

Hunger Pains

## HUNGER PAIN

Hunger pain may be defined as that which occurs at the end of the digestive tract and is relieved by the taking of food or drink.

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to describe the frequency of the condition in which it is found, especially with regard to the various organs, the associated symptoms, with the explanation of the nature of the pain and general appearance, with a list of the organs in which it is found.

It is noted that severe recurrent pain is frequent in the stomach and is relieved by the taking of food. The pain is relieved by the taking of food and is relieved by the taking of food.

The following is a list of the organs in which it is found: Stomach, Small Intestine, Large Intestine, Gallbladder, Pancreas, and Spleen.

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## Hunger Pain

Hunger pain may be defined as that which occurs at or about the height of digestion and is relieved by the taking of food or alkalis.

The object of the following is to discuss its frequency, the conditions in which it is found, especially with regard to duodenal ulcer, the associated symptoms, with the explanations which modern research and personal experience suggest, with brief remarks on the situation created by the assertion of Moynihan and his school that severe recurrent pain is duodenal ulcer and necessitates operation and by the equally emphatic statement of Hertz, Mansell Mouillan, Lane and others that he takes too much for granted.

The term was coined by Mayo Robson in 1906 (1) and familiarised by Moynihan in 1907 (2) and has been generally ~~accepted~~. It is not intended to be descriptive as if so it would be apt in and applicable only to some cases. The time

of origin, just when another meal was about due led to its adoption. Great controversy has raged round this symptom especially during 1909 and 1910 and many facts have since emerged. For the general practitioner and for myself, as a sufferer, it has been a most interesting and engrossing question, for the material is not scanty though unfortunately the solution of the more difficult problems associated cannot rest in his, the practitioners, hands.

We now recognise that in seeking for references to this condition both in modern and older literature that we must seek under various headings especially Duodenal Ulcer and it is noteworthy that under this latter we find that Abercrombie in connection with a specimen in the Royal College of Surgeons' Museum Edinburgh made the following significant statement "The leading peculiarity of disease of the duodenal part of the intestine so far as we are at present acquainted with it seems to be that the food is taken with relish and the first stage of digestion is not impeded; but the pain begins about the time the food is passing out of the stomach or from two to four hours after a meal!" Thus modern workers have been anticipated by over eighty years, (3) though Abercrombie would imply that food does not begin to leave the stomach almost at once. That it does so we now know to be a fact (4)

Up to five or six years ago references were most disappointing and though the symptomatology is well described yet the diagnoses and explanations given were far from satisfying. Previous to that time recourse had to be had to continental and American authors chiefly. For example Ewald(5) referring to the symptomatology says "The nervous nature of these disturbances is also shown by the fact that in some cases food moderates them" Riegel (6) more cautiously states that the only reason for the use of the term neurosis is that he is unable to give any other explanation while Einhorn is equally frank (7). Osler as late as 1905 appears to recognise the relation of hunger pain to gastric and duodenal ulceration but makes little attempt to differentiate and regards hyperchlorhydria, gastralgia etc. too much as clinical entities. Indeed in his own words "We physicians have been caught napping!"

#### Incidence.

Hunger pain in its widest interpretation is one of the very commonest of symptoms. In fact I am inclined to say that most people at some time or other have experienced it. It may not, it is true, amount to more than mere discomfort, it may not fulfill the conditions demanded by Moynihan for duodenal ulcer and it may be so slight as to practically escape observation. It may remain at this stage but most histories agree that this

is the early condition of affairs. If any medical men take the trouble to enquire casually as to the nature of indigestion from which are suffering his friends, the friends of his patients or the patients themselves who are being treated for something else, it is astonishing to realise how many cases never seek advice. In my experience women suffer largely from the milder and atypical forms but it is a fact that men more often require medical attention and that the severe recurrent form is commoner with them. No explanation is forthcoming but it is worthy of note that the usual answer from women whom one meets casually, for example in patients homes as to why they never need or seek advice is that they always take more food or "soda" and rarely allow the pain "to gather". One wonders whether in view of what we now consider the etiological factors of duodenal ulcer if herein we have not atleast a partial explanation of its greater frequency in men.

#### Symptomatology.

We may briefly quote from the discription given by Moynihan "After food is taken the patient is free from pain. At a time varying from one and a half to four hours there is a sense of uneasiness in the upper part of the ~~the~~ abdomen. A burning gnawing sensation develops and there is a bitter taste in the mouth with, it may be, eructations

of food and gas bitter and acid in taste. The pain which gradually increases may be relieved often considerably by belching and pressure!---"As all patients discern for themselves food relieves the pain"---"In several cases upon which I have operated the pain has been more severe than this in fact indistinguishable from a mild form of hepatic colic!"

---"The interval of relief after a meal varies chiefly with the character of the food taken the more substantial the meal the greater the interval. The appetite is generally good!"

---"It is not unusual for a patient to say 'I can take anything and I never vomit!' (8)

To the above one must add that pyrosis with an astringent sensation at the angle of the jaws often heralds an attack. A feeling of tightness of the pharynx is not infrequent compared by some to the "lump in the throat" and this raises a desire to eructate; water-brash may be present.

Smoking aggravates and male patients can often thus induce an attack while with myself and others there is strong distaste. The commonest period of suffering I have found to be after a meal like afternoon-tea.

In severe cases one or two o'clock in the morning is the worst time. In my own case if called on to get up it usually became intense. Pain may radiate to the back and as in many gastric conditions mental depression and disinclination

for work is not uncommon. Many after eating complain of a sense of fulness.

#### Conditions Leading to Hunger Pain.

Defective dentition in my experience stands easily first. No teeth are better than a few isolated teeth which are serving no useful purpose and dirty carious teeth are worse than either. It is an undoubted fact that the edentulous have very often efficient gastric mechanism and it can be noted that with removal of the offending teeth improvement sets in, long before artificial denture has been inserted.

Worry has long been associated with hunger pain.

Cold is often blamed. Patients state that they are usually worse in cold weather or if they have allowed themselves to become chilled.

Sedentary occupations or those requiring exercise,

Patients confined to bed or chamber,

Constipation,

Septic conditions, and of course

Burns,

all lead to the symptoms described.

With regard to those confined to bed I am gratified to find this corroborated by Hertz (9) who explains it as being due to the strict diet usually enforced in such

cases-----an explanation which is not altogether satisfactory (see under Theories and Explanations)

Morbid Conditions in which Hunger Pain is found.

These are chiefly duodenal ulcer, gastric ulcer, gall stones, and appendicitis. Leaving out of account that large class which we may simply call hypertonic or put under the head of preulcerative, functional or for the most part organic but without diagnostic features----according to one's views, there remain the various inflamm<sup>m</sup>atory conditions of the stomach. I have noted it in gastroptosis and gastric dilatation.

But hunger pain may be incidental in numerous diseases and may confuse. Such cases we find reported e.g. Moynihan (10) misled by the symptoms and haemorrhage operated for duodenal ulcer in a case of haemophilia; Lane (11) calls attention to duodenal kinking; Eve (12) reports cases with ulceration of the lower bowel, while we know that gastric crises and acute thoracic diseases may simulate the severer forms.

Duodenal Ulcer

Objectors to the idea that duodenal ulcer is common were fond of quoting statistics on hospital postmortem examinations e.g. Perry and Shaw (13) found only seventy cases in seventeen thousand postmortems, deaths being from all causes. But obviously the argument is not strong as duodenal ulcer has always been largely treated

outside and these figures are from 1826 to 1892 when even perforations were given up as hopeless and never sent in for operation.

A proof of this is that the same authorities state that from 1904 to 1910 there were more than half that number, that is thirty eight in only three thousand eight hundred cases.

Again there is the question---is ulcer always demonstrable? According to Bolton (14) the closest scrutiny is required, the mucous membrane being carefully washed, stretched out and pinned on a board before an ulcer can be said positively to be present or absent. It is conceivable that in superficial ulcers no white scarring will appear outside the bowel and when we consider the highly vascular membrane concerned very little trace may be left. I have removed warty growths from the buccal mucous membrane which in after years almost defied detection.

Mayo Robson (15) explains the apparent increase in the proportion of duodenal to gastric ulcers 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 ninety five per cent of all duodenal ulcers extend up to the pylorus or to within threequarters of an inch of it. As adhesions often obscured

the field the true facts were slow in coming forward.

In my own series of cases I have noted ten, nine since 1905 and one previously. Of these ten six were undoubted and four had occult bleeding. This list though small is sufficient to convince me that duodenal ulcer is not an uncommon disease. But there is no need to labour the point as the number of operations performed by modern surgeons speaks for itself.

Duodenal ulcer is first mentioned by Travers (16) in 1817. In 1830 Abercrombie published five cases and made the statement already recorded. The British and Foreign Medical-Chirurgical Review in 1864 gives a list of all recorded cases. In 1887 Bucquoy (17) suggested making the diagnosis alone from symptoms. In 1891 and 1894 appeared the theses of Oppenheimer and Collin respectively the latter of whom is mentioned by Moynihan as giving valuable information regarding the **signs** of duodenal ulcer---usually in the first part (18) In 1894 Dean records the first case of perforated duodenal ulcer operated on (19). In 1900 Moynihan's first case was operated on and he in 1901 reported seven cases only in one of which was a positive diagnosis made previous to the operation. Since then hundreds of cases have been operated on simply on the anamnesis alone, the diagnosis however not always being confirmed but sufficiently often to give Moynihan the credit for making a great advance in our medical knowledge.

But it should be said that as is the case with most special-ists there is too much of the fixed idea, a loss of perspective, as it were, so that many have taken on themselves to show that even the study of living pathology may on occasion lead to erroneous conclusions.

### Symptoms of Duodenal Ulcer.

The subjective symptoms may be taken to have been detailed previously. (page 4). But regard has to be taken of the recurrent nature of the attacks. That attacks recur in duodenal ulcer is generally admitted but in three of the ten cases of my own two were practically never free from the symptoms and one was acute having never suffered before. In a total of twenty two other cases of doubtful duodenal ulcer seven were practically never free from attacks and three had intervals of relief only for a few weeks at a time.

On the whole therefore recurrent pain would in my experience be said to be a prominent feature.

### Melaena.

This is perhaps the chief diagnostic point that practitioners rely on to distinguish duodenal ulcer. The search

for occult blood is as a rule disappointing and those who state that they find haemorrhage in a hundred per cent of cases are gifted with remarkable perseverance. Probably the best time to seek would be after a severe attack of pain but in my own case and others I was unable to confirm this.

#### Reliability of Melaena.

It would be observed that in dealing with the frequency of duodenal ulcer I stated that in ten cases six were undoubted and four had melaena.

The reason for so putting it is that doubt may arise as to even this symptom being diagnostic and some state that even the greatest care will not exclude all sources of fallacy, e.g. we noted that Moynihan was misled by bleeding from the bowel in a case of haemophilia.

We may have bleeding from the nose, gums, or occasionally injury in passing the stomach tube; we may have ulceration of the bowel quite apart from duodenal ulcer and these may be simple, tubercular, syphilitic or malignant; we may have haemorrhoids, fistula, fissure, or polypi; and we may have acute gastritis supervening. Parasites may also be a cause. Lastly those who have seen the gastric lining in life

with its marked vascular appearance are inclined to credit that there is the condition of gastrotaxis and though this might lead more probably to haematemesis there is nothing to prevent the bleeding passing into the bowel and evincing itself as melaena.

A point I observed in two cases is deserving of notice. A stout plethoric woman recovering from erysipelas complained of pain about three hours after eating. She was relieved by food and alkalis. This lasted for four or five days when she seemed to turn restless with a sense of oppression and headache. This was followed by a severe flow of haemorrhage from the bowel----in fact a blood diarrhoea which fortunately was got under control. But though weakened she experienced great relief. A second case was in a man also stout and plethoric who had allowed a simple ulcer to get into a bad state. After being three weeks in bed he also complained of hunger pain symptoms and also became oppressed and ill at ease. Recollecting the former case I requested him to keep his motions and sure enough there was bleeding. Both were constipated but had no sign of fissure, haemorrhoids or any other source of haemorrhage that I could discover.

#### Hyperacidity.

That this is common Moynihan denies (20) but Hertz

and others are agreed that it is the rule (21).

In a series of twelve cases up to 1908 I found ten had over .2 % of hydrochloric acid or expressing the total acidity, over 50. In a further series of twenty in five cases examined only one showed an acidity over the normal. But since 1908 I have strictly limited the use of the stomach tube to cases with anomalous symptoms, e.g. one was a case of dilated stomach and therefore the results were not unexpected.

Personally I have no doubt that hyperacidity is the more frequent.

In books on diseases of the stomach of a few years ago such as Riegel's, Ewald's, Hemmeter's, Einhorn's, Reid's, or in fact any book, one can see how emphatic they are on this point in their chapters on so called hyperchlorhydria and hyperchlorhydria is distinguished by having in a marked degree the symptoms concerning which we are dealing.

More recently Craven Moore (22) and Willcox (23) two excellent English authorities have insisted on the frequency of hyperacidity in gastric and duodenal ulcers.

## Physical Examination.

### Tenderness on Pressure.

In contradistinction to the experience of others I have found this to be chiefly central, in fact as in most gastric conditions.

It occupies an area of two or three inches just below the xiphisternum and may extend more if anything to the right side. It is much worse some two or three hours after a meal when the pain is present.

I have never made out any dorsal points of tenderness.

A strictly localised tender spot may be present just over the right rectus accompanied by some rigidity. This is explained as being due to peritoneal extension.

### Skin-pinching.

This elicits tenderness and if subcutaneous tissue has of necessity to be included between the fingers, the tenderness may be extreme. In my own case the line of marked tenderness began under the xiphisternum and extended, slightly arching towards the right to a little above the umbilicus.

## Differential Diagnosis.

## Gastric Ulcer

Confusion only arises in gastric ulcer with late pain. Dawson (24) has shown that late pain may occur when an ulcer is even on the cardiac portion and that a pyloric ulcer may have immediate pain. In 1906 Mackenzie (25) first drew attention to the fact that ulcers of the cardia had pain just below the xiphisternum and pyloric ulcers had the pain nearer the umbilicus. Hertz (26) without mentioning Mackenzie with regard to this point confirms the fact and adds that duodenal ulcer as well as cholelithiasis and chronic appendicitis have the pain in the latter situation. He goes on to say that it may be occasionally to the left in gastric ulcer and occasionally to the right in duodenal. In contrasting gastric and duodenal ulceration the chief distinctions are;-

gastric ulcer is chiefly in females and anaemia is common; vomiting is more frequent; haematemesis is more prominent than melaena; the pain as a rule is not so late; the recurrent history is not so usually given and the desire to eat is not so great.

But cases are met with where from the symptoms it is impossible to say whether we are dealing with gastric

ulcer, duodenal ulcer, or any of the conditions giving rise to hunger pain symptoms, and confusion is intensified from the fact that as we now know ulceration may both be in the duodenum and in the stomach Moynihan (27) gave this as existing as high as 40 % in a series of cases.

### Gall Stones.

It is here that difficulty in diagnosis is perhaps greatest. The chief distinctions are firstly the character of the pain. In gall stones it may be unendurable. If one may so describe it, it is the handle of a knife that acts in duodenal ulcer while it is the point in gall stones. Again, the tender point is as a rule more distinctly to the right and pain may be complained of in the right shoulder blade. Vomiting is more frequent. Relief may be for a time obtained by alkalis or oil, and then these quite fail to act and the pain becomes intense. It is interesting to note that Camidge (28) in this connection says that true gall stone colic is rare. Shivering turns with sweats are also more common.

One can again be always on the lookout for the early signs of jaundice and the usual train of grosser symptoms.

### Appendicitis.

This should always be considered in examination of patients. It is frequently observed that the conditions arising from ~~chronic~~ chronic appendicitis may give rise to symptoms resembling those of duodenal ulcer. There may be some tenderness and rigidity in the right iliac fossa or there may be nothing to go on but the previous history of an acute or subacute attack.

On the other hand we well know acute conditions of the duodenum and thoracic viscera give a remarkable mimicry of acute appendicitis.

### Hyperchlorhydria.

This is the large class of case where in the absence of proof we can only use a term such as this. A better way perhaps would be to call them simply hypertonic cases or we may regard them as preulcerative. Whether a primary secretory neurosis is possible is a matter of dispute, but certainly operation shows that there is sometimes no sign of ulceration or organic mischief even when all the typical symptoms were present.

Hutchison (29) is strong in this point and Moynihan in his more recent writings is careful to say that

an ulcer must be demonstrable. Lauder Brunton (30) goes so far as to base his diagnosis of duodenal ulcer on the relief obtained from the mixed carbonates of soda lime and magnesia. Childe (31) states that operating practically on the anamnesis alone his error was only about five or six per cent.

Therefore the great possibility of the existence of duodenal ulcer must always be kept in view.

Mention must be made here of nervous dyspepsia which may be very puzzling and the intelligence of the practitioner will be tested in sifting various vagaries of the neurasthenic and hypochondriac.

#### Dilatation of the Stomach.

This may give rise to symptoms undoubtedly, in my experience. The double powder method is the best aid in diagnosis. The symptoms are not as a rule typical e.g. food not relieving so much and the alkali giving only partial relief. These cases may show hypoacidity and the alkalis may be doing harm by lessening the antiseptic power of the hydrochloric acid or it may be by diminution of motility. The views of our elders seem to be borne out when they condemn the indiscriminate use of alkalis for many of these chronic cases, chiefly women, are confirmed "soda" takers.

### Gastroptosis.

I have seen women give in this condition a history which might confuse. A general visceroptosis may be found. Inflation, especially noting the position of the lesser curvature, gives the required information. It is with this class of case that I have seen marked relief obtained from the use of a supporting belt.

Loening (32) referring to this condition says that hypermotility is the rule, against the usual belief, and as we shall see hypermotility leads to hypertension and pain.

As already mentioned there are numerous conditions where some uncertainty may arise. Examination by the approved methods and time will help to clear up most. In addition to the cases cited viz:- haemophilia (Moynihan), kinking (Lane), tubercular ulceration (Eve), we can have the hunger pain symptoms as we shall see later in numerous abdominal conditions e.g. pancreatitis, cirrhosis of the liver, malignant ulceration, splenic anaemia, etc., while the gastric crisis of locomotor ataxy may simulate acute attacks. Acute thoracic conditions may also for the moment deceive,

## Theories and Explanations.

It is under this heading that we come to what is most puzzling and interesting, puzzling because we are dealing with what seems on the surface elementary facts capable of easy explanation and interesting for the very reason that none of the explanations really explain. Mere speculation would be futile but one can show considerable grounds on which solutions may be based.

Hertz in his book on Sensibility of the Alimentary Canal treats of gastric pain and settles once and for all that hunger pain is due to hypertension. Mackenzie (33) in a measure anticipated him in 1906 when he speaks of it as being due to a viscerosensory reflex, but when this has been definitely settled controversy now arises as to the part played by hydrochloric acid and as to how food and alkalis relieve.

Hertz in the above chapter explains that excess of acid causes excess of peristalsis and this rapid peristalsis causes the painful hypertension. He quotes Edelman as his authority for stating that the peristalsis is proportional to the amount of acid. But Bolton (34) states that hydrochloric acid does not cause an increase. In any case it is well known that excess of hydrochloric acid cannot cause pain and it may be drunk in large quantity even when an ulcer is present and

no pain arise. As we have seen pain exists when there is no excess of acid and again people in perfect health have been found with hyperacidity.

Many fall into the temptation of offering the solution of the difficulties by assuming that excess of free hydrochloric acid is the direct and active-agency in the cause of symptoms, an assumption that renders explanation easy.

Their views would seem to receive strong support in duodenal ulcer from the fact that the inhibition of the relaxation of the pylorus is protective as shown by Cannon and Murphy (35) and that it closes when acid enters the duodenum as shown by Pawlow (36).

But Hertz shows that an ulcer is quite insensitive to hydrochloric acid.

Let us take the case of insufficient chewed food. The pylorus rejects it, the pyloric portion of the stomach gets into a state of irritation which leads to increased peristalsis which in its turn leads to the hypertonus and pain. A hyperaemic condition naturally follows which may extend into the cardia, and so the increased flow of hydrochloric acid takes place. That such hyperaemia may be assumed is strongly supported by the fact that proliferation and hypertrophy of the oxyntic cells has been repeatedly observed by Hemmeter (37).

The increased secretion is therefore secondary to increased peristalsis.

This increased secretion leads to early fluidity of the gastric contents and hence in spite of protective cramp and spasm the hypertonic stomach is usually rapidly emptied, a fact particularly emphasised by Riegel (38), Sahli (39) and others.

#### How Food and Alkalis Relieve.

Here again if we could take hydrochloric acid as the scapegoat the explanation would be that the food combines with the excess of free acid and so relief is obtained. In the case of duodenal ulcer when food is taken there would be a closure of the pylorus and so the ulcer is protected. The acid hypothesis receives further support or would appear to by the fact that the proteids give greater relief than the carbohydrates the former being strongly acid-combining. But a more important factor is that the carbohydrates leave the stomach rapidly, the rapid motility leading to earlier tension and pain. Further support is lent to the acid hypothesis from the time relation in gastric, pyloric, and duodenal ulcers. The first comes as a rule early the pyloric later and the duodenal last and Hertz explains this by the fact that acid bathes the cardia

early and gradually gets incorporated into the food mass in the pyloric portion so that in the latter situation while there is at first actually alkaline material, it takes some time before the acidity becomes enough to act as an irritant more so since "The gastric juice is greatly diluted by the large quantity of food with which it is mixed" Hertz thus believes that an ulcer can be irritated and though not sensitive to acid can give rise by this irritation to the conditions leading to pain.

But the first objection to this is that the time relation is by no means a constant factor. Dawson as already noted has conclusively proved this, but the greatest objection is furnished by radiographers. In the case of duodenal ulcer when there is considerable material in the stomach and when more food is taken the x rays show that the top portion does not mix intimately with the food already in the stomach or reach the pylorus for some considerable time and yet relief is obtained almost at once. Obviously the acid at the pylorus cannot be effected and as food is passing all the time into the duodenum (except during actual spasm) it is difficult to see what part the neutralization of the acid plays.

The only explanation that appears to cover all is that there is a disturbance of the existing hypertonus when more food is taken, a diversion of the nervous stimulus, a rearrangement of the muscular fibres, and relaxation occurs.

On the question of relief by alkalis Hertz after reaffirming that free hydrochloric acid does not of itself cause pain on membrane or ulcer says "But the relief afforded by alkalis suggests that the pain is in some way connected with the presence of free hydrochloric acid. I believe that this apparent contradiction is due to the fact that the stimulating effect of the acid on peristalsis and the abnormally prolonged inhibition of pyloric relaxation, which occurs when the hyper-acid chyme reaches the duodenum can only cause pain when food is present in the stomach as no rise in internal pressure can occur when there is nothing in the stomach upon which the muscular coat can contract!"

Disregarding the examination of gastric contents after a test-meal I waited until the stomach should be about empty and then examined when the pain was still present. No free hydrochloric acid could be found. I verified this in my own case and in another male patient.

So that relief by alkalis is not necessarily on account of free hydrochloric acid.

In the next sentence Hertz suggests that the stimulating effect of the acid on peristalsis and the fact that the pylorus contracts to protect the duodenum from the free acid only causes pain when the stomach contains food.

But emptying the stomach does not always relieve.

In the two cases above the pain continued though the stomach was emptied and I have been told by other patients that vomiting does not always bring immediate relief. The uneasiness is not so diffuse but the gnawing continues. It must be noted that the stomach tube is not to be inserted into the stomach for this purpose as it will of itself at this stage sometimes relieve.

Emesis had to be obtained by straining after touching the pharynx or partly inserting the tube. Hertz perhaps founds his opinion on the experiment of Sick<sup>(40)</sup> who showed that no rise of pressure could be noted on an empty stomach. But instrumental interference is apt to vitiate the result and with pressure recording instruments a stomach is not empty.

It is pointed out that when alkalis relieve in a stomach with a fair amount of food that the relief obtained can scarcely be due to neutralisation of acid in the duodenum as the alkali has had no time to reach the pylorus. Further it is observed that the pain may be worse after taking the alkali until gas comes away and not before even although it would seem that the acidity must have been reduced previously. Lastly pain sometimes continues after the stomach is emptied.

This leads us to the first hypothesis that the alkali acts by distending the walls and then allowing them to

contract by release of gas, this process having much the same effect as in the case of taking more food.

Mansell Mouillin (41) in a recent article expresses somewhat similar views.

But in my opinion the better way is to look on the duodenum as the great factor in causing the hypertension of the pylorus and pyloric vestibule. The fault may be gastric or it may be intestinal, in fact anything that upsets the balance between the gastric juice and the alkaline juices of the bowel. Proceeding on these lines we see that hyperacidity need not necessarily be present. The ordinary acid chyme by entering the duodenum too rapidly may set up the hypertension. But of course the greater the acidity the more likely will actual spasm take place.

Barclay (42) in opposition to the above view demonstrates by x rays that a bismuth fluid will flow round food in the stomach to reach the pylorus, by way of the rugae, he thinks. If this is so then the alkali reaches the duodenum, reduces the acidity and so causes relaxation of the pylorus and pyloric portion of the stomach, with relief of pain.

In the two cases I referred to I observed that the gnawing did not cease, but immediately on taking an alkali relief was obtained; that is to say although the stomach was for

all practical purposes empty, the condition of the duodenum was such that it required reduction in acidity before relaxation could take place.

Sick (43) has given charcoal after a gruel and waiting fifteen and even twenty five minutes has found no admixture in the pyloric portion. This of course is strong proof that more food relieves by some other means than neutralisation of acid. In the case of alkalis matters are different. These even in powder form will soon permeate the gastric contents though relief is noticeably longer in coming than with an alkaline solution. Even water relieves and O. Cohnheim (44) states that it may pass through a stomach filled with acid contents and reach the duodenum neutral. Add some alkali to the water and relief is more complete and more prompt.

Reference should be made to the peculiar suddenness, with which all symptoms will depart. This a fairly common story and shows in itself that acid is unlikely to be an important factor in the cause of symptoms.

The marvellous effect of oil in some cases can only proceed in a slight degree from its acid-repressing power.

Coming now to the more direct aetiology of duodenal ulcer we may repeat the expression that it is closely wrapped up in the upsetting of the balance between the acid gastric juice and the alkaline juices in the duodenum. The membrane of the latter is readily digestible as we know. Katzenschein (45) infolded the ~~bowel~~ into the stomach and showed how feeble its resistance was to the gastric juice which, however little it may lead to pain is closely connected with the formation of ulcer.

Other factors seem to play their part of which sepsis may be the most important, though the necessity for such additional factors is hardly so apparent to explain the aetiology as in the case of gastric ulcer. In the latter however the work of Bolton (46) shows that the hydrochloric acid is a decided factor in ulcer formation.

We have already mentioned defective dentition, cold, worry, sedentary occupations, constipation, sepsis, and confinement to bed as all leading to hunger pain symptoms. Defective dentition is usually explained by the improperly chewed food acting as an irritant and leading to the train of events as already described so that the gastric contents often with hypersecretion is poured in too frequent and too forcible jets onto the susceptible duodenal membrane, usually

at the first part. One must here emphasise the fact that the removal of carious teeth even denuding the mouth of effective powers of mastication is nearly always marked by early improvement. It has been stated that the hydrochloric acid can kill oral organisms, but bacteriology shows that even delicate organism can survive and flourish in the stomach and we are more probably dealing with secretions, internal or external, of bacteria, that is, toxins.

Bacteriological work has been done in America by Türk (47) who by feeding with the coli bacillus and the coli toxin brought on gastric and duodenal ulcers. He argues that a cytotoxin is produced which causes lysis of the gastric and duodenal cells, in short autolysis. Bolton (48) of this country has done considerable work in connection with gastrotoxin produced by injecting gastric cells into an animal of a different species and so forming a cytotoxin or cytotoxin for the species from which the gastric cells were taken. Since he shows that this toxin is not specific for the cells of the gastric wall but is a general protoplasmic poison it is evident that any hope that has been entertained of immunizing by means of a serum must be abandoned.

Cold in my experience leads to recurrences and exacerbations rarely to primary attacks.

Worry as causing attacks and leading to ulceration is explained by Hertz (49) as due to Bolting of food but anequally reasonable explanation is afforded by the experiments of Cannon (50) on cats, When in anger or excitement gastro-intestinal movements almost ceased. We have thus a condition of affairs which we would expect to find arising in those with sedentary occupations, with constipation, or those confined to bed. That a reduction of motility may aggravate symptoms is evident when we consider that the severe pain of early morning is explained in this way. In this connection Arbuthnot Lane (51) writes as follows, referring to intestinal stasis----"The strain exerted by the overloaded small intestine blocks the duodenum at its termination, obstructs the escape of its contents, dilates and expands this portion of the small gut and especially the first portion which yields much more readily to the tension since it lies free in its peritoneal covering while the second and third part escape because they are buried firmly behind the peritoneum. These tension changes aided by chemical and bacteriological developments in the contents of the intestine and by depreciation of the vitality of the tissue by the toxic material present in excess in the circulation result in the engorgement of the mucous membrane of the first part of the duodenum, later, in

its abrasion and finally in ulceration and perforation. The same causes produce infection of the biliary and pancreatic ducts-----" "The stagnation of material in the duodenum obstructs the flow from the stomach and an accumulation takes place in that organ in which bacteriological and chemical changes arise. The strain exerted upon the lesser curvature by the gastric contents increased materially by that transmitted through the great omentum from a loaded transverse colon aided by the chemical changes in the static gastric contents and the condition of autointoxication results in an engorgement, abrasion and ulceration or in cancerous or other infections of its mucous lining!"

Later on he would appear to give to intestinal stasis a prominence in the aetiology which experience scarcely warrants.

Before leaving this section one is forced to confess that there is yet much to be learned especially with regard to the exact mechanism of the stomach, the part played by hydrochloric acid and the other factors in the cause of hunger pain. The desire for further information is stimulated when the seemingly well assured fact, that heartburn was due to acid in some form, is entirely erroneous and that it is probably due to alcohols as shown by Hertz in his book.

## Brief Remarks on Methods.

The stomach tube is essential but a too frequent use is to be deprecated. It appears somewhat barbarous to the patient and it is now being recognised that the information derived is less valuable than formerly supposed. Personally I now restrict its use to anomalous or doubtful cases. For estimation of acidity an ordinary hundred c.c. buret is used, normal soda solution diluted to decinormal with phenothalein as the indicator. One would suppose that methyl-orange would be much more accurate as an indicator since the latter unlike phenothalein is unaffected by organic acids but the error is infinitesimal and the end reaction is very distinct. For strict accuracy again with regard to the soda solution, it should be obtained from an analytical chemist as standardisation is necessary or preferable against oxalic acid. The test breakfast may be a moderate slice of toast and a large cup of tea. The test for free acid may be by congo-red paper confirmed by methyl-violet, the test for any mineral acid. I have also done the more accurate Gunzberg reaction but have not yet tried dimethyl-amido-azobenzol which is now regarded as the best.

The double powder method for ascertaining dilatation is very valuable, that is the giving of equal weights of tartaric

acid and sodium bicarbonate. It is to be noted that the older loose style of giving a teaspoonful of each leads to unsatisfactory results as the tartaric acid requires two parts by volume of the bicarbonate. From the formula it will be found that a drachm of each by weight will give roughly one litre of carbonic acid gas, or eleven hundred c.c. when raised to body temperature, 37°C. As the capacity of the stomach is from over a half to one litre this may be sufficient but if good definition is not obtained then one and a half drachms may be used. The objections are, some cannot retain the gas long enough and some vomit.

The saliuric reaction for ascertaining motility is indefinite but easy. On an empty stomach a gramme or fifteen grains of salol may be given in milk and in a typical case the reaction in the urine may appear under ten minutes. Sahli (52) suggests that the better way would be to see if all trace has gone after twenty seven hours indicating that there is no stasis. At least it is a good "show" method in the surgery as the patient is impressed by the deep violet colour in the urine when the ferric perchloride is added, more so if a control specimen has been passed and tested just previously by way of contrast.

There is the method of giving sips of water till a tumblerful has been drunk. Dulness, if any, is only obtained in dilatation with atony.

There is a method mentioned by Fuld (53) of giving an alkali and auscultating, listening for the sound of "snow being crushed," the evolution of gas. He states that it is only got distinctly when there is free hydrochloric acid, which if true would be helpful, especially towards the end of digestion, but I have obtained good evolution of gas from vomitus where no free hydrochloric acid was indicated.

The test for blood I have used is Weber's modification of the turpentine-guiae, as given by Sahli (54).

For motility, obstruction and noting the digestive powers a meal at night as mixed and complete as the patient can stand, with the addition of raisins, currents, or similar dried fruit with skins, and withdrawn in the morning for examination is very valuable.

#### Brief Remarks on Treatment.

One cannot dogmatise on matters of treatment. We may be unwittingly treating some occult condition.

Sodium bicarbonate is as good as any alkali. It seems foolish to add ginger and peppermint oil as is done in so many tablet preparations since these stimulate acid secretion, Heavy magnesia in spite of its high neutralizing power does not act so well. Liquor potass I have found of service.

I prescribe tincture of belladonna but cannot say whether it is helpful or otherwise.

Oil e.g. olive oil combined with malt or alone often helps remarkably and ofcourse has to be kept up. It may be an absolute failure and cause bad vomiting. It seems to act best in bad cases with high hypertension especially where there is constipation., and worst where there is some tendency to dilátation. An emulsion of petroleum may be used with success in its stead.

Antilytic serum I have used in one case with no success.

Morphia I rarely use but have had recourse to it in cases of acute spasm where the pylorus cramps in protect-  
-ing against ill-chewed indigestible food. Such cases are very common in the elderly edentalous.

#### Diet.

Unquestionably carbohydrates should be restrict-  
-ed even sometimes to replacing sugar by saxin or saccharin. Nothing gritty or with coarse residue should be allowed, meat to be preferably in the form obtained by prolonged scraping with a knife, or pounded, no soup or beef juices, salt to be limited and only milk, eggs, jellies, and similar articles of food to be allowed.

Rest is essential in bad cases but the average patient will not go to bed unless obliged to and in my practice it happens that the worse cases have been working men who could not afford to lie up.

Massage and gentle stroking under the left ribs I have tried tentatively in mild cases but cannot speak definitely as to their value. In typical cases it would appear to be negatived.

Abdominal belts I have mentioned already in regard to gastroptosis and have found them of great use especially where the abdomen tended to be pendulous.

#### Remarks on Results.

This is pre-eminently a question for the practitioner. Although something is to be said in seven years practice yet one feels that a longer time is necessary as cases have long intervals of relief.

Taking the ten cases referred to as undoubted duodenal ulcer two are dead. Of the two cases of visible haemorrhage the woman referred to, interviewed in April of this year stated that she "Never had anything like it before or since."

Her case was acute following an erysipelas of the leg and this gives her four years of freedom from symptoms. The other case that of the man with the bad ulcer of the leg is practically well but he says he now and again has the "nasty gnawing" which is easily relieved. That was three and a half years ago.

The next two cases were burns one from petrol and the other chemical, myself. The former has had five years complete freedom and I have had attacks in 1901, 1905, 1910, and slightly in the spring of this year.

The next two I ascribed to bad teeth. One is a painter and questioned last March said he suffered "now and again but nothing to speak of". The other was a propagator who has gone abroad. The next case is that of a field-drainer and I have not seen him for six months. He said he was no better and required to take olive oil continuously. He has suffered for years. Another case is that of a farm servant whom I last saw in April of this year. He informed me that a bad attack had recommenced after six months freedom. Both these cases are of the typical recurrent type, are men of good physique, with teeth in perfect condition, with no vices. In fact nothing can be found to account for their condition and yet they are amongst my worst cases. Of course rest has been out of the question and suggestion of operation laughed at.

It is to be noted that for the purposes of this paper the cases have had to be looked up. Of the last two cases which showed occult haemorrhage one is quite well since over a year and one states he still carries "soda!"

I have called on one or two cases with the typical symptoms but diagnosis uncertain. An elderly man, aged 69 who suffered for two years previous to 1909 tells me he has now had three years complete freedom. Asked if the oil was the cure said he did not know but "the trouble lasted off and on three months" after he started the oil, suddenly left him after that and never returned. A retired farmer tells me he suffers every few weeks but he is careless in his diet and habits. Another patient of no occupation with a typical recurrent history states that he finds the best cure is to go to a first-class hotel and "give his stomach a d---d good fright!" He seems to get relief it is true but not for long.

Where one can rely on getting the patient to do exactly as told the relief of symptoms is usually fairly rapid but they soon fall into their former habits and recurrence takes place. I have no doubt if care was observed this troublesome feature would not arise so frequently but it happens now and again that one longs to have gastro-jejunostomy performed. In my own case I could fairly well assign a

cause for recurrence.

The only case of operation I know was not fortunate. It was that of a man of 50, a retired "Scotch draper," an early patient of mine who had had several gastric analyses and who told me that he had excess of acid. I verified this and found a total acidity of over ninety. He left the district and returned to England. Meeting him two years ago he told me he was persuaded to have a gastro-jejeunostomy performed with the idea that duodenal ulcer was the cause of his symptoms. No ulcer was found and in his own words he was neither better or worse.

### Types of Cases.

#### Perforation.

J.N. joiner, aged 35 had the typical symptoms relieved by food and alkalis. Two examinations in his case gave high acid values, over eighty. There was no history of blood and no melaena was found. Some three months after I had seen him he sent for me on Nov. 15 1906 as he had an acute attack of pain unlike anything he had had before. Rigidity and tenderness were not limited to a particular part. Hot fomentations failed to relieve and I was obliged to give morphia, doubtless a mistake.

There was no collapse, pulse and temperature were normal. When the second day he was easier. On the third day the pulse was rising, the friends had given him a large dose of salts, and rigidity was marked all down the right side. Appendicitis was simulated. The late Dr. Ramsay, Carlisle, was sent for and examination of the right lung revealed at the base diminished breathing and slight dulness. He had pain in breathing.

Diaphragmatic pleurisy was the diagnosis and my suggestion of perforated duodenal ulcer did not meet with approval. He continued to get worse and then pain suddenly left him. Tinkling was heard at the base of the lung. I sent him into Carlisle Infirmary. Foul pus and gas was evacuated from the pleural cavity. He did not survive. Dr. Ramsay was good enough to write, while deploring the result, to congratulate me on my diagnosis. No postmortem was allowed but the case was undoubtedly one of perforating duodenal ulcer.

#### Duodenal Ulcer from Sepsis.

Mrs K. aged 57, a stout woman took erysipelas of the right leg. It was a large patch extending from the tibial tuberosity downwards for about two thirds of the leg. Her condition was bad and at first, <sup>she</sup> lost ground rapidly. At the end of three weeks when beginning to feel much better she

complained of indigestion, of a boring pain coming on just before she was about to partake of another meal. This made her call for food earlier and oftener. At my suggestion she tried an alkali and of course relief was prompt. She informed me that she never had had anything like those symptoms before. Olive oil was useless and only made her very sick. There was tenderness all over the epigastrium. On the third day after my attention had been drawn to the indigestion she informed me that she was feeling miserable, had a severe headache and felt a sense of fullness in the abdomen. The same evening she began to pass blood per rectum which continued to an alarming extent till the next afternoon when it gradually ceased. Local tenderness was noted just under the ribs on the right rectus. But the haemorrhage relieved her headache and oppression and from that day she never looked back. Further the erysipelas cured a thick corrugated patch of very itchy eczema which had defied treatment for years. This woman suffered at this time from constipation but there was no sign of any anal cause of the haemorrhage which as stated before came as a "blood diarrhoea!"

Gastric Ulcer ---probably prepyloric---dilatation  
---tetany.

Mrs O. aged 35 consulted me early in 1907 for pain coming on about two hours or so after eating, directly under the breast bone. Eating eased her and she was in the habit of taking baking soda. She did not vomit and was not sure about the colour of her motions. She rather thought they had been decidedly blackish but this might have been due to bottles. Her appetite was poor. She was very constipated. I tested for acidity and found it slightly over 60. She could not at this time retain the gas from the double powder very well but there was no dilatation as far as I could see. Under treatment she improved. In May she sent for me for copious vomiting, the second time within two weeks. Inflation showed decided enlargement. Again she improved under treatment, the greater curvature of the stomach rising definitely above the umbilicus. Some four months after she became careless in her diet, going back to her strong stewed tea etc., and I found she had vomited a very large quantity of dark blood. She was sent into Carlisle Infirmary where they opened her at the sigmoid flexure under the impression that there was something accounting for the constipation. Nothing was found. She was dieted and sent home

with instructions to return in a month for gastro-jejunostomy. Dieting had so far improved her that she refused to go. Again she turned worse and went to Edinburgh Infirmary of her own accord, but as she had allowed herself to become very emaciated nothing was done as malignant obstruction was feared. That was four years ago. In December of last year I was called to her and found her suffering from a sudden attack of tetany of a most painful type, both hands and feet especially of the right side being affected. The agony was acute and left her but slowly. The left foot was very slightly affected and was soon all right. Next the right foot and then the left hand became easy but three weeks after, the left hand was still somewhat stiff and sore. As for the right hand at that time the fingers were still claw-like and the hand useless. Calling on her in April she said the fingers of the right hand were still stiff and that "it must have been a stroke!" She now suffers according as she is careful or not. She could eat heartily but dare not.

#### Gall-stones.

Mrs J. aged 56 had suffered for years from typical hunger pain symptoms. Periods of relief varied from six, nine, eighteen months to two years. Constipation was marked and vomiting not uncommon. Very strict dieting and

and rest with alkalis usually brought rapid improvement, but periodically I was obliged to give morphia. No distinctive symptoms could ever be made out, but last August a yellow tinge was noticable on the conjunctiva. The jaundice became distinct the urine porter coloured, and the faeces slatey grey. The severe attacks came at intervals of one, two, then one and lastly three weeks, Sometimes an alkali gave relief and sometimes recourse had to be had to morphia. The stools were carefully sifted and some three months after a stone was found of which the centre was hard very small ragged and surrounded by soft friable deposit. She is now well though the skin is still stained and states she has never felt better in all her life.

#### Appendicitis.

The only case I have observed, probably because attention has only been called to the connection with hunger pain within the last two years or so, is that of a young man R.U. aged 24 who sent for me with a pain in the right iliac fossa. It was a mild case of appendicitis and on the subsidence of his symptoms I sent him on to Carlisle Infirmary where he would be about the last case operated on by the late Dr. Ramsay. This was in the beginning of this year. On enquiring casually he told me that he had occasionally suffered

from indigestion, and once or twice had severe cramp. He was a compositor and was told by his comrades that he had compositor's colic. From his description however there was no doubt that he had had hunger pain and that the cramp was pyloric spasm. He tells me he has now no indigestion.

#### Duodenal Ulcer from Burn.

I have seen two cases but perhaps it will be best to give a rapid sketch of my own. It represents more a recrudescence brought on by a chemical burn. My first attack began in the spring of 1901 when studying for the "final".

There was undoubtedly some carious teeth. No advice was sought but I soon discovered that by carrying biscuits and a small bag of sodium bicarbonate that I could obtain relief. There was some fullness after eating but I recollect the relief was a pleasure after a meal. Water-brash often heralded an attack. The pain was just under the xiphisternum and sometimes proceeded into fierce spasm. I well remember the sweat breaking over me as if the gut were being crushed in some powerful hand. It was just bearable and no more. I had recourse to a milk diet with no effect. In July I visited the dentist and going on a voyage symptoms completely left me. The attack would last about three months. In 1905

I received a severe burn all over the face and eyes. After three weeks illness the old symptoms manifested themselves. It was then my interest was aroused in this question. The attack was long in leaving me but was never so severe as before and spasm never intervened. In 1910 I had another attack following a course of decidedly indiscreet dieting. And in the spring of this year a slight return was promptly dispersed by careful treatment. The chief features were----the pain was diffuse between the xiphi-sternum and the umbilicus; skin-pinching showed very marked sensitiveness in the area noted, but extending more to the right and both this and tenderness on pressure gave rise to a much more marked condition during the height of an attack; examination of the faeces showed on two occasions a reaction for occult blood; no definite spot of tenderness existed; the pain was worse about seven o'clock in the evening except on one or two occasions when it came on in the early morning; having to answer to a night-call intensified it; smoking aggravated and was quite impossible except after a meal; indeed a foolish attempt to smoke at any other time induced an attack earlier; in the third attack the pulse was irregular for several weeks.

The diagnosis was based on the recurrent history, the second attack following on the burn and the melaena

in the search for which I expended considerable time and took care to exclude all sources of error, in diet or otherwise. The reactions were not strong but distinct.

With regard to Hyperchlorhydria, Nervous Dyspepsia etc. the patient I quoted as having a gastro-jejunostomy performed is doubtless a good example of the former type, while the gentleman who believed in giving his stomach a fright may be classified somewhere under the head of the latter. Acid Gastritis would appear to be the diagnosis in the case of the retired farmer of careless habit, already quoted, since his gastric contents contain considerable mucous, but we use all these terms with a sense of dissatisfaction.

## CONCLUSION.

Duodenal ulcer has hunger pain symptoms, but hunger pain symptoms do not necessarily mean duodenal ulcer.

After eliminating as far as possible all other conditions, in view of the high excellence of modern surgical results and of the relief brought to patients it is important that the question of surgical interference should be considered.

Cases with repeated attacks, undoubted haemorrhage and which do not yield readily to medical treatment should have the conditions explained.

The duodenum seems one of nature's weak spots and the hunger pain of many of the so-called neuroses is due to its call for protection, in other words a pre-ulcerative stage exists.

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