RECENT ADVANCES IN THE DIAGNOSIS AND TREATMENT OF SOME DISEASES OF THE STOMACH.

BY

L. WOLFF, M. D.,

PHYSICIAN TO THE GERMAN HOSPITAL OF PHILADELPHIA; DEMONSTRATOR OF CHEMISTRY IN THE JEFFERSON MEDICAL COLLEGE, ETC.

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RECENT ADVANCES IN THE DIAGNOSIS AND TREATMENT OF SOME DISEASES OF THE STOMACH.[1]

BY L. WOLFF, M.D.,
PHYSICIAN TO THE GERMAN HOSPITAL OF PHILADELPHIA; DEMONSTRATOR OF CHEMISTRY IN THE JEFFERSON MEDICAL COLLEGE, ETC.

THE pathological conditions of the stomach have, during the last few years, received a great deal of attention and consideration by medical investigators, with the result of placing them in a new light and a more rational position.

As pathological manifestations are but abnormal physiological functions, we will have to consider the normal gastric functions before proceeding to the abnormal.

It is a well-established fact that the stomach has distinct digestive power, and it is also well known that this is due to its secretions or to part thereof. While the secretive action of the stomach, in common with all mucous membranes, results in the production of mucus, the secretion of its acid digestive fluid from the peptic glands is one peculiar to this viscus alone,

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and to this product the name "gastric juice" is commonly given. That portion of the acid gastric juice which has a distinct influence on its digestive properties has been definitely shown by Carl Schmidt to be hydrochloric acid. The proteid ferment, which is made active only by this acid, is known as pepsin, and the result of its action on albuminoid food as peptone.

It has of late been questioned if the hydrochloric acid secreted by the peptic glands has only the power of rendering the pepsin active, and it is held by some (G. Bunge, *Lehrbuch der physiologischen und pathologischen Chemie*) that its principal value consists in rendering aseptic the ingested food, a fact which the attempt has been made to substantiate from the result of experiments which proved that 0.3 per cent. HCl prevented septic change in meat fibre, while the average normal gastric juice contained that amount of it. Normal gastric juice undoubtedly prevents and corrects effectually septic change of albuminoid food. Felix O. Cohn, in the *Zeitschrift für physiologische Chemie*, xiv., 1, July 29, 1889, ascertains that this is not attributable to the admixture of pepsin; that even small quantities of HCl prevent the formation of acetic acid, and that lactic fermentation is prevented by as much HCl as is necessary to convert into chlorides the phosphates necessary for the development of the bacillus acidi lactici. He also states that pepsin-hydrochloric acid has the same value to prevent fermentations as that without pepsin, and that HCl united with peptones fails to inhibit or prevent fer-

mentation, and, as is already known, to digest albuminoid food. He further confirms Ewald's observation that HCl is probably secreted at once with the ingestion of food, but is imbibed by and united with the albuminoids, also with the bases and salts it unites with or displaces, and that during this period there is nothing to prevent other fermentations.

The question of the production of HCl in the peptic glands from the alkaline blood and fluids, is one that is of interest and value to gastric physiology. That HCl must necessarily result from the decomposition of the chlorides of the blood and liquids of the body is quite apparent. As sodium chloride is the principal one of these contained in the blood and lymph, and as these also hold in solution sodium bicarbonate, it may readily be inferred how sodium carbonate can form with liberation of hydrochloric acid, as per following formula:

$$NaCl + NaHCO_3 = Na_2CO_3 + HCl.$$

It is, undoubtedly, the characteristic secreting power of the epithelial cell to diffuse the products of this decomposition in a manner that the more alkaline salt will be returned to the blood, while the acid becomes part of the gastric secretion.

It is a peculiar fact that the peptic glands of the pyloric region, which distinguish themselves even macroscopically by their paler color, secrete alkaline fluids, which, however, when acidulated with HCl, have marked peptonizing power. When comparing the anatomical structure of the peptic glands of the

pyloric and the non-pyloric region of the stomach, we find that the former have no ovoid, often called peptic, epithelial cells which we find in the tubules of the latter. It seems fair from this to deduce that the ovoid cells of the peptic glands secrete the HCl, as already pointed out by Heidenhain.

I have recently been able to arrive at similar deductions from experiments conducted with Dr. E. P. Davis at the Philadelphia Hospital. In a number of examinations of the gastric juice of normal infants no hydrochloric acid was found present therein, while coincident with my observations it was stated that the tubules of the peptic glands of the infantile stomach have no ovoid cells.

Ludwig and Ogata deduce from experiments on dogs on which resection of the entire stomach had been practised, as well as from some in which the pylorus had been occluded and the animals fed by a duodenal fistula, that the stomach was not absolutely necessary as far as digestion was concerned, either as a receptacle for food or as a generator of peptic fluids. Though these experiments of Czerny, Ludwig, and Ogata prove beyond doubt the possibility of nutrition without the stomach in the dog, the predigestion of food in the stomach is certainly indicated by the time it remains there, the changes it undergoes while there, and the absorption of peptonized food directly therefrom. That the stomach should only slowly rid itself of its chyme cannot possibly be only to the end of rendering it antiseptic by contact with its acid secretion, when the powerful peptonizing action of the chlorhydric solu-

tion of its secreted albuminoid principle is so well established. That different foods are much more readily acted on by the gastric juice and passed from the stomach, as shown by Beaumont, would also tend to establish the importance of gastric digestion for the nutritive process, even if impairment of the normal gastric digestion did not give rise to special pathological conditions quite characteristic as well as important in their effect upon the general nutrition of the human organism.

Before proceeding to the consideration of the typical diseases of the stomach, their pathology, diagnosis, and treatment, after having learned the special digestive function of the stomach, it is well first to study, as a general pathological condition, the perverted function which must arise more or less from all gastric disorders, and which, as an impairment or lack of the peptic act, is, therefore, termed "dyspepsia." Modern pathology recognizes the fact that gastric digestion is a purely chemical process due to various influences upon the secretory apparatus of the stomach, and the "chemical dyspepsia" has, as such, been proclaimed in the last three German Congresses for Internal Medicine, by von den Welden, Leube, Riegel, Ewald, Boas, Edinger, Jaworsky, etc.

Dyspepsia.

The chemical changes which take place in the stomach under abnormal conditions are the variable composition of the gastric secretion, owing to the increased, diminished, or totally absent quantity of

hydrochloric acid, for so far no absence of pepsin has been established in any of the gastric secretions yet examined, and all of them have answered the physiological digestive test if admixed with hydrochloric acid.

The diagnosis of dyspepsia, as practised to-day, is the consequence of, and was developed from lavage of the stomach, as introduced by Kussmaul about twenty years ago, and the close investigation of the matter thus washed out. Previously the contents of the stomach and its secretions were judged principally from the vomited matter, but as this is a pathological product, it cannot give any correct idea of the possible functional ability of this organ. The ingesta, besides largely diluting the gastric secretion, make its chemical examination very uncertain and unreliable. The withdrawal of the gastric secretion when its digestive power is the greatest, is necessary at certain periods which have been ascertained by experiment and observation. This has to be done also at a time when the presence of ingested matter shall not render the withdrawal impossible. Without dwelling on the different experiments to ascertain this period, it is now generally accepted that a special meal should be given, after which, at a certain time, the gastric secretions are withdrawn. The early meal generally employed for this purpose is known as the trial breakfast, which consists of a few pieces, altogether about two to three ounces, of bread or bun and a glass of water or a cup of tea. Jaworsky and Gluzinski, relying on the observation that a soft-boiled egg disappears from the normal stomach in one and

a half hours, use this instead of the above-mentioned trial breakfast. At times, however, and especially under pathological conditions, it is found that the egg is not peptonized in that period, and then, by clogging the tube and the now proven union of the free hydrochloric acid with the albuminoids, the withdrawal is rendered difficult and the results inaccurate. To obtain the gastric juice from four to five hours after a more copious "trial dinner" has no advantage, and the "trial breakfast," after all, is the one relied upon.

Ewald and Boas express the gastric juice by epigastric pressure, about one hour after the trial breakfast, when it is known that in the normal state it is strongly acid from hydrochloric acid, the lactic acid having disappeared, and only continuing to be present in abnormal conditions. To express the gastric juice has disadvantages which may be readily avoided by the use of a suction apparatus. Germain Sée recommends a Potain aspirator for this purpose, partial exhaustion only being made, but an ordinary syringe answers the purpose very well, as the quantity for chemical analysis need only be very small, from one to two drachms sufficing to that end. The tube employed for withdrawal of gastric juice is a small-sized, ordinary stomach-tube, having two fenestræ, and, in the cases of infants and children, I have frequently used simply a small, soft-rubber catheter.

After the introduction of the tube in the usual manner, the fluid is withdrawn by slowly drawing out the piston of the syringe. A sufficient quantity

is recognized by a small glass-tube connection in the rubber one; the tube is then carefully withdrawn and emptied into a small glass vessel. The liquid so obtained is next filtered and tested, to ascertain if acid or not, with neutral litmus paper. When the acidity is thus established, the amount of acidity is determined by acidimetry with a decinormal solution of sodium hydrate, litmus or phenolphthalein being used as indicator. Five cubic centimetres of the filtered gastric juice are neutralized with decinormal solution of soda, as stated above, and the total acidity is expressed by multiplying the result by twenty, thus giving the number of cubic centimetres of one-tenth normal soda solution necessary to neutralize one hundred cubic centimetres of gastric juice.

After this is done, the chemical character of the acid is investigated. To this end the lactic acid test of Uffelmann may be applied. This consists of a two per cent. solution of carbolic acid to which a few drops of neutral ferric chloride are added, sufficient to produce a steel-blue color. When this is added to some of the filtered gastric juice it will be decolorized if the acid is pure hydrochloric; if lactic acid is present together with hydrochloric, the color will be changed to a yellowish tint, and if the lactic acid is in excess, it may even assume a reddish-yellow; whereas, in the absence of hydrochloric acid, it will have a more greenish tint. Though this seems to answer the purpose, it only shows the presence of lactic acid, without giving a

distinct idea as to the presence of hydrochloric acid.

For the purpose of showing the latter, Laborde proposed the use of methyl-violet; a watery solution of this is promptly changed by HCl to a greenish-blue color, but is not affected by lactic nor the fatty acids. It has, however, the disadvantage of being not sufficiently delicate, and the color-changes with weak acidulous fluids are not distinct enough to judge of small amounts of HCl. To test with it, some of the filtered gastric juice is placed in a small glass vessel (watch-crystal) placed upon white paper, and the test solution is allowed to drop on the side of the vessel and commingle with the fluid, when the change of color can be plainly observed against the white background. In a series of experiments to ascertain the limit of delicacy of these tests for HCl, I have found that the methyl-violet could detect in this manner 1 part of HCl in 2500 parts of water.

The next test, superior in delicacy, is a saturated aqueous solution of tropeolin; this yellow solution, when applied for the examination of gastric juice in the manner described for methyl-violet, will change to a beautiful red, or brownish-red with HCl, is but a little darkened by lactic acid, and very slightly so by the fatty acids. I have ascertained its limit of detection of HCl to be 1 to 3500, while 1 to 500 is the greatest dilution of lactic acid that will darken its color, and it is not affected by a 1 to 100 dilution of acetic acid.

A more recently introduced reagent is the phloro-

glucin-vanillin. Germain-Sée has made extensive experiments with this, and while it does not indicate organic acids, he claims for it a delicacy sufficient to indicate as little as 1 to 20,000 of HCl—a claim, however, which I have not been able to substantiate entirely from my own experiments.

It was found by Wiesner that if a pine stick was dipped into a phloroglucin solution, and was then touched with strong HCl, it turned a dark-red color. This reaction is caused by the presence of vanillin in the pine wood; and, therefore, this was substituted for it. The test is composed as follows: 2 grammes phloroglucin and 1 gramme vanillin dissolved in 30 grammes absolute alcohol. This yellowish solution gives at once with a stronger mineral acid a dark-red color with deposition of red crystals. To apply this test to weak acid solutions, such as the gastric juice, the fluid must be evaporated at a moderate heat, not reaching the boiling-point of water, to drive off the water and leave the HCl of sufficient strength to act on the reagent. To this end a few drops of the test are mixed with a similar quantity of the filtered secretion, and then slowly evaporated to dryness. If HCl is present the dry residue assumes a red color, or if present in very small quantity may show distinct red outlines only. In my hands this test, certainly a very valuable and delicate one, failed to indicate more than 1 part HCl in 10,000 parts.

The red Congo paper, as proposed by Prof. Riegel, is a most delicate reagent, and easy of application. It is readily turned of a bright greenish-

blue color by HCl, but is also affected by lactic acid, which changes its color to a dark dirty-blue; and acetic acid will darken it with a bluish tinge. With this reagent, as obtained from Merck in Darmstadt, I was able to detect as little as 1 part of HCl in 20,000 parts of water; and while 1 part of acetic acid to 3000 water gave a slight change in color with bluish edges, 1 of this to 5000 did not affect it in any manner; 1 part of lactic acid in 5000 water was indicated by it in traces, but 1 to 6000 failed to elicit a change in its color.

It is thus seen that the latter is perhaps the most delicate reagent we have, not alone for HCl, but as an indicator of general acidity, and that when it fails to indicate the latter a total anacidity may be fairly claimed. When it indicates acidity, the application of the other tests may become necessary, not alone to ascertain the character of the acidity, but to approximate the degree to which HCl may be present in cases of subacidity. The gradation and limits of these reagents, as pointed out by me, may be readily utilized to this end by dilution of the secretion up to the point where either of the above tests fail, and computing the HCl by the amount of the dilution.

The excess, normal presence, diminution, or absence of HCl being thus established, the digestive power of the gastric juice is now ascertained by the physiological test. To this end a small piece of coagulated egg albumen of about 7 mm. diameter and 1 mm. thickness is digested at the temperature of the body, and the comparative time necessary for

its solution noted. With normal secretion this should be complete in from one to one and a half hours, whereas under abnormal conditions a much longer period and the addition of HCl may be required.

To classify the different phases of chemical dyspepsia, they can be conveniently arranged as hyperacidity, subacidity, and anacidity.

Hyperacidity is the overproduction of HCl by the peptic glands, the amount reaching as much as 0.4 to 0 6 per cent. of the peptic secretion. Its *etiology* seems still obscure, and may be attributed perhaps to the chemical condition of the blood, and to the concentric rather than peripheral stimulation of the secretory glands, and it is a fact that at times it is found irrespective of food ingestion. While its *symptoms* are quite characteristic, consisting of pyrosis, acid eructations which perceptibly affect the teeth, occasional vomiting, and ptyalism, under such conditions commonly termed water-brash, its *diagnosis* is readily made out with the tube and subsequent acidimetry, and confirming the character of the acid with methyl-violet, which is promptly changed to a distinct greenish-blue.

As a chemical indication for the *therapy* of acid dyspepsia or hyperacidity, alkalies must rank first and foremost; amongst them the sodium bicarbonate deserves principal consideration in massive doses of from 30 to 45 grains every four hours, and before meals. Alvine evacuations, which are more or less suppressed in this condition, should be freely promoted by Carlsbad salts, or by appropriate preparations of rhubarb in laxative doses Hot

potations, which are often lauded in this connection, have, in my experience, proven of little avail, but free lavage with borax or sodium bicarbonate solution gives the greatest relief. As physiologically indicated the diet should consist of proteid substances, while carbohydrates must be studiously avoided.

SUBACIDITY. *Etiology.*—While in hyperacidity it was an excess of HCl which caused rather gastric distress than suppression of the peptic act, subacidity is generally observed in the catarrhal conditions of the gastric mucous membranes which are caused by improper food, excesses in drink or chemical irritants, and where, through irritation, the mucosa is hyperæmic and secreting abnormal quantities of alkaline mucus, diminishing the ostia of the peptic glands, and admixing the former with the gastric juice proper, thus preventing its proper action on the food-bolus. The common acute indigestions of our summer months, attributable to excessive drinking of iced beverages, belong to this class.

The *symptoms* are anorexia, thirst, slight fever, a sense of pressure and fulness in the epigastrium, occasional vomiting, and belching of gas, with eructations of fermenting food. A dry and coated tongue is generally observed in this condition, together with a bitter taste in the mouth. The bowels may be loose or bound, and by a progressive advance of the catarrhal process to the duodenum it may be followed by icteroid appearance.

Treatment.—This class of dyspepsia calls for rapid evacuation of the stomach, either by tube or emetic,

followed by local sedatives, such as bismuth and soda; while after subsidence of the mucous hyperæmia and a continuance of subacidity, ten to fifteen drops of dilute HCl in water prove of service given after food. During the febrile stage small pieces of ice by the mouth and iced milk diluted with some effervescent alkaline mineral waters prove most grateful and serviceable.

Chronic Gastric Catarrh.

The persistence or frequent repetition of ingestion of the causative elements of the acute gastric catarrh cause a chronic state commonly met with in subjects, termed "chronic dyspeptics," and, as the chronic gastric catarrh, is the cause of subacid disturbances of the digestive function. This may be, and often really is, gastro-enteritis; and, again, it is dependent at times upon other diseases and, therefore, only secondary in character. The cause of disturbance of the chemical composition of the gastric secretion hence must be looked for either in the altered chemical composition of the blood or in the changed anatomical relations of the glands.

The gastric mucous membrane in this affection is heavily coated with a grayish-white material, consisting largely of cast-off epithelium; the odor from the buccal cavity is, from this cause, more or less offensive, and the mucosa beneath the coating is red and hyperæmic. According to the stage of its existence, the mucous surfaces may be smooth or atrophic, and there may be an accompanying atrophy of the peptic glands and an increase of connective

tissue between them. Not infrequently hyperplasia of the mucosa exists, diminishing the ostia of the peptic glands, the hyperplasia extending at times even to the submucous tissue.

The main result of these anatomical changes consists in the reduction of the amount of HCl in the secretion, and with it the diminished digestive power and an insufficient inhibition of bacterial development, in consequence of which fermentation of the ingesta ensues, with copious eructation of gases and the formation of lactic, acetic, and other acids. In cases where the absence of HCl is very marked, the fermentative processes may cause vomiting, and the ejecta will often reveal to microscopic inspection yeast fungi and sarcinæ ventriculi, which are, however, of no special pathological consequence.

A feature of the chronic gastric catarrh is the impaired peristalsis of the stomach. This is dependent on the diminished amount of HCl present, as we know that in the normal stomach this acid is the principal exciting cause of gastric peristalsis. In consequence thereof dilatation results, and the degree of subacidity becomes more and more marked until almost entire anacidity prevails, as I had occasion to observe in several instances of chronic gastric catarrh during a recent investigation of a series of cases of this kind at the German Hospital of Philadelphia, made by myself and the medical resident, Dr. Gerlach.

As the physical examination of the stomach offers no characteristic points for *diagnosis*, the latter cannot well be established without the chemical exami-

nation, together with due consideration of the subjective and objective symptoms and exclusion of such pathological conditions which stamp the dyspepsia as secondary to some other affection.

The *treatment* of the chronic catarrh of the stomach as a primary affection should be largely dietetic, avoiding all food that is irritant, bulky, or slowly acted on by the gastric secretions. Thus, carbohydrates should be avoided, also fats, and principally alcoholic beverages. Milk, broths with raw eggs broken and stirred into them, and toast, should, in severe cases, constitute the principal food. As the tardy gastric digestion will favor fermentative changes, which in turn act as irritants and favor the accumulation of mucous secretion, the removal of undigested food, together with such bacteria as would incite decomposition *de novo* in freshly added food, is indicated in all such cases, and calls for mechanical treatment by lavage, which, though little practised in this country so far, has often worked wonders with chronic dyspeptics.

Amongst the remedial agents the chemical indication for the use of HCl would certainly be foremost, to correct the subacidity and promote the peptic process. When lavage is practised this indication will generally hold good, but when the milder symptoms do not indicate the use of the tube it will do no good. The reason for this is the large amount of mucus which is coagulated by the acid, and by enveloping the food particles prevents them from being acted upon by the gastric secretions. In these cases the alkalies, and among them the sodium bi-

carbonate, should be used, together with the vegetable bitters to excite active secretion; of these the tinc. gentian. comp., tinct. nucis vomic., tinct. colombo, etc., will prove of good service. Gastric antiseptics and antiferments can hardly be administered in sufficient quantity to do good, and very few, if any, are germicidal enough to correct an already fermented food-bolus. The constipation, sometimes so marked in these cases, is best overcome by Carlsbad salts or by some of the laxative mineral waters, or in more obstinate cases by pil. rhei comp.

Nervous Dyspepsia.

The influence of the nervous system upon the gastric digestion has been regarded as of great importance and has led to the special classification of "nervous dyspepsia."

That the innervation of the stomach may influence the gastric secretion, and that hyperacidity or subacidity may thus arise, is not to be doubted, but that special nerve influence could pervert the chemical act of digestion with normal secretion in the stomach any more than in the retort is not reasonable. The only neuroses appreciable to our present state of knowledge must be those of either sensory or motor disturbances. The reflexes causing intense neuralgic pains of the stomach may depend primarily upon perverted digestion, with or without motor implication, and are, as such, known as "gastralgia." In neurotic patients they may develop, even without peptic disturbances, from external influences upon

a morbid mind; but that with normal gastric secretion, the chymification of food should be interfered with by nerve influence can only be explained by the interference of reflexes with the motor innervation—*i. e.*, gastric peristalsis—accompanied either by pain or emesis, or both. The many subjective symptoms of the so-called "nervous dyspepsia" are certainly pure psychoses and cannot be classed with gastric diseases. It is doubtful if migraine is of gastric origin or due to intestinal indigestion, and also if the gastric neuroses, such as vertigo, etc., do not all, more or less, arise from this source.

The *treatment* of the neuroses of the stomach, as indicated by the character of the affection, must be either anæsthetic or else directed to the motor innervation. In the pure gastralgias, morphine hypodermatically will prove all that is requisite; in increased peristalsis with convulsive contractions and vomiting the bromides or chloral, or both, will meet the issue, while in atony of the muscularis nothing will prove as serviceable as nux vomica, calisthenics and the cold douche or surf-bathing. The subjective symptoms and psychoses, electing the gastric function for the toy of a morbid mental state, will yield to moral treatment and proper hygienic conditions if at all, but not to pepsin and kindred medication.

Ulcer of the Stomach.

Of the gastric diseases not directly arising from perverted peptic function, but at times seriously affecting it, is ulcer of the stomach.

The *etiology* of this disease is as yet obscure, al-

though experimentally it has been produced by embolic processes of the vessels of the gastric mucous membrane. Though its primary cause is not known or to a certainty proven by the experiments mentioned, there seems to be no doubt that it is indirectly caused by autodigestion of the affected portion of the stomach. The fact that the normal stomach resists the digestive power of its secretions has been variously attributed to the alkalinity of the blood circulating through it and, also, to the vital resistance of the living tissue against the chemical process. The latter seems such a speculative assumption, based upon an absolutely undefined factor, that it might be far better to adhere to the more rational theory, which is certainly substantiated by experiment and the observation that the dead stomach as well as that cut off from circulation, digests itself.

The gastric ulcer is of circular outline and spreads as well to the periphery as to the fundus, so that with clean-cut edges it narrows down to a funnel shape. The fact that it is generally clean is due to the erosive action of the peptic secretion; it may be superficial only, or reach the muscular and serous coats, even perforating the viscus entirely. While generally solitary, it is multiple at times, and, though principally met with in the pyloric region, it is also occasionally found in the lesser curvature and, as a rule, on the posterior wall, which accounts for the agglutination of the stomach in such affections with neighboring viscera. The ulcerative process and erosion may affect smaller or larger vessels and thus give rise to the characteristic hæmatemesis.

Symptoms.—Though ulcus ventriculi may not in all cases be accompanied by characteristic symptoms, there are a large number of cases which exhibit phenomena to warrant suspicion of the existence of ulcer. Of these, localized pain is, perhaps, the earliest, with gastric irritability and, probably, vomiting. The pain may be localized or diffused, or of a neuralgic character, referred to the epigastrium or back, or radiating; it is often relieved by a change of position, and, if this results from recumbency on the left side, its location is most probably in the pylorus. The localized pain increases with ingestion of coarse food, and, if found circumscribed on pressure, this would also form an important factor in the diagnosis. Though the emesis accompanying these subjective manifestations is of some additional diagnostic value, it is by no means a frequent concomitant and not characteristic of ulcer, unless accompanied by blood in sufficient quantity to stamp it as hæmatemesis.

The blood from erosion of a vessel by gastric ulcer is, however, not always ejected from the stomach, but appears at times in the dejecta, imparting to them a dark and tarry appearance. The vomited blood is generally clotted, mixed with food particles, and acid reaction in contradistinction to the light, frothy-looking blood of hæmoptysis. Gastric hemorrhage may range from a few ounces to several pints, and, when very copious, may be, and occasionally is, the cause of fatal results. The dyspeptic symptoms of gastric ulcer are of no diagnostic value, and arise from accompanying conditions rather than the

disease itself; but a most general symptom connected with it is the hyperacidity described by Riegel, and since confirmed by others; this is of great importance in the indication for treatment.

The *prognosis* of ulcer of the stomach may be said to be favorable in the majority of cases, as to direct results. The indirect sequel from cicatricial stenosis of the pylorus we will consider under the head of dilatation. The incidental perforation or exsanguination are factors which make the prognosis very grave, if not necessarily fatal, but fortunately arise in isolated instances only.

The *therapy* of ulcer of the stomach, as indicated by its pathology, would preclude, before all, the ingestion of solid food and, in fact, of anything that would excite or promote peptic secretion. I consider milk diet in such cases with hyperacidity as little calculated to benefit the patient, as the firm coagula of acid casein not only stimulate peptic secretion but offer great resistance to gastric peristalsis and still further obstacles to subsequent pancreatic digestion, thereby causing intestinal indigestion and thus defeating the very object of nutrition. Broths, with raw eggs broken and stirred into them, which are capable of uniting with free hydrochloric acid and of diminishing the peptic power of the free acid, as recently proven by Felix O. Cohn, offer certainly a more rational diet, readily digested and assimilated. I have succeeded better with total abstinence from food and rectal alimentation than by any other method. Thirst is to be quenched with ice, pain controlled by morphine hypodermati-

cally, while the hyperacidity is overcome by sodium bicarbonate in massive doses.

Absolute rest is of utmost importance both physically, mentally, and as a matter of course as far as the stomach is concerned. Hot fomentations to the epigastrium arrest paroxysms of pain, while ice-pills per os and the ice-bag locally will assist in the treatment for hemorrhage. The latter is best combated by hypodermatic injections of ergotin or scruple doses of gallic acid frequently repeated. Nausea and vomiting are met with ice-pills, chloral, creasote, or Lugol's solution. Perforation and the results therefrom may possibly be benefited by surgical treatment, but too little as yet is known regarding this to base much hope on its success.

Dilatation of the Stomach.

Dilatation of the stomach is a pathological condition generally, if not probably always, accompanied by subacidity, and in a large number of cases with anacidity.

Only a minority of cases of this kind are primary dilatation—*i. e.*, that do not depend upon some other morbid affection of the stomach; the majority are secondary, in so far that they are consequent upon stenosis of the pylorus, which in turn may be either benign or malignant—the former arising from cicatricial contraction after ulcer, the latter due to the presence of carcinoma in that region. There may be also stenosis from mechanical pressure of near-lying tumors (floating kidney, Bartels), which, in

turn, like all obstructions in that locality, occasion dilatation of the stomach.

Gastric dilatation without pyloric obstruction is sometimes noted in, and consequent upon, chronic catarrh of the stomach, but never occurs then in that degree as in the variety resulting from the former cause. It depends upon paresis of the muscular coat resulting from general debility or overdistention by ingested matter, and is frequently noted in those who indulge to excess in the pleasures of the table and the cup. I have had occasion to observe it more in connection with the chronic gastric catarrh of beer-drinkers than of any other class of patients, and it is also claimed to be frequently found among diabetics.

The *pathology* of gastric dilatation is to be considered as similar to that of cardiac dilatation in aortic obstruction. The muscular fibres of the stomach, to overcome the pyloric constriction, first become hypertrophied; the accumulation of food soon produces paresis of the muscular coat and dilatation results, which increases in the same measure as the chemical peptic act is diminished, and the fermentative changes of the food-bolus produce irritation and inflammation of the mucosa. The latter, under such conditions, fails to secrete a sufficient amount of hydrochloric acid, which is the excitant to gastric peristalsis. As a consequence of overdistention, the mucous coat suffers anatomical changes, the ostia of the peptic glands—which at first appear almost hypertrophied and extend beyond the sur-

face of the mucosa—soon atrophy, and with it the secreting power of the gland becomes less and less.

It was thought at one time that anacidity was pathognomonic of gastric carcinoma, but that has been certainly disproven, and it can only be possible when pyloric cancer, and consequent obstruction, produces dilatation of the stomach. Any considerable dilatation must necessarily result in subacidity, and if existing for some time and in increasing ratio, or from complete pyloric stenosis, it must result in anacidity. This has been disputed and HCl has been claimed as being present in not merely distended stomachs but also in the overdistention or complete dilatation from pyloric obstruction, but it seems unlikely, and when we consider the diagnostic difficulties for defining distention, or even sometimes complete dilatation, the error may be readily looked for rather in the diagnosis than in the pathological condition. We may justly assume that anacidity is not a symptom accompanying any special gastric disease, but in all probability always results from complete dilatation from whatever cause this may happen. While in the functional anacidity from gastric catarrh, etc., this may exist for the time being and is, no doubt, also the result of dilatation, it being a paresis of the muscular coat rather than a paralysis of the fibre as in complete dilatation, the chemical character of the gastric secretion can be restored by a removal of the vicious element.

The *symptoms* of gastric ectasis are those of apepsia generally, anorexia, gastric pressure, eructations,

pyrosis, and vomiting. The latter is somewhat characteristic, as it takes place at longer intervals only, when the ectasis has assumed great proportions and fermentative changes exert their irritation upon the mucous membrane. According to the amount and quality of food taken, it may happen only once in twenty-four hours, or in three to four days and even longer periods. My friend Dr. J. M. Barton ingeniously ascertains the number of meals comprised in the emetic act under such conditions, by giving a number of raisins with each meal and counting their number when rejected from the stomach. To arrive at the degree of stenosis he gives both raisins and a few currants. If the latter are not ejected in the vomit he concludes very justly that the pylorus is partly pervious and deduces the amount of constriction from the size of the currants. The ejected matter is usually copious, is readily vomited without much nausea or straining, and patients feel a great sense of relief after it.

The *diagnosis* is not always as readily arrived at as might be judged from the number of mechanical means for this purpose. The objective examination by palpation and percussion leads in well-marked cases at times easily to a satisfactory conclusion, especially if the stomach is filled by fluids or gases. To that end it is often of great service to give in different potions about thirty grains of sodium bicarbonate and tartaric acid, when, as a rule, the inflated stomach may be palpated with facility. If, in addition to this, a quantity of water, about eight to twelve ounces, is swallowed and the line of dul-

ness by percussion is located below the umbilicus, the diagnosis is pretty certain. Adhesions from perforating ulcer or carcinoma may, however, embarrass the result under such circumstances. Perhaps the most satisfactory method for ascertaining the capacity of the dilated stomach in complete pyloric stenosis is to fill the stomach with water through the tube and to measure the fluid as siphoned out therefrom. In atonic dilatation or with perforating ulcer or carcinoma this, also, is valueless. The examination of the stomach for dilatation by means of the sound does not possess the value claimed for it, as displacement of the viscus may simulate the pathological condition in question.

The general condition of the patient suffering from gastric ectasis is always more or less impaired; emaciation rapidly takes place; the bowels are obstinately constipated and will not be open for days and weeks; the urine is generally small in quantity and of neutral or alkaline reaction. The total absence of hydrochloric acid from the stomach in connection with the other symptoms above enumerated would certainly point very strongly toward an existing dilatation as probably arising from pyloric obstruction. Functional dilatation may be accompanied by subacidity, and in these cases the existence of dilatation together with the degree of ectasis can be arrived at satisfactorily only in a minority of the cases.

The *prognosis* of atonic or functional dilatation of the stomach may be viewed as favorable—*i. e.*, readily yielding to proper therapy. Gastric ectasis

from cicatricial pyloric stenosis is more serious, but with proper management much can be done toward keeping up nutrition and preserving life. The recent operative treatment in this condition, as practised by Loreta in Italy, and Bull and Barton in this country, has been so successful that a great future may be promised for it. The prognosis of dilatation from carcinomatous stenosis of the pylorus is most unfavorable, though pylorectomy has produced some favorable results.

The *treatment* for atonic gastric ectasis is one that depends largely on a proper diet. Albuminous substances, such as eggs, broths, or scraped meat, should be freely exhibited, followed by dilute hydrochloric acid with strychnine or nux vomica. Carbohydrates should be prohibited, nor should milk or other beverages be taken in quantities; saline laxatives are best indicated to keep the bowels solvent; the faradic current applied to the epigastrium will prove of vast value; the catarrhal condition should receive attention, as pointed out under that head.

In the graver or secondary dilatation the treatment must depend upon the removal of the accumulated food by means of the stomach tube and the daily repeated lavage. While the pyloric stenosis is not directly benefited by this, the hyperæmiá and œdema accompanying it can be much relieved by this treatment. In a case now under my care in the German Hospital, complete stenosis no doubt existed; no alvine dejecta had passed for about two months, all of the food taken was vomited at inter-

vals of from one to two days; the patient's weight from 156 pounds sank to 89 pounds, and corresponding debility was manifest. By frequent lavage and the administration of albuminoid food with HCl, and nutritive enemata of carbohydrates (glucose), the patient gained in strength and weight. At one time during the treatment he washed out through the tube an apple-seed, though he had not eaten apples for two months past. Alvine discharges soon became regular, and are now formed, while the patient's weight to-day is 129 pounds.

Though I do not regard the stenosis in this case as cured, the obstruction arising from congestion or œdema must have been certainly relieved to admit of good gastric digestion, and the dilatation has undoubtedly been improved, as the gastric secretion is beginning to show traces of HCl, and the patient has appetite and digests food without medicinal aid.

That all of these cases should ultimately be treated in a surgical way, I fully believe, and when I consider the good result achieved Dr. Barton in his recent case, patients should be encougaged to that end. Much, however, can be done medically to relieve the patient, and this should be exhausted or done preparatory to operative interference. It has been my habit in such cases to wash out the stomach daily with 3 to 4 pints of a dilute solution of borax, to give broths with egg and scraped beef as nourishment, followed by HCl with nux vomica; and to administer enemata of from 4 to 6 ounces of glucose thinned with a little warm water and admixed with ½ to 1 ounce of beef peptones.

In the dilatation depending upon carcinomatous stricture of the pylorus, little is to be hoped from treatment of any kind other than intended to palliate the pain and relieve the most urgent symptoms.

Cancer of the Stomach.

Cancer of the stomach is the gravest and most fatal of all gastric affections. The *etiology*, like that of all malignant neoplasms, is entirely obscure in this affection as well, and though Hauser called attention to the atypical epithelium arising from cicatrices of gastric ulcer, no direct connection between the two has as yet been established. It occurs generally between the ages of forty and sixty, but much younger persons are found suffering from it at times; the hereditary tendency has been received with some credence; the duration may be from one to two years, rarely more.

Gastric carcinomata are quite frequent, and it is claimed that they comprise about one-third of the total number of malignant growths. They occur more generally in the pyloric region, next frequently in the lesser curvature, but are also found in the fundus and cardia. Arising from the mucous coat they present either as circumscribed tumors or diffuse infiltrations progressing to the submucous and muscular coats, and are sometimes the cause of adhesions to neighboring viscera. The scirrhous, medullary, and colloidal are the principal varieties observed; they are, as a rule, in the advanced state,

broken down on their surface, which is clean, owing to the digestive action of the peptic secretion.

Symptoms.—Their effect upon the patient marks a steadily downward course, leading to rapid emaciation, cachexia, and anæmia; the dyspeptic symptoms are not always alike, but are probably the first to occasion complaint—consisting of anorexia, gastric pressure increasing to pain, and in many cases, but not in all, there is characteristic vomiting. Hæmatemesis is perhaps more frequent, but not nearly as copious as in ulcer, and the small quantities of blood are generally reduced to hæmatin, which gives the ejected matter the appearance of coffee-grounds. To confirm the presence of blood the hæmin test should be made, and the Teichmann's crystals of hæmatin hydrochloride developed for microscopic recognition. The vomited matter rarely, if ever, shows cancer cells or particles characteristic of cancerous structure, and the sarcinæ, etc., are seen, as in other cases of dilatation or apepsia.

An important symptom accompanying many cases of gastric carcinoma is the anacidity frequently observed in connection with it. While it was held by von den Welden that this is characteristic of cancer of the stomach, it was found that in some cases of carcinoma there was not alone no anacidity, but even hyperacidity. The ingenious experiments of Riegel, which demonstrated the impairment of normal gastric juice by the proteid character of carcinomatous fluids, are negatived by the recent experiments, which show that all albuminous substances

will cause the same result. In my opinion only such cases of gastric cancer can produce anacidity as cause pyloric stenosis and dilatation in consequence thereof; and that carcinomatous disease in other parts of the stomach may not only not diminish the HCl acidity of the gastric secretion, but like gastric ulcer may actually increase it—an important point in the diagnosis of location.

The palpation of the stomach leads often to the *diagnosis* of tumescence directly attributable to malignant disease, but the absence of tumescence is by no means a proof of the non-existence of the former. Percussion of the stomach is of little, if any, positive diagnostic value. The alvine discharges are much inhibited in the pyloric obstruction from cancer, but otherwise offer no diagnostic points, nor does the urine. The heart and pulse bear the general character of anæmia and inanition, but have no other bearing on the pathological condition.

As we have already indicated, the *treatment* of gastric carcinoma can at best be one intended to palliate the suffering and meet the symptoms. These latter have been already referred to in the treatment of dilatation from pyloric obstruction, to which should be added morphine hypodermatically for pain; and for the obstinate vomiting, ice, chloral, creasote, or Lugol's solution. Lavage must here also be the sheet-anchor; proteid diet, rectal alimentation, and supporting treatment of all kinds, and stimulants, especially effervescent wines, are about the only means to prolong the existence of suffering to which these patients seem condemned.

It is as yet a matter of doubt if operative interference in pyloric carcinomata will prove of great value. That some of the cases so treated have done well throws out a ray of hope, which, even if it does not realize curative results, for a time relieves the sufferings incidental to this disease.

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