular.
- 2. Typhoid.
- 3. Anthrax.

C. Non-bacterial causes.

1. Thrombosis. Embolus. Plugging of the nutrient artery was the theory advanced by Virchow. It is supported by the fact that this has been actually demonstrated in some instances, & that the ulcer may present a "terraced" appearance, the latter in

accordance with the vascular supply of the wall of the viscus. It is certainly true that the lesion is common in one disease where thrombosis is common - viz, chlorosis. But in the great majority of duodenal ulcers which have been examined there is little or nothing pointing to this method of causation. In the diseases where embolism of other viscera is most frequent, gastric or duodenal ulceration is exceedingly rare. We incline therefore to the view that when a thrombosis has been found, it is usually secondary to some damage done to the mucous membrane.

2. Neuric theory of causation. Ewald + Koch^{9.} have shown by experiment that injuries of the spinal cord in the cervical + upper dorsal region may be followed by haemorrhage + ulceration of the stomach + duodenum.

3. Mechanical injury. Mayo, with a greater experience of duodenal ulcer than anyone else, thinks it probable that mechanical injury plays some part in the production of this lesion, pointing out the frequency

of ulcer in the grinding antrum of the stomach, & in that part of the duodenum which receives the impact from food leaving the pylorus. We may certainly consider this as one of the contributing causes.

There is, however, one form of injury to the stomach & duodenum that does not lead to ulceration, & this is injury produced in the course of operations on these parts. For example, the surgeon may have to incise the duodenum in order to extricate a calculus from the lower portion of the common bile duct. This form of injury to the duodenal wall has never been known to lead to ulceration. There are two cases on record ^{21. 22.} where a spoon had been swallowed, & had caused perforation.

4. Anæmia. Anæmia, usually in the form of chlorosis, has long been known to go hand in hand with gastric ulcer in the female sex. In anæmia, too, there is a diminution in the alkalinity of the blood; while in gastric & duodenal ulceration there is usually a condition of hyperchlorhydria present. When we put these facts together, we are bound to consider anæmia as a great predisposing cause.

Now, duodenal ulcer is more common in the male than in the female, the reverse of anaemia. We must look, therefore, for some cause other than anaemia to account for duodenal ulcer in men. Males, suffering from duodenal ulcer, are often anaemic, but this anaemia is brought about by insufficiency of food.

5. Sepsis. In the 141 cases of duodenal ulcer collected by Perry & Shaw¹⁷, mostly from the P.M. records at Guy's Hospital, 21 were in patients who had died from sepsis. The ulcer was invariably situated between the pyloric ring & the biliary papilla; it was usually round, but some were irregular. The authors point out that in all septic processes petechiae are found widely scattered beneath serous & mucous membranes in the alimentary canal as elsewhere, & accepting this as the predisposing cause, consider the gastric juice as the active agent in the production of the ulcer. It is to be noted (à propos of burns) that in 10 of their cases there was some sloughing condition of the skin or cellular tissue.

6.

Burns. The association between duodenal ulcer & burns of the skin was first pointed out by Curling¹¹ in 1842. He supposed there was some sort of sympathy between the skin & Brunner's glands.

It is well known, however, that congestions & ecchymoses are liable to occur in various parts of the body after burns, & the congestions found along the alimentary tract are not confined to the duodenum.

Perry & Shaw⁷ collected 29 cases illustrating this subject. The site of the ulceration was as follows:—

in 22 cases	in 1 st part
" 4 "	" 2 nd "
" 2 "	" 1 st + 2 nd parts.
" 1	the site was not stated.

It will be observed that the lesion was situated almost invariably in the first part. The ages of the patients varied from $3\frac{1}{2}$ to 70 years. Death took place from perforation in 7 cases, & from haemorrhage in 13. Of 27 cases where the age was stated, 18 were females, & 9 were males. But, as the authors point out, burns are more frequent & serious in females from the

nature of their clothing. They think that the diffuse patchy congestion of the alimentary tract is brought about by the sepsis & is the predisposing cause, while the exciting cause is to be found in the gastric juice.

At the present day, the view of pathologists is against the occurrence of duodenal ulcer after burns. We are acquainted with only one pathologist who has seen such a case. Against this we have to say that few autopsies are made in fatal cases of burns; also, the treatment adopted nowadays is stringently antiseptic.

I. Bright's disease. The association of duodenal ulcer with nephritis in its different forms is well established. It first attracted the notice of English pathologists^{12.} in 1875, but within the last few years has attracted much attention in France by Devic^{13.} & others.¹⁴ The method of production of the ulcer seems to be the same as that following burns & sepsis: first a congestion or haemorrhage into the mucous

membrane, then a superficial erosion, & later a deep ulcer which may perforate into the general abdominal cavity. The congestion or haemorrhage is the predisposing cause, the gastric juice does the rest.

Perry & Shaw⁷ collected 12 cases, with one exception, the situation whenever stated was above the biliary papilla. It is more common in the interstitial forms of nephritis. These authors point out that some form of nephritis is present in 7% of deaths from all causes; in 70 cases of duodenal ulcer collected by them, 17% showed evidences of nephritis. Devic & Charvet have made 12 observations of such cases within a space of 4 years; for the most part they were in patients with ~~an~~ advanced interstitial nephritis & who had symptoms of uraemia. The ulcer has seldom given rise to symptoms. Ulceration of the small & large intestine is known in albuminuria,¹⁵ but this usually affects the ileum, & sometimes both bowels; it rarely ascends as far as the duodenum, & is accompanied by enteritis or colitis.

8. Cardiac disease. We have now cited several diseases which lead to congestion or ecchymosis of the duodenum, apparently predisposing it to ulceration, & we might naturally expect the congestion so common in heart disease to lead frequently to the same lesion. Yet it is not so. In the cases collected by Perry & Shaw there were only 5 instances of such an association.

9. Parasites inhabiting the duodenum.

The *ankylostoma duodenale* & the *ascaris lumbricoides* are found sometimes in this part of the alimentary canal. The latter has been known to perforate the tube.

10. Hyperchlorhydria. The chief facts about hyperchlorhydria may be shortly stated here. The gastric secretion varies in quality with the food that calls it out, & the strongest & most acid juice is

produced by an animal diet. Dr Robert Hutchison¹⁷ gives it as his opinion that hyperchlorhydria is most apt to develop in those who habitually consume large quantities of meat, & he would incriminate also the consumption of salt & stimulating condiments. There can be no doubt as to which sex indulges most in such articles of diet. The subjects of hyperchlorhydria are often in otherwise perfect health. It must be added, however, that hyperchlorhydria is a common accompaniment of chlorosis.

The fact that almost every ulcer of the duodenum is situated above the entrance of the bile & pancreatic secretions leads one to the belief that the ulceration is caused by the acid secretion of the stomach. But there is further foundation for this belief. In the majority of those cases of duodenal ulcer in which the gastric contents have been examined, hyperchlorhydria has been noted.

Further light has recently been thrown on the connection between hyperchlorhydria & ulceration. It has been found that a peptic ulcer of the jejunum may develop after a gastro-enterostomy has been performed. Gosset¹⁹ has collected 31 instances of this occurrence. Most of the operations had been performed for stenosis of the pylorus. In 21 of these cases the gastric contents were examined; 2 were normal, 2 showed hypochlorhydria, & 17 hyperchlorhydria. The ulcer is identical in appearance with that found in the stomach & duodenum, & is situated usually about 1 cm from the stoma. In most cases the ulcer did not begin to give trouble until one to two years after the operation. It is significant that 29 of these patients were males, & 2 females. It occurred at all ages. No case has yet appeared where it took place after a gastro-enterostomy for cancer. It is interesting to note that one of these operations was done for congenital stenosis in an infant two months old; two months later this

patient (a female) died from perforation of a jejunal ulcer.

There are some, however, who debate whether the ulcer of the duodenum is the cause or the effect of the hyperchlorhydria. There is plenty of evidence in favour of the latter contention. We think that hyperchlorhydria is of itself able to produce ulceration, but that there is often a predisposing cause at work, & if this contributing cause be active enough, we think ulceration may be produced by normal gastric juice. The predisposing cause may be of the nature of toxic products circulating in the blood, as in the case of sepsis, or of damage to the mucous membrane, as in the haemorrhage into the mucous membrane in Bright's disease. The gastric juices is the active agent in every case, but there are many causes which permit the gastric juice to do its destructive work on a mucous membrane able to resist its action under normal conditions.

The statistics, which follow, are drawn from a careful examination of all the available cases published during the five years (1902 to 1906).

They number 314 in all, 116 of which have been separately, & on the whole, fully reported.

Acute & chronic ulceration. We have already divided duodenal ulcers in those that are indurated or otherwise, that is to say, into chronic or acute ulcers.

All authors give such a classification.

It has been our endeavour to study especially the cases in which no symptoms are averred to have been present prior to perforation.

Only 15 cases out of 116 fall into this category. It is unfortunate that the ulcers in these cases have not been described for the most part. Eight of them presented no induration nor other sign of being chronic, & of these, 2 were "uraemic". Three of them were obviously chronic. Now, a patient suffering from perforation is not in the

best position to give an account of his previous troubles; he has never been in such agony before, & is apt to minimize any previous indigestion.

On the other hand, we do believe in an acute ulcerative process, the work of a few hours or days. We have proof of the rapidity with which a duodenal ulcer may form if we refer to those which occur after burning accidents. The earliest death took place $4\frac{1}{2}$ days after the accident (from haemorrhage). [The haemorrhage here was, of course, from the duodenal ulcer].

The point that we desire to bring out forcibly here & in what follows is that in the great majority of cases there is abundant evidence to be obtained of the presence of a duodenal ulcer.

Age. Duodenal ulceration occurs at all ages, in the newly-born ^{23. 24.} as well as in the aged. It is impossible at present to account for its occurrence in infants who are but a few hours old.

In 116 cases where the age is stated the average is 36.7 years. In these cases, however, the disease had already been present for months or years before the diagnosis of duodenal ulcer had been arrived at. The average age in males is 38.4 years, & in females 35.1.

Sex. In 130 cases where the sex is stated it is found that 19.2% are females. Mayo,^{20.} who has had 188 cases under his care in America, finds that 27% of them were females. Though one is sure that the same causes are at work in both sexes, it is still an unsolved problem why the disease is more common in the male sex. It does not depend on alcohol or tobacco.

Clinical features.

The clinical features of duodenal ulcer are so well known that it will be our object here to refer more particularly to the statistics of the cases we have analysed.

Pain. Pain is by far the most important of all the symptoms. With the exception of the cases where no symptoms were observed before the onset of perforation it was present in every case. Two kinds of pain are to be noted.

1. Pain ensues when the acid chyme flows over the surface of the ulcer. It occurs with great constancy, ~~when~~ 2 hours or so after a meal, is localised to a spot about 3 inches above the umbilicus & just to the right of the middle line; it remains for hours, often until the next meal is taken. The patient often acquires the habit of taking a biscuit or a glass of milk, which, he finds, removes the pain, while others induce vomiting for the same purpose. The usual explanation of the relief

obtained by the introduction of food into the stomach is that the food neutralises the juice already in the stomach, & the pylorus remains closed until the percentage of hydrochloric acid has again reached a certain amount. Dr Robert Hutchison²⁷, however, ascribes this pain "to the acid dyspepsia which precedes most duodenal ulcers & not to the ulcer itself." This sort of pain is often most troublesome in the early hours of the morning when the patient is in bed.

2. There is also the pain produced by what is often called a chronic perforation. In a considerable number of cases the duodenum is found tacked down to neighbouring structures, e.g., gall bladder, liver, or pancreas. In such cases the ulcerative process has worked its way through all the coats of the canal & has only been prevented from actual visible perforation into the general peritoneal cavity by the unique position occupied by the duodenum, which affords every opportunity for limiting

the perforating process. The history of patientsⁱⁿ whom chronic perforation has occurred usually shows that they have suffered from attacks of severe pain in the epigastrium, lasting for several days, often accompanied by haematemesis or melaena, & recurring at an interval of months or years. Between these attacks they consider themselves, as a rule, quite well.

The pain experienced in duodenal ulcer may radiate in many different directions in different individuals & in this way the lesion is apt to be confounded with the diseases of neighbouring organs.

Pyloric spasm. Pyloric spasm is a contraction of the antrum & pyloric canal which lasts rather less than a minute. With regard to its possible relation to ulcer we shall quote from Mayo¹⁰ who has had a unique opportunity of observing this phenomenon in the course of certain operations done under local anaesthesia: "in the most

extreme degrees we have found it in connection with stones in the appendix, impacted gall stones, tuberculosis of the intestine, & chronic intestinal obstructions of various kinds.--- While it is possible that pyloric spasm may exist as the result of a mucous ulcer as believed by Eisberg & Doyen, such is not our conviction."

Appetite.- The appetite is often not interfered with except when patients, realising the relation of their pain to food, begin to limit their dietary. Is it not a fact that those who are well supplied with gastric juice have a particularly good appetite?

Vomiting.- The vomiting referred to here is not the vomiting that follows so frequently the onset of perforation. Vomiting occurred in 48 of the 116 cases we have analysed, & would therefore appear to be a frequent feature of duodenal ulcer itself. In reality it is not. The great majority of those in whom

persistent vomiting occurred were suffering from a dilated stomach brought about by the contraction of a duodenal ulcer; in them the vomiting was typical of this complication. Of the remainder, most of them vomited on one or two occasions only.

Bleeding. It is often stated that the only certain symptom of a duodenal ulcer is a profuse haemorrhage from the bowels for which no other cause can be found.²⁷ It is certainly one of the most important symptoms, but it would indeed be a misfortune if we had to rely only on the above statement.

Haemorrhage was recorded in Fenwick's²⁵ cases in 40%, either in the form of haematemesis or melaena. It occurred in 49 of our 116 cases, or 42%.

The general impression amongst clinicians is that haematemesis is more common in gastric than in duodenal ulcer, while melaena is more apt to occur in the latter, & one is able to see reason for such a belief. As a

matter of fact, this is not borne out in our cases. Out of 116 cases there was haematemesis in 37
 melæna in 32.

(Both haematemesis + melæna are recorded in 20 of these) - Haematemesis is perhaps more easily remarked than melæna. From the nature of the process it is more than probable that bleeding is a much more common symptom than these statistics show; in the majority of cases where bleeding is recorded it is a profuse hæmorrhage that is referred to.

Death from hæmorrhage is said to be more common in the case of duodenal than in gastric ulcer. It occurred in 7 out of our total number (314) of cases; two of these were in the newly-born.

Profuse hæmorrhage may ensue not only when a large named vessel is eroded, but in mucous ulcers as well. The bleeding may continue & even end fatally after a gastro-enterostomy has been performed.

Flatulence. Mayo Robson²⁶ considers that flatulence is an important symptom, that it is most marked at the time when pain is present, & by exercising tension tends to intensify the pain. Out of 116 cases we have found it recorded in 13 only.

Constipation. This is only noted in 11 of our cases; there are, however, very few remarks on the state of the bowels. Mayo Robson²⁶ says it is a very frequent symptom, that the patient is better when the bowels are open, & that it is usual to find the subject of duodenal ulcer taking a saline aperient every morning.

Stomach contents. We have already noticed how common hyperchlorhydria is in peptic ulcer of the jejunum, & all writers agree that it is usually present in duodenal ulcer, but there are exceedingly few observations on this point in our cases. Out of 116 cases it was noted 5 times; free HCl 3 times;

hypochlorhydria twice; + lactic acid once.

Tenderness. Tenderness is an important symptom. It is situated slightly above the umbilicus + to the right of the middle line. It is by no means always present, as in the case we shall afterwards relate where perforation occurred on the following day. In this connection it has to be remembered that the duodenum does not lend itself readily to palpation, being well protected in front by the liver.

Rigidity. Rigidity is often observed in the right rectus muscle, during one of the painful attacks. It will specially be referred to under perforation.

Swelling. A tumour has been felt in a few cases. It is seen in cases of chronic perforation + consists of a plastic mass of adhesions on the outer aspect of the canal. From the position of the duodenum it is comparatively seldom that any thickening can be felt.

Loss of weight- This was noted in 20 cases out of 116. It is easily accounted for in the numerous cases where dilatation of the stomach has occurred, & in those individuals who have observed that their pain was dependent on food, & who have diminished their diet in consequence.

Urine- The state of the urine is unfortunately recorded only 4 times. It contained albumen twice, no albumen once, & diacetic acid once (in a case of tetany from dilated stomach).

Intermittency of attack. This is quite one of the features of the disease, & one which deserves special mention. It is not easily accounted for. The patient has a more or less severe attack of pain, lasting a few days perhaps; this disappears, he eats anything, & considers himself quite well during a period that varies from weeks to years. Then another attack - similar to the first - ensues. This state of things may continue

for years.

COMPLICATIONS.

1. Haemorrhage.
2. Perforation.
 - (1) bacteriology of.
 - (2) leucocytosis in.
 - (3) anaesthetic in.
3. Abscess.
4. Cicatricial contraction: dilated stomach.
5. Adhesions.
6. Cancer.
7. Biliary & pancreatic diseases.

1. Haemorrhage has already been dealt with. Let us merely mention here how frequently it leaves the patient blanched. We have found only one estimation^{31.} of the blood in our series of cases. It was taken after haemorrhage had occurred. There were no nucleated reds.

R.B.C. 1,850,000

Hb. 30%

Leucs. 5000. (No post-haemorrhagic leucocytosis)

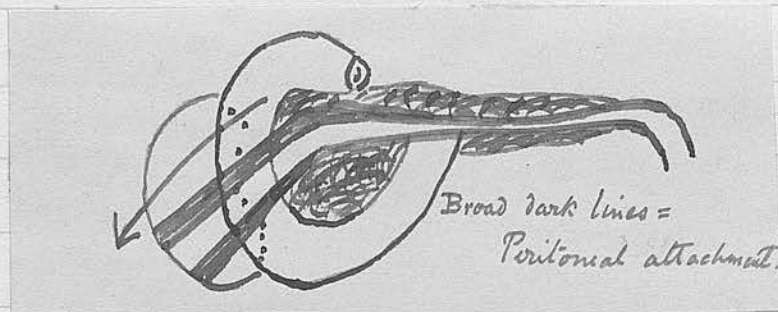
441.

2. Perforation. There can be no doubt that perforation is a very frequent complication. We shall make no attempt, however, to prove in what percentage perforation takes place, for the natural tendency is to publish interesting cases, & cases which have been successfully operated upon. Perforation took place in 45.6 of the 116 cases we have analysed, in 69% of Collin's & in only 5.3% of Mayo's cases.

Pathology of perforation. The situation of the perforation is at the spot where ulceration most usually occurs, viz, the right lateral surface of the first portion of the duodenum. Haemorrhage may occur coincidentally with perforation.

With regard to the course taken by the fluid extravasated at the perforation Maynard Smith²⁹ performed a series of experiments. He introduced a tube along the oesophagus, performed a laparotomy, & brought the tube out at the favourite site of perforation. After closing the abdominal wound, he allowed a specially prepared fluid to flow slowly down the tube.

In every case the fluid took the same direction. The diagram (after Toynehan) shows the course at starting & explains the initial course towards the right.



In no case was the foramen of Winslow invaded. The fluid first reaches the superior aspect of the transverse mesocolon, whence it is directed outwards to the right, & then finds its way down the outer side of the ascending colon, thus arriving at the appendicular region. The fluid now rises till it reaches the level of the brim of the pelvis; it then overflows into the pelvis. In cases where the ascending colon is particularly well-tacked down to the posterior abdominal wall, the fluid flows over the ascending colon before rising to the level of the pelvic brim; this occurs usually a few inches below the hepatic flexure. "Even

4.

if the fluid takes this course it still tends to flow to the appendix region—guided thither by the obliquity of the mesentery downwards & to the right, & by the slope of the abdominal wall away from the middle line. It does not directly invade the left half of the abdomen, but crosses the termination of the ileum & reaches the right side of the pelvis at practically the same point as before.

We have gone into this very fully because it has such an important bearing on the symptoms of perforation, & shows why such a large number of cases have been operated on for appendicitis, since sooner or later the symptoms become marked in the latter region.

"The amount of free fluid in the abdominal cavity is extraordinary. If the perforation has been present for several hours, many pints may be found. The fluid is thin, turbid, & odourless; there may be flakes of lymph. As a rule, there is no gas present in addition.

44

This fluid is an effusion of an alkaline nature,⁵ produced by the reaction of the peritoneum to the acid contents which escape. Comparatively little escapes, as a rule, from the perforation itself.

- (1) Bacteriology of the fluid.^{5 + 30} In four cases it was sterile. *B. coli* was found once; *B. proteus*, once; & in one case *Sarcinae*, diplococci like pneumococci, & various bacilli were found.
- (2) Leucocytosis. There are only three counts to record, viz., 16,000; 25,000; & 14,680. This would seem to show that a slight leucocytosis may take place after perforation.
- (3) Anaesthetics in perforation. We find no record of this in any of the cases. The author has given anaesthetics in three cases of perforated duodenal ulcer within a space of 6 weeks. In each case great difficulty was experienced in the patient's breathing, & this added materially to the difficulties of the surgeon. It is well known, however, that operations in this region, e.g.,

operations on the gall bladder give the anaesthetist much trouble.

Perforation has occurred quite a number of times while the patient was in the act of lifting a heavy weight.²⁹ On the other hand, it has occurred just as often when the patient was in bed & asleep. Nor does it depend on whether the stomach is full or empty.

The clinical features of perforation are usually most typical. The patient is seized with the most frightful pain in the epigastrium to the right of the middle line. It doubles him up, & completely disables him. He groans or cries out with the pain. The pain is stationary, & does not come & go. In the case which we shall presently describe, the patient, in order to locate the seat of pain accurately, placed his right hand behind his back, over the

lower ribs, almost suggesting that the kidney or pleura were at fault.

The breathing is very short, almost entirely costal, & the abdomen becomes boardlike at once, but more particularly on the right side. Change of posture does not relieve the pain, but the act of changing is agony to the patient.

The temperature is subnormal at first; the pulse becomes progressively quicker; the respiration is not much increased in rapidity.

This state of matters continues for some hours without interruption & then a reaction occurs during which the patient becomes more comfortable. It is this reaction that may mislead the medical man. The pain

becomes more general, it goes down to the region of the appendix, being always more severe over the right half of the abdomen, & tenderness becomes a feature.

Thus it is why so many cases, seen for the first time at this stage, are confused with appendicitis.

The boarding of the abdominal muscles becomes progressively greater. In some

the abdomen swells. The liver dulness varies greatly in different cases. It was diminished or absent in 15 of our series; while it was not lost at all in 10. One curious fact is that a ^{high-pitched} tympanitic note is usually obtained all over the abdomen, & yet anyone who has any experience of this affection knows there is a large amount of fluid present. We cannot expect anyone to believe this statement who has not met with a case. It is due, evidently to the boarding of the muscles.

The sooner cases of perforation are submitted to operation, the more likely with a successful result ensue. The longest period between perforation & successful operation was 31 hours.

3. Abscess. Several cases have been reported where an abscess had formed in connection with a duodenal ulcer. It may be a subphrenic abscess which may burst into the pleura or lung, or a perirenal abscess, or the pus may gravitate behind the peritoneum to the

appendicular region, etc.

4. Cicatricial contraction. This is an attempt at healing on the part of the ulcer. It often leads to dilatation of the stomach from narrowing of the duodenal channel, or, if the ulcer be situated lower down, it may obstruct the biliary passages, & give rise to jaundice & pancreatic complications.

5. Adhesions. Adhesions which form between the duodenum & its neighbouring structures may lead to intestinal obstruction, jaundice etc.

6. Cancer. Cancer of the pylorus is attributed in many cases to a pre-existing ulcer; this is a statement which meets with pretty general approval. Both these conditions are most frequently met with in the pyloric region. We might with confidence expect the same relationship to exist in regard to duodenal ulcer.

4

As a matter of fact, cancer is very rarely found primarily in the duodenum, but is known to have taken place there after ulceration. Mayo has only twice seen primary cancer of the duodenum, once developing on an ulcer base. Mayo Robson has met with the condition on three occasions; in no case, however, in connection with previous ulceration.

7. Biliary & Pancreatic disease. It is no wonder that duodenal ulcer is sometimes associated with catarrh of the ducts which open at Vater's ampulla, & the subjects of duodenal ulcer often exhibit a slight icteric tinge. When the base of the ulcer is situated over the pancreas, a localised chronic pancreatitis occurs at this point, & if localisation is incomplete the more serious complications of interstitial or suppurative pancreatitis may develop.

We have found two cases ^{32. 33.} in

67

which volvulus of the small intestine occurred as a complication of perforation. One of these was in a gastric perforation. Is this merely a coincidence?

Association of Duodenal with gastric ulcer.

In 23 cases in our series (or 19%), evidences of both gastric & duodenal ulcer were present together in the same patient. This is not surprising in view of the causes which produce these lesions, & with more careful examination at the time of operation it is probable this percentage would be higher. Mayo, indeed, has found gastric ulcer present in over 50% of his duodenal cases.

Diagnosis. Though the diagnosis is admittedly easier in the case of gastric than of duodenal ulceration, we think it should be possible for a diagnosis of the latter to be made in most cases which present themselves. In going through our 116 cases it is

surprising to find how typical the cases are, even in those where bleeding was not a feature. In most cases there is the pain delayed till two hours or so after a meal, & situated in the position occupied by the first part of the duodenum. This pain is relieved by taking food. Tenderness & rigidity of the right rectus may be present. Then there are the intermittent attacks of pain.

Bleeding occurs in about half the cases, & if suspected, might be found in more.

It is more from the history & examination of the patient, than from the establishment of the presence of hyperchlorhydria & from other laboratory work, that we are enabled to arrive at the diagnosis.

Diagnosis of perforation should not often present much difficulty, - now that the signs of this complication are so well known. The ^{primary} situation, severity, & continuousness of the pain are the most marked features. (It is important to remember that the patient may have been in quite good health up to the

moment of this accident). The tendency of the symptoms to travel towards the region of the appendix, the boarding of the abdominal muscles (particularly those on the right side), & the thoracic breathing are all typical. On one occasion we nearly fell into error; it was in the case of a painter. A typical blue margin to the gums was absent. He had taken reasonable care with his meals. For two hours we were in doubt, while he exhibited all the signs of acute perforation of the duodenum. The pain was to all appearances as intense as it is in perforation. It was unremitting & was situated in the epigastrium which was tender. There was great boarding of the abdominal muscles & thoracic breathing. Then the pain gradually lessened, & the nature of his affection — lead colic — became obvious. Had it not been for his profession, he would probably have undergone an unnecessary operation.

Treatment.

We think the treatment of this affection should be on exactly the same lines as that for gastric ulcer. We read in many of the cases in our series that prolonged & appropriate medical treatment had been tried & found inefficacious. Now, if gastric ulcer can be cured by medical means, as is generally accepted, it would seem that duodenal ulcer a fortiori would lend itself to cure by such means - rectal feeding etc. The wall of the duodenum does not distend to the same degree as that of the stomach, & it has altogether more rest from its fixed position. It has yet to be proved, however, that duodenal ulcer can be cured in this way. The facts at our disposal do not warrant us in speaking hopefully of it, but the literature of the subject from a medical point of view is extremely meagre, & those physicians who express an opinion on the subject incline to regard the disease as surgical more than we do ourselves.

Hutchison²⁷ makes the statement that whenever haemorrhage occurs, operation should be recommended; & Byrom Bramwell, after having four cases under his care within a few weeks of each other (three of which ended fatally from haemorrhage), says that one should advise operation as soon as one makes a definite diagnosis of duodenal ulcer.

While concurring more with the view expressed by Dr. Hutchison, we should like to hear the reports of a hundred cases treated medically.

Surgical Treatment— The most successful treatment so far reported is by gastro-enterostomy— thus diverting the food which would otherwise flow over the ulcer. This is certainly indicated where stenosis of the pylorus with dilated stomach are present. The danger of a jejunal ulcer forming as a result of the operation is exceedingly remote.

In the case of perforation, early operation is imperative. Suture of the rent should be practised in all cases; scarcely

a case has recovered where drainage without suture was practised. Suture is often very difficult on account of the oedematous friable margins of the ulcer; a piece of omentum has frequently to be taken to help in closing the rent. (Excision of the ulcer is not to be recommended, & the ulcer itself should be left alone unless serious bleeding is going on). It is safer, as a general rule to delay making a gastro-entero-anastomosis, which will then be undertaken at a later date if need be.

The following are the notes (taken at the time) of a case of duodenal ulcer with perforation which I had to deal with in private practice.

Male, aged 45, clerk in stockbroker's office. Very spare. With the exception of some indigestion a year ago had always enjoyed good health. Occasional intemperance. No venereal disease. Slightly constipated.

Saturday. Drank a good deal. Has not been well since. Bowels moved.

Tuesday. Called in to see him. He had had a severe attack of vomiting at 8 a.m. The vomit was dark & like coffee-grounds but had not been kept. Tongue dry & coated. I went carefully over the abdomen; there was no tenderness anywhere. I agreed with him that he was suffering from his Saturday night's performance - congestion of the liver which was just below the costal margin - & as the bowels had moved but slightly since Saturday, prescribed Calomel grs v & Sodium phosphat grs 60 every four hours.

At 9 P.M. I found him no better; the bowels had not moved, & his appetite was poor.

Wednesday. Did not sleep well. At 5 a.m. he was seized with agonising pain in his right side. He demonstrated the painful situation by placing his right hand behind his back over the lowest right ribs. The pain comes round to the front, nearly to the site of the gall-bladder. No tenderness any-

whole. Abdominal muscles like boards.

Breathing difficult, almost entirely costal.

Lungs: nothing abnormal. I gave him a soap & water enema thinking he might be suffering from severe colic caused by the purgatives. Some dark stringy stuff was the result, like a calomel stool.

At 7 a.m. I gave him a very large enema which he retained 20 minutes; the result was similar.

Urine: clear, mucous deposit, acid, albumen in considerable amount, & hyaline casts. Pain obviously limited to right side of abdomen & shoots (patient's own remark) to tip of right shoulder. No jaundice. [There was no history pointing to gall-stones].

Temperature normal. Pulse 88. Resp. 24.

The diagnosis now was chiefly between the passage of a gall stone & perforated duodenal ulcer. I passed a stomach tube up the rectum; a mixture of oil & water would not flow in.

10.30 a.m. Patient agreed to see a surgeon. In much the same condition, &

groaning with pain

NOON. Sweat on brow. Pain as bad as ever — not altered by posture, but the act of changing from one position to another gives him excruciating pain. Pulse 104. Temp. 97.6. Resp. 25, short, difficult & irregular. Abdominal muscles more board-like than ever. Definite pain & tenderness now over an area, the size of an orange, situated between McBurney's point & the umbilicus. Abdomen moves all over during respiration, but very slightly, except when the pain becomes more severe than usual — then it is motionless & rigid. The pain is constant & is strictly limited to the right side of the abdomen. (The pain, at first high up under the ribs, moved steadily in a downward direction, until now when it was at the appendicular region). Morphia still withheld. Heat, mustard, & turpentine stupes were applied on the patient & friends asking that some local remedy should be tried; they did not ameliorate the pain. No dulness on percussion of abdomen (though I was certain from previous experience that there

was fluid present.

1 p.m. 17th Alexis Thomson saw the patient, who was lying propped up in bed. He was looking better, & was able to raise a smile, & talk. The abdomen was moving slightly, but not so much on right side as on left.

Abdominal muscles & chest wall on the right side were rigid. No special tenderness at McBurney's point.

Distinct tenderness below the right ribs, but not particularly localized.

Liver dulness, about $2\frac{1}{2}$ inches vertically.

Pulse, good volume, 100. Resp. 25.

17th Thomson said that the patient had not the appearance of a man seriously ill, but that he was certain he had a perforated ulcer of the duodenum. (It will be apparent that the consultation took place during the period of reaction).

3.15. Operation in nursing home.

I gave the anaesthetic (chloroform for the most part). He took it badly.

Oblique incision, two finger-breadths below & parallel with right costal margin. Thin, opaque, yellowish fluid

escaped at once. It contained flakes of lymph; it was not coloured with bile. There was no gas. It was very difficult to secure access to the duodenum on account of distension of the colon, prolapse of omentum & small intestine, & the chloroform difficulty. The liver was pulled up, the colon drawn inwards, & after swabbing out, the duodenum was finally reached. It showed a clean-cut perforation, of the size of a quill pen, with indurated, puckered surroundings over an area of half-a-crown, in the centre of the first part of the duodenum which was lying so that its "anterior" wall looked directly to the right, its posterior wall being applied to the right aspect of the bodies of the vertebrae. Although the perforation could be seen & felt, it was impossible to get at it to stitch it up, or even to fix omentum or other structure over it. Two rubber tubes were put in at the wound, another was brought out in the loin below the right kidney, & a glass tube was placed in the pouch of Douglas - the latter yielded a quantity of turbid yellow fluid. Saline was

then squirted through each set of tubes till it returned clear. The rest of the abdominal wall was cloud in layers. Enema of saline + brandy; patient reacted well to this.

Thursday. The dressings became soaked with green bile-stained fluid; this continued throughout the day. Patient is washing out his mouth - nothing swallowed. Saline + nutrients per rectum. Blood-stained fluid was sucked from the glass tube. Loin-tube is discharging a moderate quantity of greenish fluid.

Friday. General condition good. Breathing easier, abdominal wall moving. Skin around tubes at the wound is reddened over a wide area, + immediately around the opening is a greyish area of necrosis, sharply defined. One of the tubes was therefore removed, + a Sprengel's pump apparatus attached to the other. The lumbar + suprapubic tubes were removed, a strand of gauze being inserted in their place.

Saturday. General condition good. The Sprengel pump has drawn off a quantity of brown fluid, apparently mixed

with blood; reaction of this fluid = neutral. Altered blood is coming out along the side of the tube, & once there was a haemorrhage (from digestion with the gastric juice?). Skin around wound as before, perhaps a little better. The wound was left exposed - nurse to mop up juices as they escape. In evening the tube was taken out; it was blocked with dark clot; cleaned & returned. To have meal-tea by mouth. After each portion there was an increased gush of juices from the fistula. Temperature & pulse give no cause for anxiety.

Patient is looking well, though exhausted by the pain the digestive juices cause him at wound & on the skin. Has had no proper nourishment for 5 days, & very little for 2 days before that.

Sunday - Not so well. Very tired. Pulse 123. Brandy & champagne by the mouth. Morphine, because worn out by the pain at fistula. This gave him quiet day. Dozed much. Enjoyed what was given to him. It did not look as if he would outlive the night.

63
Monday. No sickness. Died peacefully 2 p.m.

There was a greyish tinge all over the skin.

P.M. No peritonitis. The perforation was very difficult to find. Upper half of second part of duodenum was somewhat stenosed & rigid with scar tissue.

The cause of death is not clear.

For the operative & post-operative notes on this case I am indebted to Mr. Alexis Thomson.

It is difficult to understand why the bile preferred to escape at the fistula instead of pursuing its natural course down the intestine. Was it the sphincter of Oddi that prevented this?

Also, I have found no other case ^{of duodenal ulcer} in which the digestive juices played such havoc with the skin.

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