CHLOROSIS and DYSPEPSIA:
With Some Remarks On The Value Of Various Methods Of Chemical And Physical Diagnosis Of Diseases Of The Stomach.

In this paper, I do not intend to advance any original ideas, but rather to discuss and accentuate some points, which have occurred to me, in the relations between chlorosis and the dyspepsias that are so often found accompanying it; and to make some remarks upon some of the methods which have been employed in investigating the gastric functions.

I intend to divide the subject into three parts
1st. Symptomatology and Aetiology
2nd. A Discussion as to the value of the methods of examining the gastric functions
3rd. Treatment.

SYMPTOMATOLOGY and AETIOLOGY

The frequent occurrence of dyspepsia in the course of cases of chlorosis is noted in most of the ordinary text-books on general Medicine (1) and of

those wholly devoted to diseases of the stomach.\(^{(1)}\). Fagge\(^{(2)}\) and Ewald, however, do not mention any relation between the two. That dyspepsia should frequently occur in the course of chlorosis is what we should expect, when we consider the great frequency with which it occurs in all female patients; but the proportion of cases of stomach disorder is far greater in chlorotics than in other cases taken over the period at which that disease usually occurs. Thus, out of 479 women between the ages of 14 and 30 years, admitted into the General Hospital, Birmingham, during the last three years, 239 or 50 per cent were suffering from dyspeptic troubles; of girls suffering from chlorosis, there were 109 and of these 96, or 88 per cent shewed marked gastric symptoms, while only 13, or 12 per cent had apparently normal digestion. If we subtract the 109 chlorotic cases from the total, the difference is still more marked. This leaves 370 cases not suffering from chlorosis, and of these 143 or 38.6 per cent presented symptoms of abnormality of gastric function, while in 227 or 61.4 per cent there were no such symptoms. This large proportion of dyspepsias in chlorotics suggests that there is some causal relation between the two, especially when we find that in other conditions associated with lessened metabolism and

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(1) Habershon, Diseases of the Stomach. 3rd Ed. p. 127
(3) Martin, Diseases of the Stomach. 1896. - p. 212. 400
(4) Weiss, Chlorose Gum & Schnirer's Diagnostisches Lexikon. S. 569
(5) Principles and Practice of Medicine, 3rd Ed. Vol. 11.
impoverisation of the blood and which are frequently complicated by dyspepsia, the proportion is not nearly so great. Thus, out of twenty-four consecutive cases of chronic Bright's disease, dyspepsia was present only in eleven, or 41.6 per cent, while in thirteen, or 58.4 per cent it was absent.

This causal relation, if it exist, is what I wish to discuss more fully later on; but first it is necessary to examine into the symptoms of the dyspepsia and see if there is any special type which may be differentiated as "anaemic" or "chlorotic" dyspepsia.

The following cases are typical of a large number which I have been able to examine.

CASE I. E.W., aet. 20 years, single, of no occupation. She had been suffering from shortness of breath for five years. There was nothing worthy of note in the family history. The patient had always been healthy up to the present illness. The illness for which she was admitted began when she was 15 years of age (i.e. 5 years previously). Her early symptoms were shortness of breath and palpitation of the heart. These had persisted up to the time of admission, but varied in intensity from time to time, being better while under treatment, but soon relapsing when this was discontinued. About two years ago she began to suffer from pain after her food; this was only occasional at first, but gradually
became more frequent; at the same time she suffered from flatulence. There had been no vomiting. Her bowels have been constipated, and the menses have been irregular and scanty.

On admission she was well developed and well nourished, and evidently "chlorotic". There was no oedema or jaundice.

**Alimentary System.** Her tongue was large, moist and pale. There was a slight whitish fur on the dorsum. She complained of an aching heavy pain in the right side about half-an-hour after eating with a sharp pain in the epigastrium. This lasted for from an hour to an hour and a half, and was accompanied by a feeling of distension and nausea though she never vomited. She also suffered from flatulent eructations. Her bowels were constipated.

On examination the abdomen shewed nothing abnormal, and there was no epigastric tenderness. The liver dulness reached from the 4th intercostal space to \( \frac{1}{2} \) inch below the ribs in the right mammary line. The spleen was not enlarged.

**Haemopoietic System.** No enlarged glands

**Blood:**
- Red corpuscles 2,930,000 per cc
- White corpuscles 5,000 per cc
- Haemoglobin 38%

**Circulatory System:** There was marked dyspnoea and palpitation on the least exertion. The apex beat was in the 5th space \( \frac{1}{2} \) inch internal to the mammary line. The area of cardiac dulness was not increased.
There was a haemic murmur in the pulmonary area and an accentuation of the 2nd sound. The pulse was 84 per minute, with fairly high tension, but the volume was small.

Respiratory System: Healthy.

Urinary System: Urine. Specific Gravity 1030. Reaction: acid. There was no albumen, blood or sugar. The dirty reddish precipitate consisted of squamous epithelium and a large number of uric acid crystals.

Reproductive System: Amenorrhoea.

Stomach Functions: Eight ounces of acid material were removed after the test breakfast. The filtrate contained lactic acid, albumose, peptone, sugar and erythro-dextrine, but no free hydrochloric acid. The residue contained many starch granules but no yeast cells or sarcinae. The stomach contents alone did not digest egg albumen, but did, with the addition of hydrochloric acid. On distending the stomach the lower border reached three inches below the umbilicus, while the smaller curvature was about 1½ inch above that point. The fasting stomach contained a few flakes of mucus and microscopically shewed some epithelial and small cells with a few scattered starch granules.

The patient was kept in bed and given a milk diet gradually increased as she was able to bear it. At the same time the sulphates of iron and magnesium were given with Oppenheimer's bi-palatinoids of ferrous carbonate. She was also treated with general
massage. Under this treatment the condition of the patient rapidly improved, her gastric symptoms disappeared and the dyspnoea and palpitations became very much less marked. In a week the red blood corpuscles rose to 3,660,000 per cc. and the haemoglobin 48%. After a fortnight they were 4,320,000 and 56 respectively and at the end of three weeks there were 4,790,000 red corpuscles per cc. and 74% of haemoglobin. At this time the gastric functions were again examined and free hydrochloric acid was present while the greater curvature of the stomach only reached about 1¼ inches below the umbilicus, the smaller curve, however, being still depressed. On performing the digestive experiments it was found that the filtered stomach contents were able to digest egg albumen without the addition of hydrochloric acid or pepsin.

The patient was discharged from the Hospital at the end of a month apparently cured.

CASE II. E.P., aet. 17 years, a housemaid, was admitted into the General Hospital complaining of shortness of breath on exertion and vomiting. She had had the shortness of breath for three months and the vomiting for nearly a fortnight. The family history was exceptionally good and the patient herself had always enjoyed perfect health till the present illness came on.

About three months before admission, she noticed that she became short of breath on going
upstairs or walking fast. At the same time her mother noticed that she looked pale. She had no other symptoms until she had been in service as a housemaid for two or three days when she began to vomit immediately after her meals and had some pain in the epigastrium. She stated that she had eaten nothing which might have caused this. The vomiting had continued up to the time of admission and during the last three times the pain which was in the umbilical region had become worse. Her bowels had been very constipated all the time. The patient had always lived in the country until she went into domestic service.

On admission she was pale and anaemic-looking but well nourished. There was no oedema or jaundice.

**Alimentary System:** Tongue clean, moist.

She had some pain in the region of the umbilicus coming on after food and relieved by vomiting which occurred in a few minutes. No flatulence nor eructation. On examining the abdomen, all the organs appeared to be healthy. There was no enlargement of the liver or spleen, nor any tenderness in the epigastrium. The bowels were very constipated.

**Circulatory System:** No evident dyspnœa, no palpitations. The apex beat was in the 5th intercostal space in the mammary line. There was no increase in the area of cardiac dulness. There was a loud haemoid murmur in the pulmonary area and an accentuation and occasional reduplication of the pulmonary second
sound.
The pulse was 80 per minute - of medium tension
and small volume.

Respiratory System: Healthy.

Urinary System: Urine. Specific Gravity 1008 -
reaction alkaline - no blood, albumen or sugar.
The deposit consisted of epithelial cells and débris.

Reproductive System: Menstruation was regular but
the discharge was very scanty.

Blood: Red corpuscles 3,900,000 per cc
White corpuscles 10,000 per cc
Haemoglobin 64%.

Stomach Functions: About 2 oz of acid material was
removed after the test breakfast. The filtrate con¬
tained free hydrochloric acid, albumose, erythro¬
dextrine and sugar but no lactic acid or peptone.
The stomach contents digested egg albumen without the
addition of hydrochloric acid or peptone. The dis¬
tended stomach reached as low as the umbilicus in the
middle line while to the right it descended about an
inch lower. The small curvature was visible about
three inches above the umbilicus. The fasting stom¬
ach contained a few flakes of mucus, which under the
microscope were seen to consist of numerous small cells
with a few squamous epithelial cells and a small num¬
ber of starch granules. There were no yeast cells
nor sarcinae.

The patient was treated in a precisely
similar manner to the First and rapidly improved.
The vomiting almost immediately ceased and when the constipation was overcome she had no further digestive symptoms. The stomach functions were again investigated at the end of three weeks. The results were precisely similar to the first, with this exception, that lactic acid was now present and free hydrochloric acid absent. As a result of this the filtered stomach contents did not digest egg albumen without the addition of hydrochloric acid.

The patient left the Hospital apparently quite well after a month's stay.

**CASE III.** K.S., aet 21, a gilder by trade, came to the Hospital complaining of shortness of breath, pain in the pit of the stomach, with vomiting after meals - duration 2-3 years. There was a strong tubercular family history, her mother and two sisters having died of phthisis. She herself had always been healthy up to the time of the commencement of the illness for which she sought treatment.

For the last 2 or 3 years she had suffered from shortness of breath and pain after food, with vomiting. This had been very much worse at times, and at the date of admission she had scarcely been able to work for six months, and during the last fortnight she had been obliged to rest altogether. She stated that three months previously, she had vomited
some dark clotted blood, and again about a fortnight ago. Her bowels had been very constipated during her whole illness, and this had been specially marked before an acute attack.

On admission she was dyspnoeic and evidently chlorotic, but there was no oedema of the ankles. **Alimentary System:** The lips and gums were pale. The tongue large, flabby, pale, the edges indented; moist and fairly clean. She had a continuous pain in the epigastrium but this became much worse about a quarter of an hour after food and was only relieved by vomiting. She suffered also from flatulence and gaseous eructations, but had no waterbrash nor acidity. Her bowels were very constipated. On examining the abdomen the epigastrium was found to be slightly fuller than normal and there was very marked epigastric tenderness. The liver and spleen appeared to be normal. **Circulatory System:** The patient was very short of breath and complaining of constant palpitations. The apex beat was in the 4th intercostal space in the nipple line. The area of cardiac dulness was not markedly increased and there was no murmur, but a marked bruit de diable in the neck. The pulse was full but of rather low tension - 78 per minute. **Respiratory System:** Normal. **Urinary System:** Urine Specific Gravity 1006. 30 oz. reaction neutral, no albumin, blood or sugar. **Deposits:** epithelium and leucocytes. **Reproductive System:** Menstruation every three weeks,
quantity small and accompanied by pain.

**Blood**

- Red corpuscles: 4,950,000 per cc
- White corpuscles: 10,000 per cc
- Haemoglobin: 48%

**Stomach Functions**: After Ewald's (2) test breakfast 4 oz of an acid brownish yellow fluid removed. This was filtered and the filtrate found to contain free hydrochloric acid, albumose, erythro-dextrine and sugar but no lactic or butyric acid or peptone. The residue on the filter paper consisted of starch granules, and no yeast cells nor sarcinae were found. The stomach contents digested egg albumen without the addition of hydrochloric acid. On blowing up the stomach it was found that the upper limit was $1\frac{1}{2}$ inches above the umbilicus, while the lower limit was $2\frac{1}{4}$ inches below it, the whole stomach being lower down than normal. The salol(3) test shewed only slight delay.

The patient was kept in bed with a diet of milk and barley water, with the gradual addition of bread, chicken, etc., until she was taking ordinary food - at the same time iron was administered in the form of the sulphate, combined with sulphate of magnesia and also in the form of carbonate, in Oppenheimer's bipalatinoids.

The haemoglobin increased to 55 per cent and her stomach symptoms gradually diminished, although the dyspnoea and palpitations still persisted.

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(1) All blood estimations made with Gower's Instruments.
(2) See page 44
(3) See page 69
These three cases are fairly typical of three varieties of dyspepsia met with in chlorotic patients. They may be called, (a) the atonic, (b) the irritative, (c) the ulcerous.

(a) The atonic: This is characterised by a gradual onset, and usually first shows itself by a feeling of weight in the epigastrium coming on shortly after meals, together with flatulence and gaseous eructations. The feeling of weight is often accompanied by a feeling of distension, and cardiac palpitation. After a time these unpleasant sensations are followed or preceded by a definite pain in the epigastrium or on the left side, occasionally on the right side over the lower ribs or about the umbilicus. The pain may be sharp stabbing, crushing or burning in character. It is relieved by vomiting, or if the patient does not vomit it lasts for some hours, gradually dying away. The vomited matters consist of partially altered food and are of a slightly acid reaction. The appetite, at first good, becomes as a rule very poor but the nutrition of the patient is good, at any rate during the early stages. There is marked constipation of the bowels, which are generally only moved by purgatives. On examining the stomach functions it is found that there is marked delay in gastric digestion, that free hydrochloric acid is frequently absent or in very small quantities, and that there is marked atony of the stomach wall, with generally some gastrop-
but it may become so, due to the accumulation of food in it and fermentation. The irritation of the gastric mucous membrane by the delayed stomach contents and products of fermentation or decomposition may lead to the second form of dyspepsia.

(b) The irritative: This condition is usually sudden in its onset and is characterised by pain in the epigastrium of a sharp or burning character coming on some time after food, say, in from one to one and a half hours. This form of dyspepsia is generally preceded by some error of diet or by a change in hygienic conditions, or mode of living. For example, in the case cited above it came on in a girl who had always lived in the country and who had just entered domestic service in a large town. The pain is as a rule entirely relieved by vomiting which gradually occurs after about half an hour. The irritable condition of the mucous membrane may give rise to pain and vomiting almost immediately upon the ingestion of food. When the pain does not come on for some time, the time of onset corresponds with the time at which there is a maximum of free hydrochloric acid in the gastric contents. When it comes on at once it is due to direct irritation of the mucosa by the ingesta. These patients usually suffer also from pyrosis and acid regurgitations into the pharynx. Flatulence is rare. The appetite is usually good, and may be even voracious, the patient never being satisfied.
On examining the stomach functions we usually find that digestion has gone on rapidly and that the test for free hydrochloric acid gives a vivid reaction. The stomach may however shew atony and gastroptosis, while on the other hand it may be emptied earlier than normal.

It was stated above that the atonic form of dyspepsia might lead to the irritative form. We also find that the irritative may end by becoming atonic. This is brought about by the constant irritation of the walls of the stomach producing some inflammation and when this subsides leaving a weakened or atonic condition, with delayed digestion and diminished hydrochloric acid reaction.

When the acute condition has subsided, free hydrochloric acid is not secreted to excess, and in the case of chlorotics, there is a diminution as shown by its absence after a test-meal. This is brought out also in Case II.

(c) The ulcerous: I do not intend to say much about this form here, although I shall discuss some points in the aetiology of gastric ulcer in chlorosis further on. I may, however, say that it is of more frequent occurrence when there is some dilatation of the stomach. A long history of stomach trouble is not infrequent, as in Case III, where symptoms had been present for 2-3 years before any haematemesis occurred and when the type of the dyspepsia was "atonic".
The atonic form is the most common in chlorosis and may be taken as the typical form. The irritable form must be regarded as generally accidental and is then due to some error in diet or change in hygienic conditions or it is consecutive to the atonic form. We not infrequently meet with cases, such as Case II, in which a comparatively slight change in diet or surroundings will bring on a severe attack of dyspepsia, and this is not at variance with what we should expect when we consider that the functions of the stomach in chlorosis are depressed, as evidenced by the atonicity of the muscular coat, and the frequent diminution of the free hydrochloric acid, which we shall consider later. The ulcerous form generally occurs late in the disease and seems to be, in some cases at least, a sequence of long continued gastric irritation.

The question of haematemesis without gastric ulcer is one of considerable interest and importance from a therapeutic point of view. That severe gastric disease may occur without any marked gastric symptoms is shewn by a case of carcinoma of the stomach, published by Watts, in which there was no symptom except palpitation of the heart and dyspnoea until perforation and peritonitis set in, with fatal result. The appetite was good and there was no symptom of indigestion. At the autopsy an enormous destruction of the gastric parietes was found in the region of the fundus and greater curvature, with adhesions to the
spleen and other viscera, and the edges of the perforation were carcinomatous. Fagge(1) states that most of the symptoms attributable to cancer of the stomach may be absent and quotes two cases (one of Sir Thomas Watson's and one of his own) in which there were only very slight stomach symptoms during life, but in which post mortem, extensive carcinomatous infiltration of the greater curvature was found. It is a well known fact that round ulcer of the stomach may be entirely latent, until it gives rise to a severe haemorrhage or causes perforation. Thus, we see that we may have very grave organic lesions of the stomach without any of the ordinary symptoms which we are accustomed to meet with in those conditions. Gallot(2) following Hayem, states that in chlorosis we may observe haematemesis independently of gastric ulcer, and quotes three cases in support of his statement(3). In the First Case, the patient had suffered from dyspnoea and had been advised to take a sea voyage. This brought on a severe "mal de mer" accompanied by painful and abundant vomiting after which the menses were suppressed. Three days after this there was a profuse haematemesis. It is then stated that the patient became anaemic and had remained so.

In this Case, chlorosis cannot be invoked as being the cause of the haemorrhage as the patient was

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(3) Id. p. 42.
not anaemic at the time. She had suffered previously from gastric troubles, and I think we may safely say that the haemorrhage was from a pre-existing ulcer and that the determining cause was the vomiting from seasickness.

In the Second Case the patient had been chlorotic for over three years, when she met with a carriage accident which frightened her very much. After this the menses were suppressed and in addition to the digestive troubles from which she had been suffering since the commencement of her illness there was continued vomiting. The vomited matter at first consisted of food mixed with bile and then of blood in large quantity.

In this case although chlorosis had been present for three years, there were also digestive troubles which might quite well be the only signs of a chronic gastric ulcer, and the accident might easily have lead to an increase of the ulceration and perforation of a vessel sufficiently large to give rise to a profuse haemorrhage; or the initial vomiting itself might have caused a pre-existing ulcer to have increased in depth. That this might occur, is shewn by a case quoted by Christison(1), in which a youth of fourteen, after eating a Christmas feast, was attacked by violent and frequent vomiting. Next day he vomited two pounds of blood and soon afterwards died. At

(1) Treatise on Poisons, 1829, p. 89.
the autopsy the inner coat of the stomach was found to be torn in many places, and a similar condition was present in the duodenum. The rest of the alimentary canal was healthy. Ziemssen(1) also records a case of rupture following distension through fermentation processes in a stomach which was the seat of stricture arising from the healing of a chronic ulcer. I do not think, therefore, that this case of Gallot's can be put down to chlorosis alone.

The Third Case, the patient had suffered from chlorosis and dyspepsia for four years, when she had haematemesis and melaena. The haematemesis soon ceased, but the melaena continued for a month.

This case appears to me to be one of latent gastric ulcer and not to support the statement of Hayem and Gallot that haematemesis frequently occurs in chlorotics without ulceration.

I have never met with a case of haematemesis in a chlorotic patient in which there was not at the same time, some other symptom of ulcer, but I have not infrequently had girls at about the age of puberty, complain of vomiting blood and who, on strict interrogation, admitted that they were subject to attacks of epistaxis.

From the above facts, the possible latency of cancer and ulcer of the stomach, and the frequency of the blood from epistaxis being swallowed, and vomited again, I think we should treat all cases of haematemesis occurring during chlorosis as cases of gastric

(1) Sammlung clinischer Vorträge (Volkmann) vol.XV.p.100
ulcer unless we can find some other definite cause for the bleeding and while not denying that haematemesis may occur accidentally in chlorosis without ulcer, I do not think that we can accept the statement that it is frequently so observed.

We are now in a position to discuss each symptom more in detail and to point out any special features to be noticed which may be peculiar to or more common in chlorosis. I may here state that I have, in common with other observers, been unable to differentiate any definite set of symptoms as pathognomonic of chlorotic dyspepsia. In fact, I shall shew later on, that the dyspepsia of chlorotics cannot be distinguished from ordinary dyspepsia either by its symptoms or by its chemistry.

**Appetite.** This is generally poor and may indeed lead to an "anorexia" or almost complete loss of appetite. On the other hand the appetite may be good but the patient is prevented from eating, by the knowledge that pain and discomfort are certain to follow. In cases of the irritable type the appetite may be normal or even increased. I have never seen cases of pica or bulimia occurring in chlorosis.

**Thirst.** This appears to be a very frequent complaint of patients suffering from chlorotic dyspepsia. At least fifty per cent of my cases suffered from it. It does not appear to be due to an increased loss of water, as the urine and faeces are not increased and the skin does not secrete very actively.
It is probably due to there being an increased amount of fluid in the subcutaneous tissue, which is in larger amount than normal, and a corresponding drain upon the cerebral centres. This is only a suggestion and I have no facts to prove or disprove its correctness.

**Pain.** This has been partly considered in the description of the various types of dyspepsia occurring in chlorotics. It is almost invariably present, and may present all varieties of kind, intensity, position, time of onset and duration according to the type of dyspepsia and lesion present. The commonest seat of pain is the epigastrium; it is also frequently felt below the apex of the heart and over the lower left ribs, more rarely on the right side, in which case there is probably some lesion of the pyloric region of the stomach as in a case reported by Bercioux (1) in which the pain was principally in the right hypochondrium. At the post mortem an ulcer was found on the smaller curvature of the stomach close to the cardiac orifice but the pyloric portion was immensely dilated. The most frequent site of the pain in chlorotics is midway between the umbilicus and the base of the ensiform cartilage or rather below this point. The position thus described corresponds to the area between the lower edge of the liver and the upper border of the stomach, when gastrophtosis is present, which we shall see later is always the case in chlorotics. McInert (2) considers that the pain produced

(1) *Bulletin de Société anatomique 11e série.* 1857, p. 263
(2) Vide infra p. 234.
here is due to stretching of the fibres running from the solar plexus of the sympathetic which is situated behind this space. That it should only occur after food is explained by the fact that while the stomach is empty it is situated nearly in its normal position, and that it is only as it becomes distended that the stretching takes place. Gastric pain in chlorosis is sometimes complained of in the back to the left of the spine over the false ribs, but this pain is rare without the occurrence of ulcer. Intercostal neuralgia especially of the 5th left intercostal nerve is frequently spoken of by French writers as being caused by dyspepsia; Melle Porojniakov(1) in her thesis for the Faculty of Medicine of Paris quotes Peter, Beau, Dujardin Beaumetz and Leven as describing its occurrence in dyspeptics and states that it may be one of the causes of palpitations in such cases. The causes of gastric pain in these cases are various. When there is flatulence the pain is due to over distension of the organ, when a sort of colic is set up, as is shewn by the fact that eructation of the gas relieves the pain and that pressure often has the same effect(2). With a hyper-secretion of acid the pain is due to irritation of the mucous membrane by the hyper-acid contents(3), but it is also possible that it may be due to spasmodic contractions resulting from distension by gas evolved(4). Soupault (5) states that

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(2) Wilson Fox. loc.cit. p.61.
(4) Fox. loc. cit. p.25.
there may be a hypersthenic condition of the gastric muscle associated with excessive fermentation and that the pain in these cases and in round ulcer described by Cruveilhier are due to a struggle between the contracted pylorus and the muscle of the stomach itself. The ingestion of food itself may cause pain if the wall of the stomach is in an irritable condition, as shewn by those cases in which the pain comes on immediately after eating and before there is time for the formation of gas or excessive acid. Stretching of the sympathetic nerve fibres has been referred to above.

Patients often complain of a bitter taste in the mouth, especially in the morning. The cause of this does not seem to have received much attention from writers on the subject. It seems probable however that it may be due to fermentation in the mouth which has been able to go on during the night without the products being washed away by aliments. Bouchard thinks that it may be due to an excretion in the saliva of part of the toxic substances which have been absorbed from a dilated stomach or an intestine in which fermentation is going on abnormally.

**Constipation.** This condition is nearly always present in cases of chlorotic dyspepsia. Thus, out of thirteen cases of undoubted chlorosis associated with dyspepsia only one had regular alvine dejections. In all the others there was very marked constipation. This appears to be due to the general weakening of the
muscular apparatus of the body, lessening the contractability of the intestine and its power to drive on the contents.

Various other symptoms are met with, not primarily referrable to the stomach, such as neuralgias in various nerves, general states of mental and bodily lassitude, dyspnoea and cough, palpitation and cardialgia, disturbances of the menstrual function, etc. These, however, I have not been able to find more frequently in chlorotics who exhibit stomach symptoms than in those who do not, and they therefore may be referred to that disease rather than to dyspepsia.

I shall now give an account of the result of seventeen examinations of the stomach functions in chlorotic girls, all except two of whom suffered also from dyspepsia. The methods employed are those more fully described in part 2. In the fifteen cases which presented stomach symptoms I found that free hydrochloric acid was present in nine, and in one of these in excess, while in six it was absent. Lactic acid was present in eleven, and absent in four. Albumose was present in all, but peptone was absent in all but six, and in three of these the reaction was very slight. Sugar was only absent once and erythro-dextrine was always present. In the digestive experiments it was found that in every case except the one in which hydrochloric acid was in excess in the stomach contents, that the digestion had gone on further when free
acid had been added, as shown by the more brilliant reaction of albumose and peptone.

These facts show that in these cases the digestion is generally somewhat delayed, but that free hydrochloric acid is being secreted in fair amount.

As to the motor functions, as tested by means of salol they were found to be delayed in two only out of seven cases; but, as is pointed out later(1), this test is fallacious. Out of five cases in which the stomach contents were examined after fasting for twelve hours some remains of food were found in all but one. This is a much truer test than the former and shows that the motor power of the stomach is below normal in these cases.

The most distinct and constant change in the stomach is found on distending it with air or carbonic acid gas. When the stomach is thus blown up a tumour is seen arising in the umbilical region of the abdomen which has very marked limits not only laterally and below but also above. The lower margin of this swelling comes always below the umbilicus and may reach nearly to the pubes in advanced cases, but as a rule it comes about 2-3 inches below the umbilicus. It is usually, also, lower on the right side than on the left, so that the lower border runs from above downward and to the right and then takes a sudden turn upwards to form the right border. The upper border is also distinct and forms a sharp curve with the convexity downwards, the left limb running up under the

(1) See p. 70
left costal cartilages and the right joining the right border of the tumour. The right limb is much shorter than the left, shewing that the pylorus is somewhat depressed. This condition caused by distending the stomach I found in ten out of eleven cases. In the case in which I was unable to demonstrate it, so much pain was caused on injecting the air that the organ could not be fully dilated, and therefore this case must be left out of account. This condition, while occurring in a very large percentage of, if not in all, chlorotics with dyspepsia and even when there is no dyspepsia, as was shewn by two cases, also occurs in other conditions, especially in atonic dyspepsia in women and in so-called neurasthenia and hysterical conditions; but in many of these there has been at some previous period, an attack of chlorosis. The stomach never recovers from this condition, at least not in my limited experience and Meinert (1) bears me out in this. The relations of this condition and its aetiology will be considered later.

We have now discussed in some detail the gastric symptoms and conditions met with in chlorosis and are in a condition to answer the question propounded at the commencement of this paper: Is there any definite type of dyspepsia that can be called chlorotic or anaemic dyspepsia? And this question must be answered in the negative, because as we have shewn there are three types occurring in chlorotics and these types may also occur in other diseases, or exactly similar

(1) Meinert. vide infra p.228
conditions may be produced in otherwise healthy subjects as the result of improper use of foods or condiments or by the introduction into the stomach of irritants. The most constant condition which is found in the class of case we are considering is that of gastropostosis or falling down of the stomach and this also occurs in numerous other conditions and may be present with apparently perfect health(1).

We have now to consider the aetiological relations existing between chlorosis and dyspepsia. And here several paths open themselves for us. We have to inquire, 1st, whether the one is an accidental complication of the other; 2nd, whether chlorosis is the cause of dyspepsia or dyspepsia the cause of chlorosis; and 3rd, whether they are both the result of some underlying condition which may at times cause the symptoms of chlorosis alone, at others of dyspepsia alone and in the third place of both chlorosis and dyspepsia.

1st, Is dyspepsia an accidental complication of chlorosis, or chlorosis of dyspepsia?

That dyspepsia may occur as an accident in the course of chlorosis cannot be denied, for chlorotics are no less liable to gastric derangement from errors of diet etc than other people. But we have already seen the large preponderance of dyspeptics among chlorotics when compared with females of the same age(2),

(1) Meinert. vide infra p.216
(2) p. 2
and these figures, embracing as they do 479 cases, seem to me to be pretty conclusive evidence that the occurrence of these two sets of symptoms together is not entirely accidental. Moreover in a large number of cases it is impossible to trace any exciting cause for the dyspeptic symptoms, which generally come on gradually and increase slowly if not treated.

2nd. Is chlorosis the cause of dyspepsia or is dyspepsia the cause of the chlorosis?

It undoubtedly appears at first sight that chlorosis is frequently the cause of dyspepsia, because many patients who are suffering from chlorosis only develop gastric symptoms after the abnormal blood condition is well advanced. But there is no definite evidence that this is the case and it is rather a post hoc propter hoc argument that patients with abnormal general metabolism may develop all the symptoms of atonic dyspepsia is well known and is shewn in cases of diabetes mellitus, chronic nephritis, tuberculosis, certain nervous diseases, etc. Thus, out of six cases of diabetes mellitus in which I examined the stomach functions there were symptoms of chronic gastric disorder in five and all these five shewed on further examination a deficient secretion of gastric juice and a lessened motility of the stomach muscle. In these cases, perhaps, the diet, which as a rule is not calculated to promote a healthy condition of the stomach, may be a prominent factor in the production of the symptoms and the stomach condition probably depends
more upon the constant irritation of improper and too bulky food than upon the underlying general condition. In chronic nephritis again the symptoms appear to be rather due to some body, possibly urea, as was well shewn in one case under my care, or some other by-products of metabolism\(^1\) being excreted into the stomach and setting up chronic irritation there.

In tuberculosis the condition is probably one of simple atony of the stomach wall, occurring as part of the general state of lowered vitality of the organism which also shews itself in loss of weight of the body generally and diminished tone of the skeletal muscles.

The dyspepsias of nervous diseases such as neurasthenia, are probably either due to simple neuroses of the stomach\(^2\) or perhaps of the colon\(^3\) or to an atony of the muscle of the stomach due to faulty innervation. That the dyspepsia of chlorosis is not a pure neurosis will be shewn below.

Here then we have four of the most common diseases which are frequently associated with dyspepsia of the atonic type and in only tuberculosis does this appear to be due to the general debility of the system.

Why then should we put the dyspepsia of chlorosis down to that cause?

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Trousseau, who considers that chlorosis is to be classed among nervous diseases(1), states that chlorosis not only engenders dyspepsia, but also a capricious appetite, a disgust for the most substantial fare and an insensate craving for things generally regarded as very bad(2). All these last conditions are not generally met with among chlorotics as we see them in this country but are rather put in the category of neuroses. Trousseau does not bring any facts to bear further on this subject but appears rather to be trying to prove the nervous origin of chlorosis. He, however, notes several cases of neuralgia(3) referable to the stomach and the internal organs.

In the absence, therefore, of any direct proof and of any proof from analogy we are not justified in believing that chlorosis is the cause of dyspepsia.

3rd, Is dyspepsia the cause of chlorosis?

This is answered in the affirmative by Stockman(4). He states that he has found in chlorotic girls that there is a very large proportion who suffer from dyspepsia(38 out of 63) and that those who do not complain of gastric symptoms have learnt to use a very restricted diet. On analysing the food of healthy persons and of chlorotic girls, he found that the amount of iron ingested by the latter was very much diminished (6 to 11 millegrammes in healthy people and

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(1) loc.cit.vol.iv.p.106
(2) loc.cit.vol.v.p.iii.
(3) loc.cit.vol.v.p.106.
only 1·2 to 3·2 millegrammes in chlorotics). He also estimated the amount of iron in the food of a healthy dog with 6,000,000 red blood corpuscles per cubic centimetre and over 90 per cent of haemoglobin. The iron was diminished to less than one third in the food and at the end of 46 days he found that while the red corpuscles had slightly increased, the haemoglobin had fallen to 64 per cent. From these facts he concludes that dyspepsia is a cause of chlorosis.

It may be pointed out that while a condition resembling chlorosis was produced in a dog by the withholding of iron in the food, the experiments upon human beings were only performed upon those in whom the disease was already advanced and there is no evidence brought forward to shew that the dyspepsia or diminution of iron ingested occurred before the chlorosis was set up. Neither does he state that in any of his 63 cases the dyspepsia appeared first. On the other hand we have the testimony of Gallot(1) who states that "chlorotics, in spite of their nervous condition, are not dyspeptic in fact, but they are potentially; bad regimen, faulty hygiene, heroic treatment wrongly administered may make the symptoms break out". From this it is evident that Gallot does not consider that dyspepsia causes chlorosis, and my own experience agrees with him for in the great majority of cases which I have been able to examine the

symptoms of chlorosis were the first to appear. Another objection which may be taken to Stockman's theory is that it does not account for the constant occurrence of gastroptosis in anaemic dyspepsia. Such a marked and peculiar condition demands some explanation and if this is not forthcoming any theory which does not take it into consideration falls to the ground.

We have now shewn that the occurrence of dyspepsia is not accidental, and that the two conditions are not dependent one upon the other, and we therefore come to the conclusion that there is some underlying general state or abnormal fact which is the cause of both.

The question now arises, What is this pathological factor?

This has been sought for in two directions:

a. In abnormal metabolism.

b. In abnormal anatomy of the abdominal viscera.

a. Haig considers that chlorosis is due to an increased amount of uric acid in solution in the blood. He states (1) that, in experiments upon himself, he found that the blood decimal curve (by which he means the ratio between the haemoglobin and the red corpuscles) fell in proportion as the uric acid secretion rises above the normal, proportionately with the urea. This excess he considers to be an index of the amount of uric acid in the blood. He also states (2) that the administration of uric acid in chlorosis reduces the blood decimal. Further he states (3)

(1) Uric Acid as a Factor in the Causation of Disease 2nd Ed. 1894. p. 238.
(2) loc. cit. p. 242.
(3) loc. cit. p. 244
that the administration of uric acid by the mouth in those, who, like anaemic girls are of rather low nutrition, is more likely to remain in solution in the blood, because their blood is probably more alkaline than that of better nourished individuals; therefore, the effect of giving uric acid is likely to be more marked in poorly nourished individuals than in those who are robust and well fed, and, dose for dose, the effect on the blood curve will be greater in the former than in the latter. He then goes on to say(1): 'The rapid metabolism of girls of 13 causes high urea per pound, high acidity of the urine and low alkalinity of the blood and resulting from this a storage of uric acid in the organs of the body. But the activity of metabolism is not continuous — this results in a fall of urea and acidity in the urine and a rise of alkalinity in the blood, causing increased solubility and excretion of uric acid previously retained.

Menstruation is established just as these changes are progressing, which upsets digestion and further lowers metabolism, producing more or less uric acidaemia, the signs of which, slow high tension pulse and headache or mental depression are among the most common accompaniments of menstruation. The uric acid already in the blood tends to keep the metabolism depressed, contracting the arterioles and generally interfering with nutrition.

In young women then, we have a storage of

(1) loc. cit. p. 249.
urate in their active metabolism at 13, and a passage of this urate through the blood as nutrition falls in later years, and especially with each occurrence of menstruation. It is little wonder that they should suffer from anaemia. The wonder is that any of them should escape it.

He also states (1) that "Dyspepsia may undoubtedly be produced by contracted arterioles" (as occurs in uricacidaemia) in the stomach and intestines, which inhibit gastro-intestinal digestion and allow putrefactive processes to take place.

Against the acceptance of this theory the following arguments may be urged.

Firstly, Although these observations have now been published for some years, they have not been corroborated by other workers, and we ought to receive with considerable hesitation a theory built upon new facts, quite uncorroborated and only embracing a few cases.

Secondly, The constant state of gastroptosis is not taken into account and is not even mentioned by Haig in his book. According to his theory, indeed, the stomach ought to be small and contracted rather than atonic.

Thirdly, Very similar conditions may be met with in youths about puberty who for some reason or other have some special strain put upon their metabolism e.g. severe work with an insufficient diet, and

(1) loc.cit.p.81.
in these the symptoms of chlorosis are not produced.

I do not wish to deny that an increased amount of uric acid circulating in the blood may produce a certain amount of anaemia, but I do not think we can believe that this in itself is sufficient to produce chlorosis, although perhaps when that condition is present, it may be rendered more intense by a state of uricacidaemia.

Bouchard\(^1\) states that the diseases arising from putrefaction going on within the alimentary canal are diseases of debility, because the alimentary material, incompletely digested and undergoing putrid fermentation is no longer sufficient for nutrition. He considers that dilatation of the stomach is among the causes of these diseases and states that it is an exceedingly common condition\(^2\) being very frequent in the sick, and relatively common in the healthy. On the same page he states that dilatation may exist without inducing anomalous sensations and without dyspeptic or gastralgic symptoms in two-thirds of the cases. He then goes on to state\(^3\) that in his cases of dilated stomach he has found the following conditions: 1. Phthisis (in two-thirds of the cases) 2. Chlorosis (in four-fifths of the cases) 3. Nervous and hypochondriac symptoms and 4. very varied other symptoms such as cutaneous eruptions, albuminuria and peptunuria, etc. He also states definitely\(^4\) that chlorosis

\(^1\) Bouchard. Lectures on Intoxication in Disease. English Translation. 1894. p.158.

\(^2\) loc. cit. p.156

\(^3\) loc. cit. p.159.

\(^4\) loc. cit. p.172.
in young girls is often induced by dilatation of the stomach.

As to the manner in which dilatation of the stomach produces chlorosis he says that even normal bacteria may produce a considerable increase of toxic substances in the stomach if free hydrochloric acid is deficient\(^1\). Also, quoting Brieger, alkaloids are produced in the process of peptonisation\(^2\).

The substances which have a toxic effect when absorbed into the blood are besides the alkaloids, acetic, butyric, valeric and sulphuric acids, ammonia and its compounds, leucin, leucaine, tyrosin, indol, skatol, cresol, phenol and the hydrocarbons\(^3\).

Against the acceptance of the above fact that "dilatation of the stomach frequently induces dyspepsia", we may make the following observations.

1st, Bouchard considers that the "splashing" sign is the only true evidence of a dilated stomach\(^4\). This, as will be seen in Part Two of this paper, cannot be accepted and renders it more than probable that he has missed some genuine cases and has included others in which there was no real dilatation.

2nd, He states that 30 per cent of all patients in Hospital have dilatation of the stomach\(^5\). If this be so how is it that much less than 30 per cent of the females suffer from chlorosis?

\(^1\) loc.cit.p.160
\(^2\) p.94
\(^3\) p.93
\(^4\) p.157.
\(^5\) Sammlung Klinischer Vorträge - von Volkmann.
Leipsig.Jan.1895
3rd, He does not state how it is that the special form of anaemia and its concurrent symptoms which occur in chlorosis are produced; except in the general statement, that it is a disease of debility due to incomplete digestion. Now, there is no reason to believe that, until very grave dyspeptic symptoms have supervened, there is any very marked diminution of digestion in chlorosis, the only alteration which occurs, at least so far as the stomach is concerned, being that digestion is more or less delayed, and we know that chlorotics do not as a rule lose weight as the disease develops. Therefore, I do not consider it to have been proved that dilatation of the stomach in setting up putrefactive processes and auto-intoxication, is a cause of chlorosis, although undoubtedly it may cause dyspeptic symptoms.

b. We have now considered the metabolic theories of chlorosis and dyspepsia and come to the anatomical.

This view is fully set forth by E Meinert(1) in a paper entitled "Uber einen beigewohnlicher Chlorose des Entwicklungs-alters anscheinend Konstanten pathologisch-anatomischen Befund und über die Klinische Bedeutung desselben", which ought to be regarded as a distinct landmark in our progress towards the knowledge of one of the most common of diseases.

He commences by quoting the result of 100

post mortems (p.209) made by Dr Neelson of Dresden who found that the stomach frequently has an almost vertical position and in extreme cases may be almost parallel with the left abdominal wall. While the cardiac orifice was in no case altered in position, the pylorus was depressed to a greater or less extent in nearly every case. As a rule, when a more or less vertical position of the stomach is maintained, both the larger and lesser curvatures were elongated. He describes the characteristics of gastroposis (p.213) as being the appearance of the small curve below the level of the liver and a remarkable elasticity of the pyloric portion of the stomach when distended.

He states that in considerably over 100 cases of chlorosis he found gastroposis in every one, even when the blood condition was unimportant (p.214). He draws attention to the frequency of moveable right kidney which he says occurred in 15% of his cases. He also points out that the stomach is neither atonic nor dilated in these cases, for although, when distended its volume is greater than normal, after the distending force has been removed it goes back to its former size. He thinks that this is due to an increase in the elasticity of the arterial coats, as pointed out by Virchow(1) which is specially marked in the pyloric region.

In five years he only found a normal position of the stomach 35 times in young females (p.216) and

in none of these was there any anaemia. On the other hand out of 400 cases of gastroptosis in women over 30, only one half had suffered from chlorosis. These facts, coupled with the 100 cases of chlorosis above mentioned lead him to conclude that in girls typical chlorosis never occurs with a normal position of the stomach, and that the age of puberty gastroptosis occurs very frequently without being complicated with chlorosis (p.217).

He describes his experiments upon 31 girls, about 14 years of age, who were just entering a training home for servants. He was unable to carry out the experiments in two cases; in one case the stomach was normal, in 28 there was gastroptosis. Of these 28 there were 17 who shewed slight chlorosis.(p.218)

As to the cause of this abnormal position of the stomach, he states that it cannot be a congenital condition because in the foetus, although the position is vertical, the small curve is to the front and the large curve recedes, a condition not found in gastroptosis. He also controverts the theory of Glenard (9 v.p.81) that it is due to a pulling down by the hepatic flexure of the colon, as in 34% of Neelson's cases this was normal (p.219) Having disposed of these theories he states that the only possible other conclusion is, having regard for the natural firmness of the attachments of the stomach, that it can only be displaced by pressure from neighbouring organs, especially the liver. There are
two factors in the production of this pressure (p.220) 1st a long, narrow, rickety thorax, 2nd, a tightly laced thorax.

The pressure thus exerted acts upon the liver (p.222) which cannot pass upwards on account of the diaphragm and therefore must be pushed down, and especially the right lobe, which in its turn acts most directly upon the pylorus.

He believes that in Germany corsets and chlorosis began at the commencement of the 18th century; and he quotes Hantzsche and Alber who state that in Persia the women in no way constrict the thorax and that there chlorosis is unknown, and Scriba, who says that in Japan it is only those who have appropriated European dress who suffer from this disorder (p.226)

Chlorosis is only one of the abnormal conditions which is brought about by gastroptosis (p.229) and he cites among other affections which may occur gastric ulcer, enteritis, local peritonitis, hernia, phlebitis, irregularities of menstruation, uterine fibroids, cholelithiasis, and most prominent of all, nervous derangements (p.230) and especially among these may be mentioned hemicrania with vomiting and even transient melancholia but not hysteria.

He states that nervous disorders arising from gastroptosis take place in paroxysms which may last for an hour or for weeks or months (p.231) and in those of long duration vaso-motor phenomena rarely fail to present themselves.
He considers chlorosis to be an enteroptotic crisis of long duration (p.233) and that many definite causes acting along with the pre-disposing cause of gastrop-tosis, are capable of bringing on the crisis.

There is an increased excitability of the abdominal sympathetic system caused by stretching of the fibres from the solar plexus by the displaced stomach and also by the exposure of the solar plexus itself, in the gap between the lower border of the liver and the smaller curvature of the stomach, to direct traumatism. (p.236)

He quotes von Middendorf and Glass, who shew that the production of haemoglobin in the spleen comes under the influence of the great sympathetic (p.237). And as the sympathetic nerve fibres are stretched in gastrop-tosis, he considers that this is the cause of the diminution of haemoglobin. And it is only when those fibres which supply the spleen are affected to a sufficient degree that anaemia is produced (p.240)

He states that any cause which produces an increased weight of the abdominal organs will bring on an enteroptotic crisis (p.241) and among these he enumerates constipation, colds, excessive meals, and other conditions producing abdominal plethora, such as typhoid and dysentery. Pregnancy, also, by bringing on a hyperaemic condition of the abdominal viscera and sympathetic, is able to cause an enteroptotic crisis (p.242)

He sums up the result of his experience as follows (p.253)
Chlorosis in its narrowest sense, i.e., paroxysmal resembling anaemia in its nervous symptoms, appearing at the critical age of puberty in the female sex, never occurs with a normal position of the abdominal organs.

Among the anomalous situations of the abdominal organs so prevalent among females, gastroptosis seems to constantly accompany chlorosis. Gastroptosis may be mentioned as the local disturbance at the root of chlorosis only, because this anomalous situation could not be mentioned without the condition which accompanies it, of an undue irritability of the sympathetic centre of the same, viz:– Plexus solaris sive coeliacus – under whose influence comes the production of haemoglobin in the spleen.

Chronic anaemias whose type differs more or less from ordinary chlorosis may owe their origin to the descent of other abdominal organs, e.g., the transverse colon and the nerve irritability consequent upon it.

Considered as a blood disease, chlorosis stands as a secondary anaemia, but as regards its origin it stands as a neurosis. It is no morbus sui generis, but only one of the numerous complex symptoms, by which the insidious anomaly of position of the abdominal organs, in the form of Glenards Enteroptosis, shews itself.

Incipient gastroptosis comes about merely from want of space in the hollow of the diaphragm.
"The usual cause of this want of space is an alteration in the thorax produced in the female sex by the pressure of clothing."

Meinert's theory appears to me to be the nearest to the truth of any yet formulated, resting as it does, upon a definite anatomical basis. The constant presence of gastroptosis in chlorosis I consider to be certain, for not only did it occur in all of Meinert's cases, but also in the whole of my own. The relation to chlorosis can be easily understood when we remember the close relation of the sympathetic in the abdomen both to the stomach and to the spleen. It is evident that any alteration in the innervation of an organ, so very nearly associated with the formation and destruction of the blood as the spleen, must have a marked influence upon the haemoglobin. The various dyspeptic symptoms which are found in this condition are chiefly referable to the altered position of the stomach affecting the neighbouring nerves, for as we have seen as a rule there is no alteration in the gastric chemistry.

Against Meinert's theory is the fact that it does not explain why the haemoglobin should be reduced so much more than the blood corpuscles except by supposing that there is a diminution of the total quantity of blood, and there appears to be no reason for doing so.

The preceding facts lead me to the following
conclusions:
1st Chlorosis and dyspepsia are very frequently present in the same individual.
2nd, There is no definite type of dyspepsia occurring in chlorosis.
3rd, Both chlorosis and the dyspepsia causing it are produced by the same cause.
4th, That cause is an abnormal position of the stomach, generally produced by constricting the lower part of the thorax, and known as gastroptosis.

METHODS OF EXAMINATION

The methods employed in the examination of the stomach functions in these observations have undergone some modification as one or other of them proved to be more or less unsatisfactory. I shall, therefore, describe shortly the methods used, and state what I consider to be their respective drawbacks or merits. I may say that nothing more elaborate has been attempted than can be easily performed at any time with the intelligent co-operation of the patient.

In the earlier series of experiments the method of procedure was as follows: - At 8 a.m. a test meal, consisting of from two to four ounces of
white bread and a pint of weak tea, without milk or sugar was administered, as suggested by Ewald (1).

This was removed by means of a soft tube at the end of an hour, pressure being exerted over the region of the stomach if necessary. Usually about two to four ounces of stomach contents were obtained in this way. Before withdrawing the stomach tube a Higginson's syringe was attached to the free end and air pumped into the organ, the limits of which could then generally be easily seen and verified by palpation and percussion. The stomach was then washed out and the tube withdrawn. After the mid-day meal, ten grains of salol were given in a capsule, and the urine collected after 75 minutes, and again after 24 hours, and every six hours subsequently, until no reaction was obtained with the liquor ferri perchloridi. The stomach contents were then examined as to reaction and the presence of free hydrochloric, lactic and butyric acids, albumoses and peptones, erythro-dextrin and sugar. Finally, their digestive powers were tested by means of a series of digestive experiments upon coagulated egg albumin. The residue remaining upon the filtre paper was examined microscopically.

The test meal has been the same in all cases for although a more elaborate repast has been advocated by Leube, (quoted by Sydney Martin (2)) consisting

(2) Functional & Organic Diseases of the Stomach. p.149
of soup, beef steak and bread, and a more simple one by Hayem(1) consisting only of cooked semolina, the breakfast recommended by Ewald closely answers all requirements and is very easily manipulated. Bread contains proteids and carbohydrates, together with a certain amount of saline matter and these are quite sufficient to test the activity of the digestive juices of the stomach. Frequently, also, this meal may be given with impunity where a meal such as that of Leube would be followed by pain and perhaps, vomiting. It may be urged that an excess of carbohydrates is introduced into the stomach, but this does not prevent the secretion of the gastric juice nor diminish its activity as is shewn by the complete manner in which the albumin is turned into albumose and peptone.

The gastric contents having been filtered, the filtrate was examined for the substances noted alone.

1. Free Hydrochloric Acid. For the determination of free hydrochloric acid the test recommended by Gunsberg(2) in 1887 was used. It consists of

Phluroglucin 2 grammes
Vanilin 1 gramme
Alcohol 30 cubic centimetres

The chief value of this test lies in the fact (1) that it is extremely delicate (1-10,000) and (2) that it is not interfered with by any of the other stomach contents nor by mineral acids.

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2. Lactic and Butyric Acids. Uffelmann's reagent was used for showing the presence of these substances. It consists of a weak solution of carbolic acid and perchloride of iron giving a pale amethyst blue colour. On pouring a few drops of the filtrate into this, if lactic acid is present a bright canary yellow is produced, while butyric acid gives a metallic sheen. Lactic acid may be detected in quantity so small as 0.01%.

3. Peptone and Albumose. Fehling's solution was used as the test for these substances. A violet pink colour is given on the addition of a drop or two of the reagent to the filtrate if either of the substances is present. To distinguish between them it is necessary to precipitate the albumose by shaking the filtrate with ammonium sulphate until no more will dissolve and allowing it to stand for 24 hours (2). The fluid is then re-filtered and the test re-applied. It is necessary to have the reaction of the filtrate neutral or slightly alkaline for this test or the Fehling's solution simply turns green. This is best obviated by the addition of a drop or two of a 40% solution of caustic soda or potash (3).

4. Erythro-dextrin: A weak solution of iodine was used as the reagent for this substance, the fluid turning a dark brownish red if erythro-dextrin be present. The best method of performing the test is to place two or three cubic centimetres of the filtrate in a

(1) Quoted by Ewald loc. cit. p. 245
(3) Martin Sidney loc. cit. p. 138
test tube and add the iodine solution gradually, because if iodine be in excess the reaction cannot be obtained (1)

5. Sugar. The filtrate was boiled with Fehling's solution and the usual reddish yellow precipitate produced if sugar were present.

The digestive properties of the filtrate were then tested in the following way: Four test tubes were taken and labelled respectively A, B, C, and D and into each were placed half a dozen discs of white of egg.

To A was added 5 cc of the filtrate
To B 5 cc filtrate and 5 cc of a 2% solution of hydrochloric acid
To C 5 cc filtrate and 2 grains of pepsin
To D 5 cc of water, 5 cc of 2% solution hydrochloric acid and 2 grains of pepsin

The tubes were then placed in an incubator for 4 hours at a temperature of 39°C. The condition of the egg discs was then noted, the contents filtered and the filtrate tested for the presence of albumose and peptone.

The above methods of examination of the stomach contents have been used throughout the whole of my observations and I now intend to say a few words upon their clinical value.

(1) Ewald. loc. cit. p. 261
1st. Microscopical Examination: (see also p. 75.)

This is extremely important in some cases as shewing the presence of undigested food and of micro-organisms. After the test breakfast there are always numerous starch cells present in a more or less altered form. Sometimes they appear unaltered — at others they are much swollen and seem to have entirely lost their structure. In no case have they been absent. No relation could be detected between the state of the starch granules and the intensity of the erythro-dextrin and sugar reactions.

In no case of chlorosis were any remains of meat eaten the previous day found but in a case of extreme dilatation with hour-glass contraction in a case of simple pyloric obstruction altered muscle fibres were detected; and also in a case of diabetes mellitus with faulty digestion and a stomach reaching down to the umbilicus.

Out of 85 cases of all kinds examined, sarcinae were found only in two; one being in a case of chronic gastritis with considerably dilated stomach, in a woman aged 27 years whose gastric juice was fairly active, and the other the case of simple pyloric stricture above referred to. They were found in none of the anaemic cases nor in several cases of marked dilatation in males.

Yeast cells were found more frequently, in nine cases altogether, in five of which the stomach was distensible and in four of which it was not; while in 40 cases in which the lower limit was below
the level of the umbilicus were found. The presence of these organisms in the stomach must therefore be considered as entirely accidental, although shewing that the motor functions of the organ are deficient, as all the cases in which they were found shewed, either by the salol test or on washing out after a stated time, a marked delay in the passage of food through the pylorus (vide infra p.69.) A few red blood corpuscles may occasionally be seen, and, indeed, the fluid may be tinged with blood. This has generally occurred in cases in which the patient was suffering from other diseases and in elderly men. It is probably due to the rupture of some small dilated vessel about the cardiac end of the stomach or lower part of the oesophagus and although sufficiently alarming does not appear to be of any importance, except as shewing the presence of a varicosed condition of the vessels in those regions.

2. **Free Hydrochloric Acid**: The presence of free hydrochloric acid in the stomach contents depends upon three factors, (a) the length of time which has elapsed between the meal and its withdrawal; (b) the nature of the ingesta and (c) the state of the acid secreting apparatus in the stomach.

As regards (a) and (b) von Jaksch(1) found that in healthy children and adults the results were the same; the greatest quantity of effective hydrochloric acid was secreted with a milk diet alone.

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(1) Clinical Diagnosis: English Translation 2nd Ed. 1896. p.137
(0.1615%) a smaller quantity with a meat diet (0.1583%) and the least with carbohydrates (0.1102%). The maximum is generally attained in from one to three hours after a meal. With milk the increase is slow; it is more rapid with nitrogenous food, slowest, but with the greatest initial rapidity, with carbohydrates. He also found that when 200 grms of ham have been taken, there are in 100 cc of the gastric contents 0.0643 grm of HCl in thirty minutes, 0.1529 grm in 45 minutes, and 0.0992 grm in an hour.

Soltau Fenwick(1) speaking of infants says, that when fed upon a farinaceous diet the total acidity of the gastric contents is invariably diminished, and may not exceed half the normal. In many cases where a trial meal was composed of oatmeal gruel, the contents of the organ extracted at the end of an hour were found to be neutral or only faintly acid to litmus paper.

Therefore, in undertaking any series of observations, it is necessary, if they are to be of any comparative value, that the test-meal and time which is allowed to elapse before it is withdrawn should be identical in all. Even if no free hydrochloric acid be detected after the lapse of an hour or more it does not follow that it is being secreted, because it very readily combines with any bases which have been introduced with the ingesta and with the albumin and until the chemical affinities of all these have been as far as possible satisfied, it is evident that no acid will

remain in the free state. The absence of free hydrochloric acid however does denote that its secretion is slower than normal and this is always accompanied by slow digestion.

(C) The Condition of the Secreting Apparatus:
The physiology of the secretion of the hydrochloric acid of the gastric juice is not fully understood. It is secreted by the cells in the glands situated in the cardiac end of the stomach (1). The source is a matter of conjecture. Martin (2) following Maly states that it is formed from di-sodium hydrogen phosphate in the blood. This salt is decomposed by carbon dioxide producing sodium hydrogen carbonate and sodium dihydrogen phosphate, thus:

\[ Na_2HPO_4 + CO_2 + H_2O = NaHCO_3 + NaH_2PO_4 \]

and that the sodium dihydrogen phosphate reacts on sodium chloride forming free hydrochloric acid, thus:

\[ NaH_2PO_4 + NaCl = Na_2HPO_4 + HCl \]

Maly also suggested that it is formed by the action of lactic acid in the stomach contents acting upon sodium chloride, thus:

\[ 2NaCl + C_3H_6O_3 = NaCl + NaC_3O_3 + H_2O + C_3H_6O_3 + HC \]

This latter might undoubtedly occur if lactic acid were present in the stomach, but hydrochloric acid is frequently found in the absence of lactic or other acid (see cases 4, 5, 6 and 9. Appendix). Also the different parts of the acid secreting cells have different reactions (3), the deep parts being alkaline.

(1) Foster Text-book of Physiology, Pt II, 5th Ed. 1889, p. 419.
(2) loc. cit. p. 20
(3) ib.
and the superficial parts acid during the time of secretion, and there is no reason to suppose that lactic acid is absorbed by these cells. Sodium chloride is the most plentiful salt in the blood\(^1\) and it has been found that it is much diminished in the urine during digestion\(^2\) and therefore we may conclude that it has some relation with the formation of hydrochloric acid. But sodium chloride is an exceedingly stable compound, and it is not likely that it is immediately split up in the act of secretion. More probably some unstable and highly complex chlorine compound is formed in the blood and that from this the hydrochloric acid is formed. According to von Jaksch\(^3\) the quantity of sodium chloride in the blood is constant, whether much salt is taken in the food or not and, an even more important observation, Schenk\(^4\) found that in pneumonia, in which disease chlorides in the urine are much diminished, their quantity in the blood was practically unaltered. From this it appears, that any deficiency or excess in the quantity of hydrochloric acid in the gastric juice depends upon other factors than the amount of chlorides in the blood.

According to Hofmeister and Schutz's experiments on the innervation of the stomach quoted by Martin\(^5\) secretion or stimulation of the vagi or sympathetic nerves supplying the stomach produced little or no effect upon the secretion of the gastric juice. Therefore seeing that the secretion of hydrochloric acid does not depend upon the blood nor upon

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\(^1\) Kirke Text-book of Physiology. 12th Ed. 1888. p. 88
\(^2\) Päster loc. cit. p. 419
\(^3\) loc. cit. p. 78
\(^4\) Schenk Anatom-physiol. Untersuchungen. p. 10
\(^5\) loc. cit. p. 15
the external nerve supply of the stomach, we are driven to the conclusion that when this secretion deviates from the normal the cause must be looked for either in the glands themselves or in the nerve plexuses in the walls of the stomach.

Thus, when we find a case presenting a deficiency or an excess of hydrochloric acid one hour after a test meal, we may assume that there is some alteration of function of the secreting apparatus in the stomach itself. But upon this we are unable to found any definite diagnosis, for it is found that at one time no free hydrochloric acid is present and shortly after, it is present in large quantities. Thus, in the case of L.W. a female, aged 19 years, who was suffering from gastroduinia with slight atony of the stomach wall, there was no free hydrochloric acid on the day after admission, but a week later it was present in fair quantity without any other obvious change in the condition of the stomach than that the atony was less marked, as shewn by its increased ability to empty itself after a meal.

In a case of Lyon's quoted by Grandjean(1) the total acidity and the amount of HCl were noted daily for a week and marked differences were found from day to day.

(1) loc.cit.p.33
<table>
<thead>
<tr>
<th>Day</th>
<th>Total Acidity</th>
<th>HCl</th>
<th>p.mille</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st</td>
<td>4.7</td>
<td>3.7</td>
<td></td>
</tr>
<tr>
<td>2nd</td>
<td>3.67</td>
<td>Traces of lactic acid</td>
<td></td>
</tr>
<tr>
<td>3rd</td>
<td>2.25</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4th</td>
<td>3</td>
<td>No lactic acid</td>
<td></td>
</tr>
<tr>
<td>5th</td>
<td>3.646</td>
<td>HCl</td>
<td>2.9</td>
</tr>
<tr>
<td>6th</td>
<td>2.21</td>
<td>No lactic acid</td>
<td></td>
</tr>
<tr>
<td>7th</td>
<td>3.8647</td>
<td>HCl</td>
<td>3.127</td>
</tr>
</tbody>
</table>

Here the quantity of HCl varied from 3.7 to 2.9 per mille or over 20%. This was a case of hyper-acidity and it is evident that if the amount of HCl be small it may easily be completely absent on one day and give the phloro-glucin-vanillin reaction the next.

That the presence or absence of free hydrochloric acid depends upon the proper action of the gastric glands is also shewn by the observations of W. Soltau Fenwick(1). He found in a series of post mortems upon marasmic infants that there was dilatation of the stomach and varying degrees in inflammatory infiltration of the mucosa, leading to cirrhosis, in which the sub-mucosa and muscular coats participated; also degeneration and detachment of the epithelial cells covering the villi and later proliferation of the cells lining the glands, with eventual destruction.

of the whole of the glandular structures. A similar condition to this is also described as occurring in elderly people and called atrophy of the stomach by Samuel Penwick(1). W. S. Penwick(2) found that in the early stage of the disease there was no free HCl an hour and a half after a test meal of milk and water, and the quantity of combined acid reckoned as HCl varied from 0.094 per cent to 0.135 per cent. At a rather later stage the reaction was still acid, no free HCl was present, and the combined HCl seldom exceeded 0.026 per cent. In advanced cases, the reaction was scarcely acid at all, and the amount of combined HCl too insignificant to estimate. Thus, we see that the amount of free hydrochloric acid varies pari passu with the destruction of the epithelium lining the villi and crypts of the stomach.

From the foregoing facts it is clear that before any deductions can be drawn from the presence or absence of free hydrochloric acid in the stomach contents, it is necessary to note the quality and amount of the test meal and the time which has elapsed between its ingestion and withdrawal, and at the same time to remember that variations may occur from day to day in the same individual without any apparent cause. The presence or absence of free hydrochloric acid cannot be said to occur invariably in any of the diseases of the stomach except the atrophy just referred to.

The following table shews the results of my own observations upon the presence or absence of

(1) Samuel Penwick, "On Atrophy of the Stomach" 1880 pp. 2-5
(2) loc. cit. p. 832
free hydrochloric acid in various abnormal conditions

<table>
<thead>
<tr>
<th>Disease</th>
<th>Free HCl present</th>
<th>Free HCl absent</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic Gastritis</td>
<td>15</td>
<td>11</td>
<td>26</td>
</tr>
<tr>
<td>Dyspepsia with Anaemia</td>
<td>8</td>
<td>5</td>
<td>13</td>
</tr>
<tr>
<td>Hysterical &amp; Neurasthenic conditions</td>
<td>9</td>
<td>0</td>
<td>9</td>
</tr>
<tr>
<td>Gastroodynia</td>
<td>4</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Atonic Dyspepsia</td>
<td>1</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Dilatation of Stomach</td>
<td>1</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>Ulcer of Stomach</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

In addition to these, observations were made in several cases in which there were no, or only very slight, stomach symptoms. Free HCl was present in two cases of diabetes mellitus and absent in four. It was present in one case of chronic nephritis and absent in two of acute nephritis. It was present in a case of carcinoma hepatitis and one of Grave's disease; while it was absent in cases of leucocythaemia, Hodgkin's disease and cirrhosis hepatitis.

In one case of phthisis, with well marked symptoms of gastritis, it was present, and in another it was absent.

3. Lactic and Butyric Acids: The formation of lactic and butyric acids in the stomach is due to
Fermentations arising from articles of food or from the decomposition of their alkaline or other salts (1) (Foster). Starch is converted into sugar and from this lactic acid is formed by the bacillus acidi lactici. At the same time a large amount of CO₂ is given off (Martin) (2). Lactic acid in its turn, is converted into Butyric acid by the bacillus butyricus. This bacillus has also the power of directly converting carbohydrates into butyric acid, with the evolution of methane, carbonic acid and sulphuretted hydrogen (Martin) (3). Decomposition of the carbohydrates is, however, not necessary for the presence of lactic acid in bread digestion, as lactic acid is formed in the process of making the bread (Martin (4)). If however it is found with a purely milk diet, fermentation of lactose must have taken place.

Ewald and Boas (5) found in healthy subjects that after a meal of 60 grms of white bread, lactic acid appeared in the stomach contents in ten minutes, that the quantity increased for 30 to 40 minutes and that after that time it decreased and hydrochloric acid was also found and after about one hour and three quarters the lactic acid entirely disappeared.

The disappearance of the lactic acid is due either to absorption of which no evidence has been adduced or to combination with salts e.g. chlorides, and the formation of lactates.

The same remarks therefore apply to lactic

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(1) loc. cit. p.365
(2) loc. cit. p.73
(3) Ib.
(4) Ib p.32
acid as to hydrochloric acid when considering the clinical value of the test; namely, that the kind of food must be taken into account and the time which it has remained in the stomach.

Accepting the statements of Ewald and Boas as correct, we may say that, if there is a marked reaction with Uffelmann's reagent at the end of an hour, there is an abnormal production of lactic acid and that this is due to fermentation of the carbohydrates in the test meal which has been prolonged beyond the normal time. A moderate amount however is compatible with a healthy condition of the stomach. In my own cases I found that there was hydrochloric acid only in eleven, lactic only in thirty-five, both hydrochloric and lactic acids in thirty-one and no reaction either with Gunsberg's or Uffelmann's reagents in six. It was also noticed that when both the reactions occurred in the same specimen, as a rule if the reaction of hydrochloric acid was very brilliant that of lactic acid was correspondingly faint, thus shewing that with an increase of hydrochloric acid the formation of lactic acid is inhibited.

Among the thirty-five cases in which lactic acid occurred alone twelve were in cases of chronic gastritis, six in cases of anaemia with dyspepsia, four each in cases of dilated stomach atonic dyspepsia and diabetes mellitus; and one each in gastric ulcer, gastrodynia, acute and chronic nephritis and Graves disease. Although these conditions are so very
different one common factor was found in all but five of them, namely, a marked distensibility or dilatation of the stomach and three of these occurred in cases of diabetes mellitus, in which perhaps there may be some alteration in the carbohydrate digestion as part of the disease, separately from any abnormal gastric condition. In eight cases however in which there was some dilatation, there was no lactic acid found. Hence we find that the presence of an excess of, or the presence alone of lactic acid points to some degree of gastric dilatation.

Butyric acid is not normally found in the gastric contents. It is found when there is marked stasis of food in the stomach from whatever cause and is generally associated with an absence of free hydrochloric acid, the immediate cause being fermentation of carbohydrates by the bacillus butyricus (vide supra). It is perhaps most commonly met with along with lactic and acetic acids in cases of cancer but unfortunately I have only had one such case under my care while studying this subject. I have only detected this acid in five cases, in four of these no free hydrochloric acid was present, and in one there was excessive dilatation of the stomach.

4. Peptone and Albumose: The importance of testing for these bodies is to determine the digestive value of the gastric juice. The quantity present may be roughly determined by noting the brilliancy of.

(1) Martin. loc. cit. p. 479
the reaction with Fehling's solution. If there is no albumose present at the end of an hour, we may state that the proteid digestion is exceedingly slow and I have only found this in four cases, one being in a case of Hodgkin's disease, another in a severe case of Grave's disease, a third in a case of atonic dyspepsia, while the fourth was in a case of diabetes mellitus, another case of the same disease giving only a very faint reaction.

Peptone was found absent in six cases of dilated stomach, in five cases of atonic dyspepsia; it was also absent in five cases of diabetes mellitus and in very small quantity in a sixth. It was also absent in three cases of gastric ulcer. Out of thirteen cases of anaemia with dyspepsia it was absent in nine and present in small quantities in four. Out of twenty-five of chronic gastritis and dyspepsia, peptone was present in fifteen and absent in twelve. Out of thirteen cases of neurasthenia and nervous stomach troubles peptone was present in seven. Peptone was absent from cases of leucocytthemia, Hodgkin's disease and Grave's disease.

From the foregoing figures we see that the digestion of proteids is most affected in those cases in which there is a general lowering of the vitality of the body; for example, diabetes mellitus anaemia and atonic dyspepsia, and that marked organic lesions of the stomach, such as gastric ulcer and dilatation have the same effect, while simple dyspepsias and purely nervous affections are not so apt to produce
any change in the proteid digestion.

5. **Erythro-dextrin and Sugar**: The starch introduced into the stomach with the food is acted upon by the amylolytic ferment in the saliva and is converted first into dextrin (erythro-dextrin and achro-dextrin) and then into sugar\(^1\) A neutral or slightly alkaline medium is necessary for this action to take place, and a very small quantity of free acid, especially of free hydrochloric acid will absolutely put a stop to it\(^2\) Therefore, as free hydrochloric acid is present in the stomach after about half an hour no more starch is converted after that time. If however the acidity of the stomach contents is much diminished, the action may go on for a longer period. Von Jaksch\(^3\) states that there should be neither starch nor erythro-dextrin in the filtered juice an hour after a meal, and that if it be otherwise some cause tending to delay the amylolytic process may be inferred, either a deficient quantity of diastase in the saliva or an excessive acidity of the gastric contents early in digestion. In my observations however I have always been able to demonstrate the presence of erythro-dextrin except in six cases, and in none of these was free hydrochloric acid present. This fact fully bears out von Jaksch's statement as sugar was present at the same time shewing that the amylolytic action had taken place. In the cases in which erythro-dextrin was found, free hydrochloric acid was as often absent as present, and

\(^{(1)}\) Poster, loc.cit.p.360
\(^{(2)}\) Ib p.361
\(^{(3)}\) loc.cit. p.142
we must therefore infer that a diminution in the diastase of the saliva is not an uncommon condition.

Sugar was found in all cases but two and in these there was a marked excess of free hydrochloric acid.

As to the value of the digestive experiments with egg albumin not much need be said. As a rule those cases in which free hydrochloric acid was absent digested much better when \(0.02\%\) HCl was added to the filtrate while when pepsin only was added no difference could be detected, thus shewing that in the majority of cases the pepsin in the gastric juice is active and in sufficient quantity, but that when digestion is deficient, it is the hydrochloric acid which is at fault. In a case of Hodgkin's disease, however, in a man aged 34, in which there was some dilatation of the stomach and exceedingly feeble digestion while the addition of HCl produced no increase in the digestive power, when pepsin was added the albumin was fairly well turned into albumose but no peptone was formed.

**General Value of the Chemical Examination of the Stomach Contents.**

By carrying out an investigation into the chemistry of the stomach in any particular case, in the method described above, we are able to say: 1st, whether digestion is being carried out normally or not.
2nd, whether the cause of any irregularity is in the gastric secretion or not.

3rd which factor in the chemistry of digestion is at fault.

This however is only true for the time at which the observation is made; for we have seen already (p. 53.) that in the same case one or more of the constituents of the gastric juice may be absent at one time and present at another and that digestion may have gone further in the same time at one period than at another.

Again, when we have ascertained the above facts, and have shewn that there is some defect in the gastric chemistry, we are able to found no basis for treatment upon them, for it seems probable that the actual digestion which goes on in the stomach is not of such paramount importance as is generally supposed. In support of this statement I may quote Ogata's experiments upon dogs, in which he fed them with minced meat and beaten up egg through a fistula in the duodenum and found that the nutrition was unimpaired, and again van Noorden who found that patients were as well nourished with complete suppression of hydrochloric acid as those whose gastric chemistry was normal. Moritz conducted numerous experiments on dogs with duodenal fistulae, and from these he concludes that "the effect of the stomach digestion for a great part of the food, is simply to convert it into

(1) Ogata. Du Bois Raymond's Arch.1883.p.89.
"a smooth-like fluid and so to facilitate its passage "through the pylorus. For example, when milk is "swallowed it at first curdles, and only the whey "passes; but later the curd follows through the "pylorus taking the shape of little sausages; while "out of 200 grammes of minced raw meat, 58% passed into "the intestine undissolved in the form of little soft "fragments. The passage of chyme commences after a "lapse of three quarters of an hour.

From the above facts it appears that even when the gastric digestion is markedly abnormal no morbid symptoms need be produced and that when we find divergences from the normal chemistry we cannot found any diagnosis upon them alone. Nevertheless, an investigation into the activity and secretion of the gastric juice should not be omitted as it may throw considerable light upon the general state of the patients metabolism.

Having disposed of the chemistry of the stomach it is necessary especially in the light of the facts last mentioned, to make further investigations into the state and functions of the organ. The points to be investigated are,

1st. The absorptive power
2nd. The muscular activity
3rd. the shape position and size

1st. The Absorptive Power. Landois and Stirling[1]

state that salts, grape sugar, peptones, poisons,

and in a still higher degree alcoholic solutions of poisons are absorbed in the stomach. They classify the stomach however following Beaunis, as coming after the large intestine in absorptive power. Tappeiner however found that very little water was absorbed by the stomach and von Mering found that the whole quantity of water which was given to dogs to drink was expelled through the pylorus in gushes. Brand found that only 5% of 150 grms of a 10% solution of peptone was absorbed after two hours; while solutions of grape sugar and potassium iodide behaved in the same way. Von Mering and Moritz found that out of 44.5 grms of meat albumen ingested 41.5 grms passed through the pylorus; and of 11.6 grms of milk albumen 13.5 grms passed, the excess due being to admixture with gastric juice. These facts show that in health or at least in conditions as nearly approaching health as the formation of fistulae permit the absorptive power of the stomach for the products of digestion is very small and therefore any diminution of the function is not of any great importance, except perhaps in the case of sugars which are liable to undergo fermentation (see p. 57). On this account and also because of the objections to the

(2) Ueber die Functionen des Magens. Verhandl.des xii Congress f.innere Med.zu Wiesbaden 1893.Quoted by Martin.loc.cit.p.27
(3) Quoted by Moritz.loc.cit.p.112
(4) Ib id
method employed in determining the rate of the absorption by the stomach, I have not made any observations upon this point. Zweifel in investigating the absorptive power of the stomach gave 0.2 grms of iodide of potassium in a gelatine capsule 100 cc of water being drunk at the same time. He then tested the saliva for the presence of the salt from time to time. The objections which may be made to this method are the following:

1st. The gelatine capsule may not be easily dissolved. This depends partly on the activity of the gastric juice, which forms from it a gelatin-peptone therefore the appearance of the iodide in the saliva depends large upon the activity of the gastric juice, because the more active it is, the sooner will the gelatine capsule be dissolved, and the sooner will the potassium iodide come into contact with the wall of the stomach.

2nd. The appearance of the iodide in the saliva also in part depends upon the excretory power of the salivary glands. If this is diminished it is evident that the appearance of iodide will be delayed.

2nd. The Muscular Activity of the Stomach. is perhaps its most important function and one which is very often affected in disease as I hope to be able to demonstrate below.

(2) Landois & Stirling loc.cit.p.299
The nerves supplying the stomach are:

a. The vagi
b. The sympathetic branches from the solar plexus.

Along the course of these nerves are small ganglia forming plexuses, one between the longitudinal and circular muscle fibres (Auerbach's plexus) and the other in the sub-mucous coat (Meissner's plexus).

Openchowski also describes certain automatic ganglia which are to be distinguished from the above. They are represented by definite centres in the brain and preside over the cardia, the pylorus and the body of the stomach respectively. These centres are closely allied to one another and in a peculiar manner for stimulation of the centre for opening the cardiac orifice closes the pylorus. Hoffmeister and Schutz found that section of the vagi produced momentary contraction of the pyloric and cardiac orifices diminution of the ordinary movements and pallor of the gastric mucous membrane but no inhibition of gastric secretion. Stimulation of the gastric end of the cut vagus produced slight movements of the stomach after a time and stimulation of the sympathetic produced no effect but that when the stomach was removed and kept under appropriate conditions it shewed regular automatic contractions.

From these facts we see that Auerbach's plexus and the automatic ganglia are chiefly concerned.

(1) Landois & Stirling loc.cit.p.213
(2) loc.cit.p.274
(3) Martin loc.cit.p.15
in governing the muscles of the stomach.

The usual description of the movements of the stomach is that they are of two kinds circulatory and churning (Martin (1) or rotatory and peristaltic (Landois and Stirling (2)) The first mixing the food and gastric juice and the second driving it on towards and through the pylorus.

More recent experiments (3) have shown that the process is rather more complicated than this. An oesophageal sound was introduced into the stomach and attached to a manometer. It was found that no positive pressure was exerted by movements of the stomach in the fundus, but when an elastic ball was introduced through the pylorus into the pyloric portion of the stomach very distinct evidences of pressure were found corresponding to the periodic discharge of fluid through the orifice. These experiments which were performed upon dogs were afterwards confirmed in men, and shew that the pyloric portion of the stomach is the active part and that the fundus acts merely as a reservoir. The presence of a sphincter antri pylori is described by Hoffmeister and Schutz as cutting off the antrum pylori from the rest of the stomach. These facts appear to offer an explanation of the fact that only food which has been properly broken up by the stomach is allowed to pass through the pylorus. We may suppose that the sphincter antri pylori to a certain extent constricts the stomach at the opening of the antrum, and indeed this is very

(1) Ib p.19
(2) Ib p.273
(3) Moritz loc.cit.p.113 et seq.
frequently seen at post mortem examinations especially in young subjects. This being so there will be formed on either side of the ring so produced a fossa capable of being filled with the ingesta. As the food becomes more and more turned into chyme its specific gravity will fall and the solids will gravitate into the fossa on the proximal side, the fluid portion overflowing the constriction into the pyloric fossa. When there is sufficient fluid here, the pressure on the mucous membrane will be increased and this will cause a reflex or perhaps direct (by affecting the plexus of Auerbach) contraction of the sphincter antrii and the pyloric portion of the stomach, which will, press the contents through the pyloric opening. The contraction of the sphincter antrii preventing the regurgitation of the fluid into the stomach. This will also explain the great delay which at times occurs in dilated stomachs without pyloric stricture, for the sphincter not being able to shut off the antrum the fluid regurgitates into the stomach when the pyloric portion contracts.

No entirely satisfactory clinical method has as yet been devised for estimating the motor power of the stomach. Huber(1) administered 15 grains of salol in gelatin capsule immediately after a meal and noted the time which elapsed before any reaction with ferric chloride appeared in the urine. The test depends upon the fact that salol in the presence of an alkaline breaks up into phenol and salicylic acid but

(1) Landois & Stirling loc.cit.p 273
that it remains stable in the presence of an acid. The contents of the stomach after a meal quickly becoming acid the splitting cannot take place there, but when the salol reaches the duodenum and meets with the pancreatic and hepatic secretions it reaches an alkaline medium and splits up. The products become absorbed into the blood and are excreted in sulphuric acid combinations with the urine where they give a deep purple coloration on the addition of a solution of ferric chloride. Huber found that in health the reaction began in 30 to 60 minutes and disappeared in 24 hours; while in motor insufficiency it did not appear for 3 hours and disappeared up to 24 hours later. Ewald^{(2)} as the result of a series of experiments gives the time for the appearance of the reaction in health as 75 minutes.

The theoretical objections to this method are similar to those urged against the potassium iodide test for the absorption from the stomach. Namely, that the rate of solution of the capsule may vary and that other factors enter into the process. For if from any cause the alkalinity of the food in the duodenum be lowered, it is evident that the salol will not be so readily split up and if the epithelium of the villi be damaged or diseased the absorption will be slower. Again, the state of the kidney must be taken into account, for if the secretion of urine be small, it will take longer for the salol derivations to be excreted.

(1) Von Jaksch loc.cit.p.344
(2) Ewald loc.cit.p.264
The clinical results from this method are also not of much value. Thus, out of 106 cases examined, suffering from all diseases, the reaction was found in 82 after 75 minutes. In these there were digestive symptoms which would lead one to expect that motor insufficiency would be present in 13 or 15.8 per cent, while in 69 or 84.2 per cent there were no such symptoms. Again out of 24 cases there was no reaction obtainable after 75 minutes only 3 or 12.5 per cent exhibited such symptoms, while 87.5 per cent were apparently normal. Again out of six cases which were proved to have their stomachs reaching well below the umbilicus, the reaction was present in four at the end of an hour and a quarter, while in two it was delayed in one for two and a half hours and in the other for four and a quarter hours.

From these figures we see that the proportion of cases in which stomach troubles such as to lead us to expect some atony of the stomach, is practically the same whether the salol be excreted in what is considered the normal time or not. And again out of six cases in which there was marked gastric distensibility and therefore we may infer motor insufficiency the appearance of the salol was only delayed in two.

When we look at the disappearance of the reaction, however, we find rather more definite results. Thus, out of 106 cases the reaction persisted for more than 24 hours in 44 the longest time being 72 hours in a case of gastric carcinoma in which there was no digestion in the stomach; gastric symptoms were present
in 12 cases or 27.7 per cent while there were no such symptoms in 32 cases or 72.3 per cent. On the other hand there was no delay in 62 cases and in these were gastric symptoms in 4 or 6.4 per cent only; while there were 58 cases or 93.6 per cent with no such symptoms. In the six cases above referred to the reaction persisted in all beyond the normal time, for periods varying from 6 to 48 hours.

The following table shews the above results in tabular form: First taking the 106 cases.

<table>
<thead>
<tr>
<th>No of Cases</th>
<th>Delayed appearance of Reaction</th>
<th>Reaction present in 75 minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>24 = 22%</td>
<td>82 = 79.4%</td>
</tr>
<tr>
<td>With Digestive Symptoms</td>
<td>3 = 12.5%</td>
<td>13 = 15.8%</td>
</tr>
<tr>
<td>Without Digestive &quot;</td>
<td>21 = 87.5%</td>
<td>69 = 84.2%</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>No of Cases</th>
<th>Delayed Disappearance of Reaction</th>
<th>Reaction Disappeared in 24 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>44 = 41.5%</td>
<td>62 = 58.5%</td>
</tr>
<tr>
<td>With Digestive Symptoms</td>
<td>12 = 27.7%</td>
<td>4 = 6.4%</td>
</tr>
<tr>
<td>Without do do</td>
<td>32 = 72.3%</td>
<td>58 = 93.6%</td>
</tr>
</tbody>
</table>
Thus, we find that while in cases of marked dilatation of the stomach there is considerable delay in the total excretion of the salol, a similar delay may occur in apparently healthy conditions of the stomach, and that in many cases in which we should expect to find a motor insufficiency the reaction does not persist after 24 hours. The results of this test cannot therefore be considered sufficiently uniform to be of much value in diagnosis.

Another method is that of Klemperer\(^{(1)}\) who injected 100 grams of olive oil into the stomach through a tube and removed the remainder by washing with water and aspiration after 2 hours. The oil present in the washings he dissolved in ether, evaporated the ether and weighed. Seventy to eighty grams of the oil were found to have been discharged from the stomach in healthy people.

The objections to this method are the following:

1st, It is very troublesome to the patient in the majority of cases to have the stomach tube passed and the double operation increases the discomfort.

2nd, The chemical processes to which it is necessary to subject the washings are too long and difficult for ordinary clinical work.

3rd, Olive oil is not an ordinary article of diet at any rate in this country, and the stomach would probably be stimulated either to increased movements or vomiting might be produced in irritable conditions. Either of these accidents would render

\(^{(1)}\) Deutsche Med. Woch. Leipzig 1888 vol. 47. Quoted by Martin loc. cit. p. 152
the results useless and misleading.

I have had no personal experience of this method, but the above objections appear to be sufficient to condemn it for ordinary clinical work, though with plenty of time and apparatus at one's disposal, it would undoubtedly be of great service in the investigation of disease.

A somewhat similar proceeding has been devised by Matthieu and Hallot(1) in which the following emulsion is administered with Ewald's test breakfast. The gastric juice is removed and the quantity of oil determined as before.

The emulsion consists of:

Sweet Almond Oil 10 grammes
Powdered Gum Arabic 5 "
Simple Syrup 5 "
Weak tea to make the quantity up to 250 cc

This removes the third and first objections to Klempcerer's method and is said not to affect the secretion of the gastric juice but the difficult manipulation of the washings remains.

The third method and the one which appears to me to be the most free from objections is based upon the fact that normally the stomach should empty itself completely of food in a given time. Martin(2) found that the stomach was completely empty in seven hours after Leube's test meal of soup beefsteak and bread. Kirke (3) states that under ordinary circumstances

(1) Quoted by Grandjean loc.cit.p.19
(2) loc.cit.p.150
(3) loc.cit.p.291
from 3 to 4 hours may be taken as the average time occupied by the digestion of a meal in the stomach and Foster (1) says that in man the stomach probably becomes empty between the usual meals. Bouchard (2) states that the presence of food in the stomach 5 hours after digestion is pathological.

The method which I have employed has been to give the patient a pint of bread and milk at 9 p.m. and to wash out the stomach at 9 a.m. the next day. The washings are allowed to stand for a few hours, a drop of the precipitate placed upon a slide with a little weak iodine solution and starch cells looked for.

In cases in which the stomach functions are normal the washings from the fasting stomach are slightly opalescent and contain a few flakes of mucus which when stained with iodine and examined under the microscope are seen to consist of mucous shreds in which are imbedded numerous small round cells with occasionally an epithelial cell from the gastric mucous membrane. There are also usually seen more or less numerous squamous epithelial cells from the mouth and oesophagus which have been swallowed and which may be single or in groups of from 2 or 3 up to 12. Occasionally the fluid is a little bile-stained but no starch cells should be found. Out of 30 cases examined by this method all of which presented fairly severe gastric

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(1) loc. cit. p. 472
(2) Lectures on Intoxication in Disease p. 184.
symptoms starch cells were found in 19 and of these 16 or 84.2 per cent had considerable distensibility of the stomach. In 3 cases in which this amounted to pretty severe dilatation the number of starch cells were markedly increased, while in others in which the lower curvature only reached to the umbilicus there were very few.

Of the 11 cases in which there were no starch granules found, seven or 64.5 per cent had some distensibility of the stomach, but in only one of these was it very marked, and in another it appeared to have been depressed without being unduly distensible.

These figures are not very conclusive, but it must be kept in mind that distensibility of the stomach does not necessarily mean dilatation and that when that organ is not overfull the normal movements can take place even if it be distensible; and again there may be delay in the passage of food through the pylorus without any marked distensibility of the organ.

A fallacy may also enter into this method, namely, the early or late acidity of the stomach contents. If the secretion of hydrochloric acid be much delayed, the digestion of the starch will go on much farther than if the contents rapidly became acid; and that although the motility of the stomach be really impaired no starch cells may be found in the fasting stomach if there be an initial deficiency of hydrochloric acid secretion. This will account for the absence of starch in the case of dilatation mentioned above, and also for 4 of the other cases in
which there was a slight distensibility of the stomach but no starch found. Thus, removing these 5 cases, we only have 2 or 18.1 per cent left in which there was distensibility but no marked delay. Therefore taking into consideration the presence or absence of acid in the early stages of digestion we have a fairly exact test for the motility of the stomach which can easily be applied at the bedside.

3rd, The Shape, Position and Size of the Stomach.

The methods which I have employed to demonstrate these points have been 2 in number. In both, the method depends upon the inflation of the stomach by gas, in the one case air being pumped through a stomach tube by means of a Higginson's syringe and in the other carbonic acid gas being generated in the organ by the administration of two powders consisting respectively of 130 grains of bi-carbonate of soda and of 100 grains of tartaric acid. The first method recommended by Runeberg(1) and Oser(2) who however used a spray producer instead of a syringe, is in my opinion the more useful and the safer for the following reasons: Firstly, the rate at which the gas is introduced into the stomach can be regulated exactly. Secondly, the quantity of gas can be regulated and the introduction stopped at any time that it may become necessary e.g., if much pain is caused. Thirdly, the gas can be removed as soon as the required facts have been

(2) Oser. Die Neurosen des Magens. Wien.1886 p.10
ascertained. Fourthly, if from any cause the gas escapes from the stomach too soon re-inflation can easily be practised. As this examination is made as a rule immediately after the removal of a test breakfast, no special introduction of the stomach tube was necessary. I was led to use the syringe instead of the spray producer because air could be introduced more rapidly and the syringe does not get out of order so easily as the spray. My objections to the method of forming carbonic acid in the stomach are as follows:

In the first place this method is objectionable because the full quantity of gas is generated almost immediately, and is said to cause spasm of the cardia(1). I have, however, never met with this complication, vomiting usually occurring to the discomfort of the patient and often of the investigator. Secondly the quantity of gas evolved cannot be regulated. Thus, in a small stomach too much is produced with over-distension, discomfort and pain and at times passage of the gas through the pylorus into the duodenum. On the other hand the amount of gas may be too little and the organ not fully distended. Thirdly, the gas can only be removed by eructation or by passage through the pylorus thus causing increased discomfort to the patient. I have never seen any dangerous symptoms follow the use of this method but alarming symptoms have been noticed by Saundby and others.

I may here say that the usual quantities of

(1) Ewald.loc.cit.p.269
the powders used is very much less than I have tried; thus Martin(1) recommends from 15 to 30 grains of each. The larger quantities are those used by Meinert.

The chief advantage of this method is that it can be used without passing the stomach tube in cases in which it is not desired to give a test meal.

The stomach having been distended in either of the above ways, the limits of air determined by palpation and percussion and marked out the result giving the exact shape size and position of the stomach.

These details are very important especially taken with the motility of the stomach. Very frequently we find there is a delay in the passage of food and that the stomach may be distended below the umbilicus. This is specially marked in the case of anaemic girls and shows that the muscle of the stomach has lost its tone. Again, the stomach may be found dilated without any delay. In these the stomach is large and the muscular power is good. If we find that there is neither distensibility nor delay, we may conclude that any dyspeptic symptoms are from the side of the secretions or of neuralgic origin. As a rule however in cases which appear to be pure neuralgias I have found atony of the stomach wall.

In some cases the distension takes peculiar forms either that it is more or less irregular or the upper limit is depressed as well as the lower. In

(1) loc.cit.p.387
the first class we have the well known hour glass contraction in which there is a constriction in one part of the stomach with dilatation on either side. In a case of simple stricture pylorus this was very clearly shewn and diagnosed, the constriction apparently occurring at the position of the muscleus antri pylori and being caused by a fibroid thickening of its fibres as no other cause could be found in the post mortem room. In the second class we find two sets of cases, those in which the whole stomach appears to be lowered and 2nd, those in which the pylorus has remained nearly in its normal position but in which there was a sagging of the upper curvature. The differentiation between these two classes is important. The first in which the pylorus has descended is generally found in anaemic girls and constitutes gastroptosis (vide p.37.) The pyloric portion is usually very distensible and descends lower than the rest of the larger curvature. It may or may not be accompanied by true dilatation. The second of the above classes is always accompanied by true dilatation; it does not shew any marked distensibility of the pyloric portion and generally occurs in more elderly patients.

This falling down of the stomach or gastroptosis may be part of a general displacement of the abdominal organs (enteroptosis) or it may occur alone. Enteroptosis was fully described by Glenard of Lyons

(1) Lyon Medical March 1886.
in 1885 and again in 1887 (1) Eereol in the same year read a report on the condition before the Medical Society of the Paris Hospitals. Glenard considers that the first portion of the abdominal viscera to be affected is the hepatic flexure of the colon which pulls upon the pyloric portion of the stomach, the transverse colon then pulls upon the greater curvature further to the left and the splenic flexure upon the spleen. The pulling down of the pyloric end of the stomach, the pylorus itself remaining fixed causes a kink to occur at the first part of the duodenum and this produces a certain amount of stricture with delay in the passage of food. The small intestine passes down into the pelvis and the kidneys and liver also participate. The causes of this condition are enumerated by Pourcelot (3) and he mentions the following: sedentary mode of life especially with mental work, bad alimentary hygiene, pregnancy falls blows, muscular efforts and the abuse of the corset. He also states that any lesion capable of causing a stenosis of the intestine such as typhoid fever or dysentery may bring it on. He states that Glenard found the condition in 400 out of 1300 cases which he examined.

This condition must be distinguished from a single gastroptosis such as occurs in anaemic girls in

(1) Enteroptose et Neurasthenie. Revue de Medecin Jan. 10th 1887; and Expose sommaire du traitement de l'enteroptose. Lyon Medical 19 June 1887.
(3) De l'enteroptose. These Paris 1889 p. 19
which as a rule the colon is not displaced although the right kidney often (1) and also from cases in which there is simple dilatation of the stomach such as occurs in large eaters and drinkers. The case of W.D. aet. 69, a butler, who had always lived well and eaten largely illustrates this class of case. The smaller curvature of his stomach in the middle line was only 2 inches above the umbilicus, while the greater curvature reached well below the anterior superior iliac spines, the pylorus being very little depressed and there being no evidence of any falling down of the liver, spleen or kidneys.

This condition is much more frequent in women than in men, thus out of 37 women the lesser curvature was found to be lower in position in 25 (or 67.5 p.c.) while out of 35 men only 10 (or 28.5 per cent) shewed the same abnormality. The average age of the 25 women was 26\(\frac{1}{2}\) years and that of the men 44 years. It was found in no man below the age of 30. It was most frequently found in cases of anaemia (chlorosis Addison's disease) 11 cases and in neurotic and atonic states 11 cases also in 3 cases of simple dilated stomach the other cases being made up of chronic gastritis, phthisis and diabetes mellitus.

GENERAL CONCLUSIONS

Data obtained from chemical investigations of the gastric functions alone are not sufficient to enable us to form a diagnosis, but they are valuable

(1) Meinert loc.cit.p.214
in that they tell us the condition of the gastric secretions for the time being.

In making any chemical investigation it is necessary to note the exact conditions under which the test meal has been administered and withdrawn. The presence or absence of free hydrochloric acid at the end of one hour is an important factor in the stomach digestion but that the presence of lactic acid is of less importance.

The presence of albumose at the end of an hour shews grave delay in digestion, but the absence of peptone may be overlooked.

The tests for the absorptive power of the stomach are unreliable.

The salol test for the motility of the stomach is unreliable but "washing out" test gives satisfactory results.

Runenberg and Oser's method for determining the shape size and position of the stomach is better than the carbonic acid method and the results obtained from the former are reliable.

When the whole of the examination of the gastric functions is completed we are able to form a diagnosis of the case.

TREATMENT

The treatment of dyspepsia with chlorosis may be divided into two parts. Firstly preventive, and
secondly curative.

1st. Preventive. Having previously stated that the theory, which gives to pressure on the lower part of the thorax a leading role in the causation of chlorosis and dyspepsia, appears to most nearly touch the truth, it follows that in aiming at the prevention of these diseases we must forbid the use of such things as are likely to cause such pressure. Among these may be mentioned the use of the corset and the practice of fixing the petticoats, etc by a tight band round the artificial waist situated over the false ribs. If the thorax be already of such a shape as to lessen the lower opening of the thorax, that is to say, long and narrow, we must take means to remedy this defect. This may be brought about by calesthenic exercises and athletics generally which, by causing a greater development of the muscles of the trunk and upper extremities, tend to expand the thorax. Fresh air, good food containing plenty of iron and freedom from over work mental or physical about the time of puberty and at the menstrual periods should also be insisted upon.

2nd. The condition having been set up, we have to treat the gastroptosis, the anaemia and the dyspepsia. In the treatment of the gastroptosis it is very important to see that the pressure upon the lower part of the thorax is not kept up. All corsets and constricting bands must be removed. This is best accomplished by keeping the patient in bed. It is
not easy to do this with ordinary patients but where they can be prevailed upon to remain at rest for 3 or 4 weeks the stomach undoubtedly returns somewhat to its normal position. By keeping the patient in bed, we also prevent the force of gravity pulling down the stomach and stretching the fibres of the sympathetic in the neighbourhood.

These stomachs which are in the position of gastroptosis never recover entirely if the condition has been present for any length of time but Meinert states (1) that they may if they are treated early enough.

The treatment of the chlorosis and dyspepsia runs side by side. The indications are: rest in bed as above, the exhibition of iron, regulation of the bowels and diet.

I have carried out these indications in the following way with I think complete success. A mixture containing sulphate of iron 2 grains and sulphate of magnesium 40 grains in an ounce of water, 3 times a day. If the sulphate of magnesium was insufficient to keep the bowels open more was added till there were one or two dejections daily. The sulphate of iron was assisted in its action by the administration of 2 or more Blaud's pills 3 times a day, or of Oppenheimer's bi-palatinoids of ferrous carbonate. The diet at first consisted of small quantities of milk and barley water, say, one ounce every hour.

(1) loc.cit.p.228
gradually increasing as the patient could take it without pain up to 5 ounces. After this, small quantities of bread and milk were given, and then milk puddings, minced chicken or mutton, until at the end of about 3 weeks the full Hospital diet consisting of beef, mutton or fish with potato, bread, pudding and tea was reached. If the ingestion of this diet were unattended by pain the patient was allowed to get up after a few days generally in from 3 to 4 weeks of the commencement of the treatment. She was encouraged to assist in the lighter work of the ward and if this were unattended with ill effect she was discharged with the iron and magnesia prescription and the Blaud's pills.

After a course of this treatment, I have only known one case recur and in that some of the typical signs of chlorosis were wanting, and the recurrence took place after an attack of scarlatina.
APPENDIX

CASES of CHLOROSIS giving their digestive Symptoms and the Results of the Physical and Chemical Examinations of their Stomach Functions.


Test Breakfast:
Torulae present in the washings
Acid reaction of filtrate
Free HCl Absent
Lactic Acid
Albumoses
Erythrodextrin Present
Small quantity of Sugar
Peptones Absent

Motor Functions:
Salol Test
No reaction for 75 minutes
Reaction present in 24 hours
Persisted for 30 hours

The addition of free HCl to the filtrate did not cause peptones to appear after 4 hours digesting.

3. R.B. Age 27. Married. No digestive symptoms

Test Breakfast:
- No torulae etc
- Filtrate Acid
- Free HCl
- Albumoses
- Erythrodextrin Present
- Sugar
- Lactic Acid
- Peptones Absent

After digesting for 4 hours peptones were present in the filtrate

Motor Functions:

Salol Test:
- Reaction present in 75 minutes:
- Last present in 24 hours.

Test Breakfast:
No torulae etc
Reaction: Acid Free HCl
Albumoses Present
Erythrodextrin
Sugar Absent
Lactic Acid
Peptones
Motor Functions:
Reaction present in 75 mins
Last present in 24 hours

Diagram of Abdominal Region: Distended Stomach
6. K.S. Age 21. Single. Has had "indigestion" for two or three years with several severe attacks of vomiting and pain. She states that she vomited blood about 3 months ago. On admission she complained of a constant epigastric pain, especially severe ½ hour after food which is relieved by vomiting. There was limited tenderness in the epigastrium.

Test Breakfast:
No torulae etc
Reaction of filtrate: Acid
Free HCl
Albumoses
Erythrodextrin Present
Sugar
Lactic Acid Absent
Peptones
The filtrate contained peptones after digesting 4 hours without the addition of free HCl

Motor Functions: Salol Test

Reaction present after 90 mins
Last present after 24 hours

Test Breakfast: Torulae present

Reaction of filtrate Acid

Free HCl, Albumose, Erythrodextrin and Sugar: present

No Lactic Acid or Peptones

Peptones were present after 4 hours digestion of the filtrate but were better marked when HCl was added

Motor Functions:

Salol Test

Reaction present in 75 minutes

Last present in 30 hours

The stomach was not distended
M.L. Age 24. Single. Had a severe attack of vomiting and epigastric pain 2 years previously. She complained on admission of "cutting" pains in the epigastrium after meals and also of "dull" pains between the shoulders and in the epigastrium:

Test Breakfast:
- Torulae present
- Reaction of filtrate: Acid
  - Lactic Acid
  - Albumoses
  - Erythrodextrin
- Sugar
- Free HCl
- Peptones

Peptones were only found in the filtrate on digesting when HCl was added.

Motor Functions:
- Salol Test

reaction present in 6 hours
last present in 24 hours.
9. P.B. Age 17. Single. Complained of pain after food in the left side of the chest and vomiting. The pain came on about \( \frac{1}{2} \) hour after eating.

Flatulence

Test Breakfast:
- Free HCl
- Lactic Acid
- Albumoses: Present
- Peptones
- Erythrodextrin
- Sugar: Absent

No torulae found

Peptones were much more plentiful after digestion for four hours with the addition of free HCl

Test Breakfast:
Torulae present
Reaction of filtrate: Acid Free HCl
Lactic Acid
Albumoses Present
Peptones
Erythrodextrin
Sugar
On digesting the filtrate with HCl a much more marked peptone reaction was produced.

Motor Functions: The fasting stomach contained a very few starch cells with some mucus, epithelial and small round cells.
II A.L. Age 21. Single. Had epigastric pain and vomiting for 3 years. On admission she complained of a constant pain in the epigastrium, made worse by the ingestion of food, but relieved by vomiting. Tenderness in the epigastrium and left hypochondrium.

Test Breakfast:
- No torulae
- Reaction of filtrate Acid
- Free HCl Present
- Lactic Acid
- Albumoses
- Erythrodextrin
- Sugar Absent
- Peptones Peptones were only found in the filtrate on digesting for 4 hours with free HCl added

Motor Functions: The fasting stomach contained a very few granules of starch with some leucocytes in masses of mucus with squamous epithelial cells.

Test Breakfast:
No torulae
Free HCl
Lactic Acid
Albumoses
Erythrodextrin
Sugar
Peptones

Peptones were only found after digesting for 4 hours with the addition of free HCl.

Motor Functions:
The fasting stomach contained no starch granules but epithelial and numerous round cells in masses of mucus.