

with a perforated gastric ulcer of 24 hours' duration. Sometimes the fatigue of the reflex appears to occur very suddenly. Certain it is that one meets many cases of diffuse peritonitis in a late stage in which there is no rigidity of the abdominal wall. The other reason for lack of abdominal rigidity is a weak, thin or fatty muscle which cannot become very rigid under any stimulus. It is often difficult to appraise the amount of parietal peritonitis subjacent to the abdominal wall of a fat subject, for three inches of fat is an efficient screen apart from the possible fatty degeneration of the muscle.

Muscular rigidity is only found in the active stage of parietal peritoneal inflammation. When the infection becomes localised the rigidity tends to diminish. It is the rule for a well-localised intraperitoneal abscess to have a lax abdominal wall over it. Peritonitis under the diaphragm leads to rigidity of that muscle which is demonstrated by want of movement on respiration. Peritonitis over the psoas may lead to rigidity of that muscle also, demonstrated by pain on extension of the leg. Irritation of the non-demonstrative area of the peritoneum does not lead to rigidity. This fact is of very great clinical importance. Peritonitis starting from the pelvis does not cause abdominal wall rigidity till the suprapubic region is involved, and not even then if the parietes be not much affected. The same absence of rigidity is sometimes seen in cases of ruptured intestine (or leakage of intestinal suture) if the leakage be amidst coils of intestine or against a silent area of the parietes. Lack of appreciation of this fact is, I feel sure, responsible for many serious mistakes. It is possible for extensive and fatal peritonitis to occur without there being any rigidity owing to the slight involvement of the demonstrative parietal areas.

#### 6. ALTERATION OF MUSCULAR REFLEXES.

The alteration of the superficial abdominal reflexes in acute abdominal disease has been studied by Gwynne Williams,<sup>9</sup> who found that the reflexes were diminished in some abdominal conditions. I have not paid special attention to this point, but from the varying response obtained in normal persons I am not disposed to lay much stress on the importance of the reflexes in cases of peritonitis.

#### THE NERVE-SUPPLY OF HERNIAL SACS.

Hernial sacs are formed from parietal peritoneum bulged through or between the other structures in the parietes. It is interesting to endeavour to ascertain what nerves, if any, are found in connexion with such sacs. It is clear that any such nerves must be derived from the nerves which supply the neighbouring parts and as the sac grows in size, since it is very unlikely that the nerves will grow, the nerves would become more sparsely found in the sac-wall, until in time the sac and its coverings are almost devoid of nerves. Round the base or neck of the sac one would still expect to find nerves in greater number. This *a priori* view is borne out by the clinical experience that well-developed and large hernial sacs are very insensitive, whereas small sacs or herniæ in the making are much more painful. An inguinal hernia is naturally the most painful variety, as the tissues round the sac are well supplied by nerves. The ilio-inguinal nerve supplies fibres to the tissues of the cord and the ilio-hypogastric and genito-crural supply the neighbouring muscular fibres and tissues. An inguinal hernia is painful and tender in proportion to the rapidity of its formation and the acuteness of its strangulation. The irritation and stretching of the nerves in these peri-saccular tissues may cause acute hyperæsthesia of the skin over the hernia, for the skin is supplied by the same nerve. This is best seen in the strangulation which sometimes occurs the first time that a congenital sac is opened up in young adults. In long-standing cases strangulation may not be accompanied by hyperæsthesia.

Umbilical and femoral herniæ are less sensitive than inguinal, and strangulation is not accompanied by so much local tenderness. This is easily understood if one considers that in both cases the sac issues through fibrous rings which have poor nerve-supply, and that

the sac itself has very few nerves. The clinical importance of this fact has long been known and taught, but the reason for lack of local pain has seldom been given.

#### CONCLUSION.

When a patient comes to the surgeon with symptoms of intestinal obstruction an almost painless femoral hernia often proves to be the source of the trouble. In such cases there is referred pain in the abdomen, but the local pain, though present, is so slight as to be overlooked by the patient. If a complete coil of gut be present in the sac there may be greater tenderness, but if only a knuckle of gut or omentum there may be little or insignificant pain. How seriously this may mislead I can best illustrate by a case. A patient was sent up to hospital ten years ago with the following note:—

"I am sending a patient whom I have been treating for intestinal obstruction for the last four or five days. She also has an umbilical hernia. This morning to my astonishment I noticed that the skin over the hernia was gangrenous so I send her up to you."

The woman had a large gangrenous mass of omentum in an umbilical hernia and was in a moribund condition. It is clearly necessary to issue a caution as to the insensibility of hernial sacs. The mortality of strangulated hernia remains so great that I fear the point is little realised.

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## DUODENAL REGURGITATION INTO THE STOMACH DURING GASTRIC DIGESTION.

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#### Historical.

IT is well recognised by physiologists that the regurgitation of intestinal digestive juices from the duodenum into the fasting stomach is an event of regular occurrence in animals. This phenomenon has also been directly observed in man, and more recently Rehfus and his collaborators, using the fractional method of gastric analysis introduced by him, have definitely established it to be an almost constant feature in the fasting human subject. Owing to the work of Boldyreff it is also known that HCl and fatty acids in the duodenum, by stimulating the secretion of pancreatic juice and the discharge of bile, bring about such a regurgitation after they themselves have been neutralised and the pylorus has therefore opened.

In a later communication Boldyreff (1915),<sup>1</sup> working with dogs, introduced mineral and organic acids of known strengths into the fasting stomach and found that their percentage of acidity was gradually reduced, as other workers have also observed. He found that this reduction of acidity was due to neutralisation in the stomach by the pancreatic juice and that it occurred to a fixed level of 0.15 to 0.2 per cent. HCl. He points out that of the alkaline fluids available the only efficient neutraliser is the pancreatic juice, which is about ten times the strength of the other fluids. He eliminated experimentally these alkaline secretions one by one, and found that the only method of preventing the reduction in the acidity of the gastric contents was ligation of the pancreatic duct. These experiments are conclusive in showing that the saliva,

bile, mucus, succus entericus, and pyloric juice all assist to some extent, but that they cannot efficiently replace the pancreatic juice in this function of neutralisation. The pancreatic secretion induced by high degrees of acidity is driven into the stomach by strong antiperistaltic waves due to irritation of the intestine, which cannot tolerate an acidity of more than 0.1 to 0.15 per cent. HCl. The most favourable strength of HCl to gastric digestion is 0.15 to 0.2 per cent. and the mechanism described above regulates the acidity to this strength. It is styled by him "the self-regulation of the acidity of the stomach contents." He also points out that the residual fluid may be bile-stained, although this is by no means always the case, as pancreatic juice alone may regurgitate.

In the year following Spencer, Myer, Rehfus, and Hawk (1916)<sup>2</sup> examined this question of duodenal regurgitation in man. As a test of the presence of pancreatic juice in the gastric contents, obtained by the fractional method, they employed the quantitative estimation of trypsin. They examined the contents of the fasting and of the digesting stomach for this ferment and also after administration of HCl, sodium bicarbonate, and water. They found that trypsin was almost constantly present in the fasting and also in the digesting stomach, that its amount varied inversely with the acidity of the gastric contents, and that it occurred not only with a high acidity but also when the gastric contents were alkaline. They regard their work as in many ways confirming the theory of Boldyreff, and adopt the view that duodenal regurgitation is a response to irritation of the duodenum, and part of an attempt to render harmless substances that would have an injurious effect upon the small intestine. In a later paper by Rehfus and Hawk (1921)<sup>3</sup> the contrast between duodenal regurgitation during the digestive phase and during the resting phase is emphasised. They state that during gastric digestion the pylorus shows heightened tonus with but little regurgitation, except that which is essential to maintain optimum acidity in the stomach, but that during the resting phase the pylorus relaxes, permitting a ready reflex of alkaline duodenal secretion.

This view is also held by Bennett and Ryle (1921).<sup>4</sup> They state that the percentage of acidity in the gastric contents is the resultant of several varying factors—acid on the one hand, alkaline on the other, the alkali being derived from saliva, duodenal regurgitation of intestinal contents, secretion of pyloric mucous membrane. They give diagrams to illustrate their meaning, showing that when the stomach is emptying there is not much duodenal regurgitation, but when the organ is emptied there is free regurgitation. Crohn (1918)<sup>5</sup> introduced HCl into the fasting stomach of a patient with achylia and found a rapid fall in the acidity, which he attributed to dilution with a secretion of watery juice containing no acid ions, and also to a secretion of alkaline mucus.

#### Observations.

It must be admitted that at the present time the views held with regard to duodenal regurgitation into the digesting stomach of the human subject are chiefly matters of surmise, as no exact observations have been made to determine when, and to what degree, the contents of the digesting stomach are neutralised by the regurgitation of pancreatic juice, or how the curve of gastric acidity is modified by such regurgitation. The object of our present series of observations is to elucidate these points. Using the fractional tube for extraction, and the gruel test meal, we have made a complete analysis of the gastric contents every quarter of an hour, estimating the total chlorides, the inorganic chlorides, and the active HCl. The estimations were made by the Volhard method, which has been in routine use at University College Hospital for many years, except that the gastric contents were not filtered before fusing. With the gruel meal the amount of solid material in the stomach contents is small, the chloride content being about 0.02–0.03 per cent. A number of experiments were made with the

gruel meal itself, and also with the stomach contents an hour after taking the meal, both filtered and unfiltered material being used. The unfiltered material nearly always gave a slightly higher chloride figure than the filtered, but duplicate estimations of the unfiltered contents gave practically the same result in every case, and we are satisfied that no real error is encountered from this source. Estimations were also made of the free HCl and the total acidity, using dimethyl and phenolphthalein as indicators, in all cases in which the amount of available fluid was sufficient. These figures are not given in the charts as they were not obtained in all cases, and for the sake of simplicity; this method is very laborious and it is not claimed that it is of general applicability in clinical work; on the contrary, it is in our opinion only suitable for a purely scientific investigation of the events occurring during gastric digestion. The individuals used for these observations were three normal students, 13 cases of functional disturbance, in which there was no clear evidence of organic disease of the stomach, three gastric and two duodenal ulcers, one case of pernicious anæmia, and one gastric cancer.\* We have found that duodenal regurgitation is not an intermittent leak into the digesting stomach in small amounts, but that at a definite point, as the stomach is emptying and the curve of acidity is rising, the pylorus relaxes and allows of a considerable reflux of intestinal juices, which rapidly brings down the acidity as the stomach empties and determines the form of the curve. Duodenal regurgitation is thus a definite link in the chain of events occurring during gastric digestion. The curve of acidity due to free HCl as obtained by the fractional method assumes in the vast majority of cases the form of a single peak, varying very considerably in the steepness of its rise and fall and in its height. Such curves occurred in 90 out of 100 normal students recently examined by Bennett and Ryle.<sup>6</sup> In the remaining nine cases there was either (1) no free HCl present at all, or (2) no descent to the curve, the percentage of acidity remaining at its highest level till the stomach was empty. Our observations are in agreement with these findings: we have also found that the curve of active HCl (free HCl + protein HCl) has a similar form.

It is quite clear that the rise in the curve is due to a continuous output of gastric juice, varying in rapidity in different cases. The fall in the curve must result from either neutralisation or dilution of the gastric contents or both, because if neither of these factors came into play the curve would gradually rise as the stomach emptied till the concentration of the gastric contents in HCl equalled that of pure gastric juice; whilst, if the secretion of gastric juice stopped at any moment, the curve would subsequently draw a horizontal straight line, the gastric contents maintaining a uniform acidity as the stomach emptied. The curve of the inorganic chlorides, however, demonstrates that the top of the peak in the acid curve corresponds with the time at which the pylorus definitely relaxes and allows considerable regurgitation of intestinal juices, because at that point as the acid curve begins to fall the curve of inorganic chlorides simultaneously rises and the two usually cross one another. The rise of the inorganic chloride curve is the result of neutralisation of the HCl. That the gastric contents are not materially diluted is shown by the fact that the curve of total chlorides remains at its original level.

#### The Curve of Active HCl (Free + Protein HCl).

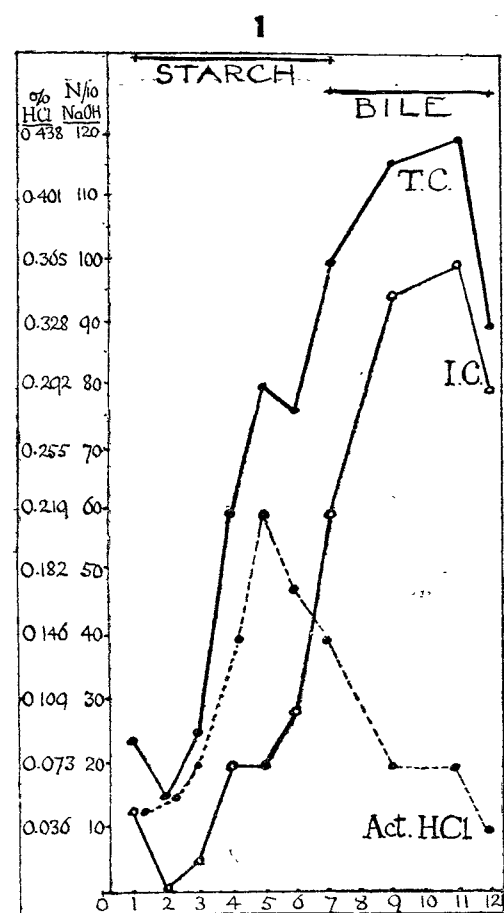
The curve of active HCl follows very closely that of the total acidity as estimated by titration using phenolphthalein as the indicator, and various types are seen corresponding to those mentioned above as being the normal curves of free HCl.

*Type 1.*—This curve shows a steady rise until the summit is reached at a level corresponding to 0.16 to 0.23 per cent. HCl in rather under one and a half hours.

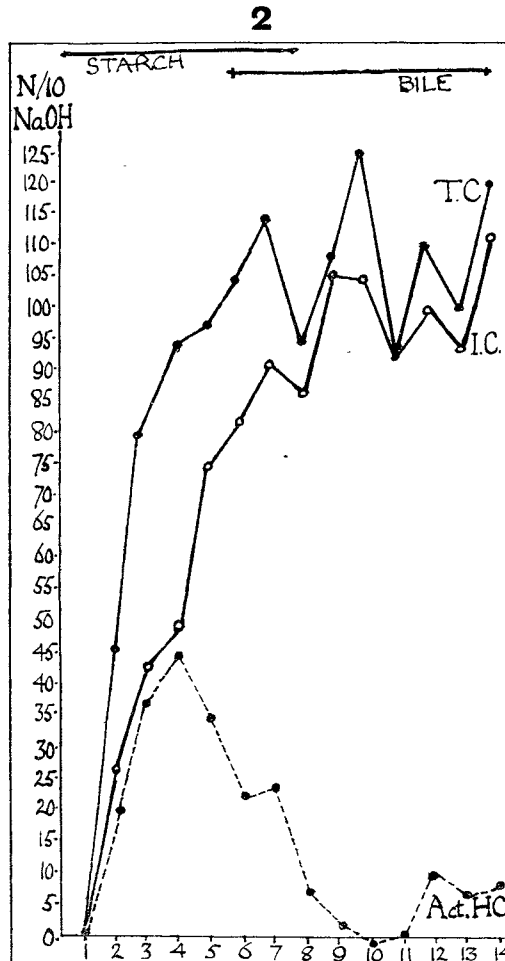
\* Up to the date of publication we have examined by this method upwards of 70 cases, all confirming our conclusions.

This rise is parallel to and only a little below that in the curve of total chlorides and is obviously, as stated above, due to a rapid output of HCl by the gastric glands. Any residue which had been found in the fasting stomach before the feed was given was estimated and the reading recorded at the beginning of the curve, which gives the appearance of an initial fall in the curve. Having attained its summit the curve then shows either a short plateau followed by a fall or else an immediate fall. In either case the cessation of the rise is coincident with or immediately preceded by a rise in the curve of inorganic chlorides, an event which can only be adequately explained by

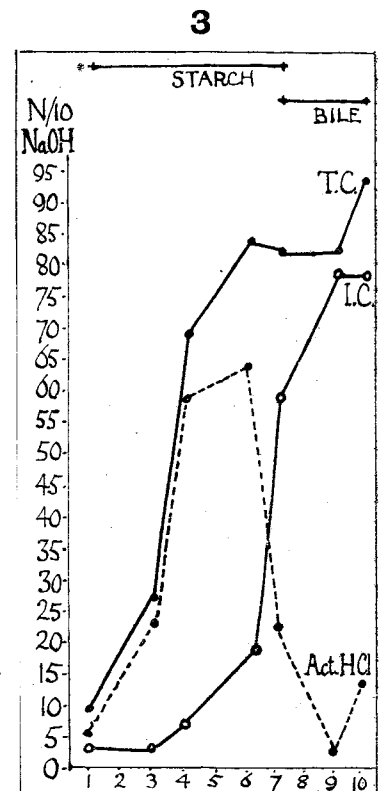
that of many normal stomachs. The fact that such curves also occur in patients who have had successful gastro-enterostomies performed and in whom the free regurgitation of duodenal contents through the stroma is the cause of the high total chloride and low acid curves, lends support to our view that duodenal regurgitation through the pylorus is similarly the cause of the fall in the active HCl curve which we describe, and of the low acid and high total chloride curves just mentioned. In illustration of this type of curve we quote eight (1-8) cases, and also three others (9-11), showing absence of free HCl with a low active HCl curve and a high total chloride curve.



CASE 1.—Normal curves of total chloride (T.C.), inorganic chlorides (I.C.) and active HCl.



CASE 2.—Functional disorder.



CASE 3.—Functional disorder.

N.B.—In all the charts the percentages of HCl corresponding to the amounts of decinormal NaOH are omitted. The figures at the foot represent quarter hours.

rapid neutralisation of the HCl owing to a more or less sudden reflex of alkaline intestinal juices. This reflux occurs whilst starch is present in the stomach, but no definite relation appears to exist between the time at which it occurs and the subsequent time at which the stomach empties, as shown by the disappearance of starch from the gastric contents. The interval between these two events, the commencement of neutralisation and the emptying of the stomach, varied in our curves from half hour to three hours. Towards the end of the curve when the stomach is empty the fall may be accelerated by diminution in the secretion of HCl and by dilution by the regurgitated fluid, as shown by a fall in the curve of total chlorides, which may at this stage descend to a level equivalent to 0.2 per cent., the amount of chloride present in the pancreatic secretion.

It is clear that this curve of active HCl is no measure of the actual gastric secretion; it represents only the balance between the two processes, secretion and neutralisation. The work of Dodds<sup>7</sup> bears out this idea. He has shown that the curve of alveolar CO<sub>2</sub> closely corresponds with the curve of gastric acidity, the rise in the CO<sub>2</sub> curve being due to the output of acid from the blood, and the fall to the output of alkali in the form of pancreatic juice.

It is also important to realise that a stomach which gives a very low acid curve with no free HCl may yet give a total chloride curve equal to or even exceeding

CASE 1 (Fig. 1).—F. R. W., male, aged 27 years, a normal medical student.

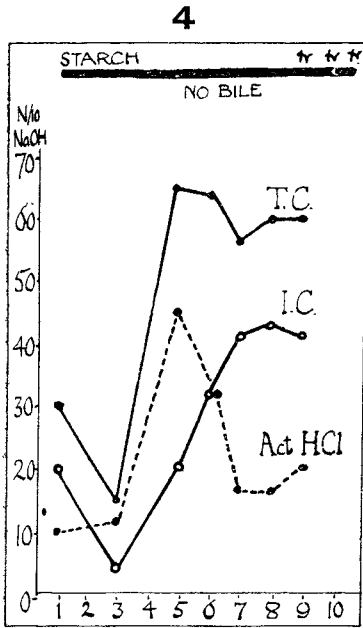
CASE 2 (Fig. 2).—F. B., female, aged 53 years, had suffered for nine years from irregular attacks of vomiting, which appeared to originate in the eructation of a clear fluid chiefly in the night, and also two hours after food. The fluid never contained food and was either tasteless or slightly bitter. The only other symptom was a feeling of fullness. For the last 18 months slight pain situated just above and to the left of the umbilicus had been superadded. It was of a dull pulling quality and occurred two hours after food, which relieved it. On examination patient was free from symptoms. By the X rays the stomach was orthotonic with active peristalsis, and emptied itself in three hours as near as possible. Diagnosis: Functional disorder.

CASE 3 (Fig. 3).—E. G., female, aged 33 years, had suffered from pain right across the abdomen and below the umbilicus for 14 years. For the last seven weeks she had had pain across the epigastrium and in the centre of the sternum more or less constant, but worse soon after meals. Vomiting also occurred after meals. There was fullness and eructation of wind but no fluid. By the X rays the stomach was long and narrow and situated considerably to the left. It was empty within two hours. Diagnosis: Functional disorder.

CASE 4.—A. B., female, aged 29 years, had had gastric attacks like the present one since 15 years of age. The onset of an attack was usually due to some indiscretion in diet. There was localised pain in the centre of the upper epigastrium with tenderness and some resistance. It occurred about 10 to 15 minutes after meals, was sharp, and radiated to the back and left shoulder. There was eructation of wind but no vomiting. She was a nervous individual and also

had pain in the right side, worse on lifting weights or at the periods. By the X rays the stomach was of normal shape and contained a small residue after three hours. Diagnosis: Functional disorder. (Analysis shows curves similar to Fig. 3.)

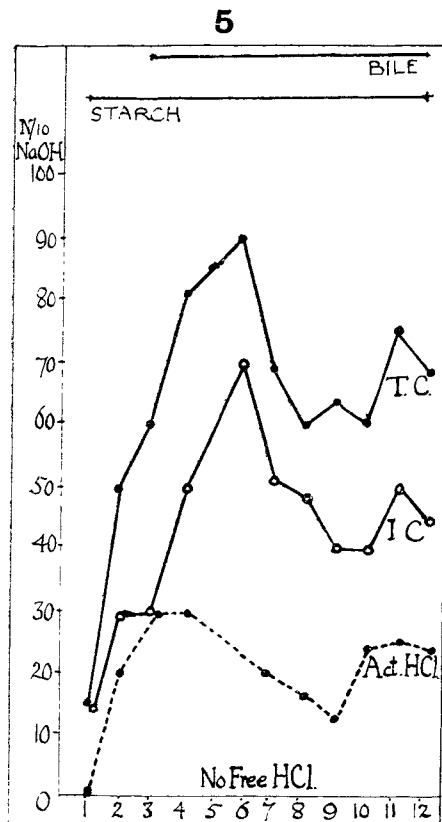
CASE 5 (Fig. 4).—A. W., male, aged 59 years, had suffered from occasional attacks of pain in the epigastrium just below the ensiform for ten years. This pain was dull and aching and came on one and a half to two hours after meals, which relieved it. He also suffered from fullness and loss of appetite for six months. He had lost some weight. There was no vomiting or bleeding. There were some decayed teeth and pyorrhoea in the lower jaw. There was rigidity of the left rectus and slight tenderness below the ensiform. X ray examination showed slight atony and a small residue after three hours. After a few days in hospital he had very little pain. He had his teeth extracted and left in a month quite well. Diagnosis: Functional disorder.



CASE 5.—Functional disorder.

the epigastrium in a band and sometimes to the back. It was usually constantly present and relieved to some extent by food for two or three hours only. There was vomiting of a "slimy greenish material" and some loss of flesh. The patient suffered from aortic regurgitation and his Wassermann reaction was positive. By the X rays there was an appearance as of defective filling of the pyloric end of the stomach, the body being of a rotund shape; there was some residue at the end of three hours. Diagnosis: Functional disorder.

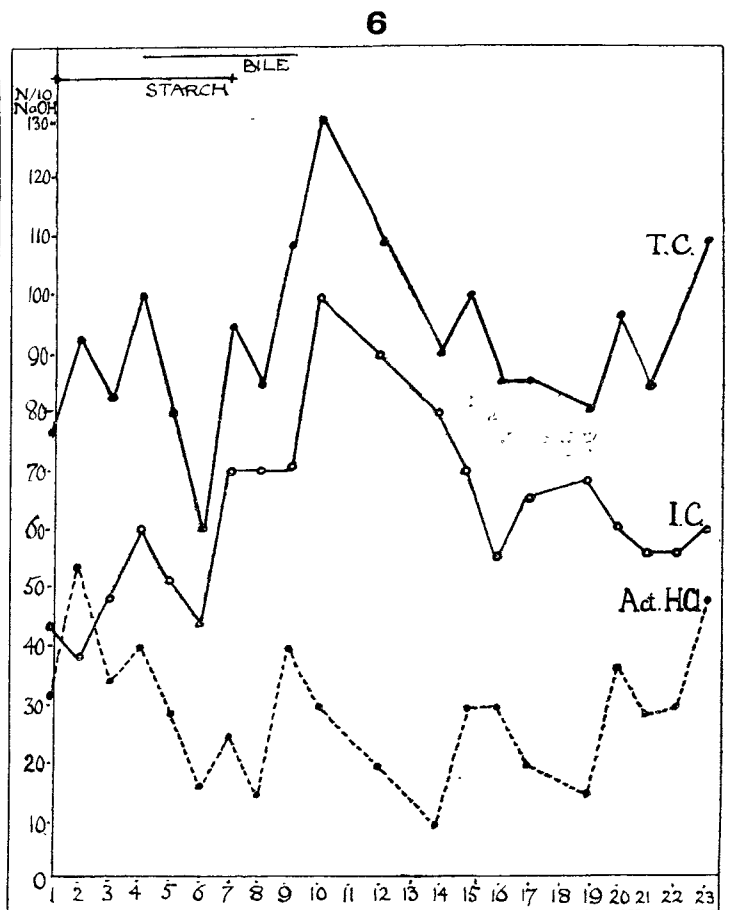
CASE 7.—A. G., female, aged 25 years, had suffered at intervals for a year from gastric attacks of the following



CASE 10.—Functional disorder.

nature. Four hours after her tea more especially, but also after other meals, a sharp shooting pain occurred in the left side of the chest below the breast, which lasted a short time and was succeeded by pain in the upper part of the epigastrium, which was dull and aching and lasted half an hour or till the next meal. This was followed by general coldness and shivering. There was also tightness in the chest, fullness, and eructation of wind with the pain, but no vomiting,

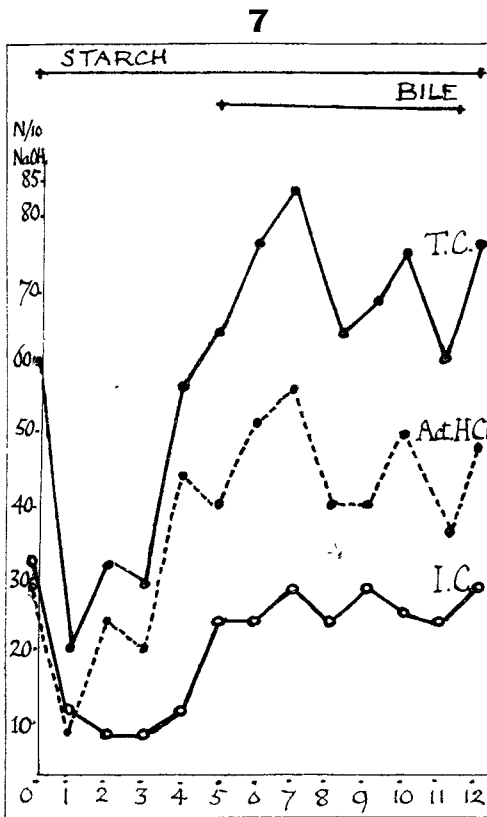
CASE 6.—B. N., male, aged 45 years, had had gastric pain since 1918. The pain extended across



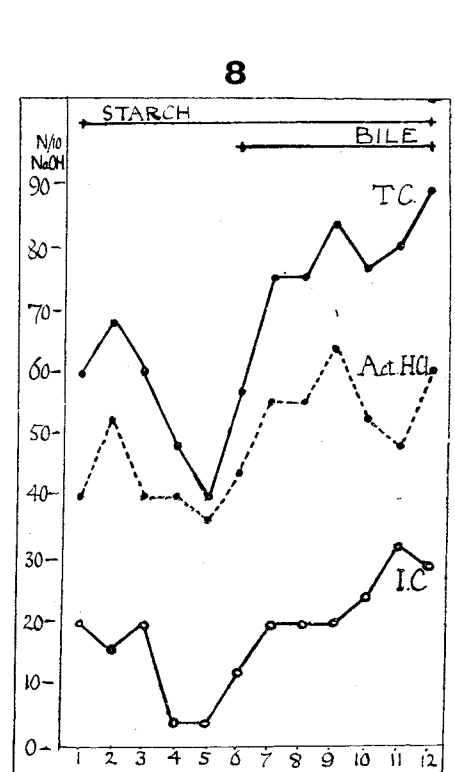
CASE 13.—Duodenal ulcer.

although nausea was felt. The patient was thin. There was slight epigastric tenderness, but no rigidity. Diagnosis: Functional disorder.

CASE 8.—T. P., male, aged 46 years, was suffering from aortic regurgitation. His illness commenced in 1916 and since this time he had had attacks of pain and vomiting, and on three occasions hæmatemesis. By the X rays there



CASE 14.—Duodenal ulcer.



CASE 16.—Pyloric ulcer.

was food in the stomach after six hours. At the operation a pyloric ulcer was found. (Analyses of Cases 6, 7, and 8 show curves similar to preceding.)

#### Low Acid and High Total Chloride Curve.

CASE 9.—W. C., male, aged 47 years, had suffered for a year from pain and vomiting. The pain was mid-epigastric, gnawing, and occurred half to one hour after food. Vomiting

occurred about one hour after the pain, which it slightly relieved. There was fullness, relieved by eructation of wind. Some loss of weight. By the X rays there was a small residue after three hours. The pain ceased in a few days and vomiting never occurred whilst under observation. Diagnosis: Functional disorder.

CASE 10 (Fig. 5).—J. L., male, aged 31 years, had been ill for seven months. The pain was just above and to the right of the umbilicus; it came on three hours after meals and lasted about half an hour when it was relieved by eructating wind, but if not the next meal relieved it. It did not wake him at night. There was no vomiting or loss of weight; no bleeding. By the X rays the stomach was empty in three hours. He only had pain on three occasions whilst under observation. Diagnosis: Functional disorder.

CASE 11.—L. H., male, aged 40 years, had been ill for 18 months with occasional eructation of bitter fluid a few minutes after meals. For six months he had had an aching pain a few minutes after meals, in the epigastrium, behind the sternum, and a feeling of a lump in the throat. There were fullness and bitter eructations leading to vomiting. These symptoms came on three or four times a week chiefly after the evening meal, and on rising in the morning there were often bitter eructations. By the X rays there was a small residue in the stomach after six hours. There was slight loss of flesh, but the patient was healthy-looking and in good condition. Diagnosis: Functional disturbance. (Curves of Cases 9 and 11 are similar to Fig. 5.)

*Type 2.*—Sometimes the curve above described is varied by a subsequent rise occurring at about the third hour or later, when the stomach has emptied. This rise is coincident with a sharp fall in the curve of inorganic chlorides and sometimes by a rise in the curve of total chlorides. In all such cases which we have examined the stomach has emptied early, but has contained a free secretion in the interdigestive phase and the last rise has been accompanied by the disappearance of bile. This rise in the curve we believe to be due to a cessation of duodenal regurgitation due to hypertension of the pylorus. The HCl at this period is almost entirely free. We quote two cases in illustration of this type of curve (12 and 13).

CASE 12.—A. H., male, aged 31 years, had had attacks of pain since 1919 lasting five to six weeks, with free intervals of 5–13 months. There was a heavy pain in the front of the chest and epigastrium half an hour after meals temporarily relieved by food. Vomiting occurred three hours after food, consisted of bitter fluid, and temporarily relieved the pain; the vomit never contained blood. An X ray examination showed an orthotonic stomach, almost empty in three hours. Diagnosis: Functional disorder. (Curve similar to Fig. 6.)

CASE 13 (Fig. 6).—M. M., male, was operated on and a duodenal ulcer found. The X ray examination showed a low orthotonic stomach and a low colon. At the end of three hours there was considerable food residue.

*Type 3.*—A third type of curve corresponds to the "climbing type" of free HCl curve described by other observers. Here the curve of active HCl begins more or less slowly, climbs steadily, and remains high for at least three hours. This type is seen in a variety of clinical conditions, the one factor common to all being defective duodenal regurgitation due to organic obstruction of the pylorus, or spasm or deficient relaxation of the sphincter pylori. In these cases the total HCl secreted may be normal or increased, but the inorganic chlorides remain low owing to defective neutralisation. This type of curve we also obtained in a case of hour-glass contraction in which the material examined was obtained only from the upper sac. In some cases the curve of active HCl shows one or more dips corresponding to slight rises in the inorganic chloride curve, no doubt due to a partial relaxation of the pylorus with only slight regurgitation (Cases 19 and 20). Sooner or later, however, unless there is an organic stricture of the pylorus, the sphincter relaxes and neutralisation occurs.

The following eight cases (14 to 21) illustrate this type.

CASE 14 (Fig. 7).—W. H., male aged 54 years, had been ill for 20 years with attacks of pain lasting a few weeks to a year. The pain was dull and aching, occurred two to three hours after meals, was situated in the mid-epigastrium, passing to the back and right shoulder and was relieved by food. Vomiting commenced three years ago and relieved

the pain, the vomited matter containing coffee-grounds material on one occasion 10 years ago. There was fullness and eructation of wind. By the X rays the stomach was found to be dilated and atonic, and after six hours about one-third of the contents still remained. The patient was operated on and a duodenal ulcer found.

CASE 15.—H. T., male, aged 46 years, had had attacks of pain for 15 years with intervals as long as 18 months. The pain occurred over the whole abdomen, but was most intense in the upper zone. It was dull and heavy and had no relation to meals, although occasionally it was relieved by food. Vomiting occasionally occurred. The patient was thin and had a few bad teeth. A test-meal of meat showed: Active HCl 0.31, free HCl 0.25, total acidity 0.45, total Cl 0.41, and inorganic Cl 0.1 per cent. The teeth were extracted and patient rapidly lost his symptoms. Diagnosis: Functional disorder. (Curve similar to Fig. 7.)

CASE 16 (Fig. 8).—O. T., male, aged 51 years, had suffered from attacks of a dull aching pain situated just below the ensiform. It came on about 3 P.M. and was relieved by vomiting or eructating wind. Fullness and discomfort appeared a few hours after breakfast and got worse towards evening. If he starved he suffered no discomfort. Eructations of wind and fluid caused a scalding sensation in the throat. The vomit was watery and sour, but never contained blood, the vomiting usually occurring towards evening or in the early morning. The patient was operated on and a pyloric ulcer found.

CASE 17.—W. S., female, aged 29 years, had exophthalmic goitre two years ago but now simply had slight staring of the eyes. For seven years she had been subject to pain and vomiting. The pain affected the left hypochondrium and epigastric region spreading to the right. It was not related to food, but might be slightly relieved by the latter. It might last only an hour or be continuous all day and was dull and aching, sometimes stabbing. It was increased by working. The vomiting was very irregular and the vomit consisted of food and a slimy material and relieved the pain. There was fullness soon after meals, but no eructations. The appetite was bad and the bowels constipated. She had lost flesh during the last five years. The X ray examination showed gastroparesis and an atonic stomach, with considerable residue after six hours. Diagnosis: Functional disorder. (Curve similar to Fig. 7.)

CASE 18 (Fig. 9).—S. S., male, aged 42 years, had had indigestion since childhood. This became worse five years ago with almost constant pain and irregular vomiting, the vomit on one occasion being of the coffee-grounds type. By the X rays the stomach was dilated and atonic and at the end of six hours the shadow had not diminished much in size. At the operation pyloric obstruction due to a chronic ulcer was found.

CASE 19 (Fig. 10).—A. S., female, aged 39 years, had been ill since 1917. She complained of a heavy dull pain at the level of the third costal cartilage passing to the back and causing a sensation of choking in the throat. It often came on half an hour after waking and then lasted all day, and she had been awakened at night by it. Food has no effect on the pain, although at the beginning she had had epigastric pain half an hour after food. This had now disappeared. For the last four months she had had attacks of swelling of the face, evidently urticarial. There was abundant gaseous eructation but no vomiting. The only physical sign was slight epigastric tenderness. By the X rays the stomach was orthotonic with active peristalsis, the lower pole being lower than normal. After five hours there was still a small residue at the pyloric end. The pyloric cap was normal. The patient was operated upon and the stomach found to be normal. Diagnosis: Functional disorder.

CASE 20 (Fig. 11).—O. B. L., male, aged 25 years, a normal medical student.

CASE 21.—S. E., female, aged 29 years, a normal medical student. (Curve similar to Fig. 7.)

#### *Curve of Inorganic Chlorides.*

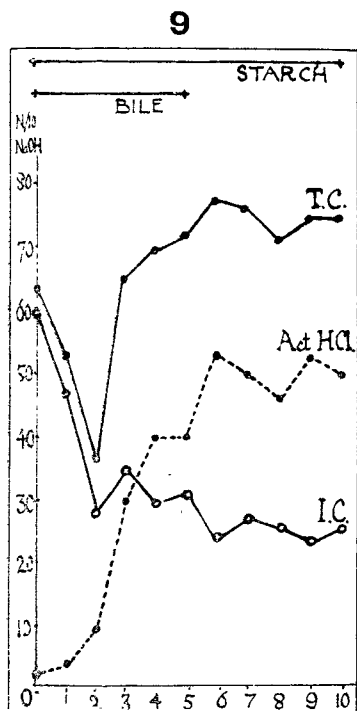
The curve of inorganic chlorides varies in each of the types of curve of active HCl described above and a reference to the illustrations of these cases will elucidate the following points.

*In Type 1.*—The curve of inorganic chlorides gradually rises from the beginning with that of the active HCl to more or less the same level. This rise is due to neutralisation of some of the HCl secreted by what alkali happens to be present in the food, or swallowed in the saliva, or contained in the gastric mucus or pyloric juice; it is also due to decomposition of organic salts in the food by the HCl. At the point at which the curve of active HCl falls, the curve of



inorganic chlorides continues to rise sharply following that of the total chlorides at a somewhat lower level. This second rise is due to neutralisation of the HCl by regurgitated duodenal juices.

In the three curves of low acid readings (9, 10, 11), it will be observed that the curve of inorganic chlorides closely follows that of the total chlorides all the time. In these cases we believe that duodenal regurgitation is active almost from the



CASE 18.—Pyloric ulcer.

“climbing type” of HCl curve, the curve of inorganic chlorides remains uniformly low owing to the absence of duodenal regurgitation and consequently defective neutralisation. In some cases, however, the curve of inorganic chlorides may show irregular rises corresponding with depressions in the active HCl curve. This suggests neutralisation by duodenal regurgitation, insufficient to bring down

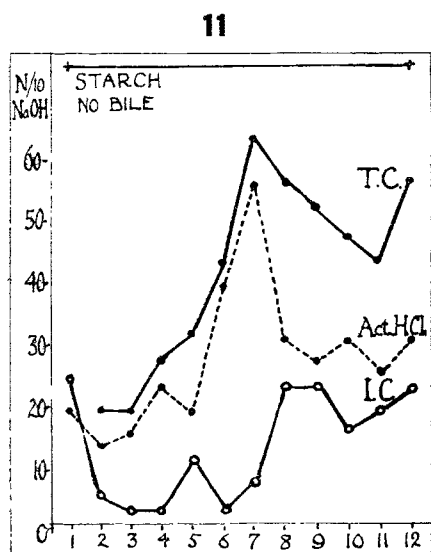
the acid curve, and no doubt due to a partial relaxation of the pylorus with only slight regurgitation of intestinal contents.

CASE 22 (Fig. 12).—A. E., cancer of the stomach.

CASE 23.—G. B., pernicious anaemia. (Curve similar to Fig. 12.)

In Type 2.—The curve of inorganic chlorides follows the same course as in the previous type, but later when the acid curve rises it coincidentally falls, owing to dilution by the freshly secreted gastric juice.

In Type 3.—In the



CASE 20.—Normal.

therefore represents the secretory curve of the gastric juice. The latter is most nearly represented by the curve of total chlorides, which maintains a more or less constant shape of various heights in all curves. When this curve rises the stomach is secreting, and when it falls the gastric contents are diluted by a fluid containing less chloride than is present in these contents.

#### Conclusions.

1. Duodenal regurgitation occurs at a definite period, but not always at the same time, during gastric

digestion, and must be considered as a definite link in the chain of events occurring in the digesting stomach.

2. It determines the shape of the curves of hydrochloric acid and of inorganic chlorides, the former rapidly falling and the latter rising.

3. This regurgitation depends upon relaxation of the pyloric sphincter; and its presence or absence, as determined by the shape of the curves above mentioned, is a measure of the degree of tone or spasm of this sphincter, or of obstruction of the pyloric orifice.

4. The curve of gastric secretion follows closely that of the total chlorides, but at a somewhat lower level.

5. The absence of free HCl or the presence of a low active HCl curve are no indications, when taken alone, that the gastric secretion is correspondingly diminished, for the total chlorides may still be at the normal height; these cases are due to free duodenal

regurgitation and are similar to those of successful gastro-enterostomy cases.

6. A curve of the “climbing type” does not necessarily indicate hypersecretion, but rather a delayed and deficient pyloric relaxation. Hypersecretion during the digestive phase is indicated by a rapid and high rise in the total chloride curve in the early part of that period; and during the interdigestive phase by the presence of an excessive amount of residual fluid containing a high percentage of total chlorides.

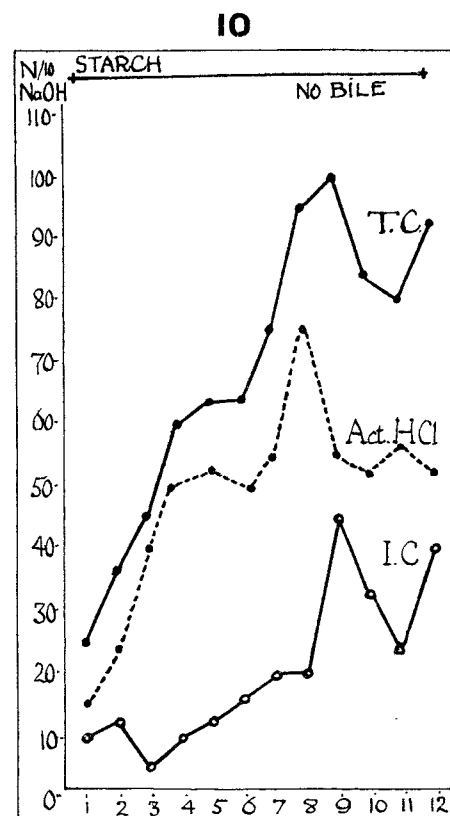
7. The two great factors contributing to the form of these curves are: (a) varying degrees of pyloric relaxation occurring at varying times, and (b) hypersecretion. The part played by deficient or excessive pancreatic secretion remains an open question.

8. The curves of free HCl and total acidity at present in use show, in most cases, the general shape of the curves we have described in this paper, more particularly when the acidity is high; but they fail in this respect when free HCl is absent.

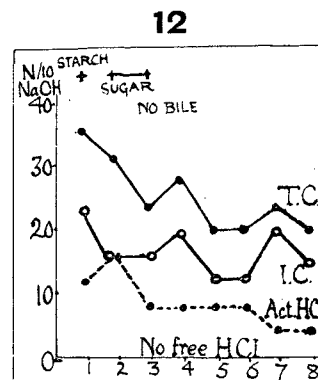
We wish to thank the Medical Unit of the Hospital for certain facilities granted us.

#### References.

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CASE 19.—Functional disorder.



CASE 22.—Cancer of Stomach.