THE DIAGNOSIS OF DYSPEPSIA IN GENERAL PRACTICE

with notes on a simple

method of

classification.

by

CHARLES MANN FLEMING, M.A., M.B., Ch.B.

Thesis for the Degree of M.D.

August 1932.

ProQuest Number: 13905403

ProQuest.

ProQuest 13905403

VOLUME 1.

C O N T E N T S.

VOLUME 2.

APPENDIX AND CONCLUSIONS.

INTRODUCTION.

DEFINITION.

Dyspepsia shall be taken to include all cases, functional or organic, in which discomfort of any kind occurs during digestion.

OBJECT.

(1) To formulate a routine procedure, suitable for General Practice, in the examination and diagnosis of cases of dyspepsia.

(2) To classify the various kinds of dyspepsia in a way which shall be useful and simple; and which shall describe each condition adequately for the purpose of rational treatment.

(3) To examine the results in 170 cases.

The diagnosis of many diseases has become increasingly more accurate in modern times with the progress of Medicine and Science. Unfortunately this has been attended by little or no simplification of diagnostic methods; but rather the reverse. Thus comes it about that these advances can but seldom be utilised in General Practice. The natural consequence of this has been the growth of Specialism. Much good has no doubt thereby resulted but it has not been an altogether unmixed blessing. One undeniable ill-consequence is the present

uncertain position of the General Practitioner. At one time there was no doubt as to what limitations should be imposed on him: there were none. Now a change has taken place and neither in the profession nor among the laity is there unanimity as to his exact function. This we propose to examine.

Whatever view be held regarding the functions of the General Practitioner it cannot be denied that he has, and must always have, more frequent opportunities than any of his professional brethern of seeing disease in its early stages. Sir Humphrey Rolleston, (1) in discussing the future of general practitioners writes:-- "Under any antici- pated change in their present independent position they would still be in the best possible position to detect and correct the earliest stages of ill-health, when prevention --- the ideal of modern medicine --- is most easily effected." On the General Practitioner, therefore, there must rest the great responsibility of sorting out the really trivial from that which is trivial only in its superficial appearances. And, in so far as he does achieve this or does not, should he be judged as having succeeded or failed. For this is his main function at the present time. And it is by this standard that he judges himself, either consciously or unconsciously. Success in the realm of treatment

may provide a pleasant titillation to his sense of self-esteem. Failures he accounts as little or no reflection; rightly regarding them as often outwith his own province and belonging to that of the consultant. But in the sphere of early diagnosis, whether in diseases such as acute appendicitis or cancer in its incipient stages, he cannot but feel the responsibility is peculiarly his own. We do not wish to convey the impression that we think the consultant has no place in diagnosis. un the contrary it is just here that he is most needed in many cases; but the responsibility of recognising that need is the duty of the General Practitioner and it is one which he must discharge himself.

The complexities of modern diagnostic methods is a great difficulty for a doctor engaged in General Practice. He naturally feels that he should avail himself of every ancillary method that is useful and practicable. But how is he to decide which methods exhibit these qualities? The literature concerning them is so enormous at the present time that he cannot keep abreast of progress. Such text-books or articles as he may consult are all too often written from the viewpoint of hospital or consultant work. It is left to the individual to decide what diagnostic aids he will himself employ. This is not infrequently a matter of much difficulty.

Moreover, many methods and appliances are too obviously time-consuming or expensive; and many call for a degree of skill which can only be acquired by intensive practice.

There is no doubt, for example, as to routine in hospital as regards the diagnosis of a case of dyspepsia. That is common knowledge to all physicians and students. Often these methods, and such appliances as they demand, are used for instructional purposes when their employment is not necessary to complete the diagnosis. It is but natural that, on entering General Practice, the young doctor is in doubt as to what laboratory aids are necessary in a given case. How many General Practitioners have a routine at their fingertips for every problem which presents itself? To have this, is doubtless a counsel of perfection; but an attempt to have such a routine is something more than desirable. Without it, diagnosis is a blind groping in the dark. So much that is trivial is encountered in General Practice that the application of a routine system of investigation is impossible in all cases. There remain, however, those, a not inconsiderable number, where it is vital. Hospital routine, even in such, is often unnecessary and often impracticable. One suitable for General Practice must then be elaborated and it is our main purpose to formulate

such a routine for Dyspepsia and demonstrate its application in 170 cases.

The classification of diseases presents parallel difficulties. For example, such a term as Hyperchlorhydria, apart from other objections with which we shall deal later, presupposes the use of the test-meal. This has a restricted use in practice as compared with hospital. Consequently, it makes the use of the term Hyperchlorhydria unsuitable for that reason. There are many similar instances and our subsidiary purpose is to construct a classification compatible with the means of diagnosis suitable in General Practice and one which at the same time is accurate for all practical purposes.

We have chosen dyspepsia because of its prevalence and importance. No complaint is so common or widespread. In a series of 1,000 consecutive persons presenting themselves to us for consultation,190 had symptoms which they, rightly or wrongly, referred to their digestive tract. This is an experience of all practitioners.

In importance also, these cases rank high. Even the minor ones often have an importance out of proportion to their superficial manifestations, being not uncommonly evidence of serious general ill-health, harmful septic foci, or serious disease

of the stomach or other organs in its early stages.
And it would appear that they, more than any other
group, predispose to the diseases of which the bulk
of the population die.   MacKenzie (2) compared two
series of cases.   The first was constructed from
1,000 cases which were well enough to attend the
consulting room.   The second was one composed of the
proportion of deaths from the principal causes to the
total deaths per 1,000.   In the first series 25 per
cent were diseases of the digestive system.   In the
second, only a little over 6 per cent.   The
suggestion arises, as MacKenzie pointed out, that dis-
ease of the digestive system, by weakening the body,
predisposes to other disease.   The importance of
these cases of early ill-health being dealt with
adequately and immediately is therefore obvious.

It is not our endeavour in this Thesis to
deduce new methods of diagnosis but merely to
examine those available as regards their employment
in General Practice.   Special stress is laid on
clinical methods as these must always be the
General Practitioner's sheet-anchor in diagnosis.
In point of fact, we are trying to build up a system
of investigation which makes the minimum call upon
laboratory or other aids, without excluding these if
their employment is necessary or desirable to obtain
an adequate finding.   We shall be able to compare

(in certain instances) early diagnoses with what were later shown correct and that should not be without diagnostic value.   This has been possible because of record cards which have been kept in our Practice and have therefore enabled comparisons of recent findings to be made with previous ones.   We shall refer to this system later.

The cases we have investigated are, almost entirely, drawn from a mining population.   Doubtless the incidence of the various types of indigestion is different from those which would be found in another grade of society.   The factors operating in these matters, such as social conditions, bad housing, poverty and local habits of living and feeding will be considered at another stage.

Certain unique opportunities in this investigation have been ours and these have not been without advantage.   The Practice is an unopposed one extending over a very wide area and including an extremely large number of patients.   We have thus been enabled to have information regarding almost all the cases of dyspepsia in the district.   Not all those were, in the first instance, personally encountered;   some having been brought to our notice by our colleagues.   In almost every case a personal examination has been made, as indicated in Volume 2.   For the other cases, which we have

included when they showed points of special interest, and for bringing cases to our notice, we take this opportunity of expressing our thanks to our former colleagues, Dr. Gordon Flint and Dr. Charles Seward.

The population, in our area, is unusually static and this also has been advantageous. It has enabled us to have records extending over a number of years of all illnesses necessitating medical attention. In the keeping of these records we have only been personally concerned in the past three years; but they have provided much useful information beginning in many instances as far back as eight years ago.

As regards facilities for investigation, we have been hampered only by our own limited time. Hospital resources have been readily available in necessary cases for aid or confirmation in diagnosis. Almost always these have been obtained in the Edinburgh Royal Infirmary. One advantage, so often taken for granted that it is apt to be forgotten, is the readiness to submit to re-examination, even if free from symptoms, which is perhaps peculiar to this type of practice. This has often made information available which would otherwise have been most difficult to obtain.

Another advantage, not confined to any type of practice but open to all general practition-

ers, has been that of seeing the early beginnings of disease.   It is in this field that the most valuable fruit is to be found but it calls for much careful observation.   Moynihan (3), writing on the treatment of cancer of the stomach and pointing out that the limit of operative measures has now been reached, says that from early diagnosis only is further improvement likely to result.   As he goes on to say, the inaugural stages have passed before appeal is made to final authority (the consultant). He then expresses his belief that from a text-book by a general practitioner, dealing with the meaning, development and interpretation of early symptoms, much needed knowledge might result.   However ambitious a venture for General Practice one may consider this, it at least illustrates an undoubted truth, viz., that the opportunity is there if it can be utilised.

The significance of early symptoms cannot be appreciated unless the mechanism of their pro-duction is understood.   Only then can their future import be realised.   We have therefore dealt at some length with this question in Chapter 2. Observation from the commencement to the end of the disease, and possibly in diseases subsequently occurring in the same patient, is necessary.   And this is possible more often for the General

Practitioner than for any of his professional brethern. We regret that our opportunities have not extended over a sufficiently long period to yield anything of great value in this respect. Perhaps it is also because we have failed to recognise what may have been obvious. But we feel that we are one step nearer to being able to recognise the significance of what may daily be before us when we have put our investigations on a systematic and ordered basis. And it seems to us that before much can be accomplished in the unrivalled but fleeting opportunities of General Practice, the methods employed will have to exhibit these qualities.

CHAPTER 1.

THE STOMACH IN HEALTH.

(1)   Introductory.

(2)   Anatomical.

(3)   Physiological.

        (a) Motor.
        (b) Secretory.
        (c) Sensory.
        (d) Digestive.

(1) INTRODUCTORY.

We include below a short account of the physiology of gastric function because upon it so largely depends the correct diagnosis and treatment of diseases of the stomach. Some anatomical details have been given first to make subsequent description more intelligible. Equally important from the point of view of diagnosis and treatment is the study of abnormal function but this has been dealt with in a separate chapter. An adequate appreciation of symptoms, so important in the study of the early stages of disease, can only be obtained by full consideration of their mechanism of production. Obviously this can only be attained by a knowledge of normal and abnormal function.

(2) ANATOMICAL:

(a) Clinical Sub-Divisions.

The cardiac and pyloric parts of the stomach are the main divisions. The former is again divided into the fundus and body; the fundus is the portion above the level of the cardiac orifice. The body lies entirely to the left of the mid-line. Its axis is directed downwards and slightly forwards. In the erect posture, the body is more or less vertical and somewhat inclined to the right, especially in the male sex. In recumbency, it lies more obliquely.

The division between the body and the pyloric portion is marked by the <u>Incisura angularis</u>, a depression often, though not always, present on the lesser curvature. The pyloric part is divided into the pyloric vestibule and pyloric canal, the termination of which is the pylorus proper. The axis of the vestibule runs upwards and slightly backwards and to the right. The pyloric canal, which begins usually just to the right of the mid-line and is approximately 3/4 of an inch long, points more directly backwards. Its termination, the pylorus, is indicated on the outer surface by the pyloric veins. Occasionally a small constriction can also be made out. Internally it projects into the duodenum and its appearance has aptly been likened to the <u>cervix uteri</u> in the vagina. The sphincter muscle is a thickening of the circular muscle beginning at the pyloric canal and most developed at the pylorus itself. It is not continuous with the circular muscle of the duodenum but is marked off by a septum of connective tissue. The longitudinal muscle is also thickened at the pyloric canal. The superficial fibres continue into the duodenum but the deeper fibres invade the circular muscle for varying distances, even to the sub-mucous coat, thus forming a dilator muscle.
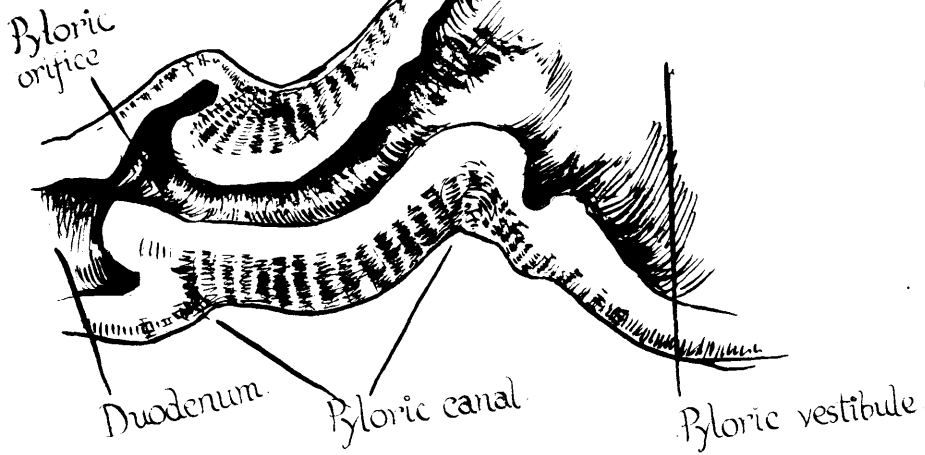
FIGURE 1. Section through the pyloric part of the stomach of a child aged 2 years.
(after Cunningham).
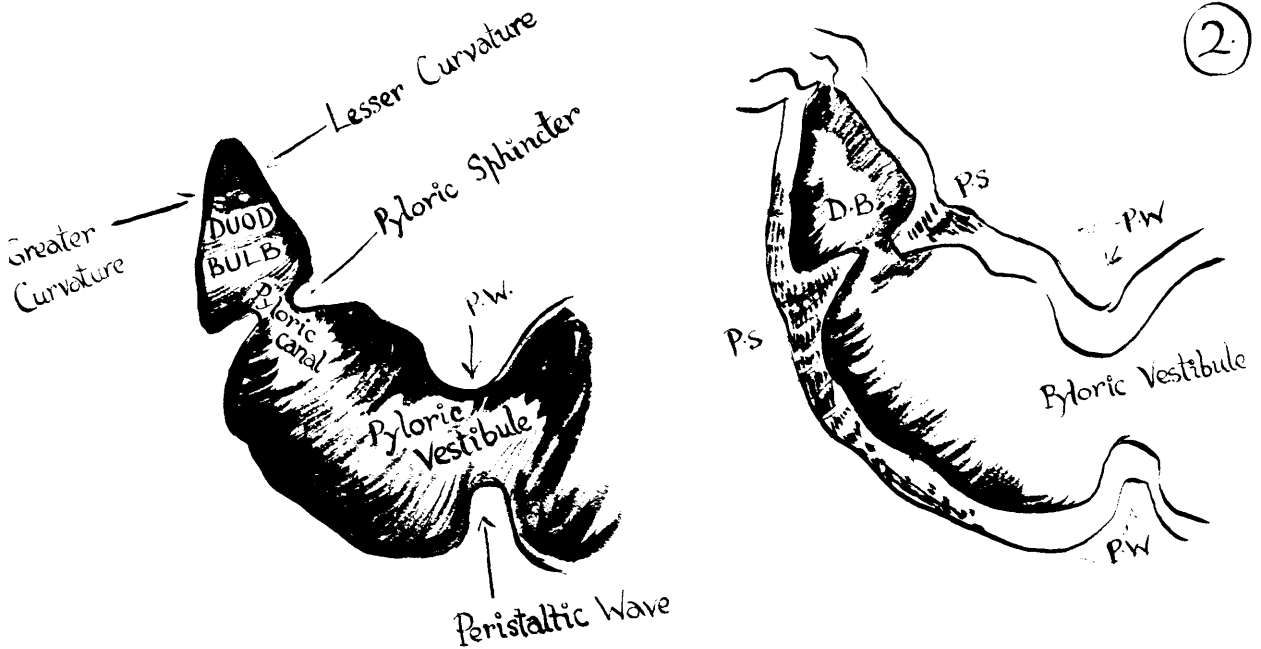
FIGURE 2. Pyloric vestibule and duodenal bulb.
(after Hurst).

(b) Position of the Stomach.

When empty, the upper third of the stom-
ach contains gas and has an inverted pear-shape.
The lower part is tubular, the muscle being con-
tracted and the mucous membrane thrown into folds.
In the recumbent position, the pylorus lies just to
the right of the mid-line, at the level of the
transpyloric plane, i.e., half-way between the
upper border of the manubrium sterni and the upper
border of the symphysis pubis. The greater
curvature reaches a very slightly lower level than
the pylorus. The stomach lies more obliquely than
in the erect posture, in which position the greater
curvature lies at or slightly above the interiliac
line.

When the stomach is partly filled, the
body is uniformly distended and lies in the same
position as when the organ is empty. The fundus
is somewhat greater in diameter and is marked off
by a slight constriction. The pyloric vestibule
narrows as it approaches the pyloric canal. The
canal is just patent when the stomach is empty;
but when it contains food, the canal is practi-
cally obliterated except when chyme is passing
through into the duodenum. The greater curvature
reaches just below the interiliac line in the erect
position and just above in the horizontal position.

The pylorus is lower in the erect than in the
recumbent position because it is mobile, but the
junction between the duodenal bulb and the descend-
ing part of the duodenum, which is retroperitoneal,
is fixed.

When full, the greater curvature
stretches more than the lesser curvature and there-
fore alters the axis so that it points more down-
wards.   No constriction can be seen between the
fundus and the body.   The pyloric vestibule is
almost as wide as the body in diameter but narrows
towards the canal.   The vestibule reaches beyond
the mid-line and hides the canal from the front.
The canal now points directly back, or even some-
what to the left, although the pylorus goes very
slightly to the right.

Variations from the average, as described
above, frequently occur.   The extremities of the
stomach, the fundus and pylorus, are more or less
constant and therefore differences of position will
depend upon the length of the intermediate part.
The short stomach, mostly seen in men, will be
higher; the long stomach more common in women, will
be lower.   Usually the former lies in a diagonal
line; the latter has the body of the stomach
vertical and meets the pyloric vestibule at an
acute angle as it bends up and to the right.

These variations were first described by Hurst many years ago. More recently, a similar investigation by Moody, Van Nuys, and Chamberlain (4) gave similar results. Their findings were:--

Average position of greater curvature.

Erect   2.5cms. below interiliac line in males.
4.5cms.   "   "   "   " females.

Recumbent   9 to 10cms. higher.

Average position of lesser curvature.

3cms. above interiliac line in males.
1.75cms. "   "   "   " females.

The so-called short stomach was found in 17 per cent of men and 7 per cent of women; the long stomach in 3.6 per cent of men and 15 per cent of women. No relation was found between the position of the stomach and the muscular development of the individual.

     (a)   (b)   (c)
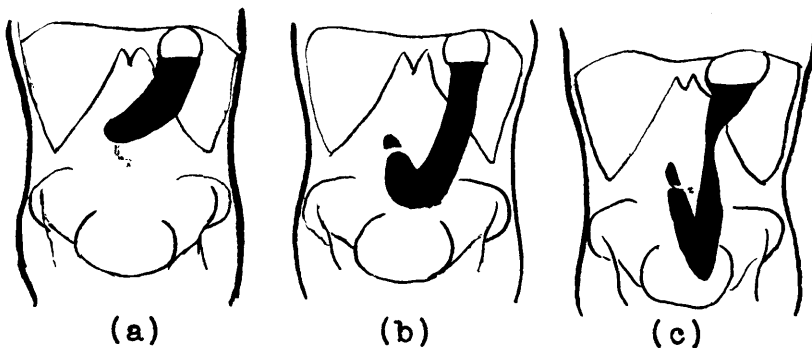
(a)   Short stomach (so-called "hypertonic stomach"). The duodenum is hidden behind the pyloric vestibule.
(b)   Average stomach (so-called "orthotonic stomach").
(c)   Long stomach (so-called "gastroptosis").

                                       (after Hurst).

(c) The Duodenum.

The pylorus projects into the duodenum, as has been said, like the cervix uteri into the vagina. The beginning of the duodenum, known as the duodenal bulb, has therefore a concave base, formed by the end of the pyloric canal and the projecting part of the pyloric sphincter. Hurst (5) suggests the names greater and lesser curvatures for the convex outer and inner sides respectively. It seems quite appropriate as they are continuous with the similarly named curvatures of the stomach.

The mucous membrane of the bulb is smooth and contains only a few longitudinal fibres, in contrast with the remainder of the duodenum which has transverse valvulae conniventes like the rest of the small intestine. The muscle is poorly developed and the submucous plexus of arteries is composed of few vessels and their anastomoses are not so numerous as in the remainder of the duodenum.

The bulb points upwards, backwards and slightly outwards. The angle it forms with the stomach depends on the type of the latter. As previously indicated, the apex is more or less fixed, though the base moves a little with the pylorus. It moves to the right as the stomach fills and may therefore point slightly inwards.

It is unnecessary to deal with the remainder of the duodenum as it has no bearing on our subject except in duodenal ileus which is dealt with elsewhere.

(3) PHYSIOLOGICAL.

(a) Motor.

The muscular tone of the stomach varies according to the contents of the organ. The introduction of food results in an increase of intragastric tension but the muscle quickly adapts itself and by lessened tone restores the tension to its former state.

Peristalsis is commenced as soon as food enters the stomach and continues until the stomach is empty. Now this is, contrary to general belief, only so for a few hours before breakfast. It follows therefore that peristalsis is an almost continuous process. The peristaltic waves begin before free acid is present in the stomach and are active even in complete achylia; but the activity increases with the amount of free hydrochloric acid present. The stimulating effect of meat extractives is said to be due to the amount of acid called forth. The waves occur every 15 to 20 seconds and two or three are present simultaneously. They begin near the centre of the stomach and deepen as they pass towards the pyloric canal; so that

shortly before they reach it they almost cut off
the part behind them from that in front.   In
consequence, the pressure in the distal portion of
the pyloric vestibule rises to a height consider-
ably greater than that in the proximal part of the
stomach.

As already stated, the pyloric canal is
obliterated, or almost so, at all times when food
is present in the stomach, excepting only the times
when chyme is passing into the duodenum.
Hurst's (6) explanation of the control of the
pylorus is as follows.   The approach of each
peristaltic wave causes relaxation of the pyloric
sphincter, the canal however remaining very narrow.
The increased pressure in the distal segment of the
pyloric vestibule, resulting from the advancing and
deepening peristaltic waves, is sufficient to
squeeze some chyme through the canal to the duodenal
bulb.   The remainder is, naturally, forced back
into the stomach again.   In addition, then, to
evacuating the stomach the peristaltic waves churn
the contents of the pyloric vestibule. Peristalsis
being absent in the fundus, where the muscular coat
is much thinner, there is no churning there.
Relatively little takes place in the greater part
of the body except when most of the contents have
been evacuated.   This is due to the shallowness of

the waves in this part in proportion to the diameter. It is apparent that the vestibule is the grinding mill while the body and fundus form a reservoir to supply it with material.

Other views of pyloric relaxation may be mentioned. The "acid-control" theory of Cannon (7) held the field for a long number of years. Briefly, it was that acid of a certain concentration in the stomach caused relaxation of the pylorus and acid in the duodenum caused contraction. When the alkaline secretion in the duodenum neutralised the acid in the duodenum, relaxation could again occur when the necessary concentration was reached in the stomach. Meals taken with sufficient alkali to prevent free acid during digestion would, if this theory were correct, remain in the stomach; which of course they do not. Similarly, in cases of achylia gastrica food should not be able to reach the small intestine. It is well-known that there is no difficulty in the emptying of the stomach in this condition and indeed it seems to exist in 4 per cent of normal people, according to Ryle, (8). These clinical facts and certain experimental evidence, which it would not be profitable to elaborate here, compelled the abandonment of Cannon's theory.

Apperly (9) suggested that the pylorus

relaxed when the total chloride value approached that of the blood. This theory does not account for the passage of water through the pylorus almost immediately after being swallowed. MacLean (10) found solutions of sugar containing respectively 4 per cent sodium chloride and no sodium chloride gave an equally rapid rise of blood sugar; and the curve was almost identical with that when sugar in 0.8 per cent sodium chloride --- the "optimum" concentration --- was ingested. As sugar is not absorbed in the stomach, each must have reached the small intestine equally rapidly.

It seems reasonable to us to accept the view advanced by Hurst. It conforms to radiological observations and is in accordance with the general principles of peristaltic action in the intestines, viz., that stimulation at any point produces contraction above and relaxation below.

A further factor concerned in the control of the pylorus is that inhibition of the muscle occurs when solid masses come into contact with the mucous membrane of the vestibule. Cannon recognised this but considered the main factor the acid concentration. The value of this reflex is evident in protecting the delicate duodenal mucous membrane. The fluid contents are thus evacuated first and more time is obtained to break up

solid masses by enzyme and muscular action, which
have escaped proper mastication.   A similar pro-
tective mechanism comes into play, it is said, when
very hot or very cold foods enter the stomach.
The practical importance of proper mastication is
apparent from a consideration of these facts.
Moreover, the main function of the stomach appears
to be its muscular action, as digestion cannot be
completed chemically by the gastric secretions;
and on the other hand, chemical digestion may be
entirely absent without causing symptoms.   It
follows therefore that adequate preparation by
chewing will very materially aid the stomach
properly to discharge its functions.

        The pyloric sphincter relaxes when the
stomach is empty;   and bile regurgitates into the
stomach then, especially in the recumbent position,
when the action of gravity no longer reinforces
peristalsis.   According to some observers, this
regurgitation also occurs during the later stages
of digestion.   MacLean (11) brings evidence to
show that this is not an essential part of digestion
and when it does occur it is a fortuitous
occurrence.   It had been argued that the cessation
of the rise of hydrochloric acid was associated
with a rise in sodium chloride.   This was said not
to be due to dilution by secretion of liquid in the

stomach as the total chlorides did not alter.
It was therefore supposed that the chlorides were
formed by the alkaline duodenal contents regurg-
itating into the stomach. A relation between the
concentration of hydrochloric acid and sodium
chloride was established: when the one was high
the other was low and vice versa. From this,
several important deductions followed. High
acidity was not necessarily an evidence of increas-
ed secretion but possibly of defective neutral-
isation. Several other similar and attractive
theories emerged. MacLean however showed that
both hydrochloric acid and sodium chloride were
secreted; and in general when more hydrochloric
acid was secreted, less sodium chloride was
secreted and vice versa. The results of his
experiments were in brief:--

(1) No evidence of increased CO_2 in
the gastric juice was found dur-
ing digestion --- as would be
expected if neutralisation were
taking place. On the other
hand, when bile was present, a
definite increase in CO_2 was
found.

(2) Trypsin was present only when
bile was detected. Trypsin
should have been present when
acid decreased if it were due to
chloride formation from neutral-
isation.

(3) Digestion was frequently com-
plete without bile being found.
When it did appear it often did
so after the acid concentration
had been reduced.

(4) H2SO4 in a test meal was given
and acidity was reduced without
appreciable neutralisation of
acid.

(5) A Pavlov pouch, in which regurgi-
tation was impossible, gave
similar results in regard to
HCl and NaCl --- a rise in acid
followed by a fall and a rise
in NaCl.

Some do not accept MacLean's argument (Halli-
burton & McDowall, 12) and observe that it takes no
account of hyperchlorhydria nor explains how the
mechanism breaks down to allow of this condition.

The duodenal bulb is more or less filled
throughout digestion. It further maintains its
resemblance to the stomach by being subject to
peptic ulceration. The rest of the duodenum is
never full as the chyme passes through it rapidly.
When chyme is pressed into the bulb, its previous
contents overflow into the next part of the duo-
denum. This is aided by a peristaltic wave which
passes from base to apex. The chyme is then
carried by a single wave throughout the next part
of the duodenum into the jejunum. The bulb may,
in the erect posture, contain some food when the
stomach is empty. With recumbency it empties.
No segmentation takes place in the duodenal bulb.
This, and the nature of the peristalsis causes no
rubbing of the contents against the mucous mem-
brane to occur. Hurst (13) considers this may be

a partial explanation of why cancer almost never occurs in the duodenum.

The Nerves of the Stomach.

The stomach is supplied by the vagi and sympathetic and intrinsic nerve plexuses. The right vagus is distributed over the posterior surface of the stomach and the left over the anterior surface. The intrinsic plexuses pass to the muscle fibres and mucous membrane. The vagus is the motor nerve, stimulation causing contraction. The opening of the pylorus seems probably to be due to vagal action. The sympathetic is inhibitory, stimulation causing dilatation. The general state of well-being or otherwise which so obviously affects the stomach probably acts to some extent through these nerves. The intrinsic nerves are capable of carrying on the functions of the stomach but in an erratic way; and it apparently requires the control of the vagus and sympathetic to obtain efficient action.

(b) Secretory.

The glands of the fundus and body secrete hydrochloric acid and pepsin. Those in the vestibule secrete an alkaline fluid without enzymes and probably similar to that secreted in the intestine. Mucus-secreting cells are present all over the gastric mucous membrane.

Pure gastric juice has a concentration of hydrochloric acid of 0.4 to 0.5 per cent. During meals, the concentration varies from 0.15 to 0.2 per cent or slightly more. This diminution is due to dilution with food, neutralisation by saliva, the combination of some acid with protein and possibly the alkaline secretion of the stomach.

In the early stages of digestion the secretion of gastric juice is due to psychical stimuli - sight, smell and taste. This was shown long ago by Pavlov in animals and took place as long as the vagus was intact. The reflex therefore is through that nerve to the glands of the fundus and body. This psychical secretion continues for some time but is only a subsidiary process, though an important one, in the initial stages of digestion.

The main secretion is due to the presence of certain foods in the stomach --- proteins, carbohydrates, their digestive products and meat extractives. It seems that these act on the pyloric vestibule and do so by absorption or by the formation of gastrin (Edkins 14), a hormone which is absorbed. They are conveyed by the blood to the glands of the fundus and body and cause secretion to occur. The products of protein cleavage and meat extractives are said to act also

in a similar way from the intestine after a latent period of two or more hours.

The stomach, according to MacLean (15), possesses an automatic regulating mechanism for hydrochloric acid. In normal individuals, after ingestion of food, the concentration of hydrochloric acid approaches 0.2 per cent. It then acts on this mechanism and stops the formation of more acid. In MacLean's view, this is explained by the H-ion concentration which at a certain point acts as a stimulus to inhibit further secretion; and this is probably effected through the gastric nerves. After the cessation of acid secretion, the stomach continues to secrete sodium chloride and fluid. This further explains the claims made above in regard to duodenal regurgitation. He goes on to state that the pepsin secreted is related to the sodium chloride secreted and not to hydrochloric acid. Estimation of the pepsin, he says, can be made with accuracy from estimation of the neutral chlorides.

Irritants, chemical, thermal and mechanical, (alcohol, hot drinks, ices and unchewed food) cause a secretion of mucus --- presumably a protective reflex. Some alkaline fluid is secreted by the mucous membrane of the duodenal bulb. It is probably insufficient to neutralise

the chyme as none of it remains in the duodenal
bulb for much longer than half a minute or so,
(Hurst 16).   In the remaining part of the duo-
denum, the bile and pancreatic juice are of course
present and the chyme is there more or less com-
pletely neutralised.

(c) **Sensory.**

To Hurst (17) we are indebted for most of
our knowledge of the sensibility of the alimentary
tract.   He showed that from the upper end of the
oesophagus to the inner end of the anal canal the
mucous membrane was insensitive to tactile
stimuli; and to thermal stimuli also, though in
this regard the oesophagus is excepted.   Chemical
stimuli, including hydrochloric acid in concentrat-
ions greater than is ever found in hyperchlorhydria,
and the various organic acids found in the stomach
in disease, had no effect from oesophagus to anal
canal.   Alcohol was the only substance which
evoked a response.   It acted on the mucous mem-
brane of the lower end of the oesophagus and stom-
ach.   This will be further referred to when con-
sidering heartburn.

As mentioned above, all forms of gastric
pain appear to be due to increased tension.   This
applies also to the oesophagus and intestine.   When
an ordinary-sized meal is taken, the stomach

musculature adapts itself and no sensation is
experienced, intra-gastric pressure remaining con-
stant.   Too rapid ingestion of food causes a sense
of fullness as the relaxation of the muscle tone
cannot keep apace with the increased internal
pressure.   An abnormally large meal causes a feel-
ing of distension because the limit of muscular
relaxation is exceeded.   There is only a differ-
ence of degree in the production of actual pain.

Satisfaction occurs, according to Ryle
(18), when the stomach musculature is adequately
relaxed to enclose a meal of appropriate bulk and
consistency.   When the stomach empties, satis-
faction continues until tonic and peristaltic con-
tractions awaken consciousness to the sensation of
Hunger.   Repletion, as is obvious from the fore-
going, is due to over-filling or too rapid filling.
Appetite differs not merely in degree from hunger.
It is due to the tonus of the muscle, but peri-
stalsis is required in addition to produce hunger ---
hence its rhythmical character.   There is of course
a psychical factor in appetite.   It is partly due
to memory and partly to smell, taste and sight.
(d) Digestive.

The Mouth. When food is introduced into
the mouth, it is, of course, ground down and pre-
pared for swallowing by the process of mastication.

With this process, important though it is, we are not now concerned.   The chemical part of digestion in the mouth is due to the action of saliva. This substance has also a mechanical function in moistening the food and preparing the bolus for deglutition.   The active chemical principle of saliva is known as Ptyalin.   It is an amylolytic or starch-splitting enzyme.   Starch is first of all split into dextrin and maltose; the dextrin being subsequently converted to maltose.   The process is more rapid in the case of erythro-dextrin than with achroo-dextrin.

The optimum action takes place at body-temperature in a neutral or weakly alkaline medium.   A small amount of additional alkali makes but little difference.   Acid, except when exceedingly dilute, stops its action.   Hydro-chloric acid in even 0.004 per cent, is sufficient to kill it.   The action of ptyalin is necessarily therefore, a very temporary one for the acid con-centration in the stomach is far higher than is necessary to inhibit the process.   The enzyme is entirely destroyed and cannot therefore resume its work in the alkaline medium of the duodenum.   It must be observed that ptyalin does, however, con-tinue to act on starch for some time after the food enters the stomach.   This is due to two

factors.   In the first place, the hydrochloric acid secreted by the gastric mucous membrane is neutralised by the alkaline food mass, which is in contact with the stomach, so that the interior of the mass remains alkaline.   Secondly, the relative absence of movement in the fundic portion of the stomach during the early stages of digestion allows the food mass, more or less mixed with saliva, to remain undisturbed.   It is noteworthy, also that in ordinary diets, starch is usually accompanied by fat, (bread with butter, pastry with cream, porridge with milk etc.).   The effect of fat is of course partially to inhibit gastric secretion and presumably thereby to prolong salivary digestion.

The production of sugar by ptyalin is extremely rapid.   In fact, bread or potato when ingested, provide sugar almost as quickly as if pure glucose had been swallowed.   The blood-sugar curve, after a fair quantity of bread has been taken, often rises just as rapidly as after glucose;   and in many cases gives an identical curve.

The Stomach.   When food reaches the stomach, it is subjected to the action of the gastric juice.   The only important enzyme present is pepsin and its function is proteolytic.   It

requires an acid medium and is associated in the stomach with hydrochloric acid. The amount of acid usually present during digestion, 0.15 to 0.2 per cent, secures the optimum activity. Protein hydrolysis to the stage at which protein can be absorbed (amino acids) cannot be effected by the gastric juice. Pepsin is only capable of the early stages of cleavage. Beyond the production of proteoses and finally peptones no further action of importance takes place. It is therefore necessary, before protein can be utilised by the organism, that the peptone and other intermediate bodies be further digested. This is accomplished by the pancreatic and intestinal juices. It is apparent from these facts that gastric digestion is not an essential process. It merely prepares the protein for the further action of more powerful enzymes which are in themselves capable of the full process of protein cleavage from start to finish.

An enzyme which coagulates milk, rennin, is present in the stomach. The curd of casein formed is later digested as are other proteins. Halliburton and McDowall (19) state that Pavlov, advanced the view that the milk-curdling property in gastric juice is due merely to another activity of pepsin; and that the opinion of eminent physiologists is divided on the question.

It is asserted by some that a fat-splitting enzyme is present. The contention is that, apart from fat-splitting due to possibly regurgitated duodenal contents, a certain amount is effected by lipase secreted in the stomach; because this takes place when the pylorus is ligatured to prevent regurgitation. At any rate, it is of little or no importance in digestion because its action, if such an enzyme does exist, is very slight.

Cane-sugar is inverted into glucose and fructose by the acid of the juice. This is assisted by enzymes contained in the vegetable food swallowed.

The hydrochloric acid exerts an anti-septic action and prevents putrefactive processes from taking place normally in the stomach. This it does by destroying the micro-organisms which produce such processes.

As indicated above, some ptyalin digestion is continued for a variable time in the stomach. No other action on starch takes place.

The Intestines. Three digestive juices are secreted into the intestine --- the pancreatic juice, the bile and the succus entericus. Intestinal digestion begins at the duodenum and is practically completed by the time the contents

reach the large bowel.

The Pancreatic Juice.   This is by far
the most important of the digestive secretions
and without it, experiments on animals have shown,
life is maintained with difficulty.   There are
three pancreatic enzymes --- trypsin, amylase and
lipase.   A fourth, milk-curdling enzyme, has been
described.   This can hardly ever be called into
action as the rennin will have already curdled
any milk.   Moreover, it has been alleged, and
there is no decisive evidence to contradict the
assertion, that this action on milk is only a
further activity of trypsin.

Trypsin.   This is a very powerful
enzyme and is capable of breaking down proteins
from the beginning to the stage at which they can
be absorbed.   It effects this through the stages
indicated above as occurring in the stomach.   It
then breaks up these substances to polypeptides
and finally to amino acids.   Its action is
different from pepsin in that, besides carrying
protein digestion further, it acts more rapidly
and does so in an alkaline medium.   It should be
noted that trypsin can act in a neutral or faintly
acid medium.   It follows from these considerations
that pepsin is not essential and in its absence
no symptoms need occur.   The preliminary action

of pepsin is not, however, without purpose or benefit as it appears that trypsin digestion proceeds more quickly after the hydrolysis effected by pepsin.

Trypsin exists in the pancreas in an inactive form known as trypsinogen. It is activated by a substance derived from the mucous membrane of the duodenum and jejunum. This substance or proenzyme was described by Pavlov and is called Enterokinase. It is said not to be specific and other substances may produce the same effect. Willstatter (20) and others have contended that this proenzyme merely increases the activity of trypsin and makes it able to attack the more resistant proteins. This may be an error due to the difficulty of obtaining trypsinogen free from trypsin. The action of enterokinase is explained by Mellanby and Woolley (21) thus:-- Enterokinase is a proteolotic enzyme which adsorbs and then digests the protein moiety to which trypsinogen is united; thus liberating trypsin.

Amylase. This converts starch into maltose which is the most rapid action of the pancreatic juice. It completes this very quickly and almost all the conversion is done in the upper intestine. Amylase completes the work of saliva

and is a much more powerful enzyme, acting on even unboiled starch. The small amount of this enzyme in infants explains their comparative inability to deal with starch. Maltose is the sugar formed: this is rapidly changed to glucose by maltase in the intestine. Some observers state that there are small amounts of maltase in the pancreatic juice.

Lipase. The action of this enzyme is to split fat into its constituents --- glycerol and fatty acids. The fatty acids unite with the sodium carbonate of the pancreatic juice and form soaps. These emulsify the fat by forming a film over the fat globules and preventing them running together. This continues until the process is completed and the fat entirely split up.

The importance of bile in assisting the action of lipase has been recognised for some time. If a glycerol extract be made of the pancreas and filtered, the filtrate has no action on fat nor has the material left on the filter. When these are united again, the lipase is active. The substance in the filtrate is not destroyed by boiling and is a co-enzyme which is necessary to activate the inactive lipase which is not filterable. Bile also acts as an activating substance and this is the explanation of its

utility in fat-splitting.   Bile itself has no
action on fat.

The Mechanism of Pancreatic Secretion.

In 1902 Bayliss and Starling (22) discovered
Secretin, a substance which, when injected into
the blood, causes a flow of pancreatic juice.
They obtained this substance in an extract made
with hydrochloric acid from the mucous membrane
of the duodenum.   According to them, Secretin
is present in the mucous membrane of the duodenum
and to some extent also in the jejunum, in an
insoluble parent form, Prosecretin.   The hydro-
chloric acid of the gastric juice when it enters
the duodenum changes the Prosecretin into soluble
Secretin which is carried to the pancreas by the
blood.   The effect is to cause a flow of pan-
creatic juice into the duodenum.

This theory held the field for some
time.   It was at length shown by Dodds and
Bennett (23) that neutral or alkaline fluid
introduced into the duodenum caused a secretion
of pancreatic juice.   This confirmed suspicions,
already aroused by clinical observations, that
there was no apparent interference with pan-
creatic activity when patients were taking
alkalies or in Achylia Gastrica.   Mellanby (24)
showed secretin was present not only in the

duodenum and jejunum but in the ileum and even to some extent in the large intestine; and that it was present in active solution, not as prosecretin, since it could be extracted by water, alkali or even dilute alcohol.   He also showed that many substances, especially bile, when introduced into the duodenum caused a flow of pancreatic juice. From these facts it was concluded that secretin was carried into the blood by bile salts when the bile is absorbed from the duodenum.   Hence the secretion of pancreatic juice is related to the entrance of bile into the duodenum.   This in turn is related to the passage of peristaltic waves from the pylorus down the small intestine. Each peristaltic wave is preceeded by a wave of inhibition releasing the common bile-duct sphincter as it passes through the duodenal muscle and this allows a few drops of bile into the duodenum (McDowall 25).

It had of course long ago been shown by Pavlov that the secretory nerves of the pancreas were the vagus and to a lesser extent the splanchnic.   Mellanby (24) found that the secretion produced by secretin became poorer and poorer in ferments yet alkali was very constant. This was not due to exhaustion of supply because stimulation of the vagus increased them to their

former value.   The conclusion he draws is that the specific work of the pancreas, which is enzyme formation, is under vagal control.   Secretin plays a subsidiary part and calls forth a secretion which carries these enzymes into the duodenum and secures for them the optimum reaction for the performance of their function.

Succus Entericus.   We have already mentioned Enterokinase and Secretin.   The Succus Entericus also contains a proteolytic enzyme, Erepsin, which acts on intermediate products of protein cleavage --- proteoses and peptones --- and brings them to the amino-acid stage.

The intestinal juice also possesses the power of converting disaccharides into mono-saccharides, e.g. maltose to glucose by maltase. There are two others of these, lactase and sucrase, which act on lactose and sucrose.

The Bile.   The bile has no digestive action of its own.   As indicated, it assists pan-creatic secretion and plays a part in the splitting-up of fat.

The Absorption of Food.   Practically no absorption takes place in the alimentary canal until the small intestine is reached.   A very small amount does take place in the stomach.   No water at all is absorbed there; but alcohol is to

some extent.   Salts in the concentrations of ordinary diets are not absorbed and sugar very little. Proteins and fats are ,of course, not even ready for absorption.

The small intestine is the site par excellence for absorption.   Its area is enormous when its villi and folds are taken into consideration and it has been estimated at 42 square metres. The large intestine has some slight absorptive power, mainly for water.

CHAPTER 2.

THE STOMACH IN DISEASE.

(1)   Gastric Pain.

     (a)   General.
     (b)   Pain in Gastric & Duodenal Ulcers.
     (c)   Pain in Cancer of the Stomach.

(2)   Fullness and Discomfort.

(3)   Flatulence.

(4)   Vomiting and Nausea.

(5)   Heartburn.

(6)   Waterbrash, etc.

(7)   Disturbances of Appetite.

(8)   Reflex Pain and Other Somatic Phenomena
     of Abdominal Visceral Disease.

(1) GASTRIC PAIN.

> "every pain has its distinct
> and pregnant signification,
> if we will but carefully
> search for it".   HILTON (26).

Increased tension of the stomach wall seems to constitute the only adequate stimulus for the production of gastric pain.   Poulton (27) showed that the successful passage of a peristaltic wave actually relieved pain in that region.   The presumption is that the muscle fibres take up the strain and relieve the nerve-endings of the pressure to which they have been subjected.   He considers, however, that peristalsis may in cases cause pain as, for example, when a contraction wave is attempting to force on a foreign body, there must be stretching of the muscle through which that body is being forced.   This is exemplified in the pylorus in certain cases, the cervix uteri in labour, and the bile passages in cases of calculus.

As McDowall (28) observes, it is quite unphysiological to have prolonged pain caused by peristalsis since the pain, if sufficiently severe, would cause sympathetic stimulation with resulting paralysis of the gut.   He suggests this may account for the intermissions of colic.   It is not, however, an argument against the theory of gastric pain set out here as we hope to show below.

For an analysis of the causes of gastric pain we must, then, consider what factors induce increased tension of the muscular wall of the stomach. We shall do this under two headings:-- (a) Increased Pyloric Resistance. (b) Distension and Atony.

(a) <u>Increased Pyloric Resistance.</u>

Leaving aside congenital muscular hypertrophy of the pylorus, which is not within our present scope, increased resistance may result from cicatricial contraction, adhesions, inflammatory swelling, spasm and achalasia of the pylorus. The first three are mechanical conditions and are well recognised. Spasm and achalasia require further consideration.

Ordinarily, relaxation of the pyloric sphincter results as each peristaltic wave approaches the pylorus. In ulcer of the duodenum this does not occur. It has long been known that in duodenal ulcer the stomach empties rapidly. We now know, however, that this refers only to the early stages of digestion before pain ensues. Probably as a result of irritation by the very acid chyme, the ulcer sets up a protective reflex whereby the chyme is not allowed to pass from the stomach (Hurst 29). At any rate, in the late stages of digestion, the pyloric sphincter fails to relax to

all peristaltic waves.   Peristalsis, in turn, is stimulated by this obstruction and becomes deep and rapid.   It is however, not successful in opening the sphincter and the rapid evacuation of the early stages is followed by abnormally slow evacuation.

Spasm of the pylorus probably results when inflammation from an ulcer spreads into the pyloric canal.   Hurst states that in these cases, nothing can be squeezed through the pyloric canal by pressure on the stomach under X-rays.   In simple achalsia, such as we have described above, this is possible.   After a while the sphincter relaxes and achalasia disappears;   and the stomach is able to empty.   Juxta-pyloric ulcers act in the same way. Hayem (30) has shown that ulcers on the lesser curvature, at some distance from the pylorus, occasionally affect the sphincter in a similar way. Gall-bladder disease, and much less frequently, chronic appendicitis, may have the same·results. When organic obstruction occurs (as in cicatricial healing) the reflex achalasia does disappear and relaxation of the sphincter follows each peristaltic wave so far as this is mechanically possible. In active ulceration the achalasia of course occurs.

Moreover, the protective reflex of the duodenum comes into action when insufficiently masticated food is compressed against the entrance

of the pyloric canal.   This is in part the
explanation of the pain in indigestion from the
gulping of food.   The too-rapid filling, with
resultant stretching, is also a factor.   Again, in
cases of inflammation further down the digestive
tract, the same reflex may operate.   It may also
be called into play in emotional emergences.   From
these considerations follows the explanation of the
reflex dyspepsias simulating duodenal ulcer and
those associated with emotional states when the
latter take on the features of ulcer.

The application of what is called the
"sympathetic-parasympathetic balance" to gastric
function may conveniently be considered at this
juncture.   Many organs and groups of organs in the
body seem to be, in health, maintained in a balanced
state by the action of the sympathetic and para-
sympathetic nervous systems.   The vagus is one
of the principal nerves of the parasympathetic
group.   Balance is a convenient term to use, though
it is not known how far a true balance exists;
and neither side of the balance appears to be
essential.   It seems that the stimulation of the
sympathetic and the reduction of vagal tone which
take place after severe exercise, to facilitate
the supply of oxygen and fuel to the muscles,
result in delayed evacuation of the stomach.   This

act the vagus normally hastens. Mental strain accentuates the condition. Moreover, salivary, gastric, and pancreatic secretions are reduced. This is important in considering dyspepsia which results from over-excitement, exercise, mental pre-occupation at meal-times and so on. Dyspepsia associated with anaemia is affected also by this principle. In anaemia, increased sympathetic action occurs to compensate for blood deficiency. A similar explanation could be extended to acute diseases where indigestion occurs. It is interesting to consider in conjunction with the above certain explanations given by Hurst (31) in regard to mental and physical strain and their effect on digestion. He states that worry and over-work, mental and physical, are far more commonly factors in duodenal than gastric ulcer. To his mind, the most probable explanation is that the exhaustion exaggerates the vagal hyperactivity of the gastric diathesis (in this case presumably the duodenal type). Moreover, it is not improbable that this is effected through the suprarenals, which Crile showed become exhausted and produce less adrenaline as the result of physical and mental strain. In this way, the continuous inhibitory influence of the sympathetic is damped down. These views do not seem to us so mutually exclusive as

they at first appear.   It is a matter of everyday observation that the same set of abnormal factors, or the same stimuli, do not produce the same response in different individuals.   Just what determines the response is a difficult matter to decide.   Undoubtedly the main factor in determining the response, as far as our present knowledge goes, seems to be the physical, or perhaps more correctly the constitutional,type of the individual.

(b)   Distension or Atony.

In distension, the stomach is stretched but still retains the power of tonic contraction.   The condition usually follows obstruction from one of the conditions described above.   It may result from over-eating, although this, like dilatation of the heart from over-exercise, is doubted by many.   Dietetic errors,such as the consumption of enormous quantities of bread and potatoes, with the consequent production of gas, commonly leads to indigestion.   The gas interferes with the propulsive power of the stomach and leads to distension and perhaps atony.   It has been suggested that the coarser vegetables, to which flatulence is commonly attributed, cause the protective pyloric reflex to operate; and delayed evacuation with carbohydrate retention and ferment-

ation results.   Relief is obtained by belching of gas but vomiting is rare owing to the muscular weakness.   A vicious circle is established because satisfaction only follows the ingestion of a large meal.   Atony is a further stage of distension and here the muscle tone is lost.   The pain is due to stretching of the muscle which no longer has the power of contracting and thereby relieving the pressure on the nerve-endings.

(B)   Pain in Gastric and Duodenal Ulcers.

This requires special consideration though the principle of pain production is identical with that already enunciated.   It was at one time believed that the pain of gastric ulcer was due to the acid in the stomach and occurred when the con-centration of the acid was sufficiently increased during digestion.   In the case of duodenal ulcer, the supposition was that food did not enter the duodenum, due to a protective reflex, until the time of pain production --- i.e., late in digestion --- and the pain was due to the acid chyme.   This theory collapsed when it was demonstrated by X-rays that the stomach, far from emptying slowly did so rapidly and from the beginning of digestion.   The pain in duodenal ulcer occurs when the stomach is partly emptied and continues until it is entirely emptied.   It should here be noted that it is a

popular error to suppose the pain lasts till the next
meal.   It ceases spontaneously when the stomach is
empty, though it may be aborted by the taking of
food.   It is not essential that food be present:
hypersecretion may provide sufficient juice to cause
the stomach not to be empty.   The point is that
pain only occurs when the stomach is partly but not
completely empty.

It is well-known that food, irrespective
of its digestibility, relieves the pain of duodenal
ulcer;   as does liquid of almost any type.   Sodium
bicarbonate results in immediate relief, as do other
alkalies though less rapidly.   The obvious con-
clusion is that the acid is neutralised.   This is
undoubtedly true but another factor has to be taken
into consideration.   The food or liquid increases
the contents.   Sodium bicarbonate results in the
production of carbon dioxide and the relief which
follows is associated with this sudden evolution of
gas, as we hope to show.

Hurst's (32) explanation is that the
relief is probably mainly due to the dilution of
acid by the food or liquid ingested;   and in the
case of sodium bicarbonate, to the neutralisation
effected.   He is of the opinion, however, that these
substances also act by virtue of their common
property of increasing the stomach contents in bulk,

whether by solids, fluids, or gas. This explanation seems to us more probable than that advanced by Ryle (33) who considers the relief due to a "simple adaptation of posture" only. He thinks the sudden evolution of carbon dioxide results in relaxation of tonus and that the greater effect of that substance than other alkalies is on account of the greater amount of gas produced by it. This is probably true as far as it goes, though the main factor appears to be the reduction of the acid concentration as Hurst suggests. A further point arises in this connection. Why is it that the greatest relief occurs at the moment when gas is belched? When this act takes place the acid has already been neutral- ised and the volume of gas in the stomach has return- ed to its previous amount or even less. Hurst thinks this is due to the fact that the sudden rise in intra-gastric pressure causes the resistance of the unrelaxed pyloric sphincter to be overcome: some chyme is forced into the duodenum and simul- taneously some gas through the cardia into the oesophagus, from which it escapes by involuntary belching. The pressure in the vestibule is thus lessened and the pain is at once relieved. Kin- sella (34) found that the administration of sodium bicarbonate was at once followed by pyloric relaxation as observed radiologically and that this took place

independently of any peristaltic wave.

When organic obstruction ensues the typical hunger pain disappears. This is because the stomach is dilated and no longer sufficiently empty at any time for pain to occur. When however there is regular vomiting in an obstructive case distension does not take place and hunger pain remains present.

That the acid is not the cause of the pain is clear from the fact that hyperchlorhydria is found in healthy persons and ulcer patients between attacks or after healing of the ulcer. Palmer (35) and Hardy (36) however, have both shown that the administration of acid after emptying the stomach causes the typical pain. The latter also produced the pain in reflex cases from appendicitis and cholecystitis. Wilson (37) observed patients under X-rays during attacks of pain and found that pressure caused the meal to enter the duodenum, though it did not do so spontaneously, and relief followed. This of course meant that achalasia, not spasm, was present; and moreover it proved that the acid did not directly cause the pain as it was at that time actually coming in contact with the ulcer. It seems reasonable therefore to explain the pain in these cases as a reflex achalasia resulting in increased tension; and acid seems to con-

stitute an adequate stimulus to produce it. It was further observed in some cases that chyme could not be squeezed through the pylorus, showing spasm to be present, and in these pain was aggravated by pressure. The increase in pain must have been due to the increased tension caused by the pressure.

It may be supposed that some pain should be present in the early stages of digestion. This, of course, is not so and the explanation lies in the fact that the peristaltic waves are not so deep when the stomach is full; but when it is comparatively empty, the waves become deeper and as they approach the pyloric canal the pressure in the vestibule rises considerably, the proximal segment being almost cut off from the distal one and the pylorus remaining unrelaxed. Moreover, this cutting off, as a result of the obstruction and the consequent peristaltic stimulation, occurs at a point further than normally from the pyloric canal.

Ryle (38) considers that the pain is due to increased hypertonus. He objects to the above theory on account of the sustained character of the pain. If it were due to peristalsis it should, according to him, be rhythmical as in colic. This view seems at first sight eminently reasonable. When however it is remembered that the peristaltic waves follow one another so frequently that there is

never time for the pressure in the vestibule to
return to normal, the objection becomes invalid.
Ryle raises a further objection in that he considers
that achalasia, if it were sufficient to permit
such a rise in pressure, would result in delayed
emptying.   It is well recognised that in these
cases the stomach empties rapidly.   Hurst has,
however, shown that the early rapid evacuation is
followed by obstruction but that later the achal-
asia relaxes and emptying proceeds.

In gastric ulcer a similar explanation is
admissible in cases of juxta-pyloric ulcer and in
those ulcers of the lesser curvature, referred to
above, in both of which cases achalasia results.
In other cases of ulcer on the lesser curvature,
spasm occurs and partial obstruction takes place
between the proximal and distal portions of the stom-
ach.   This is increased if organic hour-glass con-
striction is present.   Hurst (39) has described a
form of hour-glass constriction which he calls
"orthostatic" because it appears in the erect post-
ure.   Either of these increases the spasm and there-
fore the pain when food is taken because it is held
up in the proximal segment.   In the orthostatic
form, relief from pain and disappearance of the con-
striction follow the assumption of recumbency.
This type occurs usually in narrow-chested women

with long stomachs.   When ulcer is associated with orthostatic hour-glass constriction relief is less complete than when the condition is present without complication, as the spasm from the ulcer remains. In these cases actual pain rather than discomfort occurs.

Palmer (40) has emphatically stated that acid is directly responsible for the pain. Ryle (41) takes the view that it is not necessary even to introduce the acid factor.   He states that he has examined many cases of extreme hypochlor-hydria, and a few with complete achlorhydria, in which the pain was "just as severe and character-istic as in cases associated with a normal or high acidity".   In his judgment, given the presence of an irritative focus, the ingestion of food is an adequate stimulus for the initiation of the exaggerated tonic and peristaltic action which pro-duces the pain.   Hurst (42) makes the interesting observation that he has only had one single case of gastric ulcer in which, even after preliminary lavage, no free hydrochloric acid appeared in a fractional test-meal.   The patient had in addition chronic gastritis.   The significant point is that she complained of nausea but no pain.   The nausea could well be attributed to the chronic gastritis. This makes an interesting commentary on Palmer's

observations.

Hurst states that the relief of pain on taking sodium bicarbonate in those rare cases of gastric ulcer with achlorhydria is explained by the achlorhydria being incomplete. Though the non-stimulating gruel of the fractional test-meal or the tea and toast of an Ewald meal (meals, be it noted, which are rarely followed by pain) call forth no acid, more stimulating meals of soup, meat and vegetables and a sweet call forth acid about the time the pain develops.

(C) <u>Pain in Cancer of the Stomach.</u>

The pain in this condition must next be considered. It is a well-established clinical fact that quite a number of cases of carcinoma of the body of the stomach are more or less painless. In these there is often an infiltration of the muscular wall and a rigidly patent pylorus with rapid emptying. This is compatible with the explanation of gastric pain production given above. If the pylorus be not thus patent, then adaptation of the muscle cannot take place when food is ingested and pain results. Where it is patent, practically no adaptation is necessary as the food leaves the stomach quickly. If the pylorus is actually obstructed, then the mechanism of pain production is clear from what has been said above in regard to

simple ulcer.   Finally, just as in ulcer cases, if
the pain is very constant there is probably involv-
ment of other structures.   If constant pain is not
due to extension of gastric disease, it is usually
an extra-gastric cause which must be sought.

(2)   FULLNESS AND DISCOMFORT.

Discomfort differs from pain merely in
degree; and its production is due to the same
causes.   It is most commonly found in functional
conditions, where real pain is very infrequent.
General ill-health, nervous or toxic influences,
and in addition local causes, may affect the gastric
musculature with resulting alteration of normal
activity.   Such alteration may take the form of
an increase or a diminution of muscle tone.   A
sensation of fullness is also a common symptom in
functional disorders and is produced by the same
mechanism as discomfort.   When the tone of the stom-
ach is increased, fullness or discomfort may be felt
after a meal owing to the inability of the stomach
to adapt itself sufficiently.   When the tone is
deficient, the ingestion of food results in an
increase of tension in the already over-stretched
muscle.   A sensation of fullness may be experienced
or discomfort of varying degree up to real pain.

On the other hand, a hypertonic, rapidly-
emptying stomach may give rise to a"sinking sensat-

ion", which is relieved by food.   Hurst (43) has observed this in healthy members of "ulcer families" and some have actually developed ulcers.   He brings this forward as evidence in support of his theory of a gastric diathesis, with which we shall deal later.

(3) FLATULENCE.

It has long been a popular belief that flatulence is the result of gastric or intestinal fermentation.   Although this is true in some cases, it is comparatively rare.   By far the most frequent cause of flatulence is the swallowing of air. This is normal, to a certain extent, during swallowing and causes no symptoms.   The air is expelled later from the mouth, or passed into the intestines to be absorbed there or expelled.   When symptoms do arise they are almost always due to excessive accumulation. When the stomach muscle is not acting efficiently, gas will tend to stagnate.   The patient feels it desirable to get rid of the excess and in attempting to do so usually swallows more.   This as a rule results in raising intra-gastric pressure sufficient-ly to stimulate the muscle to expel the air.   The vicious circle may however continue and the con-dition of Aerophagy is established.   As Abrahams (44) points out, in any state of gastric discomfort there is increased salivation and consequently frequent acts of swallowing with which air is included on each occasion.   Attention has been

drawn by Ryle (45) to the dyspepsia, regarded by some laryngologists as the cause of pharyngitis and naso-pharyngeal catarrh. He regards this as more probably the cause than the result of the dyspepsia; and considers it the outcome of the frequent swallowing of air which follows from the irritation. With this, ordinary sore throat may be included. Frequent swallowing is also common in nervous indigestion. Insufficient mastication often causes air-swallowing and this is not uncommon among those taking a diet of soft food. Angina, with the feeling of retrosternal oppression, may suggest to a patient the desire to eructate and lead to aerophagy.

When fermentation does occur it is usually associated with delayed emptying of the stomach. It is however a rare occurrence, fermentation being inhibited by the hydrochloric acid. In such cases (those of fermentation) the eructations have a markedly unpleasant odour, usually of sulphuretted hydrogen, in contrast to those in aerophagy which are usually more or less without odour.

MacLean (46) does not entirely agree in the explanations we have given above and which are generally accepted. He suggests that the stomach may secrete gas, though no experimental evidence of this has been forthcoming; but, according to him, surgeons have actually seen the stomach sudden-

ly distend with gas in the course of an operation.
He feels, moreover, that aerophagy does not always
take place in those very bad cases where the patient
belches large quantities of gas. He himself has
failed to observe air-swallowing when watching these
patients. It is said in the text-books that an
explanation of what is taking place usually suffices
to cure air-swallowing. We confess that this
method of treatment has not yielded very constant
results in our experience. On the whole, it has to
be admitted that flatulence is as yet not thoroughly
comprehended.

Intestinal flatulence may be due to failure
of the stomach to rid itself of accumulated air by
belching. Fermentation however does occur in the
bowel and is usually associated with constipation.

(4) VOMITING AND NAUSEA.

Vomiting independent of food suggests
general causes such as phthisis, renal and cerebral
disease. When it occurs immediately after food, it
is usually due to oesophageal obstruction. It may
take place as a pure nervous manifestation. As
regards gastric causes, it arises from irritation of
the mucosa, obstruction of the pyloric outlet or
constriction at some part of the stomach. Again,
inflammation and obstruction at some point below
the stomach may be a cause.

During the act, the pylorus is contracted, the diaphragm descends and the abdominal muscles contract. It may be that reversed peristalsis is also a factor. Ryle (47) quotes an instance when one of his clinical clerks observed a reversed movement in a case of visible peristalsis, simultaneous with the onset of an urgent desire to vomit by the patient.

Nausea is commonly taken to differ from vomiting only in degree and as a diagnostic symptom some, like MacLean (48) regard a history of nausea as just as important in certain cases as one of vomiting. Ryle (49) observes that bile tends to regurgitate into the stomach in association with nausea and retching; so that the pylorus cannot be contracted as in vomiting. If this be true, then the two symptoms must differ in their mode of production. Nausea is induced by disgusting sights, smells and emotions. It is usually present in faintness, and Barclay (50) observed, with X-rays, the lower border of the stomach drop three inches just before fainting occurred. Similarly nausea induced by disgusting smells resulted in a drop of an inch or so. Bennett and Venables (51) noted a fall in the acid-curve and very delayed emptying when nausea was suggested to a patient under hypnosis.

(5) <u>HEARTBURN.</u>

Hurst (52) is of the opinion that heart-
burn is probably due to alcohols formed by ferment-
ation.   It was formerly generally supposed to be
due to an acid regurgitation into the oesophagus;
but as acid evokes no response here, it is difficult
thus to explain the mechanism of heartburn.   This
acid theory also breaks down when we remember that
heartburn is found in cases of achylia gastrica,
(Einhorn 53).   Hurst's theory would account for
this but it fails to explain the occurrence of
heartburn in cases of hyperacidity, in which con-
dition, the fermentation necessary to produce
alcohols would not take place in the presence of
excess of free acid.   Moreover, it will not account
for the rapid relief usually afforded by alkalies.
Hurst suggests it may possibly be, like all other
forms of gastric pain, due to tension.   There is
no proof of this but it must be admitted there are
no facts in our knowledge which militate against
such an explanation.   He considers that there are
probably two forms of heartburn.   One occurs in
acute indigestion and is relieved by vomiting but
only partly, or not at all, by alkali.   This type
is due to alcohols resulting from fermentation.
The other occurs in chronic dyspepsia and is due to
increased tension.   It is present in hyperacidity

and is relieved by sodium bicarbonate which lowers the tension. We do not think this is an entirely satisfactory explanation. As we have already observed, heartburn is experienced occasionally in chronic dyspepsia associated with achylia.

Payne and Poulton (54) regard heartburn as being associated with spasm of the cardia or lower end of the oesophagus. If this be the explanation, the relief from alkalies is probably due to the gas produced overcoming the resistance of the spasm. This would be in accordance with Hurst's theory as regards the pain.

(6) WATERBRASH, ETC.

Acid mouthfuls occur very occasionally in hyperchlorhydria but are not a reliable sign as they depend on the patient's description. Water-brash, clear tasteless fluid which regurgitates into the mouth, is said by Hurst (55) to be due to saliva and mucus in the oesophagus from excessive secretion and reflex closure of the cardia.

(7) DISTURBANCES OF APPETITE.

Loss of appetite must be clearly dis-tinguished from fear to eat because of the expect-ation of pain to follow. Genuine cases of anorexia are frequently found in carcinoma. From what has been said of normal appetite, it is easy to see how the requisite tonus will be lacking when

the muscle is infiltrated by a malignant growth.
In fatigue conditions, tonus is lacking; hence
impairment of appetite results. This may be seen
in a mild degree after excessive exercise, profound
emotional upset and so on. The mechanism has
already been referred to in this connection when
speaking of the sympathetic nervous control. Toxic
causes, in acute and chronic illnesses, account for
a number of cases. This is again due to impairment
of tonus. In chronic gastritis the excess of mucus
prevents the usual stimulation of the mucous mem-
brane, both mechanically by the food and chemically
by any acid which may be present. Tonic activity
is therefore lacking. It is well known that in
hunger strikers and in the condition of anorexia
nervosa, the desire to eat passes off after an
initial period when food has been for some reason
refused. This is due to the hypotonus, or rather
relative hypotonus, which develops later.

It would not be profitable for our pur-
pose to discuss excessive appetite and certain
perversions of appetite which are of the nature of
neuroses.

(8) REFLEX PAIN AND OTHER SOMATIC PHENOMENA OF
ABDOMINAL VISCERAL DISEASE.

In 1887 Ross (56) drew attention to
referred pain. He distinguished two kinds of pain,
one felt in the organ and the other referred to the

body wall. To the former he gave the name "splanchnic" and to the latter "somatic". The pain felt in the organ gave rise to afferent stimuli which passed along the splanchnic nerves to the spinal cord and then to the cerebro-spinal nerves which caused the pain in the body wall.

The next step in the development of the modern theory of pain came from Mackenzie (57). He denied the existence of "splanchnic" pain but supported Ross's theory of "somatic",or referred, pain. His main reason for rejecting "splanchnic" pain was because it was then well-known that the alimentary tract was insensitive to cutting, pricking or burning. Mackenzie described what he called a "viscero-sensory reflex". By this he meant that afferent stimuli passed through the splanchnic nerves to the spinal cord, where they caused an irritable focus by radiation. In this way the threshold was lowered and cutaneous hyper-algesia resulted in the skin supplied by the cerebro-spinal nerves from the irritable segment of the cord. Likewise he described a "viscero-motor reflex", by which tonic contraction of the muscles of the abdominal wall resulted by a similar mechanism through the splanchnic nerves to the anterior horn and thence along the motor nerves.

Hurst (58) in 1911 showed that "splanchnic"

or "visceral" pain did in fact exist when the
appropriate stimulus was applied, viz:-- increased
tension in the muscle fibres.   He accepted
Mackenzie's "somatic" or "referred" pain.   Recently
(59) he has modified his belief that the stomach is
insensitive to mechanical stimuli and he speaks of
"visceral tenderness".   He claims that when the
subperitoneal tissue is inflamed it is sensitive to
pressure.   In support of this he observes that
when ulcers are visualised under X-rays, pressure
over them causes pain and the seat of tenderness
shifts when the ulcer moves on change of posture or
on manipulation by hand --- the exact spot where
pain is felt being over the ulcer.   Such tenderness
can usually be demonstrated even between attacks,
though it is most marked during one, so long as
X-rays and occult blood tests show the ulcer has not
healed.   This tenderness is not to be confused with
the tenderness sometimes elicited from a point more
or less remote from the ulcer.   In such cases the
tenderness lies diffusely along the lesser curvature
and is probably due to inflammation of lymphatics
or lymphatic glands situated there, inflamed by
lesser curvature ulcers and less commonly by
prepyloric or duodenal ulcers.   This tenderness is
present all day, being due to inflammation, and is
not so closely associated with spontaneous pain as

is reflex tenderness.

Hurst's theory is not in accordance with accepted ideas. As is generally recognised, the mucous membrane, submucous tissue and muscular coats of the stomach are insensitive to mechanical stimulation; and it is usually stated that the visceral peritoneum is also insensitive. Tenderness felt over the region of an ulcer is, as McDowall (60) says, usually held to be due to inflammation of the overlying parietal peritoneum.

This view was originally due to Lennander (61) who maintained that all painful sensations within the abdominal cavity were transmitted only by the parietal peritoneum and its subserous layer, both of these being well-supplied by cerebro-spinal nerves. Kappis (62) supported Lennander but claimed that the mesenteries were also sensitive to mechanical stimuli. Cope (63) concluded that inflamed, distended intestines (and probably other viscera) were sensitive to pressure from without and considered the parietal peritoneum was the localising factor.

Very recently the unorthodox and interesting contentions of Morley have thrown new light on the foregoing considerations and seem to us to co-ordinate apparently conflicting views. Morley and Twining (64) confirmed Hurst's observations

regarding tenderness. They marked the tender points in different postures with a metal ring fixed to the skin by plaster, after filling the stomach with barium. Subsequent X-ray photographs showed the rings to be very near or actually corresponding with the position of the ulcer in the various postures. Morley (65) however denies the existence of "visceral tenderness" as defined by Hurst. In support of this he states that seizing the base of an ulcer with a toothed-forceps, when operating under local anaesthesia, failed to produce pain. Palpation and squeezing of an ulcer likewise resulted in no pain. He is of the opinion that the parietal peritoneum is the sensitive structure. He goes further and denies Mackenzie's "viscero-sensory" and "viscero-motor" reflexes. There is no evidence, according to Morley, that the splanchnic nerves are capable of transmitting mechanical stimuli and by radiation enabling them to pass to the cerebro-spinal nerves. Morley considers that a much simpler and more probable explanation is what he calls the theory of "Peritoneo-cutaneous radiation and the peritoneo-muscular reflex". His principal argument is from the occurrence of "shoulder-tip pain",which results from stimulation of the sensory endings of the phrenic nerve in the peritoneum on the under surface

of the diaphragm and causes pain in the area
supplied by the descending branches of the third
and fourth cervical nerves. "There is here", he
says, "no question of the highly doubtful hypo-
thesis of a radiation of pain from the splanchnic
to the somatic system of nerves". The rest of the
parietal peritoneum resembles the diaphragmatic
peritoneum except in one respect. Each spinal
segment which supplies nerves to a portion of the
parietal peritoneum lining the abdominal wall, also
supplies a strip of overlying skin, but in addition
takes a share in the innervation of the overlying
muscles. Morley believes that the pain produced
by stimulation of the rest of the parietal
peritoneum is referred to the superficial struct-
ures exactly as in shoulder-tip pain. It is not
appreciated as arising in the parietal peritoneum.
The radiation is through the posterior cornua or
possibly through the posterior-root ganglia. Deep
tenderness is due to stimulation of the parietal
peritoneum when it is pressed down by the
examining finger. The actual stimulation may be
a chemical one, or a mechanical one from roughening
of the surface by fibrin.

In visceral disease we have a prolonged
flow of stimuli into the cord and the affected
segment apparently becomes unusually sensitive.

Other stimuli which would not normally produce any
response now tend to evoke sensations and reflexes
when they reach the sensitive segment. Postural
tone is the result of the interaction of efferent
and afferent impulses. And in the case of a
hypersensitive segment the tone is increased, pro-
ducing thereby the well-known rigidity. Ryle (66)
regards viscero-sensory and viscero-motor reflexes,
when present apart from a severe visceral crisis,
as indisputable evidence of an inflammatory or
ulcerative lesion. This is undoubtedly true,
whatever be the mechanism by which these phenomena
are produced. Whether the afferent stimuli pass
to the cord along the splanchnic nerves or from
the peritoneum along the cerebro-spinal nerves is
controversial. It occurs to us that the common
clinical observation that pressure over the muscle
in cases of rigidity results in a further sudden
increase of rigidity is of interest in this
connection. Admittedly pressure over the stomach
will increase visceral pain and presumably the
extra stimulation would be carried along the
splanchnic nerves and be reflected in increased
rigidity. In actual practice however sufficient
pressure to raise appreciably the intra-gastric
tension need not be applied to cause the sudden
increased rigidity --- if indeed the pre-existing

rigidity would permit pressure sufficient to raise intra-gastric tension. A very slight pressure is all that is required to stimulate the production of an increase in rigidity. This, in our opinion, is in favour of Morley's peritoneo-muscular reflex and against Mackenzie's theory.

Hurst (67) at one time considered that reflex rigidity resulted from increased tension of the gastric muscle fibres. Ryle (68) disagreed and asserted that the somatic phenomena of visceral disease are symptomatic of the lesion which causes the visceral pain and not a reflection of visceral pain. In his view "when visceral pain is due to mechanical causes, or occurs independently of a gross lesion (as in ordinary colic) or in response to a distant stimulus (as in appendix dyspepsia), then there are no associated viscero-sensory or viscero-motor symptoms in the somatic zone served by the segment supplying the painful organ". Hurst (69) recently subscribed to this view. This lends support, we consider, to our suggestion in the previous paragraph that Morley's theory is correct. His explanation seems very much more sound and practical than to suppose that while the pain in gastric ulcer is due to increased tension, rigidity does not result from this stimulus but is "symptomatic of the lesion". Doubtless Ryle is correct

in his contention, and Hurst has seen fit to approve
it, but by what mechanism is rigidity symptomatic
and not a reflex from visceral pain?   In our opinion
only Morley gives an explanation.   He states that
where there is marked rigidity of the true
involuntary type operation always reveals an unusual
degree of inflammatory reaction round the ulcer, in
such a situation that it comes into immediate
relation with the anterior parietal peritoneum (70).

Cutaneous Hyperaesthesia, etc.

This phenomenon is so readily affected by
suggestion and so liable is one to be led astray
that when detected it is scarcely of any value in
gastric disorders.   Moreover, Hurst (71) is of the
opinion that it is seldom present in affections of
the oesophagus, stomach, intestines, liver and
pancreas --- too seldom indeed to be useful.

Exaggerated abdominal reflexes are rarely
of assistance in diagnosis and the same may be said
of pilomotor and vasomotor reflexes, though on
occasions they may be helpful.

Bennett (72) considers the tender spots in
the spinal region of more clinical value than the
cutaneous hyperaesthesia just mentioned.   These
were first noted by Boas and occur most commonly in
gastric ulcer, gallstones and renal disease.   They
are to be found by deep pressure in the region of

the transverse processes of the various vertebrae.
They are best located by pressing the hand under
the patient's spine while he is lying on his back.
This permits muscular relaxation.   Commencing
at the 12th. costo-spinal angle the transverse pro-
cesses are easily recognised.   Bennett maintains
that tenderness on pressure in two-thirds of all
peptic ulcers is found opposite the 8th. 9th. or
10th. dorsal vertebrae.   In ulcer of the body of
the stomach the point is usually to the left;   in
ulcer of the pyloric region it is usually to the
right.   In gall-bladder disease it is almost
invariably to the right and usually opposite the
8th. 9th. 10th. or 11th. dorsal vertebrae.
Appendix disease also gives right-sided tender spots,
often opposite the 12th. dorsal or 1st. or 2nd.
lumbar vertebrae.

CHAPTER 3.

METHOD OF INVESTIGATION & RECORDS OF CASES.

(1)   Method of Investigation.

      (a)   The History & Interrogation.
      (b)   The Examination.
      (c)   The Use of Special Methods.

(2)   Outline Scheme of Investigation.

(3)   Observations on the Scheme.

(4)   Case Records.

(1) <u>METHOD OF INVESTIGATION.</u>

The investigation can be divided into three stages:--

    (a)   The History and Interrogation.
    (b)   The Examination.
    (c)   The Use of Special Methods.

(a) <u>The History and Interrogation.</u> This is by far the most important part of the Investigation; and if done thoroughly and systematically is, in the majority of cases, sufficient to afford an accurate diagnosis. Indeed, Hurst (73) states that, with care, this is possible in 75 per cent. of cases. But the elicitation of a really good history is by no means an easy task. The physician has to contend both with the stupidity of the less intelligent and the loquacity of the better educated. And some sufferers from gastric complaints seem to be especially difficult to manage. It requires great patience, and often more than a little tact, to extract from them the desired information. In no branch of Medicine is it more important to avoid leading questions. This may seem elementary but the patient often has preconceived ideas of his complaint, the outcome of previous examinations or possibly his own reflections. It cannot be too strongly insisted upon that the patient must be allowed to tell his own story. The examiner should, as far as his skill permits, restrict himself to

guiding the patient's story and obtaining elaboration of certain points.   Patients are so often susceptible to suggestion that questioning, on the basis of a scheme, begun at the outset of an interview, is most liable to be unsuccessful.   We found that, for example, to ask a patient if the pain was worse after food, almost invariably provoked an affirmative response.   The question "How long after?" was usually answered in some vague way.   If we then fell into the trap of prompting by such a remark as "One hour or two hours?" then the immediate reply was either "One hour" or "Two hours". Subsequent questioning with reference to the patient's daily employment revealed only too often that the pain occurred perhaps half an hour or even three hours after food.   The result was then dire confusion in most cases;   or at best, a complete contradiction of earlier answers.   When, on the other hand, the patient was allowed to recount his own story in his own way, he usually did so with fair accuracy and committed himself sufficiently for us to obtain any further information required with a moderate degree of ease.   Perhaps the best way is to ask the patient to describe a typical day from waking time one morning till the same time on the following day.   This should be a narration of all that happens in that period --- when the patient

wakes, when he rises, when he breakfasts, how the
forenoon is employed, when his meals are taken and
so on. The patient will then give his medical
attendant a reliable account of two important con-
siderations: firstly, his habits; and secondly,
when and how his illness affects him. A few
questions interposed here and there should reveal
accurately, the position, nature and time of onset
of his pain and the aggravating and relieving
factors. If the patient is skillfully conducted
through his story, few questions will remain to be
answered and these can be safely approached by the
ordinary method of investigation. When an
enthusiastic enquirer sets himself up from the start
as a sort of tribunal, before which the anxious
patient is compelled to appear, the replies obtained
are almost certain to partake of the qualities of
evidence given by a poor witness.

Although questioning on the basis of a
scheme is to be deprecated, the practitioner
obviously must have in his mind some such scheme or
system. Otherwise questions will remain unanswered
which are of import in the diagnosis.

The principal symptoms upon which inform-
ation must be obtained are pain, vomiting and loss
of weight. When all of these, or even any one of
them, are prominent features, the case is almost

certainly an organic one.   Certain other associated
symptoms, of course, occur in most cases, but the
above three have usually greatest significance.
Any points regarding them which have not been fully
answered in the patient's own account of his ill-
ness, should be investigated seriatim.

      We shall give below an outline of the
scheme we have employed and follow it by comments
upon, and explanations of its various sections.

(b) The Examination.   This is of less importance
than the interrogation; but without it no diagnosis
is justifiable.   Although it is frequently barren
of result, it may reveal some lesion in another
part of the body to which the dyspepsia is second-
ary; or it may provide valuable confirmation of
the suspicions of organic disease already aroused
by the history.   The procedure we have adopted is
outlined in the complete scheme of investigation
but is roughly composed of three parts:--

    (1) General Inspection of the patient
         with reference to physical con-
         dition and type.

    (2) Examination of Abdomen.

    (3) General Examination of all systems.

We consider that this is a logical sequence.   The
general appearance is at once obvious and it seems
natural next to examine the affected region.   The
third part is a safeguard against omissions, which,

though not suggested by the history, may be of vital importance in the diagnosis.

(c)   The Use of Special Methods.   These include tests which should be done by the practitioner and also the use of X-rays, which involves the co-operation of a radiologist.   The tests necessary are:--

    (1)   The Test Meal.

    (2)   The test for Occult Blood in the faeces.

    (3)   The Examination of the blood, urine, and the estimation of the blood pressure, etc.

    (4)   X-rays.

Too much reliance should not be placed on these tests;   but as confirmatory evidence in selected cases they may prove invaluable.   Recently the tendency has been to exalt the use of X-rays and to decry chemical tests.   While the early promise of the test-meal has not been fulfilled, its use in conjunction with the test for occult blood will, in nearly all cases, give the requisite confirmation of clinical findings.   Hurst (74) says, "From the experience gained in the examination of hundreds of cases, I am convinced that the discovery of occult blood in the stools, when a sufficiently delicate method is used and proper precautions are taken to eliminate the various possible fallacies, is as valuable a sign of organic disease as the X-rays.

It is present in all cases of carcinoma of the
stomach and in the large majority of cases of
chronic gastric ulcer and duodenal ulcer.   Its
gradual disappearance during treatment is a very
valuable guide in the treatment of the latter."
When one considers such an ex cathedra statement,
the importance of it to the practitioner is at once
apparent.   It puts into his own hands the means
necessary to complete almost any enquiry into a
gastric complaint by himself.   It, moreover,
furnishes him with an accurate indication of the
progress of his treatment.   From the patient's
point of view, it has both practical and economic
advantages.   When hospital facilities are avail-
able, X-rays may be freely used;   but when these
involve private expenditure, the patient may be
loath to incur what is often a payment beyond his
means.   And the use of X-rays as a check on the
progress of treatment is a luxury outwith the reach
of most private patients.   It is just such
practical considerations as these which it is our
purpose to examine.   The wholesale employment of
even these simple methods is, of course, unnecessary
and beyond the scope of the busy practitioner.
We hope to show in which type of case their employ-
ment is essential.

(2) OUTLINE SCHEME OF INVESTIGATION.

The History and Interrogation.

(A) History.
        (1) The patient's account of an
            average day.
        (2) The history of the complaint
            from its commencement and the
            history of the present attack.

(B) Interrogation.

        (1) Pain or discomfort.
            (a) Position & radiation.
            (b) Nature.
            (c) Time, mode of onset and
                duration.
            (d) Aggravating & relieving
                factors.

        (2) Associated symptoms.
            (a) Nausea, vomiting, haemat-
                emesis.
            (b) Loss of weight.
            (c) Flatulence, heartburn,
                waterbrash etc.

        (3) Previous attacks.

        (4) Past history & family history.

The Examination.

        (1) General condition of patient
            and physical type.

        (2) Abdomen and rest of digestive
            tract.

        (3) Examination of systems.

The Use of Special Methods.

        (1) Test Meal.

        (2) Occult Blood.

        (3) Blood Count, urine and B.P.

        (4) X-rays.

OBSERVATIONS ON THE SCHEME.

The physician has the above scheme before his mind and uses it, after hearing the patient's story, to check the information obtained and supplement it where necessary.   This method we have found involves the minimum of questions and provides the maximum accuracy of response.

Pain or Discomfort.

(a) **Position and Radiation.** Mackenzie (75) pointed out that the pain in gastric ulcer was found to vary with the site of the ulcer, the higher up in the epigastrium the nearer the cardia was the ulcer; and ulcers at or near the pylorus caused pain at the lowest part of the epigastrium.   In a series of 75 cases McLean (76) found no relation between the site of tenderness and the position of the ulcer except in so far as it was more frequently right-sided in duodenal ulcer.   Pain which is slightly to the right is probably biliary or duodenal; pain to the left gastric.   A definitely localised pain is highly suggestive of ulceration.

Radiation is not so common in gastric and duodenal ulcer as in certain other conditions; and when it occurs, it may signify the involvement of other organs, for example, the pancreas.   The pain of appendicular dyspepsia is commonly situated in the epigastrium, though occasionally it may radiate to the right iliac fossa.   Gall-bladder pain is

usually right-sided and may radiate to the right
shoulder, through to the back and scapular region or
across the epigastrium.   The pain of a perforated
duodenal ulcer may radiate to the right iliac fossa
and suggest acute appendicitis. (Case No. 86).
Careful examination and interrogation, however, will
reveal the maximum tenderness in the epigastrium and
a history of pain commencing there.   The pain of
hepatic disorders is usually limited to the right
hypochondrium.   Acute pancreatic disease usually
manifests itself by pain in the epigastrium or left
hypochondrium.   This subject will be discussed more
fully under the separate diseases.

(b) Nature of Pain. The first question to decide
here is whether the pain is real or merely a dis-
comfort.   This is of great importance as it may be
the main reason for deciding if the causal condition
is organic or functional.   Real pain in nearly every
case means organic disease.   The criteria of real
pain are whether it makes the patient give up, or
wish to give up, what he is doing; whether it
interferes with sleep, or, in severe cases, causes
the patient to roll about or break into perspiration.
The discretion of the examiner is, of course, re-
quired to estimate the patient's susceptibility to
pain.   Here again is an advantage of the patient's
being allowed to tell his own story, for it gives

From Mackenzie's "The Future of Medicine".
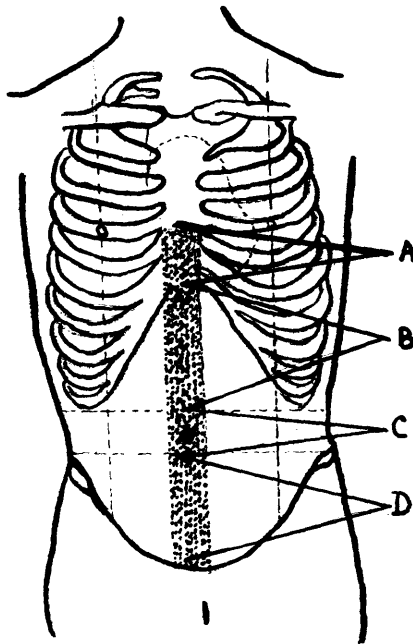
The shaded part shows the area in which pain is felt in certain affections of the digestive tube. Peristalsis passing along the intestinal tube may give rise to pain felt descending in the shaded areas.

A. Area in which pain is felt in affections of the Oesophagus.

B. Area in which pain is felt in affections of the Stomach.

C. Area in which pain is felt in affections of the Small Intestine.

D. Area in which pain is felt in affections of the Large Intestine.

the listener an invaluable insight into his temper-
ament and disposition.   Great variations in the
character of the pain are found.   There is the
sense of distension or dull weight often found in
functional conditions.   Burning pain may be ex-
perienced when an ulcer is present and in a less
pronounced form in functional conditions associated
with hyperchlorhydria.   Continuous pain, often
sharp and lancinating, usually indicates cancer.
(c) Time & Mode of Onset and Duration.   Perhaps it
is the time of onset which is the most important
point regarding gastric pain.   Until recently it
was considered almost sufficient to afford a diag-
nosis.   At any rate, it was taken as sufficient to
indicate a gastric or duodenal ulcer if the pain had
a definite relation to the taking of food.   "Hunger-
pain" was thought to be pathognomonic of duodenal
ulcer, but we now know it may occur in gallstones,
chronic appendicitis, certain functional dyspepsias;
and even renal calculus may, on occasions, cause it.
Its value however is little, if any, lessened by
these facts;   because in conjunction with other
evidence, it is usually of prime importance in the
diagnosis.

      The mode of onset is frequently of great
assistance in distinguishing between different
conditions.   A most helpful way of regarding this

is suggested by Hutchison (77) who uses the following
divisions:--

       (1)   Catastrophic.
       (2)   Episodic.
       (3)   Intermittent.
       (4)   Constant.

(1) Catastrophic. By this is meant pain of sudden
origin and overwhelming intensity --- coming "like a
bold from the blue". These are surgical cases
where this occurs and immediate operation is the
first consideration. They include perforation of
hollow viscera, haemorrhagic pancreatitis, sudden
intestinal obstruction etc.

(2) Episodic. Here the pain arises at long and
irregular intervals with periods of complete freedom
between attacks. It is to be distinguished from
Intermittent pain. Hutchison's simple simile
illustrates the distinction. "As one goes along a
street the lamp posts are 'intermittent' but the
pillar boxes are 'episodic' ---they recur at long
and irregular intervals". Episodic attacks may
become so frequent as to simulate intermittent ones.
Enquiry as to the early stages of the illness make
the episodic character apparent.

       Under episodic are to be considered the
colics. They are the result of unusually forceful
contraction of a hollow viscus and occur therefore,
in a series of spasms. Severe cases may be as pain-
ful as those of the Catastrophic class; but in the

latter, the patient is never restless, while in the former he is intensely so. Finally, episodic pain never indicates a lesion of the stomach.

(3) <u>Intermittent.</u> In this class the pain occurs daily but does not last all day. Pain of this type occurs most commonly in chronic dyspepsia and will therefore be considered fully later.

(4) <u>Constant.</u> This is a rare feature of gastric pain and might suggest malignant disease. Many cases of carcinoma of the stomach, however, are practically painless. When the colon is the seat of malignant disease colic usually ensues. Cancer of the liver, and peptic ulcer which has spread to surrounding organs, may cause it. Pressure pain, from enlarged lymphadenomatous glands, caries of the spine or abdominal aneurysm cause constant pain, commonly with nocturnal exacerbations. In cases with a history of previous operation, adhesions may be the cause.

<u>Duration.</u> This affords information of some value. In addition, the manner of cessation is of importance. For example, if the pain ceases suddenly, it indicates stone as a cause. In a doubtful case, this may be the one factor in deciding the diagnosis. McLean (78) recounts such an instance where it had been impossible to decide clinically between ulcer and gall-stones. Further questioning revealed that

on one occasion the patient was about to leave a
theatre, on account of the severity of the pain,
when he experienced sudden complete relief. This,
of course, eliminated ulcer.

Gall-bladder pain may be transient or may
last several hours. Ulcer pain varies in duration.
In gastric ulcer it lasts till vomiting brings relief
or gradually disappears before the next meal.
Duodenal ulcer typically lasts until more food is
taken.

(d) Aggravating and Relieving Factors. A consider-
ation of these is frequently of much help in cases of
dyspepsia. The effect of the ingestion of food
must be noted. This is in part included under the
Time of Onset. Any special foods which cause either
relief or exaggeration of symptoms should be ascert-
ained. In atonic conditions, fluids cause more
discomfort than solids. In ulcer, milk diet usually
brings relief. In appendicular gastralgia, which
may closely simulate duodenal ulcer, milk diet has a
much less marked effect. The effect of alkalies is
another point. Usually, considerable relief follows
in cases associated with hyperacidity. In addition,
belching of gas after a dose of bicarbonate of soda,
is a useful indication of the presence of excess of
acid. The value of this simple test is not always
appreciated. It must not be supposed, however, that

relief following alkalies indicates hyperacidity.
Even in cases of subacidity, relief may follow the
administration of alkalies. The explanation of this
is not certain but we have given one possible
explanation in Chapter 2.

Posture, including recumbency, must be
enquired into. In conditions for example, of
gastroptosis and spinal conditions simulating
dyspepsia, this may be of importance. Increased
abdominal tension, as in defaecation, weight lifting
and movements of the diaphragm as in cough, must be
noted. Movements of the trunk and lifting the legs
when lying down, put the recti on strain and may
thus help to differentiate pain of muscular origin.
Pain on running, jumping, jolting etc., may suggest
stone. Severe exertion may bring on appendicular
gastralgia.

Associated Symptoms.

These include many symptoms of major and
minor importance worthy of study and will require
fuller consideration under separate diseases.
Vomiting and haematemesis, it may here be emphasised,
are indicative of organic disease when they are
repeatedly present. Vomiting, however, may occur in
certain functional conditions; and haematemesis,
apart from such conditions as portal congestion,
does not necessarily indicate a gastric lesion: it

has frequently been observed in dyspepsia due to appendicular disease. Loss of weight must be accurately ascertained. An exact statement in pounds should if possible be obtained from the patient. A mere impression is often misleading. When present, it indicates organic disease, subject to the provision that the patient has not been on an over-restricted diet.

Previous Attacks.

The importance of this section is obvious. Frequently, the history of the present condition may not be typical; but that of the early stages may yield the requisite information for diagnosis. For example, pain may be constant at the time of examination and have apparently no relation to food; but it may at an earlier stage have been typical, intermittent "hunger pain". A history of jaundice will suggest the gall-bladder and so on.

Family History.

Many examples are on record, a fact which Hurst was the first to point out, of "ulcer families". This, of course, is due to the relation between physical type and the nature of response to disease. It has, therefore, value in many cases. Again the family history may suggest certain general diseases.

THE EXAMINATION.

(1) General Condition and Physical Type. A brief examination of the patient's general condition

is made at the outset.   Is he cachectic, emaciated,
jaundiced and so on?   Does he belong to the hyper-
sthenic or the hyposthenic type?   Those of the
former are the broad-chested vigorous athletic
variety;   the latter the narrow-chested, poorly
developed.   Valuable help may be obtained from a
consideration of these points.   We have discussed
their significance fully under the diseases in which
they are of import.

(2) Abdomen.   The attention should now be
directed to the abdomen.   The patient should be
lying in a comfortable position on his back, with
his head supported on a pillow.   It is highly
desirable to have as good a light as possible so
that the abdomen is evenly illuminated.   The hands
of the examiner should be warm otherwise difficulty
will be experienced.   He should carefully avoid
questions, as far as possible, and rely on the
patient's expression for the detection of tenderness.
Patients always expect to be asked to co-operate in
this part of the examination and if they are not
they frequently volunteer information.   As this is
not wanted, it is wise to engage the patient in con-
versation.   Any attempt at "small talk" is often
unsuccessful because the patient's main interest at
the time is, quite naturally, himself.   A method
which seldom fails to distract his attention from

his abdomen, is to enquire again into some point of the history. Here he is usually riding his favourite hobby-horse and a very small expenditure of interest, on the part of the physician, will serve to keep him occupied. The general appearance of the abdomen has already been noted. Flatness or distension should be observed and in the case of the latter, whether it is local or general. An abdomen which is hollow in its upper part and full low down suggests visceroptosis. This should be further tested, preferably at a later stage, by making the patient stand up. A dilated stomach tends to be associated with a relative fullness in the upper part and a degree of emptiness below. Peripheral fullness may suggest distension of the colon; and central fullness may indicate distension of the small intestine, often from obstruction at the caecum. The normal respiratory movement of the abdomen should be noted. Finally, inspection is concluded by looking for visible peristalsis of the stomach or intestines. This may sometimes be rendered more obvious by flicking with the finger. Although it may be normal in thin subjects, especially elderly multiparae, it is usually a sign of an obstructive lesion. The physician may have to revert here to the history and question the patient closely again regarding the radiation.

Mackenzie (79) mentions a most illustrative case of
the importance of this point.   A man who had
suffered for four years from violent pain, usually
of one or two days' duration, every four or five
weeks, was operated on for gallstones but none were
found.   Mackenzie elicited the information, after
much trouble, that the pain commenced slightly at
the lower part of the epigastrium and descended,
with increasing severity, to the lowest part of the
umbilical region, beyond which it did not go.   The
wave-like attacks indicated colic but the persistence
and greatest intensity in the central part of the
abdomen excluded renal or biliary calculus.   The
diagnosis of partial obstruction at the lowest part
of the small intestine by an adhesion from an old
appendix operation was confirmed at operation.
Palpation.   This is the most useful and reliable
method of examining the abdomen.   It should be
commenced away from the seat of the lesion and then
the abdomen should be systematically examined over
its whole extent.   Special attention should be
directed to the epigastrium, left hypochondrium,
right hypochondrium and gall bladder region, the
right iliac fossa and the kidneys.   Great care
should be given to detecting rigidity even of minor
degree; and tenderness is of course very signifi-
cant.   A tumour may be felt and, if so, every means

must be used to determine its position. This may
be aided, if necessary, by distending the stomach
by the administration of a Seidlitz powder. Its
relations to respiratory movements should be
observed. Succussion, two to three hours after
food, suggests delayed emptying. Hyperaesthesia,
detected by scratching and pinching folds of skin,
is sometimes though not often helpful.

Percussion. This has little value except for the
detection of shifting dullness.

Auscultation and auscultatory percussion
have no place in our examination; the first, because
we know of no use for it and the second because it is
not sufficiently accurate to rely upon.

Rectal examination should always be done
and, at this stage, examination of the supraclavicul-
ar fossae for the presence of glands should be made.

The Remainder of the Digestive Tract. The mouth
should be examined and the condition of the tongue
noted. Not much reliance can be placed on this
time-honoured practice but it may be valuable, as
for example when a glossitis suggests pernicious
anaemia. Lead poisoning and Addison's Disease may
show in the mouth. The teeth should next come under
observation. At one extreme of the pendulum-swing
of medical fashion is cherished the belief than in
bad teeth is the explanation and cause of all

diseases to which the human flesh is heir.　At the other extreme, towards which the pendulum is now travelling, is the view that oral sepsis is of little or no account.　It is reasonable, however, to insist on attention to the teeth at the outset of treatment of indigestion.　There are two more considerations in regard to the teeth.　They may be too tender to allow of efficient mastication;　and the constant swallowing of septic material may set up a gastritis. Even where sepsis is absent, or of slight degree, note should be made, as Alvarez (80) points out, of their adequacy as regards grinding surface.

The tonsils are important sources of sepsis and should be examined with care.　Septic sinuses or chronic pharyngitis with a septic discharge may be the cause of the dyspepsia.

(3)　Examination of Systems.　A brief examination of the various systems of the body may reveal a cause of dyspepsia or some concomitant disease, failure to observe which might cause much subsequent heartburning.

Circulatory System.　The pulse should be examined in the usual way and also the heart.　Estimation of the blood pressure, if necessary, is conveniently performed at this juncture.　The blood pressure may indicate hypertension as the cause of dyspepsia --- not an uncommon cause, according to Alvarez (81),

who found 26 cases out of 500 dyspepsias. Such
rare conditions as Addison's disease might be
diagnosed. It is stated that the pressure is lower
than normal in cases of carcinoma,(Paterson, 82).
We fear, however, that this would only be found in
cases so far advanced as to be obvious in their
diagnosis by other means.

Respiratory System. The frequency with which
phthisis first manifests itself by dyspeptic symp-
toms is well-recognised and makes examination of the
chest essential.

Nervous System. The pupils, knee-jerks and plantar
reflexes should be examined and if necessary also
the fundi. Such conditions as Tabes Dorsalis have
to be excluded. Hypertension would probably be
recognised by examination of the fundi, as might
also be diseases such as diabetes etc.

Urinary System. The examination of the urine
should of course be a routine. Where necessary,
pelvic symptoms will demand an examination.

Blood. This is included under special methods. It
will be necessary, of course, where there is any
possibility of pernicious anaemia or secondary
anaemia, where the latter is giving rise to a
debilitated condition to which the dyspepsia may be
secondary. In the latter connection, the recently
observed achlorhydric anaemia is of interest as it
might be discovered in the investigation of a gastric

case.   (Moore, Henry 83).

THE USE OF SPECIAL METHODS.

(1)   The Test Meal.   We have employed the now old-
fashioned Ewald meal.   Although much useful inform-
ation for scientific investigation of gastric
function can only be obtained by the fractional
test meal, we consider there is no information of
major clinical importance which can not be as
easily obtained by the Ewald meal.   Moreover, the
fractional test meal has difficulties in General
Practice.   Even the Ewald meal is not without its
disadvantages for the practitioner.   We have found
the test meal of considerable help in the diagnosis
of chronic gastritis and carcinoma ventriculi.   In
other conditions (and we have made frequent use of
it in these) we have found it of limited value.
MacLean (84) says "In my experience, its use as a
routine measure is unnecessary in the majority of
the usual gastric conditions, for practically all
the essential information it affords can be obtained
by simpler means".

        This subject can hardly be dismissed with-
out observing how great has been the value of
gastric analysis in advancing our knowledge of the
functions of the stomach.   This however is no
indication for the employment of the test meal in
the routine investigation of dyspepsia.   Moreover,

of recent years much light has been shed on many

general diseases by the use of the test meal.

Speaking of the failure of the test meal to justify

its early promise, Abrahams (85) says ".....it is

interesting to note that whatever remains of the

original advantage has been greatly overshadowed by

the value and importance which attaches to the

relations more recently observed between variations

of secretions, particularly achylia, and general

disease".

The Significance of Lactic Acid.   The importance

of this question to the General Practitioner is

obvious.   It is essential that he should appreciate

the significance of lactic acid in the gastric con-

tents.   Is it worth his while to test for lactic

acid and does its presence indicate carcinoma?

It has long been held that lactic acid, in

association with the absence of free hydrochloric

acid, was suggestive of carcinoma.   MacLean (86)

emphasises the value of testing for its presence.

He states that absence of free hydrochloric acid

and the presence of lactic acid in traces may occur

in chronic gastritis but the long history will, in

most cases, exclude cancer.

Ufflemann's reagent, according to MacLean,

is for practical purposes, useless; he recommends

the ferric chloride test but with the addition of

mercuric chloride. We employed this test but found that the presence of lactic acid was frequently obscured by a precipitate. J.D.Robertson (87) states that he and Dodds, from a series of tests, concluded that the precipitate was due to bile. They also found MacLean's modification of Hopkin's thiophene test even more subject to interference and concluded that only quantitative methods were accurate. We have recently, following the advice of Murray and A.B.Robertson (88), made an ether extract and shaken this with animal charcoal. As far as our limited experience goes this obviates the difficulties due to bile.

J.D.Robertson says (89):--"It would appear useful to consider the results from two points of view; firstly, the clinical, as to whether by ordinary qualitative tests or simple reaction it is possible to state that the presence of lactic acid is a helpful sign in the diagnosis of carcinoma of the stomach. The second point of view is purely an academic one, namely, whether the presence of lactic acid in gastric contents is pathognomonic of carcinoma of the stomach ......
There is unfortunately no difficulty in answering the first. The qualitative tests for lactic acid are so easily influenced by so many substances that no reliance can be placed upon them ....It would

appear, therefore, that no help can be obtained in the diagnosis of carcinoma of the stomach by the use of qualitative tests, and it follows that any consideration of this question from either of the points of view described at the commencement of this discussion must be based on quantitative methods".   He goes on to give evidence showing the second question to be untenable, viz:- that lactic acid is a specific product of cancer of the stomach. He shows that lactic acid results from fermentation, and in a series of cases (90) claims that it was present in 47 per cent of non-malignant diseases in the resting juice, and 36 per cent in the later specimens.   Finally,(91) he concludes that lactic acid, when found in cases of carcinoma, is not an early sign;   and when it does make its appearance, the growth is usually too advanced to be amenable to treatment.

J.D.Robertson's views are upheld by several authorities, for example Murray and A.B. Robertson (88) and Bennett (92).   On the other hand there are those who believe that the presence of lactic acid is extremely important. Einhorn (93) published a series of cases of non-malignant gastric troubles in which lactic acid was present and although he says that it is "by no means a specific sign" he considers its presence, in suitable

clinical associations, as suggestive of carcinoma.
Craven Moore (94) and Davidson (95) both believe
that the presence of lactic acid is of first
importance in the diagnosis of cancer of the stom-
ach.   The former found it present in 19 out of 32
cases of cancer and only present in 7 out of 287
cases other than cancer.   The latter states that
lactic acid is commonly found in cases of cancer of
the stomach but is rarely detectable by the ferric
chloride or thiophene tests in any other condition.
He considers that the results obtained by J. D.
Robertson and Dodds were due to the use of too
sensitive tests.   He believes that positive ferric
chloride or thiophene tests occur more frequently
in cancer of the stomach than in any other gastric
condition.   He agrees that the presence of lactic
acid is in no way pathognomonic of gastric cancer
but he is of the opinion that the factors which fav-
our its production are more frequently present in
cancer than in any other gastric condition.

       We have given the evidence of both sides
in this controversy and it is manifestly impossible
for us to be dogmatic on the question.   As far as
our experience goes lactic acid strongly suggests
that cancer of the stomach is present.   It is
essential of course that this sign be taken in
association with other clinical findings;   and if

this be done we consider that the test is well worthy of performance.

(2) <u>Occult Blood.</u>   In all cases where there is the slightest possibility of ulceration, simple or malignant, this test should be performed.   We employ the Benzidine test, as recommended by MacLean.   Unfortunately we began to appreciate its immense value to the General Practitioner rather late in our investigations and we did not make as full use of it in the early stages as we now do.

It is unnecessary to discuss the technique of the test since it is well-known.   One point however is of some importance, i.e. the question of diet prior to the performance of the test.   MacLean (96) says:-- "In spite of the usual statement in the text-books that it is necessary to put the patient on a special diet in order to exclude the possibility of the meat in the diet giving a positive reaction for blood, this is not at all necessary".   He goes further and declares that a liquid diet, by lessening bleeding, defeats the purpose of the test.   So revolutionary a statement could hardly be accepted without trial. We examined twenty specimens after an ordinary diet where there was no possibility of internal bleeding and obtained positive reactions in fourteen. Accordingly we follow the usual directions as to a

meat and chlorophyll-free diet.   In addition,
patients are warned to avoid too vigorous brushing
of the teeth and if any bleeding occurs to rinse the
mouth until all traces of blood disappear.

We have referred very fully to the value
of this test under the diagnoses of the various
gastric diseases.

Blood. Examination of blood should be made in the
usual way in cases such as those indicated above.

X-rays. When should these be used?   The question
is an important one for the General Practitioner.
When hospital facilities are not available, he must
realise when they are so indispensable as to justify
the sacrifice so often necessary to afford them.
We consider that in many cases of ulcer they are
frequently unnecessary.   If any doubt remains as to
the true nature of the condition after full clinical
examination and the tests recommended above, then
the employment of X-rays is desirable.   It is
doubtful however if such a contingency exists,
especially if the physician makes frequent tests for
occult blood during the time of an acute attack.   In
all suspected cases of carcinoma, X-rays should be
employed.   It is doubtful if any surgeon would
consent to operate without this as a preliminary.
Where the condition is advanced and inoperable,
then of course X-rays are unnecessary.   It should

be realised by the practitioner that the employment of X-rays does not dispense with the necessity of full clinical examination and testing for occult blood.   Especially is this so when the radiological report is negative or gives some vague information such as "atonic condition of the stomach".   We have known of more than one case where, possibly from poor technique, X-rays failed to reveal the true condition and allayed suspicions aroused clinically of the true condition of carcinoma.   In suspected cases of gall-stones, renal stone or ureteral stone, X-rays are frequently very valuable.

Case Records.   It would be difficult to compute the loss to Medicine from failure to record cases.   If one considers the clinical acumen of some of the older practitioners, gained like Paré's knowledge of wounds from everyday experience, it can be realised what has been lost through the impossibility of their leaving written evidence of their experience.   If the knowledge of some of these men, or even a small part of it, had been available for future generations, the Medical Profession and Humanity itself would have been greatly the richer   And how much more they themselves might have achieved if their accumulated experience could have been assembled and arranged for the use of each other!

Research in General Practice is a rare

thing and it might hastily be supposed to be due to lack of opportunity.   A little reflection makes it obvious that this is not the case.   In General Practice there is not only an almost uncultivated but an exceedingly fertile field for research.   The difficulties are great, as anyone who has been engaged in practice well knows;   but more attention should be given to developing research in General Practice than has been given in the past.   Sir James Mackenzie was the great protagonist of this view. His thesis was, in general, that in the early stages of disease there lay most hope of discovery of its origin and hence the means for its prevention.   The patient who exhibited these early signs seldom if ever went to hospital but to his family doctor.   The opportunity for investigation of the beginnings of disease lay therefore with the General Practitioner.

The appreciation of early symptoms is no easy task.   This is in part due to an inadequate understanding of their means of production and it is an essential preliminary to success that these should be understood.   Medicine has to-day very efficient systems of detecting disease in damaged tissues. This is mainly achieved by the elicitation of physical signs.   But the early stages, before structural alteration has taken place, have no physical signs.   It is often through failure to

appreciate this that much disrepute is levelled at the profession of Medicine. When disease is recognisable, i.e., when structural alteration has taken place, it is frequently impossible to restore the tissues to their normal condition. All too often the patient does not present himself until this stage has been reached and then he is dissatisfied because little perhaps can be done for him. As J.B.S. Haldane (97) says, doctors are too often called to cure a scar. It is doubtful if any advance in the science of Medicine will ever render such cures possible. Hence the desirability of turning serious attention to the question of research in General Practice where the early signs of disease are daily apparent, though seldom understood.

Observations over any extent of time or of any value could hardly be made without records of cases. Herein lies one of the main difficulties. It is sometimes supposed that the present National Health Insurance Scheme imposes on doctors the necessity of attending too large a number of patients and that no time is available for any fuller records than those all too inadequate ones demanded by the Department of Health. It must be admitted that the latter body have recently introduced a system of statistical research in Insurance Practice. It is not our purpose here to criticize

such a scheme but records extracted compulsorily
from practitioners are not likely to bear the stamp
of accuracy.   Moreover, these records into the
early beginnings of disease are made from the
patient's memory, assisted by the practitioner's
interrogation.   A private system of records has the
great advantage that it is built up of written
observations made by the doctor at the time of
examination.

It might be contended that the busy
practitioner has no time to compile such records.
In reply to that we need only state that in this
Practice a system has been in operation since 1923.
Not only has it proved to be of great convenience,
and we trust value, but it has shown itself to be
even time-saving.

In regard to the question of prognosis,
after the discovery of early symptoms, case records
seem to be the only satisfactory solution of a
difficult problem.   If early symptoms are noted
their outcome will, if the records are kept for a
sufficiently long period, be easily apparent.   No
memory can record, let alone reproduce when desired,
all the facts of a past history;   and no account
from the patient is sufficiently adequate or re-
liable.   We have found great help from case records
in the everyday work of diagnosis.   Much time has

been saved by glancing over a card instead of having
to question the patient, and not infrequently has it
suggested the diagnosis.

In medico-legal cases, though these are
rare, we have found the records of great help in
giving evidence.   Similar instances could be
multiplied indefinitely but the foregoing are
sufficient indication of the value of the scheme
outlined below.

METHOD OF CASE RECORDING.

DIAGRAM I.

The above diagram shows the type of card used.
Brief notes were made of all illnesses necessitating
medical attention.   After a few years it was found
that only occasionally did a patient present himself
who had not a card named and begun for him.   After
this initial period was past the trouble involved
was negligible compared with the advantages and time
saved.   Fastened by metal clips to each card were

diagrams, charts, letters from hospital and consultants, diet sheets and any information pertaining to the case. In this connection, we might say that it has frequently been our experience, and the experience of our colleagues, to be thanked by hospital or private consultants for the history we were able so easily to supply. Seldom could we have done this without records. We realise that many practitioners have record systems but we think this is seldom, if ever, the case in industrial practice; and it is perhaps in this type of practice where hospital facilities are almost universally available for accurate confirmation of diagnoses, that most value is likely to be derived from these records.

An attempt was made to classify and index the diseases under physiological systems. After a fair trial this was renounced as it was found that many cases were of such a trifling nature --- although with perhaps serious future import! ---that they could not be diagnosed or labelled.

It was then considered best to index under the presenting or cardinal symptom. Injuries and diseases belonging to the special branches of Medicine, were excluded from the index (but not of course from the case reports). Infectious diseases, venereal and mental diseases and gynaecological

complaints were also excluded.   This was done

because paucity of these cases, and our lack of

special knowledge, made it unlikely that anything

of value could be derived from them.

A list of 14 presenting symptoms, including

one "Miscellaneous" to cover other symptoms or such

terms as Jaundice which were by their nature unlikely

to be the presenting symptom.   The index cards used

were as shown below.   Two columns were used for

such a symptom as "Cough" - one for the name of the

patient, the other for the diagnosis.   In such a

symptom as "Convulsions", three columns were used,

viz:- name of patient, symptom, and diagnosis.   In

"Pain" nine sub-headings were used.   DIAGRAM 2.

CONVULSIONS etc.		
NAME	SYMPTOM	DIAGNOSIS
J. BROWN	FIT	EPILEPSY

COUGH	
NAME	DIAGNOSIS
J. CARR.	PULMON. TUB.

PAIN : ABDOMINAL		
NAME	SITE	DIAGNOSIS
H. ALLAN	EPIGASTRIC	NERVOUS DYSPEPSIA

Indicator cards with headings were used to separate

the index cards.   On the former were such other

symptoms as might be included under the principal

heading (see diagram 3).   The indicators were as

follows:--

<u>Convulsions.</u>   (3 columns).

>   Including Coma, Fits, Faints, Shock,
>   Delirium etc.

<u>Cough.</u>   (2 columns).

>   Including Haemoptysis.

<u>Debility.</u>   (3 columns).

>   Including Pallor, Emaciation, Weakness,
>   Paralysis.

<u>Diarrhoea and Constipation.</u>   (3 columns).

>   Including Melaena.
>   Applying to cases other than Infantile, i.e.
>   over two years of age.

<u>Dyspnoea.</u>   (2 columns).

<u>Infantile Indigestion.</u>   (3 columns).

>   Including Vomiting, Diarrhoea, Constipation,
>   Malnutrition.

<u>Micturition.</u> (3 columns).

>   Including Dysuria, Polyuria, Haematuria,
>   Anuria, Frequency, Enuresis, etc.

<u>Pains.</u>

>   Including ache, discomfort or other disagree-
>   able sensation and sensory disturbances.

>   <u>Subheadings.</u>

Head and Face	(2 columns)		
Throat and Neck	(2	"	)
Chest	(3	"	)
Abdomen	(3	"	)
Back (Lumbo-Sacral)	(3	"	)
Arms	(2	"	)
Legs	(2	"	)
Joints	(2	"	)
Generalized	(2	"	)

It is obvious that certain diseases
will have more than one cardinal symptom and
will be recorded under each.   This has not been

very common in our experience.

INCLUDING:

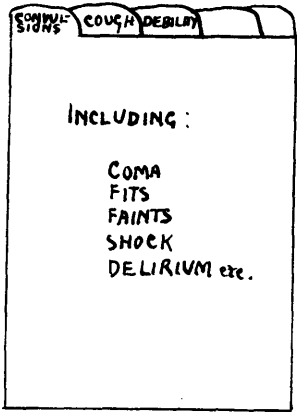COMA
FITS
FAINTS
SHOCK
DELIRIUM etc.

DIAGRAM 3.

We cannot claim any share in originating this scheme.   It was put into operation in 1923 by our former colleagues and we have willingly continued its use and proved its value.

CHAPTER 4.

THE CLASSIFICATION OF DYSPEPSIA.

In approaching this subject we are
sensible of its great difficulty.   We do not hope
to evolve a classification which will take account
of every contingency;   and we doubt if, in the
light of present knowledge, this is possible.   It
must be insisted that our object is to formulate a
scheme, suitable for General Practice, which shall
include all the important types of dyspepsia;   and
the divisions of which shall in themselves be
adequate diagnostic labels, sufficient for the pur-
poses of rational treatment.   (V. Introduction).

It is convenient at this juncture to dis-
cuss some terms and classifications employed in the
standard textbooks.   The organic diseases present
no difficulty: it is in the functional cases that
controversy arises.   Perhaps the most usual text-
book method is that used by Osler and McCrae (98).
These authors speak of motor, secretory and sensory
neuroses.   Now this division has at the present
time no longer any justification.   We have referred
elsewhere to the intimate relation between motor and
secretory function.   Further, the use as clinical
entities, which this classification employs, of such
terms as "hyperchlorhydria", "hypochlorhydria" and
"achylia" cannot now be accepted.   These states
are known to be present in many persons entirely
free from symptoms of indigestion.   In the sensory

neuroses we have such terms as "gastralgia".
Behan (99) defines it as a painful state of the stom-
ach of unknown cause.   He distinguishes as varieties
of it" gastromyalgia" and "gastroneuralgia", accord-
ing to whether the muscles or the nerves are
responsible.   It has been shown, as we have already
said elsewhere, that the viscera are insensitive to
ordinary pain stimuli and that increased muscle
tension is the only adequate stimulus for pain pro-
duction.   It can however be argued, not without good
reason, that the threshold of consciousness in some
patients is lowered and that stimuli which ordinarily
do not give rise to pain may then evoke a painful
response.   This is a common-place psychological
observation in general and it cannot be disproved
at present in regard to the stomach.   These con-
siderations might force us to admit the possibility
of such a condition as "gastralgia" really existing.
As a term for use in affections of the stomach we
prefer however to reject it.   Our main reason is
that, if such a condition does occur, it is a
reflection in the stomach of a general state of ill-
health, probably largely of nervous origin.   The
term "gastralgia" if used would only serve to focus
attention on one symptom and not on the causative
factors, which would be bad from the point of view
of both the patient and physician.

Classifications of dyspepsia have some-
times been based on the presenting symptoms; and we
have "flatulent dyspepsia" and "acid dyspepsia" etc.
It is unnecessary to discuss these in view of what
we have written elsewhere.   They could never be
accurate and seldom helpful. "Fermentative Dyspep-
sia" and"atonic dyspepsia" must also be rejected.
They probably came into being in an attempt to ex-
plain by their names the supposed underlying
pathology; but as they do not appear to serve that
or any other useful purpose they cannot be accepted.

Perhaps the best division of dyspepsias,
"qua" classification, is that enunciated by Ryle
(100), viz:-

(1)   <u>Habit Dyspepsias</u> --- resulting from
        faulty physical habits.

(2)   <u>Nervous or Psychogenic Dyspepsias</u> ---
        due to faulty mental or nervous
        adjustment.

(3)   <u>Toxic and Infective Dyspepsias</u> --- due
        to (a) tissue poisons and (b) general
        and local infective disease, and other
        and more obscure conditions interfering
        with general health and nutrition.

(4)   <u>Irritative Dyspepsias.</u> --- due to stimuli
        originating in a local or distal
        organic lesion.

(5)   <u>Mechanical Dyspepsias</u> --- due to gross
        structural disease or to surgical
        modifications of the anatomy of the
        stomach.

This arrangement has at least one very
notable feature: it does not separate functional

dyspepsia from organic. The fact that it assigns
to each group a definite cause or group of causes,
and also traces the gradual progress from healthy
to frankly morbid states, are powerful arguments in
its favour. Arranged as it is on an etiological
basis we have nothing but praise for it as a class-
ification of the pathology of dyspepsia. It does
not however fulfil the purpose which we are attempt-
ing to elaborate. This thesis has been written
with the viewpoint of the General Practitioner ever
in mind. The ideal classification for General
Practice is, to our mind, one which will enable the
doctor, when he has decided that a case is one of
dyspepsia, to search each division and allot the
case to the one which is appropriate. That done,
if the classification discharges its function, the
diagnosis is complete and he is free to commence
treatment. Ryle's classification could never be
used in such a way.

We have tried and rejected many classifi-
cations, derived from books or devised by ourselves.
The one we have formulated has at least the virtue
of simplicity. We are aware that many objections
to it can easily be lodged. The causes and
manifestations of dyspepsia are so numerous that if
simplicity is to be obtained completeness must to
some extent be sacrificed. We consider that such

sacrifice as has been made is at least compensated
by simplicity; and we do not think that any serious
ill-result could befall patients treated on the basis
of our classification.   If the duties of the General
Practitioner, as defined in the Introduction, be
borne in mind we think our divisions will not
prejudice the proper discharge of these functions.

      We give our classification below and shall
later comment on and explain each division.   They
are not mutually exclusive and in many cases must of
necessity overlap.

	ACUTE.	CHRONIC.

FUNCTIONAL.	(a) Indigestion.	(a) Habit Dyspepsia.
		(b) Nervous "
		    (1) Fatigue.
		    (2) Psychogenic

ORGANIC.	(a) Gastritis.
	(b) Ulcer.
		(A) Gastric.
		(a) Duodenal Ulcer.
		(b) Gastric Ulcer.
		(c) Gastro-jejunal &
		    Jejunal Ulcers.
		(d) Gastric Carcin-
		    oma.
		(e) Duodenal Ileus.
		(f) Gastritis.
		(B) Extra-Gastric
		    (or Reflex).
		(a) Appendix
		    Dyspepsia.
		(b) Gall-bladder
		    Dyspepsia.
		(c) Carcinoma of the
		    Colon.
		(d) Epigastric Hernia.
		(e) Spastic Colon.
		(f) Dyspepsia Assoc-
		    iated with Dis-
		    ease of the
		    Urinary Tract.
		(g)Tabetic Dyspepsia.

The divisions of the acute cases are so in accordance with convention that it is unnecessary to discuss them.

In regard to the chronic organic conditions, those of the "gastric group" are also in keeping with the usual classifications. We have chosen however to depart from convention in considering the reflex forms of dyspepsia as organic. We have done this as their cause is almost always organic disease somewhere in the body and further because we have had in mind the frequent association of certain reflex cases with ulcer. We have thus, as far as possible, reserved the functional groups for those in which organic disease is not present or where its occurrence is either entirely unassociated or very indirectly connected with the gastric symptoms.

We have retained the old division between functional and organic disease because, even though we freely grant several objections to it, it still serves a useful purpose especially to the General Practitioner. Many of the guides in diagnosis, from history, examination and special methods, are nothing more or less than means of deciding whether or not organic disease is the cause of the symptoms.

We have now dealt with the whole classification except the most difficult part --- the chronic functional disorders. If reference be made

to Ryle's classification it will be seen that the fourth and fifth groups and part of the third group are included under our organic division. We are left then with the first two and a part at least of his "toxic and infective" group. Many cases of the latter are not frank cases of chronic gastritis and we at first included a "toxic" group under functional dyspepsia to cover these. In the light of the recent work in Germany mentioned elsewhere, it seems to us that these cases are definitely due to gastritis of a mild order. We shall therefore more appropriately consider these under the section on chronic gastritis. Ryle's first two groups are similar to our own, though we prefer to subdivide the nervous cases into Fatigue Dyspepsia and Psychogenic Dyspepsia.

We propose now to deal with each group as regards symptoms and diagnosis and where there is any departure from conventional divisions to put forward a plea for the method we have adopted.

CHAPTER 5.

THE ACUTE FORMS OF DYSPEPSIA.

(1)   Functional.

    (a)   Indigestion.

(2)   Organic.

    (a)   Gastritis.
    (b)   Ulcer.

ACUTE INDIGESTION.

It is possibly rather an artificial separation to speak of acute indigestion as distinct from acute gastritis.   Many speak simply of acute gastritis and recognise a mild degree and a more severe one.   Doubtless many mild cases are actually due to irritation and exhibit an acute inflammation of the mucous membrane;   but on the other hand a considerable number owe their symptoms, at least in part, to other causes.   The ingestion of a large meal by a person in an exhausted state frequently results in acute gastric disorder.   The stomach muscle is deficient in tone and is suddenly called upon to deal with a meal of large bulk.   From previous considerations it is clear that the pain is due to an inability on the part of the gastric musculature to adapt itself.   The result is over-stretching of the fibres with consequent pain.   It is apparent that this condition would be very inaccurately described as acute gastritis.   Cases of a relatively mild order may occur in which gastritis is partly the cause of the symptoms but it seems more convenient, especially from the General Practitioner's point of view, to speak of these cases as "acute indigestion".   Even an ordinary - sized meal may be sufficient to cause acute dis- comfort if the person taking it is exhausted or in

a state of mental perturbation. Similarly a
perfectly normal person may experience symptoms
after consuming an exceptionally large meal, or an
ordinary-sized one taken in great haste, even in the
absence of exhaustion etc. Unsuitable food is
another cause though in such cases the condition may
more accurately be described as gastritis. We in-
clude it here when the symptoms are of a slight
and transient nature. Idiosyncrasy may account for
cases where the ingestion of a particular food,
which does not ordinarily cause disturbance in the
average person, is followed by discomfort or pain etc.

Clinical Features. Pain, which is not usually
very severe and is often little more than discomfort,
is present as a rule. Nausea and vomiting are
common but not invariable. The tongue is usually
furred and the patient displays anorexia for food.
Headache is sometimes experienced. Diarrhoea often
follows in a day or so if the intestines are in-
volved and is particularly common in children .

Diagnosis. The condition of acute gastritis as
described below must be considered. In it the
symptoms are similar but usually more marked and
often accompanied by more apparent constitutional
symptoms and some fever. More important is the
question of other diseases in which the inaugural
stages are more or less identical. This is dealt
with in the diagnosis of acute gastritis.

ACUTE GASTRITIS.

This condition is usually due to irritation of the lining of the stomach from errors in diet or the ingestion of slightly decomposed food. Irritant and corrosive poisons are less common causes. Chill is a frequently associated factor.

Clinical Features. Pain is usually present in a more marked degree than in acute indigestion and may be intense and burning, accompanied by tenderness. Vomiting occurs, often consisting of undigested food and sometimes showing streaks of blood in severe cases. Malaise, anorexia, headache, and depression are usually present and prostration may be considerable. Fever is usual. Diarrhoea may occur just as in the previous condition and may be very severe and urgent.

Diagnosis. Acute indigestion is readily eliminated on account of the mildness of the symptoms etc. Other causes may have to remain under consideration for a day or so.

Acute infectious diseases may first manifest themselves by symptoms which suggest acute gastritis and may be indistinguishable at the outset. The absence of any dietetic error or other cause of gastritis may suggest the true nature of the condition but most frequently time is required to decide.

Intracranial disease (tumour, early meningitis or abscess) must be borne in mind. Careful consideration of the history prior to the onset of gastric symptoms and a thorough examination will usually make the diagnosis clear. In such conditions vomiting usually occurs without relation to food and nausea is often absent. Symptoms such as vertigo or optic neuritis may be found.

Gastric crises may be a difficulty. The diagnosis is considered under that disease.

A not altogether uncommon and very serious error may occur in cases of glaucoma. The practitioner may not associate the eye trouble with the vomiting etc. and cases occur where specialist treatment of the eyes is to be arranged when the gastric condition subsides! This catastrophe has only to be borne in mind to be avoided.

Migraine can usually be distinguished by considering the history. There is a story of similar attacks and, as Alvarez (101) remarks, if attacks never occur without preliminary headache and indigestion is never present without headache, the condition is migraine.

Hysterical vomiting may simulate gastritis but the urgent symptoms of organic disease are absent. In addition indigestible food may not be vomited while easily digestible food may be rejected.

Under this category is included the vomiting of pregnancy which is readily recognised as a rule if the possibility is considered.

Poisoning, which of course causes gastritis, must be distinguished separately for the purposes of treatment. It may be detected if several members of a family are affected or if the history suggests the possibility of poison having been taken accidently or on purpose.

Certain abdominal conditions such as peritonitis, appendicitis or intestinal obstruction may at first deceive but examination of the abdomen should make the condition apparent.

ACUTE GASTRIC ULCER.

The great majority of cases of acute gastric ulcer occur in women between 20 and 30 years of age and are very frequently associated with anaemia. It would appear that the condition is much less frequent nowadays than formerly; and this has certainly been our experience. Acute ulcers are very commonly multiple and as a rule do not extend deeper than the mucous membrane or submucous layer. Occasionally perforation occurs and haematemesis may result when a vessel is eroded. According to Hutchison (102) they occur almost exclusively in the condition he calls "the painful dyspepsia of young women". By this he means a condition commonest in

young women, especially those who are anaemic, and associated with small erosions or multiple acute ulcers. He considers there is usually an element of hyperaesthesia of the stomach which justifies the term "gastralgia" often applied to it. Pain is often severe and occurs usually in the epigastrium or in the back and comes on shortly after food. It is often independent of the kind of food taken. Vomiting is frequent and relieves the pain. Haematemesis occurs in quite a number of cases but is seldom profuse. If an acute ulcer forms perforation may occur. It is doubtful however if the presence of an acute ulcer can be diagnosed in the absence of complications.

Examination reveals little of importance. Tenderness is more or less diffuse and not localized. Test meals would be unsuitable in this condition lest damage should result and could not give information of any value. X-rays are likewise of no service.

Diagnosis. The occurrence of the "painful dyspepsia of young women" is not likely to be mistaken for any other condition except, as Hutchison suggests, appendix dyspepsia and in it the bouts are usually shorter. In the former condition bouts occur but complete freedom is uncommon. Chronic ulcer may ensue and have to be considered in the diagnosis.

The method would be that described elsewhere. As
we have mentioned above the occurrence of an acute
ulcer can probably only be diagnosed when compli-
cations ensue.

CHAPTER 6.

THE CHRONIC FORMS OF DYSPEPSIA.

(1) FUNCTION AL.

      (a) Habit Dyspepsia.

      (b) Nervous Dyspepsia.

          (1) Fatigue.
          (2) Psychogenic.

(2) ORGANIC.

    (A) Gastric.

        (a) Duodenal Ulcer.
        (b) Gastric Ulcer.
        (c) Gastro-jejunal and Jejunal Ulcers.
        (d) Gastric Carcinoma.
        (e) Duodenal Ileus.
        (f) Gastritis.

    (B) Extra-gastric (or Reflex).

        (a) Appendix Dyspepsia.
        (b) Gall-bladder Dyspepsia.
        (c) Carcinoma of the Colon.
        (d) Epigastric Hernia.
        (e) Spastic Colon.
        (f) Dyspepsia Associated with Disease
            of the Urinary Tract.
        (g) Tabetic Dyspepsia.

HABIT DYSPEPSIA.

This is said by Ryle (103) to result from "faulty physical habits" and he gives as examples of these:-- Over-eating, under-eating, over-work, lack of occupation and exercise, insufficient mastication, constipation, or some combination of these factors. It is a fault of all classifications that groups can seldom be exclusive and over-lapping is bound to occur. This group is, to our mind, only artificially separable from Nervous Dyspepsia. Habits of diet etc. which are suitable to a man living under more or less ideal conditions may result in symptoms of dyspepsia in one who is worried, depressed or over-worked. Moreover, if the examples above quoted from Ryle be reviewed, it will be seen that they are mostly associated with fatigue. These considerations led us at first to exclude Habit Dyspepsia from our classification and place it under the Fatigue sub-group. It seems to us however that habits may some-times be prominent factors in the production of indigestion, when nervous causes are either absent or apparently subsidiary. We feel therefore that this group is justified and does not materially complicate the whole.

Clinical Features. The symptoms here depend to a considerable extent on the physical type of the patient and the nature of the unphysiological habits.

We have the two broad divisions of physical types,
hypersthenic and hyposthenic. As regards habits,
too long intervals between meals usually tend to
modify the symptoms in the direction of delayed
pain; while too frequent ingestion of food or too
bulky meals tend to produce the opposite effect.
It is worthy of note that, in our experience, those
habits which tend to produce delayed pain frequent-
ly occur in the hypersthenic type in whom delayed
pain is the more common result and vice-versa.
It is not worth while labouring the point but the
over-active, restless, energetic type (hypersthenic)
often neglects his meals for long periods and over-
taxes his resources. The less robust, hyposthenic
type, though this is a less common observation, may
take too little exercise for the quantity of food
consumed or, in women who are at home, eat at over-
frequent intervals.

In between these two types cases of a
mixed, indefinite variety occur.

Diagnosis. This is usually readily accomplished
by excluding organic disease by the methods indicat-
ed later, and a consideration of the patient's
habits. Our method, indicated above, of allowing
the patient to tell his own story of the events of
one typical day, we have found especially useful in
this connection. We have usually observed most

difficulty in deciding between Habit Dyspepsia and ulcer, more commonly duodenal than gastric.   In some cases we have found ulcer present at a later date (3 in our series).

NERVOUS DYSPEPSIA.

This is a term which, in the present state of confusion, connotes different things to almost everyone who uses it.   Some understand by it those cases which we shall include under Psychogenic Dyspepsia.   Others, and they are by far the greatest in number, consider the term to be synonymous with the expression "Functional Dyspepsia". This use of the term, while admittedly often due to loose thinking, is in reality not without justification.   As Hutchison (104) says:--   "I am firmly convinced that the main causes of Functional Dyspepsia may be spoken of as nervous.   By that I mean mental and emotional over-strain and fatigue". He goes on to state that in his opinion it would be no exaggeration to say that functional dyspepsia is not primarily a disease of the stomach at all, but one of the manifestations of functional disease of the nervous system.   We are in the main in agreement with this.

In our opinion there are broadly speaking two kinds of nervous dyspepsia, the Fatigue type and the Psychogenic type.   It is not always easy to

separate these for many cases of Fatigue dyspepsia have associated with them an element of mental anxiety which is of considerable importance in the production of symptoms.   Cases however do occur where dyspeptic symptoms are apparently entirely due to nervous exhaustion or fatigue.   Similarly, cases do occur where the psychic or mental element is apparently predominantly or even wholly responsible.

In regard to the mechanism of symptom production, this has been dealt with in the second chapter.   We may add here however that it seems likely that nervous excitability, through over-action of the vagus, tends to result in hyper-sthenic dyspepsia;   while depression and fatigue tend to result in the hyposthenic type and this is probably through the sympathetic.   These responses are modified by the diathesis of the individual and hypersthenic symptoms are commonly found in the Fatigue type of dyspepsia.   The functions of the stomach are sensory, secretory and motor. Variat-ions in the last two are, as we have seen, apparent-ly compatible with perfect health but when they be-come far removed from the normal or average dyspep-sia is much more likely to occur.   This, be it noted, does not mean that the departure from the normal is the cause of dyspepsia but simply, and this is a well-established fact, that dyspepsia is

of much more frequent occurrence in association with these abnormalities. The sensory function is different and as we have already stated stretching of the muscle fibres is the only adequate stimulus for pain production. This is indisputable in the normal individual; but it seems not unlikely that in some psychogenic cases the threshold of consciousness is sufficiently lowered to permit of stimuli, not normally productive of response, causing pain.

FATIGUE DYSPEPSIA.

It is difficult to estimate the frequency of this type. All functional gastric disorders are probably diagnosed more often than they exist. This mistake is, in our judgment, far more common than that of diagnosing organic disease when the condition is functional. The explanation of this state of affairs may lie in the fact that many organic cases cannot be detected as such except by exhaustive and thorough examination; and this is not sufficiently often done. Further the purely nervous dyspepsia, at least the fatigue type we are at present describing, may become organic without any very obvious change in the symptoms to mark the transition. This we have observed frequently (6 cases). Such a change is due most commonly to the presence of septic foci; and it is impossible

to say when an ulcer has developed in a case of
Fatigue Dyspepsia in which functional symptoms,
identical with those usually produced by ulcer, were
previously present, (105). Fatigue Dyspepsia may
occur in patients of great activity and consider-
able strength who overtax their resources. Many
however seem simply to be endowed with but shallow
reservoirs of nervous energy. In this latter class
we include the condition of Visceroptosis though we
shall describe it under a separate heading. Under
this class also we include dyspepsia accompanying
anaemia. We do so because it seems probable that
the gastric symptoms are due to muscle exhaustion
from deficient oxygenation. Similarly the dyspep-
sia of early phthisis seems appropriate here. Some
writers state that it is due to an associated mild
gastritis; but it seems to us that it is probably
due to deficient muscular tone. Those forms of
indigestion met with in debilitative conditions
and after acute illness are also examples of the
fatigue type of dyspepsia.

Clinical Features. There is as a rule nothing
characteristic about the symptoms. They are simply
the common symptoms of dyspepsia present in vary-
ing number and degree in each case. They may in
cases exhibit themselves in such a way as to simu-
late closely duodenal or gastric ulcer. This is

most commonly due to the diathesis of the individual but is to some extent, as mentioned above, the result of the particular causative factors.

The gastric ulcer type is usually found in women about or past middle age who are of poor physique and whose abdominal walls are deficient in tone. Any cause of fatigue which results in their being more than usually tired may provoke dyspeptic symptoms. Frequently the stomach muscle is poor in tone and, lacking support as this organ does from the abdominal muscles, ptosis is common. Since the duodenum is usually also ptosed, kinking and delayed evacuation do not often result. The retroperitoneal portion of the duodenum may remain fixed and as the pylorus drops with the stomach, the duodenal bulb is elongated so that partial obstruction may be present. In such cases, which are apparently rare, ulcer of the pylorus with stenosis may be simulated; though vomiting is never abundant and visible peristalsis is seldom observed, due to the deficient muscular tone of the internal organs, unless the abdominal wall is very thin.

The duodenal ulcer type may occur and be indistinguishable from ulcer. As we have already said, ulcer may actually develop. The occurrence of this group of symptoms is readily comprehensible when we remember that pain is not due in ulcer cases

to direct irritation of the ulcer.   This type is
especially common in excessive smokers, more
particularly those who smoke cigarettes.

PSYCHOGENIC DYSPEPSIA.

Under this term is included a somewhat
mixed group.   In a recent discussion on Nervous
Dyspepsia Hutchison (106) suggested two divisions:-
Psychoneurotic and Psychogenic.   The first he re-
served for those cases of dyspepsia due to some
emotional disturbance or anxiety state in which there
was a functional disorder present.   The second in-
cluded those cases in which no disorder of gastric
function could be demonstrated but in which the
patient complained of discomfort of some kind re-
ferred to the stomach.   We see no practical advant-
age from this further subdivision since there appears
to be no simple and certain way of determining if
functional disorder is present.

It is true however that some cases in this
class are due to a neurosis, though that is not
always easy to establish.   In these the symptoms
are the physical conversion of some psychical
pattern and manifest themselves in the stomach,
though they might equally well be projected on some
other organ.   In addition to gastric symptoms,
general symptoms are usually present --- sleepless-
ness, headache, giddiness and incapacity for physical
or mental exertion.

Those cases where no neurosis is present or discoverable are doubtless due, as Hutchison suggests, to disorders of gastric function. Secretory and motor function may show departures from the average but may quite reasonably be normal to the individual. At any rate, it would be difficult or in some cases impossible to prove that these functions were disordered. Similarly sensory function cannot easily be measured. It does seem probable however that as we have suggested these cases exhibit in their gastric sensory functions a lowered level of consciousness to stimuli which is a common psychological experience in other regions of the body.

Certain odd or more or less isolated symptoms are included in this class and are of psychogenic origin. Pain may be complained of in varying degrees up to "agony". This may occur in individuals with no previous gastric trouble or in those seemingly cured of ulcer by operation or medical treatment. Similarly, vomiting may be present for no apparent cause. It may persist after cure of some disease in which it was a natural accompaniment; or it may be present with some causative disease but in a much exaggerated form, e.g. gastric ulcer. Aerophagy is the commonest example of this type of symptom and is nearly always of psychogenic origin.

Diagnosis of Nervous Dyspepsia. The very natural habit of suspecting a nervous origin for gastric symptoms in patients of a nervous disposition in whom the symptoms are vague and indefinite is probably the commonest source of error in diagnosis. As far as we are aware the only satisfactory procedure in these cases is one of exclusion. Ulcer, cancer, disease of the gall-bladder or appendix etc. must first be excluded by the methods we have indicated under these diseases. In addition, chronic gastritis has to be considered and this in the mild form may easily be a source of error. Much support may be obtained for the diagnosis from the history if carefully taken and the physical examination; but as a rule it is necessary to resort to the use of special methods. We have found little help from test-meals but the occult blood test is very valuable and has saved us more than one bad error. When any doubt remains, and this is not an infrequent occurrence, X-rays are well justified and often highly desirable.

Dyspepsia associated with anaemia or early phthisis, which we consider as a fatigue type, would be diagnosed in the course of the routine examination as detailed in an earlier chapter.

VISCEROPTOSIS.

We have decided to include this condition

among the functional diseases. Moreover we place
it in the nervous group because nervous symptoms,
in our experience, predominate and for other reasons
given below.

Visceroptosis may exist without any
gastro-intestinal symptoms whatever (Hutchison 107).
In addition, it has been shown radiologically that
where relief of symptoms has followed the use of
belts or after operation, the viscera may occupy
the same position more or less which they did when
symptoms were present. It might very naturally be
supposed that the position of the stomach and bowel
in this condition would cause delay in the progress
of its contents but this is not necessarily so.
As Alvarez (108) says, "The bowel is a muscular
tube and I doubt very much if its position in the
abdomen has much influence on the rate at which its
contents are passed onward". Delay may however
occur in extreme degrees of gastroptosis as regards
the emptying of the stomach according to X-ray
investigations (Ryle 109).

Visceroptosis occurs in two groups of
persons. In the first it is an inborn condition,
though dropping often does not become pronounced
until adolescence. In the second it is acquired
either from stretching of the abdominal wall
(e.g. after childbirth) or from loss of abdominal
fat.

<u>Clinical Features.</u>   The symptoms fall under three
heads.   They may be dyspeptic, including anorexia,
constipation, abdominal discomfort and other assoc-
iated symptoms.   Signs of a neurasthenic condition
are usually present and consist of lassitude, pains
in the back etc.   Under the third class are cir-
culatory and Vasomotor disturbances, for example
faintness, flushing and palpitation.

It seems to us that to suppose these are
mainly mechanical effects is unreasonable.   When
the absence of symptoms in persons with visceroptosis
is considered, along with the relief following
unsuccessful (though not so in the knowledge of the
patient) attempts to correct the dropped position of
the viscera, it is more logical to regard the con-
dition as a functional one mainly of nervous origin.
Moreover, treatment of the nervous element in our
experience usually produces an improvement unknown
by other means.   Why these patients should be so
liable to neurasthenia is difficult to explain.   An
important factor is of course the knowledge, some-
times indiscreetly conveyed to them by their doctor,
of the dropped condition of the abdominal organs.
That this is not the whole explanation is obvious
from the fact that all those who suffer from symp-
toms are not aware that they have visceroptosis.
The tendency to neurasthenia is probably in many
cases inborn.

Diagnosis. This is usually simple. The condition has only to be remembered and examination will reveal it if present. Apart from the ptosis of the stomach, indeed of all the abdominal viscera, the position of the right kidney is worthy of comment. It lies low and is frequently movable, (212 cases showed movable kidney out of 240 with visceroptosis. Einhorn, 110). This, which must necessarily be found if the examination is properly carried out, is a simple method which should suggest visceroptosis to the examiner. When it is discovered the condition of the other viscera is usually readily apparent and when taken in conjunction with the symptoms described the diagnosis becomes clear. We consider this of more than academic interest because of the simplicity of palpating the right kidney as compared with an attempt clinically to delineate the borders of the stomach. An additional means of diagnosis is open to the physician. If, standing behind the patient and encircling the lower abdomen with both hands and thus raising and supporting it, he finds that the patient experiences relief, visceroptosis is indicated. This is an old and popular test and may be very useful. We ourselves have been singularly unsuccessful in producing relief by this method.

THE ULCER DIATHESIS.

The conception of an ulcer diathesis is so
much under discussion that we must consider how far
it may be regarded as proved and in what way it may
be of help to the clinician.   It may conveniently
be reviewed here before considering Duodenal and
Gastric Ulcer.   It has been asserted that a dia-
thesis exists which renders a person peculiarly
liable to develop an ulcer;   and further that
special diatheses determine whether the ulcer shall
be gastric or duodenal.

Balint (111) suggested that in the ulcer
diathesis the acidity of the blood and tissues was
high.   He found the H-ion concentration of the
blood and tissues increased in a series of cases with
gastric and duodenal ulcer.   In addition 22 cases
still showed this in which a partial gastrectomy had
been done a year or more before.   He considers
that acute ulcers in persons of this diathesis tend
to become chronic.   These results have not however
been confirmed.   Osman (112) found the alkali-
reserve in a series of ulcer cases always within
normal limits.   Gavrila (113) later found the re-
action of the blood and the urine and the alkali-
reserve of the blood showed the same variations in a
series of gastric and duodenal ulcers as in a series
of normal persons.   It must therefore be concluded

that so far this theory is not proved.

Muller (114) asserted that gastric and
duodenal ulcer patients are generally the subjects
of what he calls the "vasoneurotic diathesis". The
arterioles, the capillaries and venules in the skin
and especially the mucous membranes of the lips
show very great irregularity, as seen by the
"capillary microscope", in their course, calibre and
anastomoses. He suggested that this was probably
the case also in the gastric mucous membrane and,
with a tendency to stasis, it would be an important
factor in the production of ulcer. Later, working
with Heimberger (115) he examined fresh specimens
after partial gastrectomy (for gastric and duodenal
ulcer) and found the same condition in the gastric
mucous membrane in 100 per cent of cases. Five
out of eighteen cases of carcinoma showed it and in
four there was evidence, clinical and pathological,
that malignancy had supervened on a simple ulcer.
This contention has not yet been refuted but it can-
not be said to be of any value to the General
Practitioner as the means necessary to detect it are
outwith his scope.

Of much more practical interest and in our
opinion value, is the conception originally due to
Hurst (116) of special diatheses which render the
possessor of them peculiarly liable to gastric or

duodenal ulcer.   Campbell and Conybeare (117)
found, by the use of the X-rays and the fractional
test-meal, that hypertonus and hyperacidity were
related on the one hand;   and hypotonus and hypo-
acidity on the other.   Further, they were able to
correlate these phenomena with gastric motility and
the individual's general physique.   Thus it was
found that it was in men of the broad-chested,
vigorous, athletic type that hypertonus, hyperacidity
and a rapidly-emptying stomach were most frequently
found;   and hypotonus, low acidity and slow-empty-
ing were most frequent in narrow-chested men of less
than average physique and not taking regular exer-
cise.   The work of Moody, Van Nuys and Chamber-
lain (118) showed that hypertonus was much more
common in men and hypotonus in women --- a fact
which could be suspected clinically.   Hurst (119)
calls the one the hypersthenic t ype and the other
the hyposthenic type.   These conditions, he says,
are compatible with perfect digestion.   The hyper-
sthenic type of diathesis, he thinks, is the essent-
ial predisposing factor in the production of duo-
denal ulcer.   Although doubtless due to unusual
activity of the vagus, it is not accompanied by a
slow pulse or other signs of general vagotonia.
In support of this he brings evidence difficult to
dispute.   He found hyperchlorhydria in 61 per cent

of cases of duodenal ulcer and a high normal in
29 per cent.   In the other 10 per cent he gener-
ally found hyperchlorhydria after healing, the low
acidity at the first test-meal being due to a mild
associated gastritis.   Dealing with rapidity of
emptying, he says this is almost always found, even
though there may have been , before healing, con-
siderable delay due to interference with the
pyloric sphincter.   It is probable, in his opinion,
that the characteristics of the hypersthenic type
are present in 100 per cent of cases of duodenal
ulcer, though one or more may be temporarily
absent for the above reasons.

It is clear that when food or drink are
present in the stomach the acidity is reduced
partly by dilution and partly by neutralisation by
alkaline salts and proteins.   Now, as we have
indicated elsewhere, the normal stomach is seldom
empty except for a few hours before breakfast.
Consequently pure gastric juice seldom enters the
duodenum.   Moreover, peristalsis is only slight
when the stomach is empty unless hunger is present,
which is under ordinary circumstances not so.   In
the hypersthenic individual we have quite a differ-
ent state of affairs, for he has usually several
waking hours in which the stomach is empty.   In
addition he probably has a more continuous secret-

ion of gastric juice and one which is more acid.
It seems likely therefore that pure gastric juice
leaves the stomach of this type of individual dur-
ing several hours.   Also the duodenal bulb is much
less frequently filled in the recumbent position
than in the erect.   Hence in normal people the pure
juice, whatever small quantity does leave the stom-
ach, is less likely to remain in the duodenal bulb.
In the hypersthenic type pure juice leaves the stom-
ach in the waking hours and will therefore be more
likely to remain for some time in the duodenal
bulb --- the site where duodenal ulcer is almost
always found.

It must not be thought that this dia-
thesis necessarily results in duodenal ulcer.   It
is compatible with perfect health.   It does however
render the individual more liable in certain cir-
cumstances to ulcer.   Hurst believes that duodenal
ulcer never develops in the absence of this dia-
thesis.

Hurst admits that the gastric ulcer dia-
thesis is much less easy to define.   In a consider-
able majority of gastric ulcers the stomach, he
says, is longer than the average though normal in
tone;   and gastric ulcer never develops in a short
stomach such as is found in the hypersthenic type.
We can only observe that this statement is difficult

to reconcile with the fact that gastric and duodenal ulcer may occur simultaneously in the same person (Wilkie 120).   Hurst only found 9.5 per cent of his gastric ulcer cases with hypochlorhydria, the rest having hyperchlorhydria or a high normal acidity. Beyond the tendency for gastric ulcer to be found in persons with long stomachs it does not seem to us that Hurst leads much evidence to define this diathesis.   Writing in the Practitioner (121) however, he discusses the question in what seems to us a clearer fashion.   Dealing with the hyposthenic type he says they "have a long, low (but not 'dropped'), slowly-emptying stomach;   some have normal gastric secretion, some have hyperchlorhydria, and some --- the most extreme form of the diathesis --- have hypochlorhydria".   Hyposthenic cases, he goes on, if hyperchlorhydria be present will, under appropriate abnormal conditions, have symptoms suggestive of gastric ulcer and an erosion or acute ulcer may develop into a chronic gastric ulcer.   In those cases with hypochlorhydria abnormal conditions producing symptoms of dyspepsia lead to different results.   A mild gastritis develops and the secreting tubules become blocked with mucus so that very little gastric juice enters the stomach and any free acid is likely to be neutralised by the alkaline mucus.   This type, he says, never goes on to ulcer

as for this free hydrochloric acid is necessary.

Reviewing the evidence we consider the case for a gastric ulcer diathesis is not very definite. It is helpful however to bear in mind the facts brought forward and when other evidence points to organic disease in this physical type (hyposthenic) it is an indication at least that the ulcer is more likely to be in the stomach than in the duodenum. In fact if we accept Hurst's duodenal ulcer diathesis and an ulcer is found to be present in a hyposthenic case then _ipso facto_ it cannot be in the duodenum.   In a hypersthenic case, however, it would seem to be possible to have ulcers in both stomach and duodenum as we have observed.

DUODENAL ULCER.

By far the greater number of duodenal ulcers occur in the male sex; probably at least three or four to one being the proportion.   The average age of onset is over thirty years and the condition is comparatively rare before twenty. Cases have been recorded in two instances under ten years.   The youngest case we have encountered (No. 86) was eighteen years of age when we first attended him on account of perforation.   He gave a history which proved almost conclusively that the ulcer had been present for three years before it perforated.   This we concluded because he informed us that at that time another medical attendant had

told him he would probably eventually require operative treatment. Moreover, his history which was of even longer duration brought forth the fact that about the time operation was first mentioned to him, the pain had become definitely more severe than previously; and from that time onwards attacks were more frequent. The surgeon who operated at the time of perforation reported to us that the appearance of the ulcer was "of surprising chronicity".

Clinical Features. The symptoms commonly commence insidiously and gradually become characteristic. At the outset the attacks may be of short duration and only recur at long intervals. Later they tend to last longer and are more severe, the intervals of freedom becoming shortened. Finally, there may seldom be a time when the patient does not experience symptoms, though the intermittent character may long be maintained. Attacks appear often to be precipitated by chill or bad weather and are commonest in spring and autumn. Worry and overwork are also commonly the cause of attacks; and a period of absence from work is often beneficial, especially in the early stages. We encountered a case (No. 81) where symptoms of typical hunger pain had been present for seven years prior to the Great War. The patient was a colliery clerk whose duties

involved somewhat irregular hours and a sedentary
life in far from ideal conditions.   During the
whole period of the War while on service he never
experienced symptoms.   With the resumption of civil
duties symptoms of indigestion returned and became
steadily more severe.   In 1932, a duodenal ulcer
was demonstrated radiographically.   There was
every reason to believe from the history that ulcer
had been present for at least ten years.   Probably
the condition had been functional prior to the War
and the healthy, active life in the army had
temporarily removed most of the causative factors
of his dyspepsia.   We have observed other similar
cases.

Over-indulgence in alcohol and an unsuit-
able diet may precipitate attacks.   Tobacco,
especially in the form of cigarettes, has a marked
effect in initiating attacks.

The pain is most commonly situated in the
mid-line, half-way between the Xiphisternum and the
Umbilicus, being somewhat lower than that of gastric
ulcer in the majority of cases.   It may be situated
to the right and much less commonly to the left.
The pain may radiate to the right or, if situated
there to begin with, to the centre;   in some cases
it goes through to the back, and in these adhesion
to, or invasion of, the pancreas is usually present.

Localisation is much more defined as a rule than in
gastric ulcer.

The character of the pain varies.   It may not be
severe at first, perhaps little more than discom-
fort.   Commonly it seems to give rise to a sen-
sation of hunger or emptiness.   One of our patients
aptly described this sensation when he said that,
in the early stages, he used to come home "pain-
fully hungry".   When the pain is fully developed
it is frequently burning or gnawing in character.

The time of onset is one of the most
constant features, the pain occurring at exactly
the same time each day after meals.   The interval
of freedom after a meal varies according to the
amount of food taken, being delayed by a large
quantity of food and being then more severe.
Usually it occurs two to three hours after food.
It gradually increases in intensity, though it is
very rarely severe, for about half an hour;   it
then slowly diminishes and disappears in an hour or
two.   Ordinarily, however, another meal is taken
before the pain is gone.   This brings relief,
later followed by pain and so on the cycle goes.
A common error is to suppose that the pain con-
tinues until food is taken.

The pain in duodenal ulcer is relieved
by food, solid or liquid, and the duration of free-

dom from pain depends on the amount taken. It is not necessary for the food to be easily digestible; and spirits, though liable to make the condition worse later, bring temporary relief. Alkalies are helpful, sodium bicarbonate being the commonest used and the most effective. The belching of wind which follows leads the patient to suppose that he is better when he "gets rid of the wind".

As we have already said, spontaneous vomiting is rare though the patient may so cultivate the habit, knowing that he will be relieved, that he acquires the faculty of vomiting at will. In cases where there is pyloric obstruction, functional or organic, vomiting is common. Indeed it is often the leading symptom in organic cases. Nausea is usually absent in duodenal ulcer. When vomiting occurs in uncomplicated cases it usually consists of clear highly acid fluid.

The patient usually has a good appetite and often eats to relieve his pain. In the case referred to above we found the patient carrying indigestible fruit to his work that he might not develop the "painfully hungry" sensation. As a consequence loss of weight is not very common, though some patients restrict their diet in the knowledge that they have a "stomach complaint", and they give a history of becoming thinner. In general the

patient's physical condition is fairly good and anaemia does not usually develop in the absence of haematemesis or gross melaena, which are not very common. Heartburn is a frequent symptom and con-stipation is very often present.

That gall-bladder disease may simulate duodenal ulcer with typical hunger pain is well-known; but only recently Wilkie (122) found absence of hunger pain in duodenal ulcer in 35 per cent of a series of cases in women. This type, he says, is much more common in women than men; and as flatul-ence is usually the presenting symptom gall-bladder dyspepsia is suspected. Wilkie's explanation of the absence of hunger pain is the habit indulged in by some women of taking food between meals when at home during the day.

Tenderness is nearly always to be found, though its position varies. It is commonly a little above the umbilicus; but may be much higher and even under the sternum. If the patient is examined frequently tenderness can usually be found on some occasion, though there certainly are cases in which it does not appear to be present. Rigidity of the rectus muscle may be noted, more usually on the right side. This is a valuable indication but does not occur in many cases.

Hyperacidity is characteristically present

though in rare cases the acidity may be low or even absent. These cases are usually due to a concomitant gastritis. Occult blood is very constantly present.

Diagnosis. In characteristic cases the diagnosis is straight forward. If occult blood be found in the presence of typical symptoms the diagnosis may be regarded as practically certain. Juxta-pyloric ulcer might be the cause of the symptoms but we have decided to consider it along with duodenal ulcer as in General Practice the two cannot be differentiated. Provided the ordinary fallacies are excluded in testing for occult blood, the only condition likely to prove a source of error is cholecystitis. The error, and it would be a rare one, might be due to some bleeding from abrasion of the bile passages by stone. If occult blood is not found after repeated tests when ulcer is suspected, then the physician should review the evidence afresh and consider other possibilities. Hyperacidity is very constant and is of more value to the practitioner in this condition than in gastric ulcer; though we do not regard it as of nearly so much help as the examination of the faeces for blood.

The functional type of dyspepsia in which hunger pain is present must next be taken into consideration. If occult blood is absent after repeat-

ed examinations in a suspected case of ulcer, this diagnosis is a distinct possibility. When the complaint is of great chronicity with typical attacks and periods of freedom then ulcer is probably present. The severity of the pain and its occurrence at night should be noted. Severe pain and nocturnal pain point to ulcer; though it must be remembered that duodenal ulcer may not cause severe pain at any time and functional dyspepsia may cause pain at night. More important, in our experience, than the presence of nocturnal pain is its absence: in ulcer, it is seldom for a patient not to experience nocturnal pain on some occasion. Vomiting is important and haematemesis would be conclusive in the decision here. If any doubt remain, though it should be insisted that this is rare and unknown to us, then an X-ray would be essential.

The dyspepsia caused by appendix disease is very liable to cause error in some cases. We shall indicate the main features of that condition in the section allotted to it. Here we shall be content to speak of those cases where the symptoms may well be regarded as identical with those of duodenal ulcer. Vomiting and haematemesis are of no help; the former is even more common in appendix disease than in duodenal ulcer and the latter may actually occur, presumably through infection by the

blood stream.   Here again the great value of the
occult blood test is apparent as it may decide the
diagnosis.   On the other hand it is here also that
one of its limitations is evident, for chronic
appendicitis may be present as well as duodenal
ulcer!   Therefore, although it would prove that
ulceration was present, it would not exclude append-
ix disease.   Similarly, appendix disease may be
shown to be present by tenderness in the right iliac
fossa (the most important clinical sign and one
which is present in nearly all cases); but that
does not exclude ulceration.   It is therefore mani-
fest that X-rays are desirable in these cases for
the possibilities as shown in the table below are
too many.

Occult Blood. (repeated tests).	+	—	+	—
Tenderness in right iliac fossa.	—	+	+	—
Probable Diagnosis.	Ulcer.	Appendix Dyspepsia.	(1) Appendix Dyspepsia. (2) Ulcer.	Doubtful.

The test-meal is useless as both cases
commonly show hyperchlorhydria.   Bastedo's test we
do not consider suitable for General Practice and in
hospital X-rays are much more certain.

Almost all that has been said of appendix disease may be repeated in the case of gall-bladder dyspepsia. Many cases can be differentiated by the symptoms and these we shall describe under the heading of Gall-Bladder Dyspepsia; but some cases occur in which differentiation on these grounds is impossible.

As before, abdominal examination may be of great service. Tenderness over the gall-bladder is usually very constantly present, even weeks after an attack; and the same may be said of rigidity. Again test-meals are of no help for just in those gall-bladder cases which closely simulate duodenal ulcer, the commonest finding is hyperchlorhydria. Occult blood is distinctly in favour of ulcer but again is not conclusive as it may occur with gall-stones causing bleeding (Hurst 123). This is however extremely rare. X-rays are the only sure way to diagnose between these two conditions.

Gastric crises are comparatively rare but must be borne in mind. It seems to us more reasonable to consider them elsewhere. The diagnosis from gastric ulcer and gastric cancer have been done under their respective headings.

GASTRIC ULCER.

This disease generally occurs between the ages of twenty and fifty. Men and women are

apparently equally affected.   Hurst's and Moynihan's statistics (124) correspond remarkably in this regard and no authorities seem to differ very much from their view;   some consider women preponderate slightly and some men.

Clinical Features.   The most important clinical feature is pain.   It is generally situated high in the epigastrium in the mid-line.   Sometimes it is a little to the left or under the left costal margin just below the xiphisternum or even deep beneath the lower end of the sternum.   The pain may radiate either through to the back or round to the left.   It occurs generally from half an hour to one and a half hours after food, though it may be both earlier or later.   When the ulcer is near the cardia, the pain comes on immediately or very soon after food and vomiting commonly occurs.   In ulcers nearer the pylorus the onset of pain is later and vomiting is a less common result.   In character the pain may be dull and heavy or it may be burning.   The more indigestible the food the more severe the pain.   As a rule liquids cause less pain than solids.   After reaching a maximum the pain dies away, usually in about an hour though this varies in individual cases. A sense of uneasiness frequently remains.   It is usually gone before the next meal.   Vomiting brings complete relief and alkalies usually improve the

condition considerably. It may happen that the patient describes himself as having more or less constant pain; but in these cases he will often disclose the fact that it becomes worse at a certain time after meals. There may be periods of freedom of several weeks or, even more commonly, months and occasionally years. When an ulcer has been present for a year or so spontaneous remissions are rare.

Vomiting has already been mentioned. It is a common but not invariable accompaniment. It usually occurs at the height of the pain and never does so in the absence of pain, with which it is definitely associated. Haematemesis occurs less frequently --- about one-fifth of cases --- and varies in severity. The stools are usually "tarry" at some time or another. The appetite is often good but the patient may be afraid to eat. Flatulence is common, especially in nervous patients and relief usually results from belching. There may be a fair loss of weight as a result of vomiting and a restricted diet.

Tenderness on pressure will be found in the epigastrium almost invariably during an attack. It is usually to the left or the right of the midline above the umbilicus and may show no relation to the position of the pain. Very frequently it is limited to a small area the size of a shilling or a

half-crown. A vertebral tender-spot may be found. Cutaneous hyperaesthesia may be present at the tender area or a slight distance from it.

During an attack muscular rigidity is commonly present in the recti or in their upper portion. It may be,however, that only one rectus is rigid.

Melaena is usually present after haematemesis but may occur independently. If not, occult blood can almost always be found and blood may be detected in the gastric contents.

Friedman (125) found the haemoglobin percentage averaged 75 per cent in cases of gastric ulcer compared with 90 per cent in duodenal ulcer. The individual variations were 55 to 110 and 65 to 120 in gastric and duodenal ulcer respectively. The explanation of this is probably in part due to diathesis and probably in part due to the relative preponderance of male cases in duodenal ulcer.

Diagnosis. Certain cases present themselves to the physician which are so typical that the diagnosis is almost at once apparent. In such cases, where the history and examination are characteristic, we consider that in General Practice no special methods are necessary except the occult blood test. This should never be omitted as it is so simple and supplies such excellent confirmatory evidence.

X-rays may be employed if hospital facilities are
available or the patient can well afford the expense.
Otherwise they are not necessary as the diagnosis,
in the typical cases we are now considering, is much
more certain than many the physician is daily called
upon to make unaided in other diseases.

Apart from such cases, it not infrequently
happens that the diagnosis can be made clinically
with equal certainty.   When haematemesis occurs and
cancer of the stomach and cirrhosis of the liver are
excluded the cause is practically certain to be
peptic ulcer.   The history and examination should
supply the remaining evidence necessary.   Haemat-
emesis is not common in duodenal ulcer.   Hurst (126)
states that 75 per cent of patients with duodenal
ulcer have symptoms of active ulceration for many
years without having a haemorrhage;   and it is
usually possible to decide whether the ulcer is duo-
denal or gastric from the history excepting, of
course, juxta-pyloric ulcers.   The consequence
of a mistake between duodenal and gastric ulcer is
of little or no account as the treatment is the same.
It has been argued by surgeons that the indications
for operation are more definite in duodenal than in
gastric ulcer.   The success of modern medical
treatment, it seems to us, makes this claim invalid.

When vomiting occurs frequently in

association with suggestive symptoms, the probability of gastric ulcer is considerable; especially where this takes place shortly after meals and is followed by relief.   If in such a case occult blood is present in the stools, the diagnosis is practically conclusive.   Vomiting is rare in duodenal ulcer when complications are absent.   When present it is usually not spontaneous, though it need not necessarily be induced artificially, but may be voluntary -- the patient having acquired this power after having frequently in the past induced emesis to obtain relief.

In spite of the above considerations, the problem has most frequently to be decided from a review of the characteristics of the pain. Functional conditions may closely simulate ulcer.   Here the occult blood test is practically diagnostic when present.   If absent, after repeated tests, it is improbable that the case is one of ulcer.   The diagnosis may still remain undecided though this should be comparatively rare.   The severity of the pain is however usually greater in the organic conditions than in functional ones.   If rigidity is present it is definite evidence of an inflammatory or ulcerative condition, as we pointed out at an earlier stage.   If to this the physician can add an area of hyperaesthesia or a vertebral tender-spot,

These considerations may make it appear
that the diagnosis is never a matter of any great
difficulty.   We must confess that this has not been
our experience.   We have found most perplexing
those cases where the patient, usually a woman, is
obviously debilitated, nervous and of poor physical
development.   Quite evidently the patient's
description of the severity of the pain is unreliable.
This makes considerable inroads on the most important
part of the history which, as we have pointed out,
is usually by far the most useful factor in diagnos-
is.   Moreover the detection of "tenderness" in
these patients is all too readily elicited.   In our
opinion, it is with such patients that mistakes in
General Practice are most frequently made.   It is
exceedingly tempting to label the case "Nervous
Dyspepsia" and relegate it to the limbo of functional
cases, and to regard the symptoms as wilfully
exaggerated or not even present at all.   Nothing
could be more unjust to the patient, even if the case
be not an organic one;   and nothing is more likely
to prove a source of error for the practitioner.   In
these cases two courses remain.   The first is the
effect of treatment.   If ulcer régime be instituted
and fail to result in improvement after a reasonable
trial, it is almost certain that the patient has not
an ulcer.   The second is to see the patient in an

attack when, if the case be an organic one, it is exceedingly probable that unequivocal tenderness and rigidity will be detected.

In our argument we have of course assumed that a clinical diagnosis has to be made with the simple special methods available for the practitioner. We consider that with these patients, if any possible doubt remains, radiographic evidence is eminently desirable. This should be a comparatively rare occurrence if a good history has been obtained, a complete examination made and the appropriate special methods applied.

We have just referred to chronic functional dyspepsia. Certain other conditions have frequently to be contemplated. Tabetic crises should be borne in mind. These are considered in detail elsewhere, as is also cancer of the stomach. Disease of the gall-bladder may cause confusion, though this is more likely to be a source of difficulty when it simulates duodenal ulcer with hunger pain. Where the symptoms are like those of gastric ulcer, the detection of tenderness in the gall-bladder region is helpful. It may be noted that the position of the gall-bladder at the tip of the ninth costal cartilage has been shown to be less constant than was hitherto supposed. The tendency of dyspepsia due to cholecystitis is for

the symptoms to be more erratic than those of ulcer.
A history of jaundice is suggestive; and if the
pain at any time has suddenly disappeared it
indicates with practical certainty that the trouble
lies in the gall-bladder.   Extreme care as regards
the characteristics of the pain should eliminate
most errors and finally occult blood if present is
distinctly in favour of ulcer, though this is not
always so as we indicate under the section on gall-
bladder dyspepsia.

Cases may present themselves in which the
use of X-rays is absolutely essential.   These
however should be far from frequent after full
investigation.

Appendix dyspepsia has already been con-
sidered when dealing with duodenal ulcer.   It is
also referred to under its own section.

In regard to the differentiation from
duodenal ulcer sufficient has been said in the
previous section to make that possible except in the
case of juxta-pyloric ulcers, which as we have
already said we consider along with those in the
duodenum proper.

GASTRO-JEJUNAL AND JEJUNAL ULCERS.

The first of these conditions is meant
when the ulcer is on the anastomosis line whether it
is mainly gastric or mainly jejunal.   The second

when some healthy tissue separates the ulcer from the anastomosis. The condition does not follow operations for cancer where the acidity is low; and it is rare after operations for gastric ulcer where the acidity is usually not so high as in duodenal ulcer, to which it is most frequently a sequel. It only occurs in probably about 2 per cent of cases.

The symptoms resemble the original ulcer but the onset is less regular and is usually earlier after food. Food and alkalies relieve it less completely than the original condition. The site of pain and tenderness is most commonly about the level of the umbilicus instead of higher up and is usually to the left, instead of the centre or right as in duodenal ulcer. Perforation or haemorrhage may occur.

Diagnosis. The condition should always be considered in a post-operative case with symptoms. It has to be distinguished from a persistence or recurrence of the ulcer. In the latter the symptoms are the same as those before operation in regard to position and time of occurrence. Pain and tenderness are usually to the right or in the centre in duodenal ulcer and not to the left as in this condition. In gastro-jejunal ulcer the pain may occur immediately after food which is never so in duodenal ulcer. The relief from food and

alkalies is not so complete as in the original condition. X-rays may be used to settle the question though this does not appear to us as immediately desirable, since before another operation is considered, medical treatment as for duodenal ulcer should be given a long trial. If at a later date the question of operation arises X-rays can then be used.

GASTRIC CARCINOMA.

This is a common disease and the stomach is one of the sites most commonly affected by malignant disease. It occurs most frequently between the ages of 45 and 65, though it may appear either earlier or later. Cases have been described under thirty years of age. Osler and McCrae (127) had 6 out of 154 cases between 20 and 30: the youngest being 22. Men are much more commonly affected than women though the proportion is controversial.

The onset of symptoms is usually rapid though insidious. By far the greater number of cases occur in people with no previous gastric disturbances, though cancer may appear in ulcer cases or in those with a history of functional dyspepsia. As a rule the early symptoms have nothing characteristic about them and it is this fact which, all too frequently, allows them to pass unattended even by

the patient himself.

The early symptoms are usually discomfort or pain, loss of appetite and failure of strength. Other symptoms such as vomiting may occur. The site of the growth is responsible for variations in the clinical picture. If it is at the pylorus there may be signs of a rapidly developing pyloric obstruction. Growths at the cardia may first manifest themselves by dysphagia; these are the least common. Those in the body of the stomach are usually near the pylorus and on the lesser curvature.

Loss of appetite is a well-recognised and important feature in early cases, though it is not infrequently absent. When it is of sudden onset its significance is increased. It is commonly stated that anorexia, when it specially affects the patient's appetite for meat, is very suggestive. Abrahams (128) says that in actual fact this is not a very prominent feature in the histories of early cases. Some victims of cancer of the stomach preserve an unimpaired appetite up to an advanced stage in the disease.

When indigestion is associated with a definite loss of weight it generally spells organic disease. (It is important of course to eliminate such a fallacy as over-strict dieting). If this symptom is present in a suspected case of

carcinoma it is a valuable sign. Unfortunately
it is commonly rather late in onset to be of help
as the diagnosis may then be all too obvious.   In
advanced cases it is a prominent feature on account
of the small amount of food digested, the vomiting
and general effects of the tumour.   Perhaps a common-
er early sign is impairment of vitality and energy in
association with dyspeptic symptoms.

Pain is commonly present in varying degree,
being no more than discomfort in some cases while it
may be sharp, lancinating and very severe in others.
It is usually epigastric in position but may be
referred to the shoulder or back.   It has not as a
rule the marked relation to food that the pain of
peptic ulcer exhibits; but food usually aggravates
the pain soon after it is ingested.   The tendency is
for the pain to become more continuous as the disease
advances until it is seldom absent.   It must be
borne in mind that pain may be conspicuously absent,
even till the end;   and its absence in early cases is
more common than is generally supposed.

Vomiting, which may be occasional to begin
with, usually becomes more frequent as the condition
progresses.   The vomit is foul-smelling and may con-
tain "coffee-ground" material or undigested food
which has been swallowed many hours before.   Relief
from vomiting is not so marked as in ulcer and may be

entirely absent in advanced cases.   Nausea is fre-
quent and marked.   Flatulence is very common but
heartburn is comparatively rare.

Haemorrhage is the rule and is usually
present in the vomit and, according to Hurst (129),
always in the faeces either as melaena or occult
blood.   Indeed he declares he has never seen a case
of a growth of the stomach or colon in which it was
not present.   If this were taken as constant the
logical implication would be that failure to find
occult blood would exclude malignant disease;   but
Abrahams (130) says he has had negative results in
several cases when the diagnosis was beyond doubt.
He admits however that such cases are very rare.
Profuse haematemesis is very infrequent but does occur.
In our series of cases, we have only met with it on
one occasion (No. 109).

Cachexia is often marked in the late stages.
When it is of rapid development it is almost diag-
nostic in association with suggestive symptoms.
Anaemia is another important sign.   A secondary
anaemia with no apparent cause especially in a man
should suggest, if not suspicion of carcinoma of the
stomach, at least the necessity of excluding it.
Addison's Anaemia which commonly presents gastric
symptoms may closely simulate carcinoma.   The blood
pressure in carcinoma of the stomach is often low,

especially in the later stages.   Jaundice and
ascites may occur though usually late and are due to
secondary deposits in the liver.

Constipation is usually present.   In this
connection it is interesting to quote some remarks of
Paterson (131):-- "If the oft-repeated statement
that '60 per cent of patients suffering from gastric
cancer present a history of long-standing gastric
ulcer' were amended so as to read '60 per cent of
patients suffering from gastric cancer present a
history of long-standing intestinal stasis' it
would approximate more nearly to the truth".

Diarrhoea is rare.   It may alternate with
constipation and suggest malignant disease in the
bowel.   One of our cases showed marked constipation.
When a purgative was taken. it resulted in marked
relief of symptoms but caused rather severe diarrhoea.
A palpable tumour was felt and it was not without
difficulty that we decided the growth was in the
stomach and not in the transverse colon. (No. 111).

Fever is sometimes present though it can-
not be regarded as a regular occurrence. Einhorn
(132) claims that it is more often present than is
generally believed; and he considers it is due
either to an inflammatory process in the neighbour-
hood of the growth or toxic absorption from ulcerated
areas of the tumour.   When present it is usually a
late and ominous sign, frequently heralding the end.

We mention it though it does not seem to us of any diagnostic importance.

Oedema is occasionally present at the ankles and other signs of anaemia may be present. The urine may contain albumen and, in certain cases, acetone due to starvation.

The test-meal findings are dealt with below.

As we have said above, the clinical picture is affected by the site of the growth and its type. At the cardia dysphagia may be the initial symptom. Epigastric pain and vomiting usually result immediately food is swallowed. Growths at the pylorus give the symptoms of pyloric obstruction. Cirrhosis of the stomach or "leather-bottle stomach" causes vague dyspepsia of slow progress. Vomiting occurs, at first occasionally, but later the stomach retains only small amounts of food, having no muscular adaptability. Pain becomes continuous and wasting sets in. The stomach can be felt as a smooth sausage-shaped mass which can usually be moved. Those cases where there has been a previous history of dyspepsia, functional or due to ulcer, are best considered under the diagnosis.

Diagnosis. It is hardly necessary to point out the importance of early diagnosis in this condition. Even if operation is all too frequently out of the

question it is still the only hope of cure at present; and improved results are likely to be obtained only when the physician is able to seek surgical aid at an earlier stage. As the majority of cases must of necessity come first of all under the care of General Practitioners, the outlook of the future is largely in their hands. We hope to show below how the early diagnosis is very much dependent on the use of special methods. It is unfortunately true that gastric analysis and occult blood tests are the exception rather than the rule in General Practice. If aid is sought by the practitioner it is usually to the radiologist that he goes. While carcinoma can be diagnosed with considerable certainty by X-rays great evil sometimes results from reliance on this means alone for two reasons. Firstly, the examination may be performed by a radiologist of no great experience or the report may be based merely on the films taken. Many cases, even quite advanced ones, may show little in the way of "filling-defect" but a careful screen examination will reveal the true nature of the condition. Secondly, considerable delay may occur before the practitioner considers it advisable to put his patient to the expense of an X-ray examination. We do not mean to detract from the value of radiological evidence. In fact we con-

sider it should be obtained in all cases where carcinoma is suspected and cannot be absolutely proved otherwise; but it should be done only after gastric analysis and occult blood tests. Moreover, we consider these should be done by the practitioner himself.

In general two types of cases are found which should suggest to the practitioner the possibility of gastric carcinoma and call for a thorough investigation. The first is where a patient over the age of forty presents himself with a complaint of indigestion and a previous history of no gastric trouble. Whenever such a case is encountered in which the symptoms last more than ten days then carcinoma should be _presumed_ until adequate proof to the contrary has been obtained. These cases constitute the great majority, probably at least three-quarters, of all cases. The second type is that in which there is a more or less long history of indigestion but where some change in the symptoms has occurred. We shall describe our procedure in these two types of cases.

In the first, the usual method of taking a full history and making a careful examination often yields a good deal of suggestive evidence. The nature of the pain or discomfort and its persistent character is worthy of note. Anorexia has always

been recognised as an important early sign. It is
however of little account if it be absent as it "is
sometimes delayed until a comparatively late period"
(Einhorn 133). The patient may however complain of
failure of strength and energy. Vomiting or haemat-
emesis if present are very significant, as is also
melaena. The family history may also be suggestive
if a case or cases of carcinoma have occurred.

Clinical examination may give almost con-
clusive proof. A tumour may be found but may of
course be due to a chronic gastric ulcer or other
cause. This must be considered in association with
the other evidence and would hardly be possible in
the type at present under consideration. Enlargement
or irregularity of the liver should be sought but may
be due to cirrhosis etc. Finally, rectal examinat-
ion may reveal a metastasis in the recto-vesical
pouch.

It must be admitted, however, that in early
cases the history and examination are frequently
barren of result. With the exception of advanced
cases, or those in which there is a rapidly produced
pyloric stenosis, it is seldom that a definite diag-
nosis can be reached clinically. It is therefore in-
cumbent on the General Practitioner to employ the
necessary ancillary methods and in these he posseses
very valuable aids.

The test-meal will very frequently supply

all the additional information necessary.   As

Bennett (134) observes:- "Gastric analysis is possi-

bly of less value than the X-rays in arriving at a

positive diagnosis, but it is a method much more

readily available to all physicians, and experience

leads me to believe that it is exceedingly rare for

a case of carcinoma of the stomach to present itself

for examination without the diagnosis being definite-

ly established as soon as a gastric analysis is per-

formed".   This dogmatic statement might well be

pondered by those practitioners who apparently do not

feel it their duty to employ the stomach tube in

diagnosis.   MacLean (135) endorses this opinion and

adds that any patient with a suspicion of gastric

cancer whose test-meal shows no hydrochloric acid but

a definite amount of lactic acid has got cancer.

Much has recently been written as to what reliance

can be placed on absent hydrochloric acid and the

presence of lactic acid.   Einhorn (136) says they

cannot be regarded as pathognomonic but are frequent-

ly of help in the diagnosis.   We have dealt with the

question of lactic acid separately on account of its

importance in the chapter on Method of Investigation.

It appears to us important however to consider care-

fully MacLean's assertion.   No one to-day, we imagine,

would assert that these findings are pathognomonic

of carcinoma.   But would anybody deny that, in the

type we are considering, (patients with a sudden on-

set of gastric symptoms in middle or later life with
no previous history of indigestion; and these, be
it remembered, form the great majority of carcinoma
cases) these findings are almost certain proof?
Those who under-estimate the value of gastric
analysis seem to suggest that it is used apart from
clinical evidence.   Whatever be the truth in this
connection, the practitioner has a further means of
assistance in the test for occult blood.   It should
be added that sour-smelling fluid withdrawn from the
stomach, probably containing blood or even portions
of the tumour or cancer cells, will naturally weigh
in the diagnosis; and may in the case of tumour
cells be diagnostic.   It is important to add,
before leaving this subject, that clinically sus-
picious cases which do not show absent hydrochloric
acid and the presence of lactic acid, may still be
cases of carcinoma.

   We have already remarked on Hurst's
assertion that occult blood is present in every case
of carcinoma.   Even if this proposition be not
granted, it seems likely that if it is present in a
suspected case it is well-nigh conclusive.   If to
this can be added the evidence of gastric analysis
then no more certain diagnosis, short of surgical
exploration, could be found.

   Finally, X-rays should be employed if any
doubt remains.   Where the test-meal and occult

blood findings are in accordance with the clinical
suspicions, X-rays are hardly necessary, but may
be used if desired. So important is it however to
make the diagnosis early that if the practitioner's
tests have contradicted the clinical suspicions
then the case should not be dismissed on any account
until negative radiological evidence is also obtain-
ed.

We know of one case, not under our care,
where the employment of X-rays alone resulted in a
bad error. The patient, a man past middle life,
began to suffer from indigestion. He had always had
a good digestion and the suspicion at once arose
that he had cancer. <u>With this clearly in mind</u>,
X-ray photographs were obtained and showed no
evidence of a neoplasm. The diagnosis made was
"ptosed stomach" and treatment by diet resulted in
slow but definite improvement. In six months
discomfort was seldom experienced though symptoms
were never entirely absent for any length of time.
More than two years after the original attack con-
siderable pain was experienced for a week and then
perforation occurred. Operation was successfully
performed, a malignant growth being found, but the
patient died in a few months. The diagnosis was
confirmed microscopically. It is open to argument
whether the original attack was due to carcinoma but

in our opinion it seems highly probable.   The
lesson we have derived is that reliance on X-rays
alone in cases otherwise clinically suspicious is a
very dangerous practice.   We are very strongly of
the opinion that a gastric analysis and testing of
the faeces for occult blood would have enabled
surgical intervention to have been sought in this
case at a very early stage.

We might here record a case to show the
other side of the picture, where radiological evi-
dence sufficed to banish suspicions aroused on good
clinical grounds, (No. 132).   This patient present-
ed himself to us and it seemed very probable from
his history that he had a carcinoma.   Examination
revealed nothing beyond localised tenderness in the
epigastrium.   We failed to find occult blood.   His
stomach contents, after an Ewald meal showed no free
hydrochloric acid.   We could not be sure in regard
to lactic acid as the test was obscured by the
presence of bile.   We felt however that he was a
case of carcinoma and referred him to the Royal
Infirmary of Edinburgh.   The physician who examined
him gave it as his opinion that the patient was
suffering from cancer of the stomach.   He even
added that he was inclined to think he could palpate
a definite mass in the epigastrium, though he was
unwilling to commit himself.   The patient was

X-rayed and no evidence of carcinoma was discovered, either on the films or by the screen. The subsequent history of the patient proved that the clinical suspicions were without ground in fact.

Case No. 167 may be quoted briefly. He complained of what appeared to be intercostal neuralgia. No improvement resulted from various forms of treatment, including such drastic measures as blistering. Anorexia set in with failure of strength and we considered the question of carcinoma. He was sent to an hospital and no evidence of carcinoma of the stomach was discovered radiographically. There was no free hydrochloric acid and the lactic acid test was negative. The patient returned home with the recommendation to continue treatment by blistering. No lasting relief resulted and in a month's time a palpable tumour was discovered and he was sent back to hospital, where it was found he had malignant disease of the transverse colon. Two lessons result from this. The first is the necessity for a radiological examination to be of the whole intestinal tract; and the second is for ourselves, which we trust we have appreciated, viz. the necessity to test for occult blood. If we had done this test, the probability is that we should have been able to diagnose the condition which the radiologist had overlooked.

We come now to the second type where indigestion of fairly long standing is present. In these, a much smaller number than the first type, the practitioner's suspicions should be aroused by any sudden change in the symptoms. This may be an increase in the severity of the pain, or less regularity in its onset, it perhaps occurring soon after food instead of after an interval; or it may be relief does not follow so readily from measures formerly successful. Obviously these points are only likely to be brought out by a very careful history. When they are observed in a man over thirty-five then the possibility of carcinoma should not be dismissed until adequate proof of its non-existence has been obtained. Examination as indicated when discussing the first type may yield much information but recourse to the aid of special methods is usually necessary and always advisable.

The test-meal should be used though it is less likely to be of help here. Hurst (137) states that in cases where a test-meal had been performed before an ulcer turned malignant there was no change in the character of a test-meal taken subsequently. When such a case becomes advanced changes in the gastric contents would in all probability be observed but at that stage diagnosis would probably be otherwise obvious.

Occult blood should be sought in the faeces. Manifestly this may be present in a case of long standing indigestion and be due to ulcer. Hence there is little value from this means in differentiation. If the patient is put on an ulcer régime however occult blood should become progressively less and finally disappear if the case is one of ulcer. In a growth it varies daily in amount and is very unlikely to disappear. This means daily testing over a period. It is not therefore very suitable in General Practice; but it is a valuable method in a difficult cases and is worthy of employment on the few occasions in which such problems arise.

The most useful method at our disposal in such a case is X-rays; though here again, if the case be one of malignant degeneration of an ulcer, it may be impossible to recognise the true nature of the condition in the very early stages.

It would appear therefore that in this second type early diagnosis is not likely to be made if the case is one of malignant degeneration in an ulcer. If it be one of cancer following some other type of dyspepsia it may be detected sooner by the above methods. The means at our disposal are not so efficient for this type of case as regards early diagnosis; but in consolation this type is much

more rare.   It has not been our experience to meet
such a case.

Special mention might be made of those
cases where the symptoms of gastric or duodenal
ulcer are closely simulated.   The latter is more
rarely imitated.   No special means other than
those we have described can be employed to detect
them.   It need only be said that the possibility
of such cases should be borne in mind and with it
the necessity of full investigation.   In deciding
between duodenal ulcer and carcinoma, Moynihan (138)
and others lay stress on the absence of remission,
spontaneous or following treatment, in carcinoma.
Hurst (139) reports a case in which a three month
remission took place.   He says that as far as his
experience goes these cases show achlorhydria,
which makes a diagnosis of duodenal ulcer impossible,
though cholecystitis, or appendicitis might be
present.   In his opinion no condition shows the
value of the test-meal better.   We do not consider
the use of the test-meal of much assistance to the
General Practitioner in the diagnosis of ulcer.   In
these cases, although ulcer is the most likely from
the clinical examination, we consider the stomach
tube should be used if carcinoma is a possibility.

Finally, if there be any doubt and the
symptoms do not readily clear up an exploratory

operation should be performed.   No hesitation in advising this course should be felt by the practitioner.   It is not courage which is required to advise it but courage to decline the assistance of a surgeon.

The diagnosis between gastric carcinoma and Pernicious Anaemia is sometimes not without difficulty.   As a rule the anaemia of gastric carcinoma is of the secondary type but certain cases occur where the blood picture is identical with that of Pernicious Anaemia.   Wasting is rare in the latter, the patient usually appearing well-nourished, while in the former cachexia is present and a tumour may be detected.   It might of course be that an early carcinoma would not show these features though this is probably rare.   The total acidity in Pernicious Anaemia is as a rule lower than in carcinoma, being often under ten.   Van den Bergh's test is positive in Pernicious Anaemia.   This is not likely to be useful to the practitioner and reliance on X-rays would be necessary in a doubtful case.

DUODENAL ILEUS.

Duodenal Ileus is the condition which results from compression of the third part of the duodenum where it is crossed by the mesentery.   It appears to be not altogether infrequent.   We our-

selves have only encountered one case. (No. 117).
The text-books usually state that it is commonly
associated with visceroptosis but Hurst (140) says
it is as common in well-developed men as in thin,
feebly developed women.   In this connection our
case was of some considerable interest.   It
occurred in a woman of unusually good muscular
development, of the hypersthenic type, who had two
brothers operated on for duodenal ulcer.   One of
these we had the opportunity of examining for
another complaint and we observed that he was of the
broad-chested, vigorous athletic type.   As Hurst
remarks, it must predispose to the development of
duodenal ulcer, if the other conditions necessary
for its formation are present;   and if secondary
gastric stasis occurs gastric ulcer is liable to
develop.

The symptoms are discomfort or pain one
and a half or two hours after food.   Food and
alkalies are said not to relieve the condition.   In
our case McLean's Powder produces very considerable
relief, no doubt due to the fact that she exhibits
hyperchlorhydria.   There is no reason to doubt the
diagnosis, it having been confirmed by Wilkie who
first described the condition and an X-ray examin-
ation having been made.   Vomiting brings complete
relief in most cases.   Apart from these symptoms

various others may occur though none are diagnostic
and X-rays are required to make a decision.

GASTRITIS.

        This condition, as it is generally known,
is one in which there is chronic inflammation of the
gastric mucous membrane accompanied by changes in the
gastric secretion and function. It has recently
been demonstrated by German surgeons that the con-
dition is a much more common one than was hitherto
supposed. These surgeons have made partial
gastrectomy a routine operation for ulcer. Some have
even gone to the apparently extreme length of perform-
ing this operation for "indigestion" in the absence
of evidence, before or after operation, of ulcer
being present (Hurst 141). The stomachs in the
great majority of cases showed obvious evidence,
macroscopic or microscopic, of chronic gastritis.
Whatever may be thought of this method of treatment
it has yielded information previously unknown. It
follows therefore that gastritis is not the rare dis-
ease it has been supposed to be but is a common and
important condition. It seems curious that chronic
gastritis should formerly have been regarded as pres-
ent only when a low acidity, or achlorhydria, with
abundant mucus was found. After all, in these cases
the causative factors could often be shown to have
been going on for many years, and obviously catarrh

of the mucous membrane must have been gradually taking place. Certainly the condition in these cases is usually symptomless until the gastritis is fairly far advanced; but this surely does not exclude the possibility of symptoms being present in mild and early cases. In fact surgery has, as we have just seen, at last demonstrated this to be true.

The commonest causes are imperfect mastication; over-indulgence in alcohol, tea, coffee, condiments and tobacco; and the presence of oral or oro-pharyngeal sepsis. Where food is habitually swallowed in an imperfectly-chewed state it remains longer than usual in the stomach and tends to promote increased vascularity and secretion. Such cases commonly result from defective or septic teeth and then a toxic element is superadded. Tea and coffee when taken strong or in excessive quantity irritate the gastric mucous membrane. Spices and other condiments provoke increased secretion but their long-continued use in large quantities results in inflammation of the mucosa. Tobacco, especially if chewed, is an important factor. It results in increased secretion at first, and is important in the etiology of ulcer as already mentioned, but after a long time gastritis may result. Alcohol causes vascularity and stimulates the mucosa at first but finally gives rise to catarrh.

It will be seen that the above factors usually result at first in increased secretion and it is probable that the acid itself is a factor in promoting catarrh eventually. At any rate many cases show an increased acidity to begin with but later the mouths of the secreting tubules become blocked with mucus and diminution of acidity results. The mucus probably also prevents the stomach receiving the natural stimulus from contact with food and this is another factor in lessening secretion.

Chronic gastritis also occurs secondary to diseases in other organs. Among these may be mentioned diseases of the heart, lungs, and kidneys. Venous stagnation occurs and catarrh is the eventual result.

Clinical Features. The symptoms are ill-marked at the beginning but become more definite later. The appetite is poor, especially in the morning, and meals may be followed by a sensation of fullness in the epigastric region sometimes amounting to pain. Severe pain is not as a rule found in this condition. A furred tongue and bad taste in the mouth is common, while regurgitation of "acid" or sour mouthfuls may occur. Flatulence is sometimes present. Diarrhoea, due to achlorhydria and/or an accompanying enteritis may be found. More frequent is constipation which may however alternate with diarrhoea. Morning vomiting is especially common in those cases due to alcohol.

General symptoms are usually present, such as loss of mental and physical energy, heaviness of the limbs and headache.

On examination, some increased sensitiveness to palpation may be found but real tenderness is uncommon. The test-meal, in the fully established condition, usually shows low or absent free hydrochloric acid and excess of mucus. The low acidity is due to the lessened secretion explained above and also to a slight extent by the alkalinity of the mucus. If the mucus be removed, by washing out the stomach, a considerable increase in acidity can usually then be obtained. In early cases the acidity may be increased.

Diagnosis. We shall consider first of all the fully established cases (i.e. the classical "chronic gastritis") and then later the diagnosis of early mild cases.

The history, including symptoms as described, and the long course of the disease are important. When combined with absent or very low free hydrochloric acid, the probability of gastritis is increased. The absence of severe pain is useful in excluding ulcer and cancer, as is also the lack of circumscribed tenderness. The only positive physical sign, according to Bennett (142), is the presence of considerable quantities of mucus in the

gastric contents.

Ulcer and cancer can be eliminated by the methods described for these diseases. Functional conditions can easily be excluded by the history, the use of the test-meal and a consideration of the individual.

The diagnosis of early cases of chronic gastritis, it seems to us, must be largely a matter of exclusion and in some cases there may possibly be an element of conjecture. When acid is present in fair quantity, mucus may be found and suggest the diagnosis when considered in conjunction with the history and examination, where these have revealed a toxic or infective source. On the other hand, mucus may be digested in the presence of acid and in these cases there seems no certain way of diagnosis. We have followed the plan of excluding all other organic causes and when there appears to be a toxic or infective source present, treating the patient for the gastritis. This of course is subject to the condition not being obviously nervous dyspepsia but where other organic diseases are definitely excluded and the most prominent and likely cause is a toxic one. In the absence of any improvement the question of a purely functional condition must be reviewed. Focal sepsis has all too often been blamed for diseases for which it has

been in no way responsible.   If the toxic cause be
removed and no improvement results in a reasonable
time, then the diagnosis has obviously been wrong.
The procedure is, we confess, not a very satis-
factory one.   It seems to us however essential to
recognise the early development of gastritis as it
is in the early stage that cure is most likely.
And no serious harm is likely to ensue as a result
of the temporary delay in deciding between early or
mild gastritis and functional dyspepsia.

APPENDIX DYSPEPSIA.

        When chronic appendicitis gives rise to
gastric symptoms, with or without pain in the right
iliac fossa, it may reasonably be called appendix
dyspepsia.   Two groups can be distinguished.   In
the first the dyspepsia is irregular in type and
does not suggest ulceration.   The symptoms are
practically continuous during attacks and these are
of short duration, usually just a day or so.   They
recur however more frequently than ulcer attacks.
Exertion and the occurrence of diarrhoea seem to be
common precipitating factors in this condition;
whereas in ulcer weather and mental anxiety more
commonly bring on an attack.

        The pain is situated in the centre of the
epigastrium and may radiate to the umbilicus or to
the right iliac fossa.   Pain in the latter position
may occur independently of that in the epigastrium.

The pain has generally no very definite relation to food though it may be worse immediately after a meal.   Vomiting is not uncommon but does not result in very marked relief, though some improvement is usual.   Alkalies are not very successful in producing relief.   Nausea is frequently present unlike ulcer conditions.   Haematemesis may occur from an acute gastric or duodenal ulcer, probably caused by the presence of the septic focus in the appendix, and prove very deceptive.

On examination tenderness on pressure in the right iliac fossa may be felt.   It may result in pain being experienced in the epigastrium as well but there is always tenderness over the diseased organ.

A test-meal shows normal or increased acidity.   Hypochlorhydria or achlorhydria in chronic appendicitis is present in most cases where pain is confined to the right iliac fossa (Bonar 143).   These cases do not concern us.

Certain cases occur where the symptoms are identical with gastric or, more commonly, duodenal ulcer.   This constitutes the second type.   In these cases not only is the pain related to food but relief follows vomiting or the taking of alkalies.

Diagnosis.   If this condition be borne in mind in cases suggestive of ulcer then careful investigation,

on the lines indicated as a routine, should reveal
the true nature of the condition.   X-rays are
commonly necessary however as we have said in the
section on duodenal ulcer where the differential
diagnosis from gastric or duodenal ulcer is fully
considered.

In the first type the irregular nature of
the symptoms combined with careful examination of
the abdomen should be sufficient to avoid error.
It is seldom that the condition is over-looked if
due care be exercised.   The opposite mistake is
more common to-day when the appendix is frequently
blamed for symptoms for which it is not responsible.
Walton (144) states that in 2,237 upper abdominal
operations, changes in the appendix sufficient to
call for its removal were present in only 4 per cent.
The test-meal is of no help and occult blood tests
only eliminate ulcer.   This may be helpful but it
must be noted that both conditions may exist to-
gether.   This however only applies when the con-
dition simulates ulcer.

GALL-BLADDER DYSPEPSIA.

The symptoms of dyspepsia which arise in
connection with gall-bladder disease are due to
cholecystitis and not to gall-stones.   As in the
case of appendix dyspepsia two groups may be dis-
tinguished, one in which the dyspeptic symptoms are

of an irregular type and the other where they simulate ulcer very closely.

In the first group the irregular symptoms combined with the prominence of a feeling of distension and the consequent belching of wind is the most usual picture. There is nothing characteristic about the symptoms although flatulence is seldom absent and commonly predominates.

In the second group the symptoms resemble those of ulcer, especially duodenal ulcer, very closely. In fact, they may be identical and a consideration of the symptoms gives little help as a rule. Periods of complete freedom, such as are present in the early stages of ulcer, are usually absent.

Common to both groups are such symptoms as distaste for fatty food, eggs etc. which may be suggestive and a full history may point the way to a diagnosis. It is however usually necessary to rely more upon the examination in this condition than in true affections of the stomach.

Diagnosis. On examination, tenderness may be elicited in the region of the gall-bladder and is frequently present several weeks after an attack. In ulcer of the duodenum the tenderness is usually only present over the duodenal bulb when spontaneous pain is being felt. Rigidity of the upper rectus

when present gives the clue to the condition being
an inflammatory one but does not distinguish between
ulcer and cholecystitis, though it persists longer
after an attack in the latter.   Tenderness is as a
rule nearer the midline in duodenal ulcer than in
cholecystitis.   Deep inspiration causes the pain to
be greater in cholecystitis and indeed it may only
be present when inspiration brings the gall-bladder
below the ribs and into contact with the fingers.
In this connection it should again be emphasised
that cholecystography has shown the position of the
gall-bladder to be much less constantly in the
classical position, at the tip of the ninth costal
cartilage, than was formerly supposed.

The test-meal is of no help here and what
has already been said of that procedure in appendi-
citis applies equally in this condition.

The presence of occult blood in the stools
is a very useful sign as it excludes cholecystitis.
It must be remembered that this statement does not
hold absolutely because occult blood can occur,
though only occasionally, in cholecystitis, pre-
sumably from damage by a gall-stone resulting in
bleeding.

In doubtful cases X-rays must be used.
An opaque meal may be sufficient to exonerate the
stomach and may show the presence of gall-stones.

Where no stones are present, or if present are not
visible to the X-rays, it may be necessary to out-
line the gall-bladder by using sodium tetra-iodo-
phenolphthalein.

We have considered the diagnosis mainly
in relation to ulcer as it is probably there that
the greatest difficulty lies.   As a rule the diag-
nosis is readily apparent from the history and
examination with perhaps in addition the use of the
occult blood test.

Although not a common finding, it is con-
venient here to mention what Wilkie (145) calls the
"trinity of abdominal lesions".   By this he means
the simultaneous presence of cholecystitis, appendi-
citis and peptic (usually duodenal) ulcer.   He
found 22 such cases out of 814 cases of chole-
cystitis, i.e. approximately 3 per cent.   They
frequently first showed symptoms after an attack of
influenza and Wilkie considers they are the result
of a blood-borne infection.   As regards diagnosis
the condition does not involve any extra responsi-
bility for the general practitioner since the
recognition of the appendix disease makes it
desirable to have surgical aid.

CARCINOMA OF THE COLON.

Cases of this condition have usually to be
considered in connection with malignant disease of

the stomach, if indeed they simulate dyspepsia at all.   Hurst (146) however reports two cases simulating duodenal ulcer.   Unless the tumour is palpable the history and examination are not likely to provide the key to the diagnosis.   Test-meals and occult blood tests might establish the malignancy of the condition but X-rays would probably be necessary to locate the site in the absence of a palpable mass.

EPIGASTRIC HERNIA.

A small protrusion of extra-peritoneal fat through the linea-alba may give rise to reflex dyspepsia.   It is usually acutely tender on pressure.   An impulse on coughing may be obtained . Usually the symptoms are those of distension; and though food may aggravate the condition it is usually never absent.   Cases have occurred which simulate duodenal ulcer closely.   Very commonly these herniae seem to develop after a rapid loss of weight.   It need only be said that when they resemble ulcer in their symptomatology complete examination should be made to exclude the latter condition since there is no reason why ulcer should not occur in a person with an epigastric hernia. Generally speaking however the condition has only to be borne in mind to enable the diagnosis to be made.   A careful examination should seldom fail to

reveal the presence of hernia. At any rate if several examinations fail to reveal the presence of a protrusion in the mid-line the consideration of this condition may be dismissed.

SPASTIC COLON.

This condition has been well described by Ryle (147) and also Eggleston (148). According to them, an irritable state of the nervous system is the principal predisposing condition. Women are affected twice as frequently as men and it occurs usually between the ages of thirty and fifty. Constipation is common. In some cases there is an over-secretion of mucus. Attacks are often brought on by the use of purgatives in an attempt to treat the constipation, or by worry and exposure to cold. Irritability of visceral muscle in other parts is sometimes present and causes frequency of micturition, dysmenorrhoea, vascular spasm or pyloric spasm. As a rule, the cardinal symptom is lower abdominal pain which is usually gnawing in character and never rhythmical as in ordinary colic. It may be slight in severity but is sometimes, though rarely, so severe as to simulate renal colic. The pain may last for several hours or even days and the patient is commonly depressed or irritable and lacking in energy.

On examination the colon is unusually

palpable.   In the left iliac fossa the colon is
ordinarily palpable but in this condition it may be
felt as a hard, narrow, cord-like structure.   The
ascending and transverse parts of the colon cannot
as a rule be felt in health, but in this condition
they are palpable and similar to that described for
the portion in the left iliac fossa.   Tenderness of
the palpable portion may or may not be elicited.
In the group of cases formerly described as
mucomembranous colitis, excess of mucus is present
and in some cases casts of the colon.

X-ray examination is not always conclusive
as the spasm is very intermittent and may be absent
at the time of the examination.   A barium-meal is
usually more successful than an enema as the latter
may overcome the spasm.   In typical cases the
appearance is of a thin line of barium sometimes
sharply cut off from the adjacent portion.   The
normal haustrations may be completely obliterated in
severe cases but in lesser ones they are exaggerated.
Diagnosis.   This involves a great number of abdomin-
al diseases, e.g. renal and biliary calculus,
appendicular disease, peptic ulcer and pelvic dis-
ease in women.   For our purpose, confusion is most
likely to occur with ulcer or functional dyspepsia,
though the possibility of carcinoma of the stomach
should not be forgotten.   A routine examination

should suffice to eliminate organic disease of the stomach. Difficulty, which may be considerable, may remain in regard to functional dyspepsia. In such cases, the use of X-rays is desirable since the type of patient in these two conditions may be closely similar.

DYSPEPSIA ASSOCIATED WITH DISEASE OF URINARY TRACT.

It is unnecessary to consider at any length such conditions on account of their comparative rarity. They must however be borne in mind. Distension of the bladder from enlarged prostate, stricture or other cause, not infrequently results in persistent dyspepsia. Similarly, chronic nephritis may be the cause of gastric symptoms. No doubt these conditions could be regarded as forms of gastritis for the mucous membrane is probably the seat of catarrh. We prefer however to regard it as due to a reflex mechanism of nervous origin, just as uraemic fits originate from toxic effects on the brain. As Einhorn (149) says, diseases of the kidney associated with gastric symptoms are due either to excretion of urea through the gastric mucous membrane or by retention of urea in the circulation and irritation caused thereby on the brain. Diagnosis. This is dependent on careful routine investigation of the systems including examination of the urine and the habit in obscure cases of

performing a rectal examination.

TABETIC DYSPEPSIA.

We follow Ryle's classification in considering this condition as a reflex dyspepsia. Like the last condition, this does not require very full description as it is fairly rare.

Clinical Features. It should be noted that two forms of gastric crises occur:- (1) Painful Crises. (2) Vomiting Crises. These are usually independent but may occur at different times in the same individual or even together at one time.

The pain is usually extremely violent and may be mistaken for an acute abdominal emergency. It is often of sudden onset and may cease in an equally abrupt manner. Nothing except morphia brings relief.

In the second type enormous quantities of highly acid fluid are vomited and the attacks as in the first type usually last three or four days. The nature of the food taken has no effect.

Diagnosis. Gastric Crises may resemble gastric or duodenal ulcer though this is rare. As a rule the condition is perplexing from the outset. The great difficulty usually arises from the fact that the usual signs of locomotor ataxia are almost or entirely absent.

There are several important points to bear

in mind in the absence of typical signs. The ankle-jerks usually disappear before the knee-jerks and this may suggest the diagnosis. The pupils seldom exhibit the Argyll-Robertson reaction but commonly there is a certain sluggishness. The periodic nature of the attacks, a history of lightning pains or signs of an old chancre should be useful.

In cases of doubt, the Wassermann reaction in the blood and cerebro-spinal fluid should be done.

S U M M A R Y.

In the Introduction we defined dyspepsia
so as to include all cases, functional or organic,
in which discomfort of any kind occurred during
digestion; and we stated our object as follows:-

   (1) To formulate a routine procedure,
        suitable for General Practice,
        in the examination and diagnosis
        of cases of dyspepsia.

   (2) To classify the various kinds of
        dyspepsia in a way which shall be
        useful and simple; and which
        shall describe each condition
        adequately for the purpose of
        rational treatment.

   (3) To examine the results in 170
        cases.

In Chapter Three we have explained our
procedure.   This is more or less in accordance
with convention as regards the history, interro-
gation and examination, i.e. the purely clinical
part.   That part must be done as fully in General
Practice as in hospital work and in it there is no
curtailment desirable or justifiable.   It is in
regard to Special Methods (more particularly the
test-meal, the test for occult blood in the stools
and the use of X-rays) that we have endeavoured to
discriminate and decide in which forms of dyspepsia
one or more of these is necessary.   The conclusions
reached are referred to in detail under the sections

on the various diseases.

We have made a classification of dyspepsia
which seems to us to fulfil the purpose we have
indicated above.   The difficulties have been dis-
cussed and reasons advanced for the arrangement we
have formulated in Chapter Four.

In the second Volume notes of 170 cases
have been included.   A few conclusions and comments
regarding these are given at the end of the Volume.
The case-sheets are constructed on the lines indi-
cated in our routine procedure.   It is not to be
supposed that these notes contain all that was used
in the diagnosis.   They contain the salient feat-
ures and, we trust, make clear the method by which
we investigate a suspected case of dyspepsia.
Much that is important has of necessity been ex-
cluded on account of exigencies of space.   For
example, the examiner's impression of the patient's
temperament has often been summed up by such a word
as "nervous".   To deal adequately with questions of
this nature is impossible in tabular notes such as
we have been obliged to use.   It is apparent how-
ever that they may be of considerable importance in
the diagnosis.

Above we have indicated briefly the way
in which the object of this Thesis has been con-
sidered.   Below we give a summary of the chapters

in order.

INTRODUCTION.

Having defined dyspepsia and stated the
object of the Thesis we deal with the present
position of the General Practitioner.   We then
indicate how the multiplicity of modern diagnostic
methods has been a powerful factor in the growth of
specialism and has resulted in confusion in regard
to what methods it is incumbent on the General
Practitioner to perform.   We examine the duties of
the latter at the present time and find that his
main function is sorting out trivial complaints
from more serious ones which deceive by their
superficially trivial appearance.   We comment on
the necessity for a definite routine specifically
adapted for General Practice.   We indicate our
object to formulate such a routine for cases of
dyspepsia.   This subject we chose because of its
prevalence and importance.   Reference is then made
to the opportunities of General Practice and the
special advantages for investigation in our own
particular practice.

CHAPTER 1.

The necessity of understanding the normal
function of the stomach, before approaching the study
of morbid gastric conditions, has made us include a
short account of the anatomy and physiology of the

stomach.   This seems to us especially important as
it is only by at once appreciating departures from
the normal function that early disease can be
detected.   And it is this which it is the special
duty of the General Practitioner to realise.

CHAPTER 2.

This chapter is merely a continuance of
the previous one but deals with the stomach in dis-
ease.   It has to be appreciated that pathological
physiology is really the study of normal function as
deranged by morbid processes and no progress in
early diagnosis is possible unless the mechanism of
symptom production is understood.   We deal at some
length with these questions in this chapter.

CHAPTER 3.

The methods employed in investigation are
described here and the full scheme is given in
tabular form.   Some general observations on the
scheme are then made and finally the system of case-
recording in our practice is described.

CHAPTER 4.

This deals with the question of classifi-
cation.   The unsuitability for General Practice of
many existing schemes is pointed out and our own
classification is stated and explained.   Some of
the reasons for the divisions adopted are reserved
until the two subsequent chapters in which the

various forms of dyspepsia are considered in detail.

CHAPTER 5.

The acute forms of dyspepsia are described briefly. The clinical features are mentioned shortly and the differential diagnosis indicated. This chapter exhibits no special considerations for General Practice but is included to make the survey of dyspepsia complete.

CHAPTER 6.

The chronic forms of dyspepsia are dealt with in this chapter. It has been deemed necessary to include the clinical features in considerable detail in certain instances, to make clear the grounds upon which the diagnosis which follows is based. A short account of the modern views on the ulcer diathesis precedes the sections on Gastric and Duodenal Ulcer.

B I B L I O G R A P H Y.

1. Rolleston, Sir H.: Brit. Med. Journ. 25:10:30,
   p.700.

2. Mackenzie, Sir James: The Future of Medicine,
   1919, p. 6.

3. Moynihan, B.G.A.: Practitioner 1928, CXX1,
   p. 137 and 138.

4. Moody, R.O.; Van Nuys, R.G.; and Chamberlain,
   W.E.: Journ. Amer. Med. Ass.
   1923, LXXX1, 1924.

5. Hurst, A.F.: Gastric & Duodenal Ulcer by Hurst
   & Stewart, 1929, p. 24.

6. Hurst, A.F.: See Ref. 5, page 21.

7. Cannon, W.B.: The Mechanical Factors of Digest-
   ion, N.Y. 1911, Chapter 1X.

8. Ryle, J.A.: Gastric Function in Health & Dis-
   ease, 1926, p. 34.

9. Apperly, Modern Views on Digestion and
   Gastric Disease, by H. MacLean,
   1928, p. 11.

10. MacLean, H.: Modern Views on Digestion &
    Gastric Disease, 1928, p. 11.

11. MacLean, H.: See Ref. 10, pp. 35 and 36.

12. Halliburton, W.D.; & McDowall, R.J.S.: Hand-
    book of Physiology, 1930, p. 411.

13. Hurst, A.F.: See Ref 5, p. 26.

14. Edkins, J.S.: Jl. of Physiol., 34, 133, 1906.

15. MacLean, H.: See Ref. 10, p. 36.

16. Hurst, A.F.: See Ref. 5, p. 26.

17. Hurst, A.F.: Goulstonian Lects. On the
    Sensibility of the Alimentary
    Canal, London 1911.

18. Ryle, J.A.: See Ref. 8, pp. 8 & 9.

19. Halliburton, W.D, & McDowall, R.J.S.: See Ref.
    12, p. 409.

20. Willstatter, R., & Baman, E.: Uber Magenlipase.
    Vergleich in verschiedenen Rein-
    heitsgraden mit Pankreaslipase;
    Abhandlung uber Pankreasenzyme:
    Ztschr. f. physiol. Chem. 173:
    17-31, 1928.

21. Mellanby & Woolley: See Ref. 12, p. 419.

22. Bayliss & Starling: Jl. of Physiol. 28, 325,
    1902, & 29, 174, 1903.

23. Dodds, E.C., & Bennett, T.I.: Jl. of Physiol.
    55, 381, 1921.

24. Mellanby, J,: Jl. of Physiol., 60, 85, 1925.

25. Halliburton, W.D., & McDowall, R.J.S.: See Ref.
    12, p. 416.

26. Hilton, J.: Lectures on Rest & Pain, 2nd. Edtn.
    London, 1877.

27. Poulton, E.P.: Some Visceral Sensations, Lancet
    1928, 2, p. 1223.

28. McDowall, R.J.S.: The Science of Signs and
    Symptoms, London, 1931,
    p. 255.

29. Hurst, A.F.: See Ref. 5. p. 133.

30. Hayem, G.: Bull. et Mem. de la Soc. Med. des
    Hop. de Paris, 1893, 3rd. series X,
    366.

31. Hurst, A.F.: See Ref. 5, pp. 70 and 71.

32. Hurst, A.F.: See Ref. 5, p. 155.

33. Ryle, J.A.: See Ref. 8, p. 63.

34. Kinsella, V.J.: Med. Jl. of Australia, 1928,
    1, 64.

35. Palmer, W.L.: Arch. Int. Med. 1926, XXXVlll,
    694 and 1927 XXXlX, 109.

36. Hardy, T.L.: Lancet 1929, 711.

37. Wilson, M.J.: Arch. Int. Med. 1928, XL1, 633.

38. Ryle, J.A.: See Ref. 8, pp. 61 and 65.

39. Hurst, A.F.: See Ref. 5, p. 161.

40. See Ref. 35.

41. Ryle, J.A.: See Ref. 8, p. 61.

42. Hurst, A.F.: See Ref. 5, p, 157.

43. Hurst, A.F.: Guy's Hosp. Reports, LXX1, 450, 1922.

44. Abrahams, A.: Diseases & Disorders of the Digestive Organs, 1932, p. 72.

45. Ryle, J.A.: See Ref. 8, p. 71.

46. MacLean, H.: See Ref. 10, pp. 52 and 53.

47. Ryle, J.A.: See Ref. 8, p. 82.

48. MacLean, H.: See Ref. 10, p. 54.

49. Ryle, J.A.: See Ref. 8, p. 75.

50. Barclay, A.E.: Lancet 1922, ii, 261.

51. Bennett, T.I., & Venables, J.F.: Brit, Med. Jl. 1920, 2, 662.

52. Hurst, A.F.: See Ref. 5, pp. 27 and 28.

53. Einhorn, M.: Diseases of the Stomach, N.Y. 1929.

54. Payne, W.W., & Poulton, E.P.: Quart. Jl. Med. XV11, 53, 1923.

55. Hurst, A.F.: Quart. Jl. Med. V111, 300, 1914.

56. Ross, J.: Brain, Vol. X, 1887-1888, p. 333.

57. Mackenzie, Sir J.: Symptoms & Their Interpretation, 1st. edtn. 1909, pp. 7 & 44.

58. Hurst, A.F.: The Sensibility of the Alimentary Canal, London, 1911.

59. Hurst, A.F.: See Ref. 5, p. 163.

60. McDowall, R.J.S.: See Ref. 28, p. 261.

61. Lennander, K.J.: Edin. Med. Jl. XX11, 103-111,
                         1927.

62. Kappis, M.: Die Sensibilitat der Bauchhohle,
                   Klin. Wochenschrift 1925, ii,
                   2041.

63. Cope, Z.: Clinical Researches in Acute Abdomin-
                 al Disease, 1925, pp. 26 and 27.

64. Morley, J.: & Twining, E.W.: Brit. Jl. of Sur-
                                    gery 1931, XV111,
                                    376.

65. Morley, J.: Abdominal Pain, 1931, p. 71.

66. Ryle, J.A.: See Ref. 8, p. 58.

67. Hurst, A.F.: Brit. Med. Jl. 1925, 1, 145.

68. Ryle, J.A.: See Ref. 8, pp. 59 and 62.

69. Hurst, A.F.: See Ref. 5, p. 168.

70. Morley, J.: See Ref. 65, pp. 80 and 81.

71. Hurst, A.F.: See Ref. 5, p. 166.

72. Bennett, T.I.: The Stomach & Upper Alimentary
                      Canal in Health & Disease, 1925,
                      p. 114.

73. Hurst, A.F.: Chronic Indigestion, Practitioner,
                    Jan. 1931.

74. Hurst, A.F.: See Ref. 73.

75. Mackenzie, Sir J.: See Ref. 2, p. 71.

76. MacLean, H.: See Ref. 10. p. 111.

77. Hutchison, R.: Lectures on Dyspepsia, 1927,
                      p. 25 et seq.

78. MacLean, H.: See Ref. 10, p. 109.

79. Mackenzie, Sir J.: See Ref.2, p.204.

80. Alvarez, W.C.: Nervous Indigestion 1931, p. 184.

81.   Alvarez, W.C.: See Ref. 80.

82.   Paterson, H.J.: Differential Diagnosis and
                              Treatment of Indigestion, 1929
                              p. 9.

83.   Moore, Henry: Brit. Med. Jl. 23rd. Feb. 1932,
                              p. 363.

84.   MacLean, H.: See Ref. 10, p. 47.

85.   Abrahams, A.: See Ref. 44, p. 25.

86.   MacLean, H.: See Ref. 10, p. 112.

87.   Robertson, J.D.: Gastric Acidity, 1931, p. 37.

88.   Murray, I. & Robertson, A.B.: Brit. Med. Jl.
                              April 2nd. 1932. p. 607.

89.   Robertson, J.D.: See Ref. 87, p. 41.

90.   Robertson, J.D.: See Ref. 87, p. 42.

91.   Robertson, J.D.: See Ref. 87, p. 51.

92.   Bennett, T.I.: Brit. Med. Jl. May 28th. 1932,
                              p.1006.

93.   Einhorn, M.: See Ref. 53, p. 332.

94.   Moore, Craven: Brit. Med. Jl. April 23rd.
                              1932, p. 771.

95.   Davidson, S.: Brit. Med. Jl. May 7th. 1932.
                              p. 859.

96.   MacLean, H.: See Ref. 10, p. 129.

97.   Haldane, J.B.S.: Possible Worlds, London, 1930,
                              p. 103.

98.   Osler & McCrae: Principles & Practice of
                              Medicine 1923, p. 499.

99.   Behan, R.J.: Pain 1922, pp. 409, 428.

100.   Ryle, J.A.: See Ref. 8, p. 100.

101.   Alvarez, W.C.: See Ref. 80, p. 72.

102.   Hutchison, R.: See Ref. 77, p. 52.

103.   Ryle, J.A.: See Ref. 8, p. 100.

104. Hutchison, R.:   See Ref. 77, p. 94.

105. Hurst, A.F.:   See Ref. 5, p. 422.

106. Hutchison, R.:   Proceedings Royal Soc. Med. XXV,
                3, 1932, p. 295.

107. Hutchison, R.:   See Ref. 77, p. 83.

108. Alvarez, W.C.:   See Ref. 80, p. 71.

109. Ryle, J.A.:   See Ref. 8, p. 113.

110. Einhorn, M.:   See Ref. 53, p. 444.

111. Balint, R.:   Ulcusproblem u. Saurebasengleich-
                gewicht, Berlin, 1927.

112. Osman, A.A.:   See Ref. 5, p. 58.

113. Gavrila, J.:   Arch, des Mal. de L'Appareil
                Digest. 1928, 18, 502.

114. Muller, O.:   Die Kapillaren der menschlichen
                Korperoberflache in gesunden und
                kranken Tagen.   Stuttgart, 1922.

115. Muller, O.: & Heimberger, H.:   Deutsch. Zeitsch.
                f. Chir. 1924, CLXXXVll, 33.

116. Hurst, A.F.:   Lancet 1922, 2, 1369.

117. Campbell, J.M.H., & Conybeare, J.J.:   Guy's
                Hosp. Reports, LXXlV, 354, 1924.

118. Same as Ref. 4.

119. Hurst, A.F.:   See Ref. 5, p. 59.

120. Wilkie, D.P.D.:   B.M.A. Meeting, R.I.E. Edin.
                27th. April 1932.

121. Hurst, A.F.:   Chronic Indigestion, Practitioner,
                Jan. 1931.

122. Wilkie, D.P.D.:   Lancet 1927, 2, 1228.

123. Hurst, A.F.:   See Ref. 5, p. 237.

124. Hurst, A.F.:   See Ref. 5, p. 32.

125. Friedman, G.A.:   Amer. Jl. Med. Sci. 1914,
                CXLVlll, 540.

126. Hurst, A.F.: See Ref. 5, p. 152.

127. Osler, W., & McCrae, T.: See Ref. 98, p. 489.

128. Abrahams, A.: See Ref. 44, p. 55.

129. Hurst, A.F.: See Ref. 5, p. 246.

130. Abrahams, A.: See Ref. 44, p. 58.

131. Paterson, H.J.: See Ref. 82, p. 41.

132. Einhorn, M.: See Ref. 53, p. 321.

133. Einhorn, M.: See Ref. 53, p. 315.

134. Bennett, T.I.: See Ref. 72, p. 238.

135. MacLean, H.: See Ref. 10, p. 105.

136. Einhorn, M.: See Ref. 53, p. 332.

137. Hurst, A.F.: See Ref. 5, p, 245.

138. Moynihan, B.G.A.: Practitioner 1928, CXX1, 137.

139. Hurst, A.F.: See Ref. 5, p. 250.

140. Hurst, A.F.: See Ref. 5, p. 67.

141. Hurst, A.F.: Chronic Indigestion, Practitioner,
                    Jan. 1931.

142. Bennett, T.I.: See Ref. 72, p. 156.

143. Bonar, T.G.D.: Guy's Hosp. reports, LXX11,
                      400, 1922.

144. Walton, A.J.: The Surgical Dyspepsias, 2nd.
                     edtn. 1930, p. 674.

145. Wilkie, D.P.D.: See Ref. 120.

146. Hurst, A.F.: See Ref. 5, p. 238.

147. Ryle, J.A.: Lancet 1928, 2, 1115.

148. Eggleston, E.L.: Jl. Amer. Med. Ass. 1928,
                        Dec. 29th., 2049.

149. Einhorn, M.: See Ref. 53, p. 573.

        NOTE: Ref. 34 could not be traced. It is
               quoted from Gastric and Duodenal
               Ulcer (Hurst & Stewart 1929).