On Ulcer of the Stomach.

A thesis,
submitted for the M.D. degree,
to the
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by
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Leith, 1898.
Definition: Ulcer of the stomach is a disease in which, over a circumscribed area of varying size, there is destruction of the wall of the organ to a variable depth, not extending to involve all its coats, so that an open sore is left.

The small follicular ulcers found in chronic inflammation of the stomach and the hemorrhagic ulcers found in portal obstruction and other conditions, are not considered as true gastric ulcers and will not be included in the following description. Their relationship to the true ulcers will be alluded to when discussing pathology.

Geographical distribution: It appears to be less frequent in America than in England, and according to Mr. Sodeman it is very rare in certain districts of Germany (the Rhine province and the Bavarian Alps) and in the greater part of Russia. I am inclined to think it is less common in Scotland than in England.
On the whole it is a fairly common disease. Brinley found it occurred in five per cent. of persons dying from all causes.

It exists in two forms, the acute and the chronic. In 160 fatal cases examined 4 found 52 acute and 95 chronic or 15 to 19. In the Pathological Records of the Edinburgh Royal Infirmary of 53 cases 7 were acute and 46 chronic or about 1:57. It cannot be stated that these are records of fatal cases. There is, however, no satisfactory line of distinction between the acute and the chronic ulcer. Many described as chronic have formerly originated as acute ulcers, while others are chronic from the first. Clinically, except from the age of the patient, we cannot distinguish them. Though etiologically they are distinct, the age of the patient is, however, by no means a safe guide to the nature of the ulcer, as the acute form may occur in elderly people; but it is doubtful whether...
an ulcer. Chronic form the first ever occurs in young adults.

**Etiology.**

Sex: 50 more frequent in females than in males in the proportion of about two or three to one. Brimba put it at two to one. Martini's cases included 144 women and 27 men, a proportion of 5.3 to 1; Habershon's figures are 127 females and 74 males or almost 1.7 to 1.

With regard to fatalities Kissenheim's 9 death five 58 men to 42 women and Habershon's 10 108 men to 77 women and Sclith's 29 men to 26 women. Add to these figures give 195 males to 145 females or almost 1.3 to 2. If we take the number of females suffering from jaundice twice as twice the number of males, this shows the mortality to be almost three times as great in males as in females.
Age: It may occur at any age, but it is rare before puberty.

Brinley's data on cases and cicatrices found at post-mortem is as follows:

<table>
<thead>
<tr>
<th>Age Interval</th>
<th>Total Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-20</td>
<td>45</td>
</tr>
<tr>
<td>20-30</td>
<td>39</td>
</tr>
<tr>
<td>30-40</td>
<td>36</td>
</tr>
<tr>
<td>40-50</td>
<td>32</td>
</tr>
<tr>
<td>50-60</td>
<td>32</td>
</tr>
<tr>
<td>60-70</td>
<td>15</td>
</tr>
<tr>
<td>70-80</td>
<td>5</td>
</tr>
<tr>
<td>80-90</td>
<td></td>
</tr>
<tr>
<td>90-</td>
<td></td>
</tr>
</tbody>
</table>

But, these results being taken from post-mortem examinations, now mean the great frequency in early life, as the appearance of a cicatrice gives no information as to what was the age of the patient at the time it was excited, and, as alums may exist for many years, the discovery of one on post-mortem examination, therefore, affords no clue to the date of its origin.

Martin's 271 cases were distributed as follows:
Between 0 and 20 there were 15 cases

- 20 - 30 - 75
- 30 - 40 - 38
- 40 - 50 - 21
- 50 - 60 - 14

Over 60 - 4

This being a record of actually existing cases and not taken from pre-mortem records only, gives a more correct estimate and shows plainly the great preponderance of cases in early adult life.

Hibben's table shows that the greatest frequency occurs in males between 40 and 50 and in females between 50 and 60.

The age at which death is most common differs in the two sexes as shown by the following table:

<table>
<thead>
<tr>
<th></th>
<th>Female</th>
<th>0-20</th>
<th>20-30</th>
<th>30-40</th>
<th>40-50</th>
<th>50-60</th>
<th>over 60</th>
</tr>
</thead>
<tbody>
<tr>
<td>female</td>
<td>14</td>
<td>70</td>
<td>32</td>
<td>15</td>
<td>10</td>
<td>3</td>
<td>14</td>
</tr>
<tr>
<td>male</td>
<td>1</td>
<td>5</td>
<td>6</td>
<td>10</td>
<td>4</td>
<td>1</td>
<td>27</td>
</tr>
</tbody>
</table>

We then see that in females the maximum is reached between 20 and 30 and in males between 40 and 50.
Occupation does not appear to have much influence except in the acute perforating form, which is undoubtedly most frequent in female domestic servants. The occurrence of gastric ulcer in shoemakers, weavers, tailors, etc., probably due more to the conditions under which they live and work than the nature of their occupation.

Statistics and mode of life: It is generally said to be more frequent in the poorer classes. Martin thinks this may be due to the excess of vegetable food in the diet of the working classes, but according to M. Joly, the inhabitants of Rome and the cities where he found ulcers of the stomach least common exist almost exclusively on a vegetable diet and Barker points out that in from one to seven cases of acute perforating ulcer that he operated on, the patient (all domestic servants) had only been for a short time in their situations when the accident occurred and he suggests that prevailing privation followed by a supply
of unmentionable food has played an impor-
tant part in the causation of the ulcers. It is not so much the peculi-
arity of cases as the peculiar difficulty in obtaining a cure that is the prominent feature in patients of the former class. It is difficult whether the habit of drinking alcoholic liquors in excess increases the tendency to ulcer of the stomach. There has been no suspicion of any thing of the kind in any cases I have had to deal with and astronomers seem to be seldom affected with this complaint. Brünnich thought that old age, fatigues, mental anxiety, and inconstancy, were more or less immediate causes in a large proportion of cases.

Heredity: It is seldom that any hereditary trait can be traced but Duschfield found conclusive evi-
dence of it in lighter cases; in how instances, mother and daughter were affected; in one, father and daughter, in two, his cousins; and
in one, brother and sister. But we have not enough statistics at present to show whether the occurrence of gastric ulcer in more than one member of a family is more than a coincidence.

Relationship to other diseases. There are some diseases that seem to be especially associated with gastric ulcer. Of these, cholera is by far the most common. It is seldom that all symptoms of are absent in the acute cases occurring in young females. The connection with other diseases does not seem so close. It occurs in some cases of pulmonary tuberculosis, but the simple ulcer must not be confused with the Tubercular ulcer which is rare and only occurs in advanced cases of tuberculosis. Poisson and my think that Tuberculosis was present in cases of gastric ulcer in a larger per cent. of cases than in all persons indiscriminately. Ulcers of the stomach also occasionally occur in syphilis and in aneurysm most likely the result.
of the anaemia and the arterial changes which these diseases bring about. They are not sufficiently common to warrant any definite conclusion. Because a person, suffering from ulcers of the stomach is cured by the administration of carbonate of potassium, it is not conclusive evidence that the ulcer was syphilitic, even when other signs of syphilis are present. Demich found some incision complication present in more than half of the cases recorded at the London Hospital. These included aches in the joints, muscular head-aches, cardiac disease, the rash - rashes which were not specified; marked changes in the spleen; and prostatic glands. Heals found a large ulcer, occasionally, present in ulcerative disorders, such as dyspepsia, pyelitis, and typhoid fever. They occur also occasionally in tumors of the skin, though not so often in the stomach as in the duodenum. But all these diseases occur often enough in these very signs of ulcer in the stomach and it is difficult to tell exactly the
nature of the association and how they are dependent on one another.

**Morbid Anatomy.**

Site. It is most commonly situated on the posterior surface, in the pyloric region and towards the lesser curvature.

Brinton found it occupied the various regions of the stomach in about the following frequency:

- **in the posterior surface** ..... 43 promin.
- **lunar Antraline** ..... 27
- **pyloric extremity** ..... 16
- **both anterior and posterior surface** ..... 6
- **the anterior surface only** ..... 5
- **greater Antraline** ..... 2
- **cardiac pouch** ..... 2

**Number.** It is generally single, but two or more occur in about twenty-one per cent of cases. Affected in about a case in which altogether twenty-four ulcer were present, twenty being in the stomach and four in the duodenum. They were all on the posterior surface.
and towards the organic end.

The Acute Ulcer is small, ranging in size from about half an inch to an inch in diameter, but seldom larger than a scribbling piece. When first formed its edge is sharply defined, but not thickened, and it has the appearance of having been "punched out" of the wall of the organ. When it has existed some time, there may be some slight thickening around the edge, and when the distinction process has extended more deeply, it may become more or less funnel-shaped and tend to assume the character of a Chronic ulcer. The base may show regions of slough and small hemorrhages, and congestion may occasionally be found at the edge and in the neighborhood of the ulcer. They frequently perforate. They are found almost exclusively in the pyloric region and are generally solitary and are not so frequently situated on the posterior surface.
as the chronic ulcer.

**Microscopic Anatomy**: Leith very carefully examined three cases of acute perforating ulcer, making several hundred section through the ulcer and the tissue around. He found the condition as follows. A transverse section through the Cæcum of the ulcer showed a central deficiency. The mucous membrane

at the margin covered some cilia in such a manner that the flaps turned to lie horizontally and some few even had their ends directed downwards so that any fluid that might be secreted would be directed over the floor of the ulcer. At a short distance away from the ulcer assumed the normal vertical position. The cells of the flaps were normal. There was no increase of connective tissue between them and the capillaries were not dilated, except slightly, in one case. There was considerable dilatation of the small vessels and capillaries which entered the flaps from the submucous coat in one case.
The muscularis mucosa came right up to the margin and stopped abruptly, and was somewhat retracted. There was no change in the solitary glands, which were carefully examined. The submucous tissue was not thickened at all in one case, only very slightly, so in another but in the third it was more marked. The muscular coat was not changed, but its ends were covered with more or less necrotic-looking tissue. There was slight firm-cellular infiltration between the ends of the muscular fibres in one case. The peritoneal coat was considerably thickened. The structure of the thickening was that of a young inflammatory process and numerous giant cells occurred throughout it, some of which contained blood pigment.Successive sections, passing one towards from the centre of the ulcer, showed no marked change in its character, beyond the gradual creeping in of the peritoneal and muscular coats, the ends and surfaces of which were
Covered with a thin necrotic layer. Further onwards, the submucosa crept over the floor. In one case there was considerable undermining of the mucous membrane, which was convoluted. Presently, these circumscribed margins admitted and then formed a continuous covering, below which was a slit-like cavity, that of the ulcer. The muscular

mucosa soon formed a continuous layer with healthy mucous membrane above it. The cavity became smaller and resembled an abscess cavity except that its contents were not purulent. This cavity also appeared in another case but was less marked, and as it disappeared, the position of the ulcer was represented by a complete and almost perfect mucous membrane lying upon thinned musculari mucosa, beneath which there was some cicatricial tissue which extended some distance and increased especially on one side. No bacteria were found in either case. The mo
ing feature in all three cases was the presence of an endarteritis obliterans, with a young organized thrombus in the chief arterial supply going to the ulcerated area. This condition extended beyond the first arterial twig given off, for a distance of about three-quarters of an inch. While the artery became normal as did the coils of the stomach generally, except for some slight thickening of the peritoneal layer.

Ewald thought the curving inwards of the mucous layer to be due to the contraction of the muscular coat in virtue of its elasticity. Foreman reports some experiments the results of which, he thinks, show that the funnel-shaped appearance is due to the contractile properties of the deeper tissues in response to the irritation of the gastric juice.
The chronic ulcer is generally larger than the acute and may be a number, four, five or six inches by one and a half to two inches. Its walls and floor show signs of considerable inflammatory thickening and its funnel shape is much more marked than in the acute form. This funnel-shaped appearance is due to the different coats being affected by the destructive process in a gradually lessening degree from within outwards, the mucous membrane being destroyed to a greater extent than the submucous coat, the submucous coat than the muscular and the muscular than the peritoneal. The base may be formed by the muscular or peritoneal coat or by one of the neighbouring organs, such as the pancreas or liver, which has become attached by adhesions. The muscular base is sometimes extensive. It seems to be more resistant to the ulcerative process and the circular fibres more so than the oblique. There may be some undermining or the ulcer may show
Signs of healing on one side while it is extending on the other. Sometimes granulations are seen at the base.

Microscopically the ulcer is thicker at the edge of the ulcer to be due chiefly to an increase in the quantity of fibrous tissue in the submucous coat. The submucous membrane, the muscular and the periamucous coats are also affected but not to so great an extent. In recent cases the glands are healthy but in old cases they are compressed and atrophied. The chief is always confined to the neighbourhood of the ulcer.

**Fig. 1.**

Diagrammatic sketch of a section of a chronic ulcer.


(After Martin).
Pathology.

There is perhaps no disease that has had its pathology so much discussed, and had so many theories put forward to account for it, as gastric ulcer. Vascula, Nervous, functional disturbances have each in their been suggested as responsible. Even the ubiquitous bacteria has its supporters. But in spite of all we must admit we are still very much in the dark, and that no one theory hitherto proposed will satisfactorily account for even a majority of the cases of gastric ulcers.

The fact that ulcers, with the peculiar character of a gastric ulcer, occur only in the stomach, the lower part of the esophagus and the first part of the duodenum, naturally suggests the idea that the acid secretion of the stomach is an important factor in its causation. That the stomach has the power of digesting the stomach wall under certain conditions immediately after death, is well known and can be seen at many post-mortems. But the normal gastric
juice does not digest the normal living gastric mucous membrane, and it cannot therefore be the sole cause. There must be some disturbance of the balance normally existing between the digestive action of the gastric juice and the resisting powers of the tissues. The gastric juice can digest recovered tissue or tissue of low vitality and if a small portion of the gastric mucous membrane should from any reason become recovered or have its vitality sufficiently lowered, it is easy to understand how the gastric juice could remove it and so cause an open sore. The chief question to consider is, what is the cause of the preliminary recovery or lowered vitality?

I shall first briefly discuss and point out the objections to the various theories that have been proposed and then state what seems to me the most likely cause for the acute and for the chronic ulcer.

It will be convenient to arrange them under the following headings:
(1) Injury.
(2) Pressure.
(3) Articular Inflammation.
(4) Venous Obstruction.
(5) Hemorrhagic Erosions.
(6) Blood Changes.
(7) Bacterial necrosis.
(8) Necrosis di incurvus.
(9) Necrosis ad incurvus.
(10) Inflammation.

Drying (mechanical and chemical) is a frequent cause of necrosis in other parts of the body, but injuries to the stomach seldom if ever lead to ulcers. Injury is produced experimentally, and in surgical operations heal rapidly and fiddles, liniments having been known to swallow hand and throat the chances without apparent harm to their gastric mucous membrane. The antacid taste of hot foods has been suggested as a cause of gastric ulcer in cattle, but the number of cattle suffering from this complaint is not proportionately higher than the number of domestic
Servants or even females generally.
Removal of portions of mucus mem-
brane by the stomach pump is said to be followed by no ill effects. Mineral
acids and caustic alkalies when swal-
lowed, cause extensive ulceration,
which heals very slowly, probably in
consequence of the retarding effect of
the gastric juice. But this can hardly
be looked upon as a true ulcer of the
stomach. Blows on the epigastrum
are sometimes followed by hematuria,
but I am not aware of any instance
of a gastric ulcer having been produced
in this way. As already mentioned,
ulcers are occasionally found in the
stomach after burns of the skin as
other parts of the body. The connection,
which is indirect, will be discussed
later on.

Pressure:

A tumor growing outside the stomach
at the junction with the colon, causes ulceration,
e.g., an aneurism.
Pressure of the last or sheath-maker, or of the conel or female, has probably nothing to do with the origin, though it may perhaps interfere with the healing, of gastric ulcers.

Martin describes a form of pressure neoplasms occurring in the mucous membrane of the stomach, due to the presence of small submucous fibro-myomata. These are small tumours varying in size from a half to one centimetre in length and about half that in width. As a rule their presence is of little significance but in one case a small ulcer developed on the surface of one of these tumours. But the mode of formation must be very exceptional.

Artificial obstruction: He has observed that the position and extent of a particular correspended with the distal sipphid by smaller branches of the gastric arteries and also that the bottom of the ulcer (the apex of the Cone, as it were) was not generally in the middle, but pointed
To one side, in the direction from which
the artery came, i.e., towards the greater
or lesser curvature as the case might be,
and showed that they thus presented an
analogy with infarcts found in other
organs of the body. He therefore concluded,
that an arterial obstruction first caused
damage to the tissues which were after-
wards removed by the faecetic juices and
so an alias was formed. Pan showed
by experiment that life of the gastric
arteries would produce ulceration. There
are two ways in which arterial oc-
struction can be brought about: (1) em-
bolism or by arterial change, such as
arteriosclerosis and arteritis obliterans or
jaundice. With regard to (a) em-
bolism, if it were the univocal cause
of gastric alias, emboli or sources of Em-
boli should always be discovered. But
this is very far from being the case. In
by far the greater majority of cases, no
Emboli or source of Emboli can be found
and even when Emboli are found in
other visceræ the stomach is not affected.
The embolic theory in no way explains why clots are so frequently situated in the pulmonary region. Saltan Kemrick performed various experiments on living and dead animals and on the human cadaver, with a view to determine the course usually followed by an embolus when injected into the general circulation. He employed tobacco leaves, finely powdered black wax and silver sand as emboli and as a result of his experiments found that only from two to five per cent. of the emboli injected found their way into the systemic vessels and that two-thirds of them occupied the middle and cardiac zones, and that in no instance was the pulmonary region alone affected. He also found that the clots thus produced differed essentially from the acute variety in the human subject. The immediate result was an intense congestion and extravasation in the affected areas and the clot subsequently formed usually exhibited in its edge and base some indication of its hemorrhagic.
origin. We may conclude from this
ten, that, although it is possible that
embolism may occasionally be the
cause of chronic ulcer in people suffering
from tubular heart disease, it is probably
never the cause of the acute ulcer seen in
young people.

(b) Arterial obstruction due to atheroma,
edematitis or pancreas disease, is impo-
lant more gradually than that due to
embolemia. It causes a lowering of the
vitality of the mucous membrane of the
area affected, with decreased power of resi-
rting the action of the gastric juice and so
allow of its removal. Actual death of the
part is not necessary as Parry found that
the tip of a pancreas was dissected when
inserted into the stomach. But atheroma
is essentially a disease of past middle-
age and can therefore have nothing to do with
acute ulcers occurring in young people. It
is frequently present in elderly people suf-
ferring from chronic ulcer of the stomach and
may be an important factor in the causat-
ion of the ulcer. Endarteritis obliteran-
Seldom occurs in young people except as a secondary condition and most observers have failed to find any evidence of it in the neighbourhood of gastric ulcers. Lilié, however, found it present in all three of the cases he examined but he admits that it might be secondary as it occurs as a secondary result in ulcers in other parts of the body; but he points out that the spreading of the condition beyond the first branch of the (which was noticed in all three cases) is unusual.

Gastric cancer in cases of cancer is common in advanced cases of phthisis and may be one of the causes of the gastric ulcers found in some of these cases.

An allied change, then, though they may account for some of the cases of chronic ulcer in elderly people and those suffering from phthisis, cannot be looked upon as being the prime cause of acute ulcers occurring in young people.
Vomous Restriction:

If this were a constant factor in the causation of gastric ulcers, we ought to find the latter disease a common companion of cirrhosis of the liver and heart disease. But it is not so. Hemorrhagic erosions are frequent, but ulcers do not occur in a greater number of cases than is sufficient to warrant us in concluding that it is merely a coincidence. The alkaline juices which are present in the stomach in considerable quantities would interfere with the action of the gastric juice and may perhaps account for ulcers not being more common than they are.

Fenwick found from his experiments on animals that ligating one or two small gastric veins had no effect on the gastric mucous membrane, but when he ligatured the portal vein or the main gastric veins entering it, the mucous membrane showed numerous hemorrhages and some ulcers. Vomous Restriction, then, is almost only an occasional
Cause of gastric ulcers, but there is no doubt one of the most potent causes for the delay in healing in ulcers already existing and brought on by other causes.

**Punctiform Haemorrhages:** There are present when there is obstruction in the gastric veins, either produced experimentally or as a result of disease. They sometimes precede ulceration. But the ulcers thus formed are curvariately, multiple; they occur almost exclusively at the cardia end of the stomach and under the microscope show traces of their haemorrhagic origin. They have not the characteristic nor do they give rise to the symptoms of true gastric ulcers.

When atonia is produced artificially, by injecting such substances as pyrogallic acid or toluidindiamine into the blood stream, ulceration of the stomach is found. This at first sight seems to present some analogy with the cases of acute ulcers occurring in chronic jails. But an exam...
location of the ulcers show that they are different in character from those occurring in this disease. They are multiple, occur at the fundus and their hemorrhagic origin is evident.

Rindfleisch thought the sequence of phenomena to be somewhat as follows: the act of vomiting, temporarily arresting the return of the blood, causes minute extravasations from the superficial veins, radicals of the gastric mucous membrane; the extravasated blood corpuscles infiltrate a circumscribed patch of mucous membrane to such an extent as to compress the capillaries and thus stop circulation and nutrition, the gastric juice dissolves the dead tissues and then forms a hemorrhagic erosion which in time becomes an ulcer. He finds an account of a case in which hemorrhagic erosion and an ulcer were present together and concludes this to be the usual mode of formation of gastric ulcer.

But considering the very small number of cases in which erosions and ulcers are found together and bearing in mind
the fact that they generally occur under dissociated conditions; that erosions are multiple and ulcers generally single and for the that they occur in different regions of the stomach, we are justified in looking upon a haemorrhagic erosion as only occasionally a precursor of gastric ulcer.

**Changes in the Blood:**

The frequent connection of gastric ulcers with chronic amenorrhoea, pregnancy to suggest the possibility that the state of the blood is an important factor in the causation of the ulcers.

It was thought that it was the alkalinity of the blood alone that prevented the digestion of the stomach by its own secretion. But it is difficult to see how the alkalinity of the blood could have any protective influence over the superficial layers of the mucous membrane and it has been shown that the blood may be made neutral by means of an acid and yet not lead to auto-digestion.
Of all blood diseases chlorosis is the one by far the most commonly associated with gastric ulcers, but we have no evidence to connect the one with the other as cause and effect. After all it is only a small percentage of patients suffering from chlorosis who have also a gastric ulcer and often enough we see young girls suffering from gastric ulcer who have only slight, if any, trace of chlorosis. Chlorosis and ulcer occur in the same class of patients and under similar conditions and it is more reasonable to look upon them as both results of the same cause than one the cause of the other.

I have already referred to the occurrence of ulcers in the stomach in artificially induced anemia and pointed out how they differ from the acute ulcer occurring in chlorotic girls, and in no way support the theory that chlorosis is a cause of gastric ulcer.

Blood changes, though no doubt very important and predisposing causes, are not the only cause of gastric ulcers.
Referenda will be made to this subject.

Bacterial necrosis:

As already mentioned, ulcers are sometimes found in the stomach and duodenum after burns. They generally occur during the stage of separation of the sloughs and the usually accepted explanation of them is that they are caused by septic emboli, which find their way into the circulation from the damaged skin surface. Ulcers are also found occasionally in cases of erysipelas, typhoid fever and pyelitis, diseases of recognized bacterial origin.

Martin lays great stress on the fact that ulcers are mainly found in those regions of the stomach where no hydrochloric acid is secreted from the glands and he considers that bacteria would here be less exposed to the antiseptic action of the acid. He thinks that in not a few cases of gastric ulcers, the pemphigus necrosis may be due to the invasion of bacteria, the subsequent ulceration and exfoliation of the ulceration being aided by the action...
of the gastric juice. He invariably found bacteria in the sloughs occurring in ulcers produced in guinea pigs. Leith however failed to find any syphillus bac-

Nervous disturbances.
The fact that various mental states increase the pain of gastric ulcer and that anxiety and worry appear to have some influence as predisposing causes, points to the nervous system as having something to do with the causation of gastric ulcers. This view has recei-

ed support from experiments. Salina produced ulcers by prolonged stimulation of the vagus, and Osterr produced gastric hemorrhage, corroding ulcers and even perforation by injury to the corpora quadrigemina. But it is not easy to say how far the ulcers were due directly to nerve lesions and how far to compression of the blood vessels by the contracted mus.
cousin Brille of the stomach wall.
But at present the whole question of
the relationship of the stomach to the
nervous system is so little understood
that we cannot do more than vaguely
theory on this subject.

Functional disturbances.
As we have already seen, the normal
gastric juice has no power of digesting
the healthy mucous membrane of the
stomach, and little if any effect in
preventing the healing of lesions acciden-
tially or intentionally caused.
Normally a balance exists between
the digestive action of the gastric juice
and the resisting powers of the tissues.
Before an ulcer can form there
must be some change in the character
of the gastric juice or some loss of resist-
ing power in the tissues. But if we
wish to prove that it is an alteration
in the character of the juice that
is the causative cause of a gastric
ulcer we must be able to show.
quite, that some change is always present; and, secondly, that this change can and always does produce a gastric ulcer unless prevented by some other circumstances. With regard to the first point, the only change in the gastric juice that has been shown to occur with any degree of constancy is hyperacidity. But it is by no means always present and in some cases the acidity is actually diminished. Moreover, it is quite possible that the hyperacidity, when present, is due to the existence of the ulcer and it has not been shown to precede it.

With regard to the second point, Pan, found from his experiments that a slight increase of acidity would cause ulceration when a vessel had been ligatured, when normal acidity did not do so and that still greater acidity would do so even without previous ligature.

Hyperacidity alone, then, is not the actual cause of a gastric ulcer.
It may cause an ulcer when the viability of the mucus membrane has been lowered and it is generally the most important factor in the delay in healing of ulcers however formed. Whether or not the acidity ever becomes, for some reason, as to cause circulation with previous damage to the mucus membrane as happened in Pan's experiment is difficult to say, but it must at least be very unusual.

We thus see that no one of the theories discussed so far is sufficient to satisfactorily account for more than a very small proportion of the number of cases of gastric ulcers. There are serious and incommutable objections to every one of them.

There is one other theory, that certain be di chlorane and that is that ulcers of the stomach are in some way the result of inflammation.
In many of the older text-books, ulceration of the stomach is merely referred to as an advanced stage of chronic inflammation. Curreri defines it as "inflammation of the stomach, ending by loss of substance in the form of an ulcer." In recent text-books, inflammation, if alluded to at all, as a possible cause, is invariably dismissed as having no evidence in support of it. But it seems to me to be worthy of a little more consideration.

In chronic gastric carcinoma a fibroid thickening of the mucous membrane takes place in a greater or lesser degree. Except in advanced and rare cases (atrophic ventriculitis or plasti limitis) the muscular wall is not involved. In the early stages there is small cell epithelial infiltration between the glands and the mucous membrane is demeared of its epithelium; in later stages the small cell epithelial infiltration becomes converted into
fibrinous tissue, which causes compression and subsequent atrophy of the glands. Mentin has already been made of the fact that the secretion of the glands has no power of digesting the gland cells themselves. The cells must therefore possess some inanimate power of resisting the digestive action of their own secretion. What would be more likely, then, than that when the cells of the mucous membrane have been removed, the fibrinous tissue (of low vitality, owing to the pressure on its blood vessels by its own contraction) should fall a victim to the digestive action of the gastric juice produced by the still healthy and active glands in other parts of the stomach, especially when the vitality of the system generally has been lowered by want of food, intemperance, overwork, worry, wasting diseases or the menopause?

It is said, however, that portions of the mucous membrane may be removed experimentally or accident
ally (as by the stomach pump) without ill effects following, the damaged surface readily healing. But this may be because the tissues left bare are sufficiently healthy to resist the action of the gastric juice and to fill the breach and do not consist of fibrous tissue only. Many sufferers from chronic ulcers have had chronic dyspepsia for some time, and it is often impossible to fix the time when the ulcerative process commenced or we may not suspect the existence of anything more serious than a chronic cachexia until we are suddenly and unpleasantly warned of the existence of an ulcer as well by the recurrence of a copious and alarming hematemesis. The chief arguments used against the inflammation theory are that in acute cases no sign of inflammation is discernible after death and that in chronic cases what inflammation there is is secondary to the ulcer.
and does not precede it. And if we suspect that the fibrosis is primary and the ulcer secondary, we must be prepared to explain why ulceration takes place on some fibroid patches and not on all. Why should one part of the inner membrane be affected while others escape? If there were several patches we ought to find several ulcers. On the other hand, more extensive the fibroid change the less healthy inner membrane there would be left to secrete active gastric juice, and then leading to ulcer formation. It seems to me quite as reasonable to suppose that there was one fibroid patch and that became ulcerated as to suppose that there was only one ulcer and that subsequently became surrounded by secondary chronic inflammation. Ulcer takes place mostly at the pyloric end of the stomach and this, as we have been in the region most commonly affected by ulcers.
the reason why fibrin is more marked in this region is because a form of tissue, lymphoid tissue, exists in larger quantities in this than in other regions of the stomach and that as age advances it tends to become fibrinoid tissue. The greater the amount of lymphoid tissue existing, the greater would probably be the amount of fibrinoid tissue that would place it especially if it has been subjected to constant irritation. We have here one very good reason for the frequency of ulcers in the pyloric region which all other theories completely fail to account for.

Fibrin patches are, however, sometimes in other parts of the stomach when ulcer is present. In twenty cases recorded at the London Hospital Morrison made a thickening at a distance from the ulcer. In one instance the base seemed to be excavated in a thickened tissue and had no thickened margin, the middle coat was hypertrophied a quarter of an
with thick, the tissue being hard and shining." In another case besides two ulcers there was also "fibroid invagination in patches scattered over the mucous membrane of the stomach." As already mentioned, Martin describes a form of ulcer occurring on the surface of tuberculous fibro-myxomatous. He considers the cause of the necrosis of the mucous membrane to be due to adhesion of the tumour to it and subsequent interstitial fibrosis and atrophy of the gland cells. If necrosis takes place on the surface of these small tumours, why not on the surface of larger patches of fibroid thickening? When the ulcerative process had once commenced a secondary fibroid invagination would also take place and ulcers would be further thickening in the neighborhood of the ulcer and the gradual obliteration of the original fibrosis. Chronic ulcers perforate much less frequently than do acute ulcers. This is generally supposed to be due
To the absence of inflammation found around the latter and its presence in the former. But it is difficult to understand why there should be less atelectasis in a young housemaid, healthy (except possibly for some slight anemia) than in a half-starved, intermittent over-worked woman of fifty or sixty years of age. On the other hand, if we look upon the fibroid thickening as primary it is easy enough to understand why perforation should occur so seldom in chronic cases as compared with the acute ones. But ulcers occurring in young people and apparently commencing as acute ulcers may have a certain amount of secondary thickening around them, though it is never so marked as in those which have been chronic from the first. And they must have existed for some time before this takes place. If the secondary thickening were invariably secondary, the longer the ulcer had existed, the more marked the thickening might be.
be, but this is certainly not the case. If the ulcers were secondary to the thickening then the longer the ulcers had lasted the less would be the amount of thickening unless a secondary inflammation produced fresh firm tissue as fast as the other was destroyed.

On the whole there seems to me to be less to be said against; and more to be said in favour of, the theory that chronic ulcers are due to a chronic inflammation ending in a loss of substance than there is to be said against or for any other theory yet proposed. It is also the more simple and the more natural theory.
The idea that the solitary glands were responsible for some forms of acute ulceration of the stomach had not received much attention until the appearance of a paper by Dr. Saltan Tennick. Wilson Fox had already described their nature and shown that they became enlarged in inflammatory diseases of the stomach and sometimes preceded to ulceration. He described the ulcers as being small follicular ones.

These glands vary much in size and number in different individuals. They are most marked in infancy and childhood, and after puberty they diminish in number, especially at the cardiac end until after forty; it is often difficult to find any in the cardiac two-thirds and in the pyloric region they are much reduced in size and number. Sometimes they undergo hyperplastic degeneration. They are situated in the mucous membrane and rest on the muscularis mucosae. Occasionally, the base
of the gland is in the submucous tissue. Each gland has a small nutrient arteriole and a lymphatic vessel which passes from its deeper surface obliquely through the muscularis mucosae to join the lamellar trunks in the submucosa. At the circumference of the gland the gastric tubules are slanting but toward the surface they are perpendicular. Besides the foveolar glands, lymphoid tissue exists in a diffuse form around the blind ends of the gastric tubules and spreading over the muscularis mucosae in a thin layer. In inflammatory conditions of the stomach, especially in cases of infectious diseases, such as typhoid fever, diphtheria and in acute tuberculosis, these glands show signs of acute inflammation, their tissue degenerates and they undergo rapid necrosis. The lymphatic cells proliferate and the gland is increased in size. The mucous membrane above becomes bipitted with cells and the tubules are obliterated. The cells
in the centre of the gland degenerates and a cavity is formed, the serous membrane above becomes thinned and at last gives way. Kennicott observed all these stages in the stomach of guinea pigs that had died after inoculation with Eberth's typhoid bacilli, and from these and other observations he came to the conclusion "that under certain conditions inflammation of the solitary glands may give rise to ulceration of the stomach, which by extending its area in all directions can assume the characteristic form of an acute perforating ulcer."

There are several objections to be raised against this theory, but I think they can for the most part be refuted.

1. The ulcer shows no evidence of having originated in a lymphoid follicle.

With regard to this objection, it does not seem too much to suppose that the gastric juice has removed all traces of lymphoid tissue or
at any rate made it unrecognizable as such.

(2). The healthy follicles are very much
smaller than the average ulcer.

The follicles however can be much
so large and become much enlarged when
inflamed. It is quite possible that some
of the surrounding tissue (the lesion
which has been lowered by pressure of
the enlarged gland) are removed by the
gastric juice as well as the gland itself
or two or more glands might coalesce.

(3). There is no change from the nor-
mal discoverable in the follicular gland
throughout the stomach generally.

Leith found no change whatever
in the cases he examined, but Hot-
wick found many increased in
size and the gastric secretion, who
he says, was able to trace distinctly
the various gradations between the
enlarged follicles and the true
perforating ulcer.

(4). The ulcers which occur when
anemia is artificially produced
Show no trace of connection with cover glands.

But we have already seen that these ulcer in no way resemble the ordinary acute perforating ulcer (5). It does not account for the greater frequency in the females.

We have no evidence to show that there are a greater number of, or larger glands in the female than the males.

We must suppose that the reason for enlargement are more often present in females than in males.

(6). The ulcers described by Fox as occurring in Cattle can be, if the stomach were small for circular ones.

The condition of the gastric mucous membranes is quite different in Cattle under conditions from those it is when an acute perforating ulcer occurs. The Cattle causes much alkaline influx and this would prevent the gastric juice from enlarging the ulcer.
Fig. 2.

Vertical section of Commencing ulceration of a solitary gland of the stomach in a case of tuberculosis.

(after Fox Diseases of the Stomach Plate II. fig 18)

Fig. 3.

Section of the stomach of a guinea pig which had died within 48 hours after inoculation with pure culture of抵制 bacillus, showing the formation of an ulcer from necrosis of a solitary gland.

(after Kanwick. Jour. of Path. and Bacter. Vol I. No 4. Plate XVII figs.)
In support of the theory that acute perforating ulcer of the stomach is due to an inflammation and subsequent necrosis of the solitaire glands, there are some points of analogy between the clinical aspects of this disease and some others which are, I think, well drawn if attention to.

These diseases are tonsillitis, acute lobar pneumonia, appendicitis, rheumatic fever and inflammation of the serous membranes.

The points of analogy are:
(1) They are all essentially diseases of early adult life. They do not occur in more advanced ages, but the first and most typical attack is generally between the ages of 15 and 30, which is also the age for acute perforating ulcers.
(2) They are all liable to recur.
(3) They are most common in the autumn months.

This is at least true of tonsillitis, which is a rheumatic fever, though
not so evident as pneumonia. Acute perforating ulcer is, I think, most common at this time of the year, but I have no statistics to prove it. Chlorosis, which so frequently accompanies it, certainly is so.

(4). The menstrual period appears to increase the tendency to an attack.

I have seenُ 39 says "there is some evidence to show that menstruation predisposes to the attack of pneumonia". Kellogg says "suppression of the catarrh is said not infrequently to occur at the onset of appendicitis". I have observed the same to happen frequently in tonsillitis and occasionally in pneumonia. We have already seen that the symptoms of gastric ulcer are aggravated during menstruation and in a case of acute perforating ulcer I attended the only deviation from perfect health was the nonappearance of the menst which were due at the time. The common complication of acute per-
Jorating ulcer is ammoniæa.

(5). Pregnancy and the puerperal state increase the danger of and the predisposition to an attack.

The days labor pneumonia is very fatal in the puerperal state and the danger is increased in pregnancy. Chorea, an acknowledged manifestation of rheumaæon is well known to be fatal to pregnant women. Almost the stomach may occur in successev

preparations of pregnancy or shortly after confinement. In the only case of ulcer of the stomach in a pregnant woman that I have attended, the symptoms were very pronounced and ordinary treatmænt produced little benefit.

(6) The liability of the lungs, the joints, the seræ membranes to be affected in cases of blood poisoning, and the occasional occurrence of ulcers in the stomach and duæenum in hæm may be alluded to.
There are also some points of resemblance in the minute anatomy of the organ affected.

Thus, in the appendix the most striking histological feature is its richness in lymphoid elements. It is most marked in youth.

The tonsil is made up of masses of lymphoid tissue and Bland Sutton points out the marked histological similarity between the appendix and the tonsil and shows the great resemblance in their inflammation, claiming he looks upon the appendix as an abdominal tonsil. Throughout the lymphatic system of the body we find collections of lymphoid cells which are either round or uniform, but in adults they tend to become finer. One thinks that the disorder which on a lesser scale presents the greater analogy with acute pneumonia is acute tonsillitis. Many of lymphoid tissue also exist in the serous
membranes.

It does not appear to be a prominent feature in the structure of the synovial membranes of joints. But the joint affection in rheumatic fever is probably a secondary result and not the primary disease. It does not bear the same relationship to the disease as the inflammation of the lungs, the tonsil does.

The existence of masses of lymphatic tissue in the synovial membrane of the stomach in the form of solitary glands and their tendency to disappear as a rule advanced already been alluded to.

Then facts and others that might be brought forward suggest the possibility of a 'maladies moist' common to these diseases mentioned, or in other words that the circumstances which would in one patient bring on an attack, say, of pneumonia, might in another bring on tonsillitis, appendicitis,
Rheumatic fever, inflammation of the serous membranes or enlargement of the solitary glands in the stomach. And it seems not unusual to suppose that this material modifies as an irritant to the lymphoid tissue, causing inflammation. The reason why one person is affected with rheumatic fever and others with another is no doubt due to the presence of an abnormal quantity of the tissue in the lung, appendix or stomach as the case might be. The condition of the organ too might play an important part in deciding which organ would be affected. Thus, contraction leading to appendicitis, bodily fatigue to rheumatic fever, a mental shock to chorea, indigestion to enlargement of the solitary glands and so on.

If this view is correct, we ought to find these diseases fairly frequently associated with one another and with acute perforating ulcers.
The frequent association of tonsillitis, appendicitis, pneumonia and inflammation of the serous membranes with attacks known as rheumatic fever is well known. Many writers have spoken of them as "phases of rheumatism" or "manifestations of the acute acid diathesis."

It remains to be seen whether they are associated with acute perforating ulcer of the stomach first with regard to rheumatic fever. In some cases of acute ulcer there has been a history of rheumatic fever or rheumatic pains. I cannot say whether this is a sufficient number of cases to show it is more than a coincidence.

But it is a fact that the class of patient most likely to be affected with rheumatism is the one most often affected with acute perforating ulcer. Thus Page remarks, "Young females are made up a large proportion of the cases of..."
Rheumatism seen in hospital practice, and then we find the people we find suffering from acute perforating ulcer. "A striking feature of rheumatism from is the anemia it causes, and the rheumatic poison appears to be a clinical to the red corpuscles or their hematin." Whether the rheumatic poison is it produces results very similar to what we find in cholera, which is probably due to a poison of some sort, and I have already suggested the possibility of the cause of cholera and acute perforating ulcer are being one and the same, or at least of the same nature.

Haig shows that as the amount of uric acid in the blood increases the blood decimal (i.e., \( \frac{70 \text{ of hemoglobin}}{70 \text{ of red cells}} \)) is lowered and he considers that cholera is caused by the presence of uric acid in the blood. If this is true we may add cholera to the list of diseases manifesting a "urate acid diathesis".
The close connection of Chlorosis and acute perforating ulcer leads one to think that the latter also might be a "phase of rheumatism." If this is so, we might find some of the other diseases mentioned in fairly constant association with acute ulcer, as well as rheumatism and Chlorosis.

The patient already referred to as having had gastric ulcer when pregnant had suffered from several attacks of quinsy. Her family history is interesting as showing how the different diseases supposed to be phases of rheumatism may be present in various members of the family. Her father suffered from Diminutia for many years; another sister had Chlorosis, endocarditis pecora, a brother had Diminutia, and this too had several attacks of pleurisy, and another Chlorosis. A cousin (the father's niece) too had several attacks of appendicitis (verified by operation) and also pleurisy with effusion.
Deit has an account of a case of a girl dying from hemmorhage from a gastric ulcer in which ulcerous vegetations were formed on the internal curps, and he quotes Ora who sought to prove from 17 cases, that certain endocardial and periocardial lesions might arise in the course of an ordinary gastric ulcer. Ora considered them due to stimulation of the vagus, while Deit asserts they were caused by micro-organisms entering the blood from the gastric ulcer. It appears to me more likely that they were both due to the same cause and not dependent on one another.

I know of no case of a patient being afflicted with both appendicitis and ulcer of the stomach and the family of this occurrence might be accounted for by the difference of sex in which each is most common. Kellynack however mentions a case of appendicitis in which there was distinct enlargement of
The solitary glands in the lower part of the ileum in a male aged 38, when there was no evidence of Tuberculosis.

Pneumonia and acute ulcer sometimes occur together and for 72 days that in pneumonia there is an occasional though rare severe implication of the intestine with swelling of regional patches.

Inflammation of lymphoid tissue is followed by the presence in an increased number of leucocytes in the blood. An increase may be brought about by injecting various substances, such as nuclein, urea, uric acid, bacterial products to into the blood. They act no doubt as irritants to the lymphoid tissue, causing proliferation of the cells and an increase of the number of them passing into the circulation. It is only reasonable to suppose that the solitary glands in the stomach would withstand
in this reaction and that the cells of these glands by proliferating and producing substances harmful to themselves, bring about their own destruction and, by infiltration and pressure, the destruction of the tissue around them and the mucous membrane above them. The gastric juice by removing the necrosed area would complete the formation of the ulcer. Possibly many ulcers thus formed here without producing symptoms. Others, owing perhaps to the original gland having extended somewhat deeper than usual, perforate at once. While a site preheating meal is prevented from healing by the action of the gastric juice (which may be hyperacid) and by the anemic and unhealthy condition of the patient generally. Necrosis tissue is normally present during growth and development, during necrosis and
fertility. And then are the periods
for clear of the stomach. There must
be some reason for this lean.
Most likely it is due to some substance
circulating in the blood and caus-
ing increased activity in the lymph-
oid elements throughout the body.
If to this lean we bring some other
cause for lean, we should get a
double activity in the lymphoid
elements, which might result in
an attack of pneumonia, appendicitis,
tonsillitis, peri-or endocarditis,
pleurisy, rheumatic fever orelayed
solitary glan-ds (which might pro-
ceed to ulceration) the question of
which organ would be the one
affected depending on the amount
of lymphoid present in it and on
the state of the organ at the time.
So sum up, the view I wish to put forward is that there are two kinds of ulcers, having a quite distinct pathology. One, the acute, is due to an inflammation of the solitary glands of the stomach brought about by the presence of some substance in the blood, which acts as an irritant to the lymphoid tissue, and this irritant is the same as that which at other times, or in other individuals causes the disease already frequently alluded to. This form of ulcer may heal without symptoms, or perforate at once, or it may be prevented from healing for some time, and lead to the characteristic symptoms and become one form of what is often described as the chronic ulcer.

The other form, what I consider the true chronic ulcer is due to the excessive formation of fibrous tissue in the place of lymphoid tissue as age advances and to
the viability of the lining tissue to resist the erosion of the gastric juice, especially when the viability of the system generally is lowered by overwork, worry, deprivation or when the gastric juice is hyperacid.

This view is the only one that explains why ulcers are so commonly situated in the pyloric region.

The detailed account of the microscopic anatomy given by Deitrich is just what one would expect to find if an enlarged solitary gland was the cause of the acutely perforating ulcer.
Symptoms.

The most characteristic symptoms of gastric ulcers are pain, vomiting, and hemorrhage. They are not, however, all present in every case and there is considerable variation in their relative predominance and order of appearance. Occasionally, an ulcer is discovered after death that gave rise to no symptoms during life. Or there may have been an entire absence of symptoms until perforation or the erosion of a large artery suddenly takes place, though the appearance of the ulcer as found post mortem, shows it has been left for some time.
Pain is the most constant and characteristic symptom and is usually the first complained of. It varies in degree from a slight feeling of weight or fulness at the epigastrium to paroxysms of intense agony. In cases of long standing it is seldom entirely absent. It is usually described as burning, boring or gnawing but rarely or never as lancinating, stabbing or stitching. Sometimes it assumes a throbbing or beating character, though nothing of that nature can be felt by the hand placed over the region. It may disappear altogether for a few days. It generally comes on a few minutes after taking food but may be delayed an hour or even more. Sometimes it starts immediately after the food is swallowed and is no doubt to a great extent caused by the digestion of the food and gastric juice and by the movements of the stomach. In one case I noticed the pain came on about an hour after every nutritive
Chromatogram was given. When the pain has reached its height, the patient usually vomits, which brings or less complete relief. If vomiting does not take place the pain may continue for an hour or more or until such time as he may assume the food that passed into the duodenum. In chronic cases it is sometimes continuous. Rarely it is said to be worse when the stomach is empty and to be relieved by eating food.

It is increased by taking solid and indigestible food. Beer and drinks such as tea; also by flatulence, fatigue, anxiety, and particularly by constipation. It is often a prevailing aches or lumps before and during the first days of the menstrual periods. This probably arises from some change in the blood causing either increased acidity of the gastric juice or diminished resistance in the uterus or perhaps both; and is not a simple reflex, as Fallopos suggest. In some cases pain is more complained of in certain
Photographs but it is undeniable, a fact that all pains are relieved by the recumbent position. Pressure as a rule aggravates the pain. Gentle pressure, however, is sometimes grateful when more is unbearable. In some exceptional cases, the patient obtains relief by leaning over the back of a chair, by folding his arms lightly across the epigastrium or flexing the thighs onto the abdomen and holding them firmly against it. Three cases methods of obtaining relief by pressure were induced in by a patient under my observation in aston, post-mortem, a large chronic ulcer was found on the posterior surface of the stomach, which was matted to the pancreas from an artery of which the fatal hemorrhage had taken place. Barlow suggested that perhaps pressure might increase the pain according to whether it supported the peritoneum or compressed on the surface of the ulcer.

There is generally some hyperesthesia and occasionally some anesthesia in
The region of the stomach

The seat of the pain: Pain is generally referred to a circumference about an inch in diameter. In some cases it is a mere point; in others it is more diffuse. It is usually situated just below the suprasternal notch, but it may be higher (behind the sternum) or lower (towards the umbilicus) or in one or other hypochondrium.

Pain in the back is as a rule complained of. It is not generally noticed so soon as the epigastrian pain.

Pressure in front generally increases it. It is referred according to Bum_RET 29 to a spot to the right of the spine opposite the sixth to eighth dorsal or first lumbar vertebra or it may be as high as the eighth dorsal or ninth dorsal. Duchfeld says it is situated "either over the union of the spine between the sixth and eighth dorsal vertebra or to the left of the spine and then somewhat lower, between the seventh dorsal and first lumbar vertebra." The pain does not move with the movements of the organ during inspiration and expiration. 

The pain in this case is generally associated with the region of the stomach.
It will be worth while to consider here the value of pain as a symptom and see whether there are any facts concerning the character or position of the pain which will guide us in coming to a conclusion as to the character or position of the ulcers.

In this regard to the character of the pain, there is nothing that, in the whole of Hebra's works, will help us in forming an opinion as to the character of the ulcers. There is no special kind of pain connected with any special kind of ulcer, nor is the intensity of the pain in proportion to the size of the ulcer. Many small ulcers cause more acute suffering, while some large ones are attended with only slight discomfort.

But in regard to the position of the pain, there are some facts within our knowledge that may help us considerably in localising the position of the ulcers and I think we may safely predict that when this matter has been more thoroughly worked out,
we shall be able to diagnose accurately the position of the ulcer in nearly every case.

This question has already received some attention and various suggestions have been put forward, but most of them fail when put to the test of practical experience.

1. It has been supposed that if the pain came on immediately after taking food, it was due to the ulcer being situated at the cardiac end, whereas if it was delayed some time it was inferred indicatively that the pyloric end of the stomach was affected. But there is no evidence in support of this, and the time after food at which the pain came on is no guide to the position of the ulcer.

2. Berilson thought that perhaps the position assumed by the patient during a paroxysm of pain might be some help in diagnosing the position of the ulcer. He found that in two-thirds of his cases the pain was influenced by position and he concluded after comparing the effect...
of posture during life and the appearance seen after death. That, if relief was obtained by lying on the right side, the ulcer was probably at the cardiac end, if by lying on the left side, the pyloric end was affected; if the prone position gave ease that the ulcer was on the posterior wall, if the supine it was most likely on the anterior wall. But in some cases he found that the posture assumed was no guide and occasionally, it gave contradictory results.

(3). He also suggested that if the pain was referred to one or other hypochondrium the ulcer was more likely in the corresponding region of the stomach. He found this to be true in about twenty cases.

(4). He points out that the cardia can be found that accompanies gastric distention occupies one or more of the three areas corresponding to the supply of the seventh, eighth and ninth dorsal segments and he is inclined to believe that the sixth dorsal area is associated with disease in the lower part of the esophagus and
that when it and the seventh are affected, the disease is near the cardiac orifice and that pain in the ninth dorsal area is associated with disease near the pyloric orifice. He forewarned, however, no post-mortem needs to prove this.

Cancer only rarely occurs in which one has an opportunity of noting accurately the area of tenderness during life and the position of the ulcer after death. In many of the fatal cases, the patient is far too ill to give an exact account or bear a long examination and in others no post-mortem can be obtained. In the many recorded cases in which an operation has been performed for acute perforating ulcer, the pain is generally only roughly stated to have been "in the epigastricum" or "over the region of the stomach" and it has not fallen to my lot to have an opportunity of verifying this supposition. Since reading his paper, Prof. MacKenzie gave an account of three cases in which he was able to note exactly the position of the
pains and also the position of the ulcers and I think his cerebellum-suprarenal view. In one case the ulcer was at the cardiac end and the point of greatest intensity of the pain corresponded with the 11th-12th. In the other two cases, in which the ulcer was at or near the pylorus, the point of greatest intensity, though not exactly corresponded with the 11th-12th, was situated within or nearly within the ninth-dorsal area.

It would be interesting to know whether, in those cases in which the pain in the back is referred as high as the fifth or sixth-dorsal vertebra, the ulcer is at the cardiac end, while in those in which it is referred as low as the last-dorsal or first-lumbar vertebra, it is near the pylorus. In one of Mackenzie's cases in which the position of the back pain is noted, the head opposite was the case. The ulcer was at the pylorus, whereas the pain was over the 11th and 12th-dorsal vertebra.
The pain in the back is sometimes referred to the right of the spine, sometimes to the left, and sometimes over it. But pain to the right side does not appear to be particularly associated with ulceration of the pyloric end or pain to the left side with ulcer at the cardiac end. When we remember that it is the left vagus nerve that supplies the front of the organ and the right vagus supplies the posterior surface, we might not unreasonably expect to find that when the pain was referred to the left of the spine that the ulcer would be on the anterior wall, whereas when it was referred to the right of the spine that the ulcer would be on the posterior wall. This is however mere supposition and I have no cases to prove or disprove it.

We are not at present in possession of data sufficient to enable us to discern the position of the ulcer from the position of the pain, but we see on what lines we ought to work. What we want is the collection and comparison of a large
a large number of cases in which both the position of the pain and the position of the ulcer have been accurately noted.

There is, however, I think one conclusion that we may draw from the facts alreadybefore, and that is that the pain in gastric ulcer is not localized in the ulcer itself but is referred "pain." This view is supported by the following facts:

1. The position of the pain and the position of the ulcer do not correspond. In nearly all cases the pain is referred to the epigastria region wherever the ulcer may be and even when the pain is localized elsewhere the epigastric pain is there as well.

2. There is generally some hyperesthesia at the site of the pain.

3. The pain does not move with the movements of the organ during respiration and positional.
Fig. 4.

To show the distribution of the segmental areas on the front of the body. On the right side are the maximum spots (after head).

Fig. 5.

To show the distribution of the segmental areas on the back. On the left hand side are the maximum spots. (after head).

Fig. 6.
Case 1. Shaded area shows site of pain. X corresponds to the position of the clavus (left truncus).

Fig. 7.
Case 2. Shaded area shows site of pain. X corresponds to the position of the clavus (left truncus).

Fig. 8.
Case 3. Shaded area shows site of pain. X corresponds to the position of the clavus (left truncus).
Hoemorrhage.

Martin found haemorrhage occurred in 144 of his 171 cases, a percentage of 84. He admits this figure is probably too high, as a large number of cases are admitted into hospital only when they are the subjects of an attack of hyperpyrexia. Making allowance for this, he thinks that the percentage of cases in which this symptom occurs cannot fall short of 80.

Bennett thought severe haemorrhage did not occur in more than one third of the cases in ordinary practice. He found it to be four times as frequent in males as in females.

I think Martin's calculation of 80 per cent. still too high. It is very difficult to form an exact estimate and we are more likely to err on the side of a too high than a too low percentage, as many cases of haemorrhage pass unnoticed. Whereas when haemorrhage occurs this is less likely to happen.
The hemonchus varies very much in amount. There may be only a slight capillary ooze and if no vomiting is present the hemonchus may be overlooked unless the stools are carefully examined. If vomiting is present a microscopic examination will reveal the blood corpuscles. When there is a larger hemonchus it generally excites vomiting. If the vomiting takes place immediately the blood is little altered. When it has remained in the stomach some time it acquires the characteristic "coffee grounds" appearance, due to the presence of haematin formed by the action of the gastric juice on the haemolytic matter of the blood. When hemonchus takes place from a large artery such as branches of the splenic, coronary or celiac, it is very profuse and may be rapidly fatal. The blood vomited being bright and arterial. Immediately fatal hemonchus is however rare. It may leave the mot-
Hemorrhages. Anemia, the patient being bleached and is apt to turn at intervals. Dealt from hemorrhage takes place in from 3 to 5 per cent. of all cases. Coercion hemorrhage is generally followed by diarrhea, the feces being black and tarry. In some very exceptional cases the blood passed per annum may be bright red and unaltered. This was the case in the patient already referred to who died from hemorrhage from an aneurysm in the pancreas.

Hemorrhages may occur at an early stage of the disease, frequent symptoms being absent or they may have been only those of slight dyspepsia. There is generally some feeling of faintness and dizziness and a peculiar taste in the mouth just before the vomiting of blood takes place. Death may take place without any blood being vomited, the stomach and intestines being subsequently found distended with blood.
It generally occurs soon after a meal when it is naturally brought about by the flush of blood to the stomach and its movements during digestion. Or it may follow violent emotion. It has been known to follow careless manipulation during the examination of the patient.

Such fields have seen three cases in which convulsions occurred as a consequence of melancholy.

Vomiting.

This symptom is rarely altogether absent except in some cases of perforation. It occurs next to pain in frequency but has not the same diagnostic value.

It generally occurs when the pain has reached its height and gives immediate, though not always complete, relief and it is not as a rule attended with much effort or retching. As the pain reaches its height at various periods after a meal so
the vomiting may be almost immediate or not for some time after eating. The character of the vomited matter will vary with the time that the vomiting occurs. When immediately after a meal, the food is returned little altered. When not for some hours the vomit is more and may contain an excess of hydrochloric acid. It is very rarely contains bile. Sarcina may be present when there is dilatation. Occasionally it is alkaline, this being due to the large quantities of saliva that have been produced and swallowed before vomiting takes place. Both kinds of vomiting may be present in the same patient during one attack of illness. Vomiting is more commonly associated with clearness near view of the entrance, especially if phlegm and it seems when a gastric discontinuance has developed.

Ammorhea occurs so frequently along with clear of the stomach that it is often described as a symptom, but it cannot be considered a symptom in the
true sense of the word. It cannot be shown to have any direct connection with the ulcer. Some cases especially the chronic menstruation regularly throughout the attack. It is not commonly associated with the ulcer occurring in young chronically girls, but even then it will sometimes be found to be menstruating regularly. It cannot of course be present in every case of gastric ulcer.

The bowels are generally constipated, though as previously mentioned diarrhea is common after hemorrhage. Ulcers situated in the duodenum give rise to diarrhea much more frequently than ulcers in the stomach.

The appetite is generally poor. The patient is often afraid to eat because of the pain that he knows will follow. This is generally increased when catarrh is present. The appetite
fains.

The tongue is usually clean. When there is catarrh as well, it is coated.


Flatuscence may be present and give rise to much pain and distress.

Dever is absent unless there be some complication.

General Dehydration. In the early stage, there is not much wasting, but pain, frequent haemorrhage, vomiting and the smaller amount of food taken gradually produce chronic anaemia and in long standing cases, the condition becomes one of chronic starvation. The anaemia may be very marked. Olet has found in one case the blood count as low as 700,000 per c. mm. 

Boinard thought he could recognize a peculiar physiognomy in patients suffering from ulcer of the stomach.
and that he had "sometimes been
frightened by the mere
sight of the patients in a crowded
hospital or patient room." But it
is probably not distinguishable from
the physiognomy of people suffering
from chronic dyspeptic troubles gener-
ally.

Acetone and diacetic acid may
be found in the urine of patients
suffering from gastric ulcer. They
may give rise to no symptoms or
there may be severe pain, persistent
vomiting, headache and giddiness,
or even toxic symptoms resembling
diabetic coma.
The course and duration are extremely variable. In some cases of acute perforating ulcer the duration is no doubt very short — a few days or perhaps even only a few hours; but as the absence of previous symptoms does not necessarily negative the previous existence of an ulcer, it is impossible to say in any given case how long the ulcer may have existed. In chronic cases the symptoms may be present more or less continuously for many years. Brittin had one case of 35 years, two of 30 years and several of many years duration. Haber has some cases of an ulcer which the ulcer occurred in the same pregnancy or shortly after confinement.

Many cases end in Complete Recovery. Brittin thought that about half underwent a spontaneous cure. It may prove fatal in various ways; by perforation, hemorrhage, exhaustion or some complication.
Dagge found death from perforation 65 cases in 9 ml of 20 deaths, from hemorrhage 8 ml of 20 and from ulceration 3 ml of 20. Hataiho found 11, 10, 3 respectively, ml of 24 deaths.

Many cases exhibit some prominent feature throughout the attack such as hemorrhage or dyspeptic symptoms and this has led some authors to enumerate different varieties, such as the acute hemorrhagic, the chronic hemorrhagic, the dyspeptic, the ulcerous, and so on.

But it does not seem necessary to describe any separate forms and only refers to complicated matters.
Results and complications.

The ulcer may cicatrize. This is the most frequent result. When the ulcer has been small and not deep the ulcer will be small and scarcely visible, and it is not improbable that many heal without producing visible cicatrices. But if it has been extensive or long in healing, the scar will by its contraction produce more or less deformity of the stomach and perhaps alter its relations to the surrounding organs. There may be only slight puckering but if the ulcer has been near the pylorus; hernia will result. While a cicatrix in the middle may produce "hour-glass" contraction. Perhaps pains continuing for some time after we have reason for believing the ulcer healed may be due to nerve filaments being involved in the cicatrice. Sometimes healing takes place on one side while on the other the ulcer is spreading.
Relapse as a rule follow some indiscretion in diet or it may be brought on by worry or overwork, but in some cases we can find no reason for it. It is difficult to say whether the relapse is due to the opening up of an old ulcer or to the formation of a new one. The tendency to relapse is greater in males. It may occur at varying intervals of months or years and in some cases the relapse takes place at the same time of the year as the first attack. It may recur in successive pregnancies as already mentioned.

Cancer. Pierre Marie thought that ulcer and cancer were antagonistic, but Brunton considered that ulcer had no protective influence against cancer and that one might pass into the other, and it is now an undisputed fact that a cancerous growth may take place from an ulcer. Several cases have been observed by various authors.
Adhesions may form to the neighboring organs. The position of the adhesions and the organ affected will of course vary with the position of the ulcer. As the ulcer may be found in any part of the stomach so may every organ that has anatomical relations with the stomach become adherent to it. But as ulcers are much more frequent in some parts than others so adhesions will be found between the stomach and some organs more frequently than between the stomach and other organs. We find, therefore, that the pancreas is the most commonly affected, the left lobe of the liver next and occasionally the spleen. More rarely it is the anterior abdominal wall, the colon, the duodenum, the gall bladder or the diaphragm. Sometimes adhesions break down again and allow of the escape of the gastric contents within the walls.
Perforation.

Of all the complications this is the most fatal and the most to be feared. Brinton found it occurred in 13.4 per cent. of all cases, and that of 234 deaths from it there were 160 females and 74 males (that is to say that it occurs in about the same percentage in both sexes) and that the liability to it decreased as age advanced. The oldest case of perforation he knew of was a man aged 62, and the youngest were a girl eight and a boy of nine.

It occurs much more frequently in young women and when the ulcer is on the anterior surface. Brinton calculated that one of one hundred ulcers on the anterior surface, eight out of one hundred would probably perforate. The reason of the frequent perforation in this position is probably due to the constant movement of the abdominal wall preventing the formation of adhesions.
It generally takes place suddenly and without much warning; often after a full meal or some excitement, such as coughing, sneezing, vomiting or lifting a heavy weight. Sometimes perforation is the first indication of the existence of an ulcer, the patient having been apparently in perfect health. In other cases the ulcer has been known to exist for months or years.

At the moment when the perforation occurs there may be a sensation of something having given way in the abdomen. More usually the patient is suddenly seized with acute pain, generally in the epigastrium, followed by the symptoms of shock. The face has an expression of intense anxiety, and the skin is covered with a cold, clammy sweat. The pain gradually extends over the abdomen and sometimes up to the neck and shoulders or even down the arm. It may start in the right iliac fossa and simulate appendicitis, or be confined to the lower half of the abdomen.
Barber relates the case of a girl aged 24 who suffered from a right-inguinal hernia for which she wore a truss. One night, contrary to her usual custom, she removed the truss and the rupture came down. She pushed it back. Shortly afterwards, on turning over in bed she was seized with violent pain in the right inguinal region. A provisional diagnosis of reduction en masse or appendicitis was made. But on opening the abdomen, there was no evidence of strangulation or of appendicitis. There was a perforation on the anterior wall of the stomach near the pylorus.

The subsequent symptoms depended upon whether the gastric contents are prevented from escaping by means of adhesions or whether they find their way into the general peritoneal cavity. The former is more likely to happen when the perforation is on the posterior wall as adhesions form here more readily than on the anterior wall, for reasons already mentioned.
Sometimes slight adhesions form, which breaking down again subsequently, permit the escape of the gastric contents and a general peritonitis is set up. Or again the adhesions while permitting the gastric contents to escape may be so arranged as to prevent their entering the general peritoneal cavity, a local abscess then results. This may be situated in front of the stomach and communicating externally give rise to a gastric fistula or behind the stomach causing what is known as a sub-phrenic abscess. In rare cases the perforation may take place into the pericardium, the gall bladder, the colon or some other organ, adhesions having previously formed between the stomach and the affected organ.

The immediate effect of the escape of the contents of the stomach is shock and this may be so intense that the patient rapidly succumbs to it. Possibly some cases of sudden death are due to this. If the shock be
recovered from the patient may die from toxemia. In these cases a post-mortem examination reveals no change in the peritoneum and death is supposed to be due to the absorption of toxic agents. In a general peritonitis sets in with its usual symptoms, the patient lies on his back, with his knees drawn up and his hands on his head, his features are flushed and anxious looking, his tongue dry and his excessively thin. His breathing is short and coarse, his pulse small, rapid and airy, his abdomen at first retracted, sometimes almost to the spine, afterwards excessively tender and sym pathetic. The temperature varies and is not as a rule very high. The liver and spleen dulness are nearly or quite obliterated and there may be signs of fluid in the flanks. Death usually takes place in from twenty-four to thirty-six hours.

Cases of recovery after all the
Signs of perforation have been recorded but they are rare and there is always room for doubt whether the perforation was complete or the diagnosis correct. I have seen one case of ulcer in a woman who was suddenly seized with all the signs of perforation and in whom the symptoms of a gastric ulcer had existed for some time previously. A favourable result is more likely to occur when the stomach is empty and the patient in bed or at rest at the time.

The diagnosis of perforation is difficult sometimes, especially if there have been no precursor symptoms. We have already seen that the pain may be referred to the lower regions of the abdomen and thus give rise to suspicion of appendicitis, or even strangulated hernia. It may also be referred to the chest and be mistaken for a commencing pneumonia or pleurisy. Perforation due to malignant
disease will generally be distinguished by the previous history and wasting.

But it must be remembered that perforation due to malignant disease may take place at a very early stage and before the growth has caused wasting or any other symptom. A patient suffering from an attack of renal or hepatic colic comes about instead of lying still on his back, his pulse is not quickened, he finds relief from pressure and sometimes there is a history of previous attack followed by jaundice or hematuria. There is not that sudden profound illness in which the patient is apparently dying, which is so marked in cases of perforation. Very rarely it may happen that a hyperplastic ulcer perforates in a patient who is not ill enough to be in bed or under treatment. It is common for it to be impossible to distinguish this from the perforation of gastric ulcer. Coming on as it so often does after a meal, perforation may give rise to suspicions of poisoning.
In a certain number of cases we can not do more than diagnose some sudden and grave emergency in the abdominal cavity, demanding that cavity should, if possible, be opened without delay. When the opening has been made the presence of a fluid containing hydrochloric acid, particles of food, and perhaps smelling of peppermint or other drug taken to relieve pain, indicates a perforation of the stomach or first part of the duodenum.

Dilatation of the stomach occurs under various circumstances. Haberhorn found it in 18 cases out of 60 (14 males and 4 females) the average age being 52 or 53. It may be due to narrowing of the pylorus from cicatrisation of an ulcer or, what is more common, to muscular weakness or paralytic from exhaustion of the patient. Roberts has seen ulcers follow dilatation. It may therefore occur as a result, a complication or a cause of the ulcer.
Hourglass contraction is not very common and not easy to diagnose during life. It is seen with gas upright, reveals the shape of the organ. The only point that serves to distinguish it is that there may be all the signs of dilatation, but when large quantities of liquid have been introduced into the stomach, little can be withdrawn by the pump as the fluid has passed beyond the contraction into the further compartment.

Ewald has seen a number of cases in which the patient finally died with symptoms of pernicius anaemia.

Dutchfield has seen ulcers in one case.

Parotitis may occur as a complication in many abdominal diseases and may be present with ulcer of the stomach.

Endocarditis has already been alluded to.
Scapnum.

When all the classical symptoms—pain after food, vomiting which refuses it, hæmatemesis (with or without melena)—are present, the diagnosis is easy. But the combination of symptoms is by no means always present, and we have already seen that any one of them may be absent through the great hæmorrhage and sometimes during the whole period of the illness. And again they are all common to other diseases, particularly of the stomach, so that in some cases it is only by taking into account the whole history of the attack, the age, sex, symptoms and effects of treatment, that a correct diagnosis can be arrived at.

Cancer of the stomach is perhaps the most likely disease to be confounded with it. As a rule the diagnosis is not difficult, but occasionally it is so. Cancer is a disease of middle
age and often if the patient is under to the symptoms are more likely due to ulcer. Cancer is progressive, though many, perhaps slight. Improvement at first under treatment, while cancer generally (tends) considerably under treatment. Though not readily explained. The pain of cancer is more continuous and less connected with eating food, but it may occur in severe hysteresis. The vomiting, even cancer is not so definitely associated with pain or eating, and the vomiting matter contains much hydrochloric acid and products of fermentation. Sometimes, and occasionally portions of growth. The hydrochloric acid may be taken when the cancer has developed from an ulcer. The vomiting of blood in large quantities at an early stage of the disease is more likely to occur in ulcer, whereas the vomiting of “coffee grounds” is more frequent in cancer. The presence of a palpable
Cancer is an illness of cancer, though occasionally a cancer may be felt in cancer of the cancer due to an absence or the imbalancing together of the organs by adhesion. We must guard against being misled by spasm of the bladder, which may simulate a cancer. But in some cases of cancer no cancer can be felt and it is especially likely to occur when the cancer is at the cardiac end and during those cases that present the greatest difficulty as the fund of the cancer, or immediate after taking food and so simulate the pain of an ulcer. In the case of a man I recently attended, the symptoms at first appeared to be due to an ulcer, but as he showed no signs of emaciation under treatment, I began to fear he must be suffering from cancer though no cancer could be felt. After about ten weeks from the time he first sought advice, I did succeed in learning
The case was. He died almost 6 months after. Wasting is more pronounced and rapid in cancer. If the patient live more than a year it is in favour of ulcer. Secondary cancer may occur in other parts and should be looked for. Oswald says a fluid in the ligamentum falciforme and a chain of glands near by are liable to be sympatheically contractile and sensitive in presence of cases of ulcer and they may be elicited as small tangents. The sign of hagel results at the lower edge of the stomach.

It is said that the lenovery time itce normally takes place after a meal is absent in case of farbic Cancer. If this were invariably true it would be a valuable diagnostic sign but it is not. It is absent in other diseases besides Cancer and may be present in Cancer. This Test therefore, though apparently as reliable as the hydrochloric acid test, is like the latter, fallible both in its positive and negative sides.
From depressed pepitas or stomach, in
the diagnosis may be puzzling, but
the following points may serve to di-
cinque them. Abdominal pain will be
absent. There is not the same connec-
tion between the pain and vomiting and
the taking of food as in ulcer. Gastralgia
is more likely to occur when the stom-
ach is empty and is relieved by tak-
ing food and hot drinks. Which we
have seen in exceptional cases of
ulcer. Other nervous symptoms are
generally present and heat may
does not have such good results as in
the case of ulcer.

In Gastric Catarrh the pain is not
so severe and is more diffused. The
malady is, if it occurs at all, still
small and amoeboid. The form is
not contains muscus and no exces of
acid. The condition is much Ca
dative. It must be remembered that Cataries
and ulcer may be present together.
Henshaw gives a case of
Common
take in half a teaspoon of water before breakfast. When the pain occurs the case, he thinks, is probably one of ulcer, when no pain, it is one of dyspepsia only.

The passage of a gall stone is not generally difficult to distinguish, but, as it often comes on after a meal, it may occasionally mislead. The onset and termination are very sudden and there is often no loss of a previous attack. The gall bladder will be tender. The patient is more restless, coming from side to side and perhaps rolling on the floor. The subsequent jaundice, pale face and discoloration of gall stones in the motion, decide the matter.

The Gastastis crisis of locomotor ataxia of other nervous diseases would be distinguished by the presence of other symptoms of their disorder, such as lightning pains &c.
Duodenal ulcer cannot often be
de distinguished from gastric ulcer.
Frequently there are no symptoms at
all before perforation or hemorrhage
occur and then there is no clue to the
position of the ulcer. Duodenal ulcer
may be suspected when pain comes
on to the right of the epigastrium some
time after eating food. When much
blood is passed by the subject while
little or none is vomited or when
with symptoms of gastric ulcer, the
vomits have an alkaline reaction con-
tains bile and digests food (Boos)
It occurs more often in males than
in females.

A movable kidney may produce
gastrical pains and vomiting and
so simulate a gastric ulcer. A hi-
murmed examination clean up the
diagnosis.

Hemorrhage from a gastric ulcer
has been mistaken for that from
Chlorotic girls frequently complain of acute pain coming on immediately after food and in the absence of other signs. It is often difficult to say whether the pain is or is not due to the presence of an ulcer. It often only lasts a day or two.

Habitual amenorrhea may take place in young women suffering from anemia and this bears inspection of ulcer. They can only be diagnosed by the absence of other symptoms of ulcer.
The Prognosis on the Whole is favor- 

able. The presence of any chronic 
complain, such as caries of the 
liver, heart disease, vesical disease, 
or phthisis makes the out-look more 
grave. As we have no means of 
accurately diagnosing the depth or 
position of the ulcer and the form of 
knowing how far we may be in 
haemorrhage or perforation, the pro-

gnosis should always be guarded 
as these accidents may occur when 
the patient appears to be improving 
favourably. There is little chance 
of recovery, yet the well to-do, who 
can afford to carry on the treat-
m ent.- Haemorrhage is rarely fatal 
the first time and no matter how 
severe it may have been or how 
acute the condition of the patient, 
there is always some formula for him 
for his recovery. Relapse are frequent 
even when all possible care has been 
taken, but they are generally brought 
in by overwork or undue eating in 

"For the rest, the personal elements will chiefly influence the future course. A man of phlegmatic temperament, who is amenable to reason and obeys the instructions of his advisers, will have a better chance of ultimate recovery than one of a nervous and irritable disposition, who is impatient of restraint and careless of his habits and mode of life."
Diet and Management.

In treating an ulcer of the stomach, there are two objects to be borne in mind: to heal the ulcer and to relieve the various symptoms as they arise. The treatment will be based on a theory of healing the ulcer with regularity of the daily routine and the regularity of the digestive process. The constant movement of the stomach during digestion and locomotion, the presence of other disorders of the stomach, such as constipation and diarrhoea, and the various abnormal states of the blood and circulation. Until these attacks are removed, there is little hope of any improvement. The future progress of the case will depend on the thoroughness with which treatment is instituted with a view to the removal of these attacks. If carried out, the result of the stomach and body, general.
by is essential. The patient—must be kept to bed and remain there for a month or six weeks or until the time that
there is such improvement in the symptom
as to warrant an exit concluding that
the ulcer is healed. They must be kept
there a durable pump to do good
so required to sustain life and the
endurance than when a patient is
moving about, and it is from help
in attaining one of these means, viz.
to grow the stomach a little foods as
possible.

When I am has been a recent1 hormone
than, when we believe the ulcer has
been forced or is threatening to return.
When motion is unendurable, or when
pain is severe and unrelied by ordi-
nary means, except no food whatever must
be allowed to the ulcer the stomach.
The patient should be fed by a thin
uninten. Some or five days will gen-
ally be long enough and it is not
advisable to keep it up longer than
this unless necessary or the amount
of insurgents than can be absorbed by the dream is small and not ade-
quately to the requirements of the body, the
patient soon begins to weaken and the
consuming for something to eat becomes
inconceivable. This may be relieved by
lucrative small piece of ice. Provide
direction as to time and amount of
moisture. This should be followed by a
vitamin drink be obtained to carry
them out. The emenata should be
given about every four hours and
one cup of four ounces of peplosed beef
liver or fruit or the same quantity of
pepoloised milk with a little sugar and
salt. A tablespoonful of barley may
be added if thin or any indication for
sweatiness. The emenata are to be
given with a large Jaques catheter and
a funnel. Enzal maintain that it
is not necessary to use peplosed albumen
as the lower part of the intestine can
absorb unchanged miles of egg and under
it ample in the metabolism of the body. The victim must be treated reli-
will be made warm with about fifteen minutes before each enema is given. If this is done the victim will stand. This method of instillation for some weeks without becoming irritating. He may now begin to take a little nourishment by the mouth. Pepperized milk, taken to form ounces at a time and at intervals of three or four hours or a little clear soup should be tried first. If this is well borne we may try milk and water, mixture of this. If there is any tendency to acidity it is better to add a little soda or lime water. A mineral enema may still be given twice or thrice a day if well borne. He should be left off entirely as soon as he can take the food taken by the stomach is being digested and producing residual symptoms. Some bland easily digested food, such as Bengal or Indian, and some beef-tea, such as raw beef juice, may now be ordered. At the end of three weeks or a month painless
inations. Such as an 114. 114.
or capriccioso, may be allowed and at the end of another night fish,
roasted chicken or stew meat with a little stale bread. In any type of a
relapse the food should be back to milk or Benjamin food according to
improvement. Continues a mutton
chop may be taken, but for a
month and tea had better be avoided
for some months.

This treatment is unknown to the
patient and it is as well to warn
him beforehand of the time he will have
its duration and the great risk
he runs in not strictly adhering to
the rules of diet laid down for him.

Patients who have suffered from a
febrile ulcer should be made to thoroughly understand the fear of
relapse, even years after all the
symptoms have disappeared. They should
be warned against over indulgence
in indigestible foods and drinks and
against over exertion. Women should
also be taught special care during menstrua-

li and pneumonia.

In the venous case we must begin with preparation before and proceed in the same way as already described.

On the advisability of washing our stomach there is much difference of opinion; some authors strongly recommend it, while others advise against it. It cannot be altogether unattended with risk and the question comes to be, is the benefit likely to follow its use sufficient to make it worth while to run that risk? In simple uncomplicated cases we may follow as the text, especially as they generally improve rapidly under ordinary methods of treatment. But when the ulcer has been of long standing and is complicated with enteritis or ultrabac, washing our stomach is worth, of a trial and we may expect to see some improvement following its use.
*Songs have probably no direct action on the ulcer itself and do good only by removing the already mentioned obstacles to healing. Bismuth, in the form of carbomali or subnitrate, is the most generally employed remedy. How it acts is not certain. It is usually given in doses of from 60 to 150 grains, but much larger doses are recommended by some. If the benefit is due to it forming a protective coating to the ulcer, it is difficult to conceive how the smaller dose can do any good, as the amount that comes in contact with the ulcer must be very small, but it is pretty well agreed that the small doses do act beneficially and that for this reason it must conclude that they have some other action. Bismuth can be given as a powder or suspended in syrup. Epsom salt is in favour of large doses and, on account of the high price of the Bismuth salts, suggests the use of the bicarbonate of calcium or lead.

In alkaline carbomali can been
Useful and no danger act by diminishing the acidity of the gastric juice. They probably do good to the system generally as well. Be careful of soda as it is most used and is frequently given along with the Bismuth. The mineral should be taken before meals.

Sulphur was formerly much used, but it has fallen into disuse on account of its sometimes causing a permanent discolouration of the skin. Sulphur has been administered for a long time. This seems to be the fate of most drugs. Some are put on an account of its virtue, adding a long list of "successful cases" (usually by it). It is taken up and used more or less indiscriminately. Perhaps, instead of "alarming symptoms" or anything during its administration and this is followed by a whole host of cases in which unlooked for and hasty effects are attributed to its use, until it seems as if the drug were bad merely because within some harm
being done and so we become afraid of using it at all. So with initial of silver. Because a few cases of angina occurred, we have not, if it is a really valuable drug. stop using it altogether. While taking it, the patient should be kept under observation and the administration of the drug should be discontinued after a few weeks. Often find that strongly on its former, especially he recommends it for hospital and out patients, who cannot afford to undergo the daily treatment. He has often found illustrated pain and other symptoms better than morphine and, although he has seen in 200 cases, he has never seen a case of angina. I agree that it certainly sometimes relieved pain. In other cases, however, thought it absolutely inert. It should be given in a pain dose or pill or solution. Doses are localised in cases by giving them his bracket duration. (2 to 3 grams) three times a day for about ten days and then
he substituted pills of nitral.-
Silica (or semi-precious) for two days, resuming the bismitt again for four.

On four days, after this, for three or four days, he gave on semi-precious of calcium bismitt daily. This practice he repeated for three or four months. How does

intralicl silica act? Like the bismitt balls, it has been supposed to form a protective covering at the ulcer but the amount given on one alone

do small that it would be necessary for the whole dose to find its way to the ulcer to produce such an effect.

Bromide: Considered semi-precious in some form external to the cure of an ulcer of the stomach in the same way as some physicians considered it essential to the cure of an ulcer of the leg.
The thought it had some special action beyond earning pain and sickness and he especially recommended it in cases of long standing and for patients with exhausted constitutions.

Carbonated salts in four times each and
and is certainly of beneficial. It may be given in doses of one or two teaspoonful in a tablespoon of warm water before breakfast. Its action is probably due to its relieving, purging, alkalizing and neutralizing properties. An artificial salt consisting of sulphate of soda 50 parts, calcium chloride 10 parts and sodium chloride 3 parts may be used instead of the natural salt, and obtain will equal benefit. Its chief disadvantage is that many persons are unable to use it, even in the case of long standing complications with portal congestion and jaundice, etc.

Bromate mixture of caustic soda and iodide of potassium will some times improve such cases as calamine or gelatin be successful in chronic cases.

Chlorotic cases will be improved by a Course of iron (the ammonio-citrate, or the Cr. ferri perchlor), as soon as the symptoms of ulcer have passed off, and they should be carefully watched for any return of chronic symptoms.
such as amanita or lethaire, as
these may be the forerunners of a relapse.

Relief of Symptoms:

Pain. This symptom will often disappear as soon as the patient is put to bed and properly dieted. In many cases opium is some good to near
by 6 or 8 grains sometimes even 1/2 an ounce or as a rule of 1/2 an ounce or half a hypodermic of morphia (8 gr.).

As already mentioned, calomel of sulphate sometimes has a good or even better effect. External application, in the
form of linseed poultice, warm compresses, hot steam bags or steam filled with
hot water, sometimes gives relief. When
it is very severe and unrelieved by
these means it is as well to try not
failing for a few days, but I have
seen at least one case in which the
pain was made worse every time an
enema was given. Whipple also ob-
erved this in a case of chronic colitis.

Fluctuation is often the cause of
much pain and distress.
often when the patient is being fed by the victim only. It is best relieved by peppermint or chloroform oak. I have seen pain instantly relieved by peppermint. When ordinary doses of morphine had no effect.

Vomiting should be treated according to its nature. That kind that occurs soon after a meal and when the pain has reached its height is best relieved by cinchona. The amount of food eaten, giving the victim of his smell and soda and in very obstinate cases, feeding the victim only for a few days. In chronic cases when the vomiting has not such marked relation to the digestion of food, carbonated soda and the mixture of laudanum and drops of potassium with tincture of calomel will generally produce beneficial results. If these fail to effect a lasting cure the stomach might do good by removing the outer residue of undigested food. I have been the most

-29-
**Hemorrhage.**

When this is only slight, the ordinary treatment should be continued according to the stage of the disease and the nature of the other symptoms. If a severe hemorrhage has recently taken place and the patient must be at once restored to life and ease suffered, my peregrinations of eight times may be tried but an of about five times. It is best to administer a hypodermic injection of morphia and, of course, keep the patient absolutely quiet in bed. The food of all kinds must be prohibited. Local antiseptics such as a solution of carbolic, caustic or sulfuric acid are of advantage and had better be withheld. Drs. Gaskell recommend Chamtpalin, 20 to 30 grains in a capsule or mixture of equal parts of egg, but if the patient continuing its administration had better be stopped. If the patient seems likely to succumb the loma blood transfusion may be tried. Hemorrhage is rarely fatal at the first time.
Constipation is best relieved at first by enemata. Later, Castor seed, stools, or Cascara may be tried. Strong purgatives should always be avoided. Bowls may be washed against giving them curds.

Perforation.

As soon as there is any degree of perforation, the patient should be kept under the influence of opiates. As nothing must be given by the mouth, a hypodermic of Morphine must be administered at once. The patient must be kept absolutely still, with a view to preventing any escape of the fecal contents and to allow time for the formation of adhesions, and he must be fed by intravenous enemata. A few days' no strick.-water without further treatment and this is more likely to occur when perforation is on the posterior surface, when the stomach is empty, the opening small, and the patient in bed at the time.
But, as far as the greater majority of cases, death will take place in 24 to 48 hours from peritonitis unless operation measures are immediately adopted. The best chance, therefore, lies in operating as soon as possible and the success of the operation depends a good deal on the shortness of time that is allowed to elapse before doing so. But delay is often unavoidable. There may be delay for doubts in the diagnosis and the medical attendant naturally will delay before advising such desperate measures, or he may be far away from a hospital or from any one accustomed to abdominal surgery, so that by the time the operation is undertaken it is often too late. An ulcer on the anterior surface is of course, much more danger than one on the posterior and it is fortunate from a view of operation, that the perforation, as already stated, generally occurs in the posterior. The rule used to be, to wait until the patient was covered
from the shock, but it has been noticed that the patient's pulse improved as soon as the abdomen was opened and care operated on during Colapso have done well, so that it would seem to join the patient a better chance to diminish it and operate as soon possible.

There are three methods of operating for perforation of the colon. The first is to simply close the perforation by ligation sutures. Again: this may be justified, that the colon sutures were wider area than the perforation and the sutures may fail to hold on the ulcerated tissues. The second and most radical way is to excise the ulcer and bring the healthy edges together by a continuous suture of the peritoneum membrane and submucous tissue and then by a second row of Lambert sutures. Again: this method may be urged, that it involves a considerable time, that a large portion of the stomach may have to be removed and that there
may be Cæsarean hystectomy. The third and most common way is to envisage the whole colon and stomach the healthy stomach wall together with decubitus sutures. If necesary an overlying flap may be sutured over the injured surface. Before closing the abdomen the peritoneum should be thoroughly washed out with sterilised water and the cervix removed by placing the patient in an almost sitting position and sponging out the pelvis, which it will have gravitated. One great 'trick' of the peritoneum is to be appreciable as if only covering the peritoneal surface of the bladder and uterine. The risk of subsequent peritonitis. It is essential that the operation should be done as quickly as possible so as to reduce the shock of the operation to a minimum. After the operation, the patient must be fed for four or five days at least by nutrient enemata. Some operators recommend a longer time but others
maintain that is not necessary. If peritonitis occurs subsequent to operation, Dr. Nevin recommends the use of 1:5000 ichnium hypodermically (80 to 80 to 60 to 40 to 30) and in
dawn on faint. Recommend sulphate of magnesius to be given internally to clear away any accumulation in the alimentary canal.

If operation is decided against or the patient seems to far from to far any hope of recovery he can only

pain opium and alcohol. The end.
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