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Non specific chronic gastritis

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A Senior Thesis on

NON-SPECIFIC CHRONIC GASTRITIS

by

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1933

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NON-SPECIFIC CHRONIC GASTRITIS

Introduction

A few pronounce it non-existent and the cloak of ignorance; a few consider it the ultra-ultra in factors of all gastric diseases; but the majority consider it just "an inflammation of the gastric mucosa of long duration associated with some other pathology of primary importance," ignoring the possibility of there actually being a disease analogous to diseases of other glandular organs as hepatitis and nephritis.

From a survey of the accessible literature it is readily noted that few conceptions of disease have suffered such vicissitudes as that of gastritis. Over 100 years ago Broussais, Andral and Louis considered it the cause of nearly every pathological phenomenon, but they committed the general error of using autopsy findings as their criterion, calling it "catarrhal inflammation" of the stomach. At about the same time Barras was differentiating nervous gastralgia and dyspepsia from gastritis and thus producing confusion everywhere, especially when Carswell in 1838 demonstrated definitely that the "catarrhal" necropsy findings were simple results of auto-digestion and putrefaction. With the advent of the stomach tube and test-meals and thus the physiological examination through the initiative of Kussmaul and Leube, gastritis and catarrh of the stomach were less discussed and diagnosed because mucous was rarely demonstrated and mucous was considered the criterion of catarrhal inflammation. Instead, they attached undue significance to the two outstanding secretory disturbances, hyperchlorhydria and anacidity, information which was little understood and erroneously elevated them to the status of diseases

However, opinion was considerably altered between 1890 and 1900 when it became possible to preserve the stomach by injecting fixation fluids immediately after death, either intraperitoneally or via stomach tube. This was the work of Hayem who used a potassium bichromate solution, and Faber and Bloch who used a 10 per cent formalin. By this preservation it was soon clear that inflammatory changes were frequent and important.

One should hesitate before criticizing the man before 1900. His diagnostic horizon and clinical application was limited because he was practicing at a time when basic considerations of enzyme activity were wanting, and qualitative estimations of proteolysis were crude and inaccurate; when there was an absence of such means of diagnostic refinement as the fluoroscope, cholecystogram and calorimeter; when they failed to employ such secretagogues as alcohol, histamine, and bouillon-test meals, and when they failed to employ fractional gastric analysis and lacked a means of estimating hydrogen-ion concentration. In these days where the acute phenomena associated with ulcer, gall-bladder and appendiceal inflammation have so dominated the clinical mind, there has been a tendency to overlook the sufferers in whom chronic changes in the mucosa of the digestive tract are apparent. The practitioner's failure is not so much due to lack of diagnostic refinement as it is due to lack of routine employment. True that many of the methods are not practical as yet; but why not utilize those that are and know all you can about your patient ?

The writer in limiting this paper to "non-specific" necessarily excludes linitis plastica, and that of tuberculosis and syphilis.

Etiology

As long as the patient is alive, the thing of greatest importance regardless of the pathological condition is the physiological function. Rehfuss (35) points out that the present classification is a pathological one which places it as a thing of the post-mortem room and the laboratory and not a classification likely ever to be reached before the gastric mucous membrane is bottled up and the patient dead and gone. He collected some 212 definite causes of chronic inflammation of the stomach, ranging all the way from the multitude of direct causes, including dietary indiscretions to the secondary forms of gastritis, secondary to disease of nearly every visceral organ as well as focal infections in various parts of the body.

The conclusion to be drawn then is that an etiologic cause is only of value from a therapeutic point of view, and not because it produces a specific form of gastritis. It then seems plausible to classify so that the etiologic consideration will be of value in aiding you to restore the physiological function rather than in helping to make a diagnosis.

Because of the different therapeutic approach it is of paramount importance to first determine whether the gastritis is primary or secondary. Secondary gastritis represents a large chapter in gastric-medicine. Chronic passive congestion, whether from myocardial insufficiency, cirrhosis of the liver or lung changes such as emphysema, is prone to impair the secretion, motor function and resistance to infection so that the mucosa is more susceptible to influences favoring gastritis. It is found with many chronic diseases, such as pulmonary tuberculosis, malaria, nephritis, leukemia, gout, cholecystitis, pelvic disease, and diseases of the stomach such as ulcer and cancer. Jones (24) favors the theory that

the gastritis is due to a lowering of the mucosal resistance, but Alvarez (1) feels that hastily blaming the disturbance upon the mucous membrane is to error because most of the trouble can be easily demonstrated as a breakdown in motor transportation. But ~~ir~~regardless of the mechanism, I believe it can easily be seen that the gastritis is of minor importance (yet not to be forgotten) until the primary condition has been brought under control. I feel that the rightful place for the consideration of secondary gastritis is at the time of study of the primary diseases and so this mere mention of the possibilities suffices at this time.

It is the primary gastritis that is of great interest. Faber (11) makes note of the importance of keeping in mind its dual etiology, whether due to direct irritation or to toxic-hematogenous. In the former, there will be found a more localized pathologic condition, especially in the pyloric region, as found in cases of food indiscretions, alcohol excess, etc. In the latter, there is a tendency towards a pangastritis as a result of acute infections, etc.

More and more the influence of heredity is being considered in the study of clinical medicine. Rehfuss (34) and Crohn (8) speak of familial predisposition in the form of an idiosyncrasy to certain foods. Hurst (23) supports an achlorhydric gastric diathesis which he describes as an obscure congenital deficiency. Although we must not let ourselves become "diathesis minded," it is certainly evident that there are marked variations between the digestive capacities of different individuals and that familial gastric debility should be considered along with environment. Our attention is called by Alvarez (1) to the fact that savages do not become uncomfortable when they eat large amounts of uncooked and decomposed foods. *How would he know?* Verbruycke (37) noticed that the stomach of a person

below par physically reacts quicker to fatigue producing large amounts of mucous, thus supporting the theory that the asthenic has a tendency which others do not have.

I fail to recall a single article discussing the etiologic factors of primary gastritis that did not speak of dietary indiscretions as the major offense. Some of the more common are : Improperly masticated foods due to missing or carrous teeth and badly fitting plates and bridges; improperly selected and prepared foods; unnatural heavy protein diets; snatched lunches; inordinately prolonged and heavy dinners with heavy desserts; irregularity of meal time; bolting of foods; gourmandism; emotional upset; thermal insults, too hot or too cold; excess use of spices and may others even to the excess use of coffee and tea. Theoretically, it sounds plausible but it makes us wonder if we do not lay too much importance to the diet when a man such as Alvarez (1) suggests that we must remember that primitive men used to eat irregularly, whenever they succeeded in making a kill, and that it is only during recent times and with advancing civilization that regular hours of eating, fixed volume, calculated calories and definite vitamin, mineral, etc., contents have become possible.

Alcohol, tabacco and toxic irritants, particularly medicaments, are listed as playing important roles in gastritis. Crohn (8), Rehfuss (34) and Hurst (17) consider alcohol as not only a common but the factor producing the most classical form of chronic gastritis. Hurst goes as far as pronouncing it the most common cause of primary gastritis. Although he remarks about it seeming to be the most definite cause, Alvarez (1) calls attention to the work of Hirsch. With the most careful technique,

Hirsch (16) was unable to find any signs of acute or chronic inflammation in the mucous membranes of the stomachs of a series of drunkards, some who had died of delirium tremens and some who had died of other causes. Tobacco likewise comes in for its share of criticism. Hayem (34), Lickint (29), Hurst (17) and Rehfuss (34) are all convinced that it is an active cause due to its disturbance of secretory function along with its effects on the cardiovascular and nervous systems. The toxic effects can be better realized by observing the effects of tobacco on those who have never smoked. But then you question the duration and seriousness of the effects when you consider the enormous numbers using tobacco and yet seemingly enjoying good health. Ingestion of medicines over a long period of time is considered by many as a cause but I am in sympathy with Alvarez, Eggleston and Hatcher that the upsets probably cannot be assumed to be due to the presence of such pathology; but are the effects on other parts of the body. If such pathology is present, it probably was initiated by some other condition and the drug is just adding "insult to injury." Of course, caustics such as bichloride of mercury were not included because their actions are known.

Sex, season, and climate hold little or no sway. However, age is of greater importance as the frequency is much higher during middle life when the stress and strain is first manifest. Dedichen (10) showed that 66 per cent of inmates of institutions for the aged poor in Sweden, from 67-92 years of age, had achlorhydria; that is, they had lost their secondary defense to ingested bacterial invasion. But, Kerley (25,26) presents cases of children with gastritis, evidently due to pyloric spasm with retention and to misconduct of nutrition of overly enthusiastic parents; so, it can be expected in all ages.

For two reasons the consideration of achylia and achlorhydria as etiologic factors have been reserved until the last. First, although there is acknowledged to be a definite relationship between gastritis and achylia and achlorhydria, there is a severe controversy as to which is the sequela. Is the gastritis the result of achlorhydria or is the achlorhydria the result of a gastritis? Faber (11) is a strong advocate of the latter and Hurst (17), although he admits the plausibility of Faber's view, is convinced that an hereditary, familial conditions may exist, a functional achlorhydria primarily, though it may be less common than believed at one time. So it is as yet undetermined definitely as to whether achlorhydria is an etiologic factor or a sequela. The second reason is that in their investigations of achlorhydria they uncovered a finding proposed as the basic consideration in all gastritis cases. An achylia in 1-2 per cent and an achlorhydria in 10-16 per cent were found by Bennet and Ryle (19) and Hurst (17) and many others in a large group of students and individuals presenting no symptoms or complaints, in fact enjoying good health as nearly as could be determined. Thus it was concluded that from 10-20 per cent of all individuals have a "constitutional achlorhydria or achylia" and it is these that are potential gastritis cases. They are more subject to the exciting causes enumerated above, because they do not have their secondary defense. They further assume that the other 80-90 per cent having a gastric secretion within the "normal" limits are thus sufficiently protected by the diluting and antiseptic action of the gastric juice to prevent the invasion of the swallowed bacteria of oral sepsis, purulent sinusitis and contaminated foods. It has been shown by Knott (27) that the increased alkalinity due to the achlorhydria allows

bacteria which normally are confined to the colon to ascend even to the stomach. Even though a very enthusiastic sequential picture with ultimate gastritis may be presented by these men, it must be "taken with a grain of salt;" but not to the extent that the increased vulnerableness of the mucous membrane to irritation and infection is not appreciated. It brings up the important question of whether gastritis and its sequelae would be more readily recognized or even exterminated if the hydrochloric acid percentage was determined for each and every patient irregardless and then a classification of these individuals made and prophylactic measures instituted accordingly.

Pathology

It has been suggested by Goldsmith (15) that death probably does not affect the entire body simultaneously, although in a practical way we may consider the individual has died when his respiration and heart beat have ceased. It would then seem possible or even probable that during the last hours, with waning vitality, changes may occur in the mucous membrane of the stomach while the patient is still alive and thus cloud the true pathological picture we think we are getting with the 10 per cent formalin injections. However, there is a rather definite uniformity of the findings from the best methods of technique that we know.

In general the gross examination will show the fundus nearly always normal. Only in severe toxic-hematogenous with an advanced atrophic form of pangastritis with achylia gastrica is the whole stomach uniformly diseased. The distribution of the lesion is not uniform, but decidedly uneven and patchy. No two of the abnormal areas will be identical in a given case. Hurst (22) states, "It is indeed possible to find in

different parts of one specimen, atrophy, excess regeneration, superficial ulceration of the mucosa, polypoid growth, and hemorrhagic erosions. The most constant gross finding is a thickening and a palpable hardening or stiffening of the stomach wall in the pyloric region."

On gastrotomy, the first thing to be encountered is a layer of slimy mucous adhering to the inner surface. No peptic ulcer can be found but minute superficial hemorrhagic erosions may be present, occupying the apices of slightly rounded projections, or appearing as tiny slits between the folds of the mucous membrane. "Worm-eaten patches," superficial irregularly-shaped areas of partial destruction, are often seen, especially in the pylorus and the antrum.

Microscopically are found all stages of transition, from diffuse inflammation with no glandular lesion to complete atrophy. There is a more or less pronounced lymphocytic infiltration of the upper layers beneath the mucous epithelium and between the glands, yet the glands may remain anatomically well preserved. This is important from a therapeutic standpoint because functional restoration is possible. As the inflammation progresses, the glands begin to show signs of injury, becoming less numerous and appearing separated from each other by inflammation products. Atrophy then progresses until they become mere vestiges, finally disappearing altogether. By this stage therapy is useless as far as restoration of function is concerned. The mucous membrane now consists of granulation tissue covered by the gastric surface epithelium, a complete anadenia. There are other changes seen. These lymphocytes may infiltrate until follicle-like masses of round cells may develop into typical lymphoid follicles, resulting in "follicular gastritis." In other cases,

cysts form in the degenerating glands; may even be the predominant feature. Not uncommonly the surface epithelium of the stomach is converted into intestinal epithelium, characterized in particular by goblet cells with mucous, such as are never found in the normal stomach, according to Faber (11). A fairly constant and striking feature is the apparent increase in the number and distention of the blood vessels in the submucosa, serosa, and mucous membrane but may later be obliterated by connective tissue contraction.

The above picture is essentially that of primary gastritis but may be applied to secondary with the primary lesion as cancer or ulcer superimposed.

Symptoms and Signs

Chronic gastritis rarely gives rise to gastric symptoms which are sufficiently serious to call for investigation, especially in the early case. Some other associated condition, as achlorhydria, is often the cause of various other conditions which leads the patient to seek medical advice. On the other hand, there are cases who constantly complain of their stomachs, and are inclined to become irritable and uncomfortable. Because experiments on animals have shown that an inflammation in any part of the wall of the bowel will produce disturbances of digestion and give symptoms, Alvarez (2) is of the opinion that many of the latter group's symptoms are probably due to an enteritis, associated or unassociated. We find, Hurst, Rehfuss, and others believe that in general there are usually few to no symptoms from the gastritis; but we must wait until the associated conditions are severe enough to produce the symptoms. Thus, the earlier or symptomless case depends mostly on careful stomach examination.

You might say that the so-called classical gastritis case is characterized by no characteristic subjective findings. We are dealing with a disease of insidious onset from which the patient complains of lassitude, progressive debility, loss of weight, distress, discomfort, and capricious appetite, or even anorexia. However, unlike symptoms associated with lesions further along in the digestive tract, chronic gastritis is accompanied by its active symptoms during the digestive cycle.

The pain is not characteristic, being more of a feeling of unpleasant fulness around the waist, incident to eating. The duration of this discomfort depends upon the condition of the motor function, according to Alvarez (3) and Jones (24). If the motor function is unimpaired, the pain and distress leaves as the stomach is emptied, within 1 to 2 hours. But, if the motor function is slow, the distress may persist to the next meal. There may be an aching or driving sensation between the shoulders or across the small of the back.

Nausea, with belching of gas and eructations of bad tasting and sour fluids is considered a constant symptom by Crohn (8). Hurst (20) found that nausea is more marked in the morning with an absolute distaste for breakfast. In the more severe cases, associated with this morning nausea, there is often found retching and vomiting upon arising. It usually takes place in a fasting stomach but may occur after the meal. Fenwick (8) became so interested in the vomitus that he made a detailed description. The vomiting temporarily relieved the nausea when it occurred after the meal although it never completely emptied the stomach. A marked thirstiness was noted by Work (38), occurring in both those with

and those without vomiting. Hematemesis is not found according to Fitzgerald (12), although Konjetzny reported a fatal case of hematemesis in which gastritis with no ulcer or cancer was found at autopsy.

Either constipation or diarrhea may be present or there may be an alternation of the two develop. Alvarez (3) states that Jonas has shown just as hypermotility may cause a rapid emptying of the stomach, hypomotility may slow the bowel movements and cause constipation. Occult blood is not an uncommon finding in the stool.

The physical findings are essentially negative. The patient will have a fetid breath and a coated tongue. Bassler (6) describes the tongue as being "red on the tip and margins with a triangular-shaped coating on the dorsum, or the entire tongue may be soft, pale, and flabby looking, showing serrations from the teeth and generally covered with a thin furry coating." The epigastrium may be distended and hypersensitive. If there is tenderness it will be epigastric, rarely diffuse, disappearing as the meal leaves the stomach. An atonic stomach may give the "splashing." The general objective picture may or may not be that of debility, malnutrition and even an anemia.

Not only in the above mentioned symptomless cases but also in these cases with a classical picture is gastric analysis of great value. It is a measure of gastric work and gastric work includes two factors -- one mucosal work and the other sphincteric and muscular work. The former is estimated in terms of gastric secretion in extent, quality and quantity. The latter is estimated in terms of evacuation and degree of chymification. Also, it exposes the presence of any pathological secretion or excretion.

In general there is a tendency towards a progressive lessening in secretory work, although in the beginning the phenomena may be those of irritation and excess. And with this gradually downward change appears

hand-in-hand evidence of mucous transformation and the presence of excessive mucous output.

Rehfuss (34) contends that a careful study of the gastric chemistry is the only measure we possess of determining the intactness of the gastric mucosa; because for all of the possible chances of error, repeated analyses on successive days in experimental work with students have proven to be remarkably constant as to form. The importance of gastric findings will be further considered in the diagnosis, prognosis, and treatment.

Complications and Sequelae

Probably the more immediate complications of gastritis that must be considered are hemorrhage, pyloric stenosis, and perigastric adhesions. Hemorrhage is not common, although Konjetzny reports a fatal case. The loss of blood if present will usually be in a slow seeping manner. Pyloric stenosis of a mild degree may be expected to be caused by the inflammatory spasm, hypertrophy of muscle, and contracture, according to Fitzgerald (12). It may be sufficient to cause a rather severe retention. The adhesions if present are most prominent around the pylorus.

However, the more serious and thus the sequelae of greatest interest are carcinoma, pernicious anemia, and ulcer. Recent literature indicates that some of the more prominent clinicians are becoming more thoroughly convinced that cancer results from an atrophic gastritis rather than precedes it. Hurst (9) gives it as his opinion that 75 per cent of the cases of carcinoma of the stomach are secondary to chronic atrophic gastritis. Konjetzny (9) makes it even more sweeping, stating he has found evidence of inflammation in 90 per cent of cancer cases and believes the gastritis is a cause and not an effect. Ramond (32) and Haudek (9) agree

that gastritis is a prominent precursor to cancer. In his writings, Hurst (19,17) feels he has a great deal of evidence which indicates that cancer of the stomach is never a primary disease, but is always secondary to either chronic gastritis or, less frequently, to chronic gastric ulcer. He has never seen a case of cancer in which free hydrochloric acid was present at an early stage and disappeared as the disease advanced, and he has been unable to discover any record of such a case. On the other hand, he has seen three cases of gastric ulcer in which a test meal was given both before and after the onset of malignant degeneration; in each case, the acidity, which was high in two and normal in the other, was undiminished when cancer had developed. Ensterman (10) does not agree with Konjetzny, Hurst, Ramond, and Haudek. All the facts we know go to show that Billroth's dictum is correct, cancer never arises from normal tissue. And so as Davis (9) states, whether or not we are ready to give assent to the views of Hurst and his supporters, it is amazing how neatly they fit into the known facts. "The most common pathology found in the stomachs of people past 40 years of age is gastritis; it is, therefore, the most likely cause of carcinoma."

In a paper published in 1926, Hurst (20) showed that achlorhydria, which is an essential predisposing cause of Addison's anemia, is not necessarily due to constitutional achylia gastrica, but may result from achylia secondary to gastrectomy, atrophic gastritis, or chronic gastritis in which atrophy of the mucous membrane is not necessarily present. Not only does he (20) consider the achlorhydria due to chronic gastritis as well as constitutional achylia gastrica as the cause of pernicious anemia, but an important factor in the production of all intestinal infections, chronic appendicitis, cholecystitis, and even rheumatoid arthritis.

Ramond (32) believes that ulcers as well as cancer of the stomach develop from chronic gastritis.

Diagnosis

The more gastroenterologists learn about cholecystitis, peptic ulcer, carcinoma, and chronic appendicitis the less frequently do they dare make the diagnosis of chronic gastritis. It is now a diagnosis made from exclusion which certainly means nothing but an exhaustive systematic study of the case !

Most men advocate the usual three stages of diagnosis be made in the following order : History, physical examination, and special methods. I agree with the necessity of an exhaustive application of each of the three stages, but I disagree with the sequence of their application. In belief that an increased sensitiveness to the presence of pathology may be developed if the physical examination be conducted before your diagnostic horizon becomes biased by the patient's history, I suggest the history be taken after the physical is made. In this physical examination you are attempting to find whether or not there is evidence of pathology in any part of the body and not just that related to the gastro-intestinal tract. Therefore, you not only prevent the error of permitting a history to influence you in finding physical evidence of pathology where it does not exist, but you may make a finding that will enable you to secure a more accurate history.

Hurst (18) finds that the only satisfactory method of getting an accurate history is for the patient to give a full description of everything he does, eats, and drinks in an average day. And to describe in detail the nature, position, time of onset, exciting causes, and manner of obtaining relief of every unpleasant sensation he experiences.

If in this history some pathology is suggested, which if present, should have been found during the physical, you may now go back to that region and make a thorough reexamination. If it is not present now, then you are more certain that it is probably not there.

Some exclusions may be made by the physical examination and the history, but a definite diagnosis of primary chronic gastritis can never be made without the utilization of the special methods which in general include : Gastric analysis, roentgenoscopy, gastroscopy, and stool analysis.

According to Rehfuss (33), "The stomach tube is to the physician interested in stomach conditions what the stethoscope is to the chest man." The fractional test-meal with a Rehfuss tube is accepted now by all in preference to the single analysis of the Ewald meal. However, we find Bloomfield and Keefer (7) considering the fractional analysis so inadequate that they adopted a procedure which made it possible to estimate, under uniform conditions and with reasonable accuracy, the acidity of the undiluted gastric juice, the volume of secretion, and the emptying of the stomach. Although they conclusively pointed out the shortcomings of the ordinary meal, Eusterman (10) believes their procedure too time-consuming for routine laboratory use.

In general the test meal may show a lessened degree to an absence of hydrochloric acid, a low total acidity, a juice of low enzymotic power, an increase of incorporated mucous, food more or less imperfectly digested one hour later, and the amount of extraction larger (70-150 cc.).

In moderate cases the total hydrochloric acid falls to about one-half (30°); in marked cases it is less or absent. Eusterman (10) calls attention to the statistics showing a 7-15% greater incidence of

achlorhydria with single than with fractional analysis. When the acid secretion is low, erythro-dextrin is present in small amounts and achroodextrin and sugar are abundant due to the absence of the inhibiting effects of the hydrochloric acid on the salivary content and the more complete digestion of ptyalin on the starches in the stomach.

As the acidity falls, with it the enzymotic content is reduced. Bassler (6) is of the opinion that quantitative tests of the zymogen should always be made when the acid is low; because he considers it a practical fact that upon this more than on reduction of the hydrochloric acid depends the diagnosis of the degree of the gastritis and the prognosis. His conclusion was made from considering the hydrochloric acid secretion more sensitive to gastric disturbance than are those of the ferments. Polland and Bloomfield (31) support Bassler in the belief that enzyme estimation is a more delicate index of gastric injury, but they call attention to the many difficulties besetting the ferment estimation. The effects of the ferment action vary with the time over which the reaction proceeds, the concentration of the substrate, and the concentration of the enzyme, as well as with other factors. Most methods have been based on the principle of varying one factor while others are kept constant. But some investigators have shown that the activity does not vary in direct proportion to the concentration. Northrup (30) has shown that the time required to cause a given percentage change in the viscosity of a solution of substrate is nearly inversely proportional to the amount of enzyme present. On this basis a practical method for the estimation of peptic or tryptic action has been developed. Gilman and Cowgill (14) modified Gates method, one involving the digestion of the gelatine layer of a photographic film, that is considered rapid and presumably accurate. For a simple, quick method to be used in ordinary

clinical procedure, Bassler (6) suggests the use of the simple test of quantitatively estimating the total enzymotic contents by means of the amount of milk-curdling power present. In the latter it may be taken as a standard that where the enzyme is present in 1:160 dilutions that there is probably not much advance in destruction. But the prognosis is uncertain if no activity until you get down to dilutions of 1:80 to 1:40. Eusterman (10) feels that although the usual methods of gastric analysis always will play the major diagnostic part largely because of their simplicity and universal application, that more extensive investigation should be encouraged in order to properly evaluate the method before adopting quantitative determinations of peptic activity as a routine laboratory procedure.

At this point of consideration of hydrochloric acid and enzyme estimations, that Basch (4) suggests, the more frequent employment of either the Salomon or the Wolff-Junghans tests for the differential diagnosis between malignant and benign achylia. These tests were introduced following the discovery that gastric cancer yielded a protein-digesting ferment. Smithies (36) and Friedenwald and Kieffer (13) some ten years previous found the latter test 80-90 per cent positive in later demonstrated gastric cancer cases. However, we must understand that this is not a specific test, but merely a test to determine the presence or absence of incomplete protein digestion. If it were specific it would be of unquestionable value.

The old authors considered mucous as the most important finding for a diagnosis. But now its presence or absence is not an essential point, according to Reffuss (35) and Goldsmith (15). The former insists that if

it were essential, only the type due to the ingestion of the irritants would be diagnosed. The latter points out how the mucous might be swallowed mucous or be the result of the irritation of the stomach tube, of the retching and gagging of the passage, or of a reflex from some other organ as the appendix or gall bladder. Similarly, Hurst (17) does not consider the presence and deposit of squamous epithelium as found in atrophic conditions as of diagnostic significance but Work (38) gives it detailed attention.

Histanune injection as a test of the ability of hydrochloric acid secretion is advocated by many but found to be of no diagnostic value to Hurst (17). He objects to its administration because it calls forth secretion of acid only when the achlorhydria is due to chronic non-atrophic gastritis, and therefore curable by treatment; because in several instances it has led to no secretion of acid, although treatment has led to the appearance of normal secretion; because it occasionally produces unpleasant symptoms; but most of all because with a preliminary lavage before the test meal, especially where second meal is given, much better results are obtained.

Most clinicians use the x-ray just to exclude the presence of some other pathology such as cholelithiasis, peptic ulcer, and carcinoma rather than the presence of chronic gastritis. But some use it for the latter purpose. Bassler (5) is convinced that there are roentgenographic signs of simple and common forms of chronic gastritis, ones not always present even in well-marked cases, but which, when present, are essentially pathognomonic. First, the regular outlines of the greater and lesser curvatures are much changed in appearance. Often numerous small indentures and bulgings, none of them much more than a centimeter in depth or height, are seen along the greater curvature, but occasionally along the lesser. Second, the whole stomach displays a lack of the

regular, large, sweeping peristaltic phenomena; the waves, when of some size, being irregular and in other instances the whole organ being bag-like and inactive as far as normal peristolysis is concerned. "This serration of the greater curvature outline may be definitely localized such as in the pyloric region and at the base of the cap, or as is most often seen, in the lower two-thirds of the greater curvature." Third, the irregularity of the lower outline is noted only in the films or observations made in the prone position and is completely obliterated in those made in the upright. This is due to the weight of the contents of the stomach when in the patient is in the upright position being sufficient to iron out the outline to a straight edge and overpower the fibrillary contractions which are so characteristic. Fitzgerald (12) calls attention to the statements of Zweig, Berg, and Schwarz that an increased sensitiveness to pressure over the stomach outline with multiple indentations along the greater curvature and an increase in the number and depth of the folds indicate gastritis. They are more conservative than Bassler.

Although Eusterman (10) considers it of value, Fitzgerald (12) claims gastroscopy has proved disappointing as an aid to diagnosis. He finds that although the technique is not difficult, the whole of the interior of the stomach cannot be clearly visualized; and, what is more important, it is extremely difficult to interpret the findings correctly.

It is unnecessary to mention how incomplete an examination is without a routine blood and urine analysis. Alvarez (1) and Hurst (18) consider stool examinations essential in all gastro-intestinal cases, especially for occult blood and in doubtful cases for amoebae and other parasites.

Basch (4) believes that surgical exploration may be indicated in two types of cases, either those with a positive clinical picture but with negative x-rays and showing no speedy improvement or in doubtful cases when subacidity is present with some other feature that does not fit into the classical cancer picture but may be benefited by an early operation.

Differential Diagnosis

Although the diagnostic methods provide nothing specific, an accurate diagnosis should be arrived at even in the atypical cases if a concentrated effort is made with that indescribable faculty of interpreting the findings to fit that particular individual. In conducting the differential diagnosis for primary chronic gastritis, an exclusion of the following would probably be made : Gastric neoplasm, peptic and duodenal ulcer, chronic appendicitis, gastric syphilis, cholecystitis, cholelithiasis, passive congestion, pernicious anemia, and tuberculosis. It is well to always keep in mind the possibility of pregnancy, sprue, pellagra, carcinoma of the bowel, gastro-colic fistula, gastrogenic diarrhea, hyperthyroidism, and such; but confusing them with chronic gastritis is less likely so they will not be discussed in this paper.

A good roentgenologist will disprove or confirm the presence of cancer or ulcer in 70-95 per cent of the cases and according to Bassler may make a diagnosis of gastritis. Also, he will disclose the presence or absence of a gall bladder condition or a pulmonary tuberculosis. In addition to x-ray findings in keeping with cancer, there will be found present occult blood, lactic acid, Boas-Oppler bacillus, and a more or less constant dull aching pain and evidence of constitutional disease with a history of only a short duration compared to one of 5-20 years in chronic gastritis. Ulcer patients besides having definite x-ray findings and a hyperchlorhydria in contradistinction to the usual

achlorhydria of gastritis and carcinoma, complain of more severe pain 1-2½ hours after meals which tends to be periodical and seasonal and can usually be relieved immediately with milk or alkalies. Hematemesis may be found in ulcer and cancer, especially in the former, but Work (38) claims it is never found in pure gastritis. It is seen that a good anamnesis is an important differential factor.

In every case of achlorhydria, besides cancer and gastritis, pernicious anemia must be considered routinely by a blood smear, the same as a routine Wassermann should be made to rule out syphilis. A thorough physical examination should disclose any cardiac decompensation, oral sepsis, and possibly tuberculosis. Periodicity of attacks, localized area of pain and tenderness and leukocytosis would point towards appendicitis.

It must be remembered that it is possible to have chronic gastritis associated with another condition such as appendicitis and cholecystitis, and that the patient cannot be considered "cured" but will continue to have symptoms if the gastritis is not recognized and treated also.

Prognosis

The course is chronic and when well established may become progressive. As in all diseases of numerous etiologic factors, the prognosis varies with the possibilities of the removal of each factor. Various signs have been offered as the criterion of changes of which the most stable at the present is that gotten from gastric analysis. Zweig (12) claims the amount of diminution in acidity can be taken as an index to the degree of destruction of the gastric mucosa. Hurst (21) also uses this as the index. He feels that if a normal or high curve of acidity is obtained in a second test meal after a lavage, it can be considered that little or no atrophic

change has occurred so far because the condition is mostly catarrhal. However, if no trace of acid in second meal, then there must be present either a constitutional achylia gastrica which may or may not be associated with a secondary gastritis; or it is a comparatively advanced primary atrophic gastritis. Jones (24) feels that patients with an abundance of gastric mucous have the better prognosis.

Hurst (20) and Fitzgerald (12) offer two points of probably value. The former reminds us that although the inflammation may be overcome by treatment, nothing can prevent the possibility of an abnormal mucous membrane subsequently undergoing malignant degeneration. The latter states that there is an undetermined psycho-nervous element super-added on this underlying pathology that we must consider.

Treatment

If it were possible for the medical profession to control the health program of each and every individual, prophylaxis would play a great role, especially so in prevention of gastritis. For then we would know all who belong in the 10-16 per cent group which is predisposed to gastritis, and in these we would institute prophylactic measures. But is it not true that the greater part of those who consult a physician, at least those over whom the physician has any control, have passed the point of prophylaxis and need active therapy? In other words, prophylaxis is nice to know, but mostly for your own personal use as it is the rare case that you pick up some new pathology by accident and it is still more rare to find it in a patient who will cooperate with you to prevent its progress. More important is to determine and eliminate the causative factor.

So possibly the first therapeutic measure is a gastric lavage followed by a test meal to determine just how severe your therapy must be.

For this lavage Hurst (21) suggests the use of one dram of hydrogen peroxide to a pint of water and should be continued until no further mucous comes away; the final washing should be of plain water. The nascent oxygen set free when the hydrogen peroxide comes into contact with the mucous dislodges it much more efficiently than any other fluid, and at times acts as an antiseptic. The lavage should be made daily at first and if the mucous does not all come away with the above concentration, the hydrogen peroxide should be increased until it does. As the patient improves the concentration is first decreased and then the frequency of the lavage, first to alternate days, then two times a week, and finally once a week for several months. If there are recurrences the patient should receive another course of treatment. In any case a test meal should be given once a year subsequently.

The lavage may be given before breakfast but Bassler (6) feels that since the patient should rest for two hours afterwards, that just before going to bed is a good time. He also suggests some other points worthy of note. If the residual water is 500-1000 c.c., either a marked atony of the stomach or a relaxed condition of the pyloric sphincter exists. If atony is not present the removal of the mucous is facilitated by allowing the water to run in under some pressure for which the Leube-Rosenthal method is best. But in positively empty stomachs the gastric spray douche may be of advantage. When primary atony exists, the hand syphonage apparatus is safer by delivering into the organ only small quantities of fluid at one time. He does not find the employment of massage of the stomach or changes in position during the lavage of any essential benefit. In cases where lavage just before retiring is employed, it is suggested that some

mechanical sedative to the mucosa as bismuth salts, cerum oxalate, or insoluble and bland magnesia combinations may be added. Jones (24) and Crohn (8) use sodium bicarbonate or boric acid solutions for their lavages.

Hurst (21) believes it better to restore normal secretion than give hydrochloric acid but where the acid is absent from the second meal it should be given, about 15-30 minums of dilute acid diluted in two ounces of water one-half hour after meals. Then after a lavage course, a third test meal should be given for the acid may have increased. If there is a hypochlorhydria or achlorhydria found, hydrochloric acid should be given continuously, but only in such cases.

A proper diet is important of course. It should be so prescribed that the food is of such quantity and quality that the secretory and motor powers of an enfeebled stomach are not over-taxed. Milk, buttermilk, custards, soups, broths, baked and broiled meats, eggs, zoolak, stewed fruits, and pureed vegetables are the type of foods most suitable to this type of stomach. Crohn (8) calls attention to the fact that there is no object in withholding protein foods since they stimulate acid secretion but that fats and fatty foods should be restricted in the presence of nausea and vomiting the same as any food if there is an indication of an idiosyncrasy. He suggests also the avoidance of excess fluids, especially at meals. Alvarez (1) claims digestants are not indicated.

Almost all writers claim that one of the most important points is the association of the care of the general health in the way of sufficient non-strenuous exercise, fresh air, vigorous walking, and recreative games in the open air, particularly golf. The bowels should move daily so

salines in the morning if needed are recommended by Jones, Crohn and others. Mineral waters seem to have been of benefit to some, especially those of French Lick Springs, Saratoga, Carlsbad, and Vicky. But most investigators feel that they are a treatment for the well-to-do and as good results can be obtained without their employment.

Bassler (6) made use of the faradic current in ameliorating subjective symptoms. There is a possibility of it toning up the muscularis but he admits that probably the greatest effect is psychic, especially in nervous cases. The intra-gastric proved vastly superior to the percutaneous method of administration. Along about this same line is the use of local applications of hot fomentations or turpentine stupes as suggested by Crohn (8) in cases of exceedingly annoying symptoms.

Coming as most clinicians feel that it should, as the last resort, surgery must be considered. However, Fitzgerald (12) does not think it should be placed last, especially in his "follicular" cases (cases in which the infiltration of round cells has formed follicles). He makes the statement in regards to these that, "It is unnecessary to enter into an argument of conservative vs. operative treatment because our present knowledge is built up from operative cases, the data of which are complete and available, while the case in favor of conservative treatment is yet to be made." In regards to the surgical procedure to be elected, whether gastro-enterostomy, extra mucosal pyloromyotomy, or resection of the pyloric portion of the stomach and duodenum, he finds the latter receives the greater support. It is selected by Konjetzny, Finsterer, Florcken, Elter, and Nicolaysen. Konjetzny (23) reports eight cases of Billroth I resections which put an end at once, and apparently permanently, to gastric disturbance of several years standing. He claims the expected

ulcer or cancer was conspicuous by its absence, and that a chronic gastritis or duodenitis explained alone the clinical picture. Wanke (12) gives warning that cases resected early in the disease are more likely to have past operative trouble (fundus gastritis) than those operated upon late, when the inflammation is confined to the pyloric region.

Case Reports

These cases have been selected from many presented in accessible literature, not so much to show the results of treatment as to show the indicated close relationship of chronic gastritis, Addison's anemia, carcinoma and ulcer of the stomach. One case illustrates how even our more dependable findings may be so altered as to lead to an error in diagnosis.

Case I -- Taken from reports of Fitzgerald (12)

"Mr. E. R., age 52, machinist. Admitted February 21, 1927.

"History -- Attacks of aching pain in the epigastrium for fifteen years. At first the pain came on two or three hours after eating, and was relieved by food. For the past few years the pain had no relation to the taking of food. The attacks came on four or five times yearly, with long periods of relief. Attacks were always preceded by diarrhea. No bleeding.

"On examination -- Test meal : 25 c.c., total acidity 15, free HCl. 1. Occult blood present. Discharged on medical treatment.

"Re-admission (January 20, 1929) -- Complaining of abdominal pain, loss of weight, weakness, anorexia. Slight anemia (Hb. 72%). Occult blood detected in stool. Test meal gave 59 c.c., total acidity 12, no free HCl. Occult blood present in stomach contents. X-ray showed a small ulcer of the duodenum. He was given the Sippy regime, and left the hospital in twenty-eight days without pain.

"Second Re-admission (Aug. 10). -- Felt well until a month ago when there was a return of the periodic pain, which was always relieved by food. Epigastric tenderness.

"Operation (August 13 -- Dr. C. K. P. Henry) -- Stomach and duodenum normal except for a small thickening of the duodenal wall. Partial gastrectomy.

"Pathological Report (September 29, 1190) -- The specimen consists of a portion of a stomach measuring 7 by 4 cm. Mucosa covered with a viscid secretion. There is no ulcer.

"Microscopical appearances -- The mucous membrane is closely packed with large follicles with germinal centers, and the gastric glands are separated from each other by round cells, plasma cells, and eosinophils. The muscularis mucosae is irregularly thickened and fibrosed. The submucous layer shows diffuse scarring and dilated blood vessels. There is also a follicular duodenitis.

"Diagnosis -- Chronic follicular gastritis and duodenitis.

"Subsequent Course -- The operation was followed by intractable vomiting with 48-hour retention. Abdomen opened, adhesions divided. Discharged September 21, feeling very well. Three months after the last operation there was still gastric retention (10% in 6 hours). Classified as improved."

Comment : Of interest here is not only the success of operative where conservative treatment had seemingly failed, but also the question of whether the gastritis was provoked by the effects of the duodenal ulcer or if it had existed for some time before the appearance of the ulcer.

Case II -- Taken from reports of Hurst (20).

"Mr. C., aged 70, had several attacks of severe diarrhea in Singapore in 1890. The diarrhea had none of the characteristics of sprue and ceased on his return home in the summer. From that time he had periodical attacks of flatulent dyspepsia with waterbrash. He left the tropics finally in 1910. Since 1915, but never before, each attack of dyspepsia was associated with a sore tongue. Since November, 1919, symptoms have been continuous, and for the first time an ulcer appeared on the inside of his lower lip. During the last two months he has become increasingly short of breath, lost 5 pounds in weight, and his legs have become weaker.

"He was admitted to New Lodge Clinic on January 7, 1930. He had a lemon color, and a blood examination gave the following characteristic picture of Addison's anemia : R.B.C. - 2,050,000; Hb.-41%; W.B.C.-4,600; presence of normoblasts, megalocytes with poikilocytosis, anisocytosis, and polychromasia.

"The van den Bergh test gave a delayed direct and a strong indirect reaction. His urine contained a considerable excess of urobilin. He had the typical atrophic glossitis of Addison's anemia. His spleen was slightly enlarged. A test meal showed that he had complete achlorhydria with a considerable amount of mucous in each fraction. The stools contained no excess of food residue. While on a farinaceous diet a fecal extract gave a hematoporphyrin spectrum, corresponding with the fact discovered by Ryffel that during hemolytic crises hematoporphyrin is excreted by the bile. Moderately severe pyorrhea alveolaris was present.

"As there was no family history of Addison's anemia, and as the test meal showed excess mucous with rather higher total acidity than is usual in constitutional achylia gastrica, it was thought that the achlorhydria might be secondary to chronic gastritis, caused by food poisoning in 1890 and aggravated by swallowing infective material from the teeth. In addition to giving him liver, his stomach was washed out with hydrogen peroxide and the pyorrhea was thoroughly treated.

"When examined on April 2 his blood picture was almost normal; the van den Bergh test gave no direct reaction and only a very weakly positive indirect reaction. A fractional test meal showed a normal secretion of free acid, although some excess of mucous was still present. On September 1, the number of red corpuscles had reached 5,000,000 and the hemoglobin 100 per cent; the van den Bergh reaction was completely negative."

Comment : A case of Addison's anemia probably correctly interpreted as secondary to an achlorhydria due to chronic gastritis, in which nine months of treatment of the gastritis plus the administration of liver restored normal gastric function and cleared up the anemia.

Case III -- Taken from reports of Hurst (17)

"The patient served in the East during the War, and in 1920, when 31 years old, had repeated attacks of diarrhea whilst on duty in Constantinople. He then became progressively more pale, and complained of soreness of the tongue. He was invalided home in 1921, and soon developed attacks of vomiting with loss of strength and shortness of breath. On admission to Millbank in October, 1921, his hemoglobin was 20% and red corpuscles 740,000 with a few megaloblasts, and white cells 4,000. He was admitted under me in January, 1923, having been transfused eight times. He had a

lemon-yellow color and atrophic glossitis. His hemoglobin percentage was 42 and red corpuscles numbered 1,670,000 (color index 1.26); the Price-Jones (cell diameter) curve was typical of Addison's anemia and his blood gave a positive indirect van den Bergh reaction. He was given hydrochloric acid, and his teeth, which had infected roots, though there was no pyorrhea, were extracted. He soon began to improve, and in July his hemoglobin was 75 per cent. He was then able to return to his old occupation of school master, and he has continued without any return of anemia to the present time. His hemoglobin was 110 per cent in October, 1923, and has been over 100 per cent, and the red corpuscles over 5,000,000 each time his blood has been examined; the van den Bergh has always been negative. He has never had liver. He continued to take hydrochloric acid until October, 1925, when he complained of gastric symptoms and was found to have a high normal curve of acidity. He therefore discontinued the acid." The patient had a history of alcoholism preceding treatment.

Comment : This, like the preceding case, is an Addison's anemia interpreted as secondary to chronic gastritis; but this case differs in that the anemia was cleared and the normal function of the stomach restored by treatment of the gastritis alone. It is interesting to note that Hurst considers oral sepsis (plus alcoholism in the last) as the cause of the chronic gastritis.

Case IV — Taken from reports of Hurst (17)

"In April, 1924, I saw a man of 51 who gave a history of periodic attacks of epigastric discomfort for 15 years, which he ascribed to excessive smoking. The x-rays showed a spasmodic condition of the pyloric vestibule but no filling defect. There was complete achlorhydria with excess mucous. Though the possibility of carcinoma was considered, this

seemed to be excluded by the complete disappearance of symptoms on taking hydrochloric acid and giving up smoking. He gained a stone and a quarter in weight, and remained completely free from symptoms until September, 1925, when he began smoking again and discontinued taking acid. In January, 1926, he returned to the Clinic, having lost nearly a stone in weight. The x-rays now showed a definite filling defect in the pyloric part of the stomach, blood was present in every fraction of the test meal, which still showed achlorhydria, and the stools contained much occult blood. Carcinoma was diagnosed and partial gastrectomy performed. He survived 'till July, 1930."

Comment : As Hurst states, the good health and gain in weight between the patient's two visits make it doubtful whether anything more than chronic gastritis could have been present on the first occasion; but, even if a very early growth was already present in April, 1924, the achlorhydria must at that date have been a result of chronic gastritis and not of carcinoma. It can then be assumed that the gastritis was a precursor to the cancer.

Case V -- Taken from reports of Hurst (17)

"Mrs. R., ages 61, was admitted in May, 1930, with a history that she had been unwell for 5 years. She had first consulted her doctor in 1927 on account of general weakness and dyspnea, which he attributed to severe anemia, as she was very pale. In July, 1928, she was found to have only 35 per cent hemoglobin with 3,580,000 red corpuscles; no occult blood was present in her stools. The anemia persisted in spite of treatment with iron and arsenic injections and liver extract, and she had occasional indigestion.

"On admission there was an ill-defined tender tumor in the epigastrium. The blood-count was identical with that of two years before, the Hb. 36% and R.B.C. 3,500,000. The W.B.C. 7,800 with a normal differential count. The van den Bergh reaction was negative. A test meal showed complete achlorhydria. A large filling defect of the pyloric end of the stomach was found with the x-rays. The Wassermann reaction was negative.

"The patient was given 40 and subsequently 60 gr. of iron and ammonium citrate three times a day and was transfused three times. Her hemoglobin had risen to 66 per cent by June 12, 1930, when partial gastrectomy was performed for a mass involving the pyloric end of the stomach. She made an uninterrupted recovery, and by July 23, her hemoglobin had risen to 90 per cent and her red corpuscles to 4,700,000, the color index having thus risen from 0.5 in June, 1928 to 0.95 in July, 1930.

"Histological examination showed the presence of a primary carcinoma with secondary peptic ulceration. The rest of the gastric mucosa was the seat of advanced chronic gastritis, mainly hypertrophic, but with atrophic changes in the lesser curvature region above the growth. Unmistakable tubercle follicles with characteristic giant cells in the subserous and muscular coats over part of the growth were discovered, together with an early tuberculous lesion in one of the lymph glands of the lesser curvature. The lymph glands along both curvatures were extensively invaded by carcinoma.

"It is very unlikely that the tumor had been in existence for more than two years, so that the anemia, which had probably been present for four years and was certainly as severe two years before as at the time of the operation, must have been of the simple achlorhydric type and not a result of the growth.

"Complete achlorhydria was still present after the operation. There was thus just as much reason for anemia to develop now as four years ago. The patient was therefore advised to take 40 gr. of iron and ammonium citrate three times a day after meals for the rest of her life. When seen in October, 1931, her Hb. was 78% and R.B.C. numbered 4,500,000.

Comment : Here is a case of cancer associated with an achlorhydric anemia that can be interpreted as having gastritis as the precursor.

Case VI -- Taken from reports of Faber (11)

"A young man, aged 25, who for 10 years had had pronounced gastric symptoms and had four times been subjected to severe dietetic treatment for ulcer, each time with good but transitory result. The symptoms were hunger pains, appearing about one and a half hours after meals and lasting until the next meal, which relieved them; frequent vomiting, and a dozen small hematemeses, rarely more than 1-2 tablespoonfuls. At the hospital frequent occult bleedings in feces, once melena. Hyper-chlorhydria was demonstrated repeatedly; on Ewald's test meal, free acid 93, total acidity 106. Fractional withdrawal likewise showed high figures, rapid emptying of food, protracted marked secretion of acid, which still persisted four hours after, and bile influx after $2\frac{1}{2}$ hours. X-ray showed doubtful deformity of bulbus duodeni. The diagnosis seemed clear and Professor Schaldemose did a resection of the pyloric part of the stomach and duodenum.

"Examination of the resected part revealed : Macroscopical : No chronic ulcer simplex, but in the antrum of stomach 3-4 diminutive erosions, which did not penetrate the mucous membrane and were outlined by slightly elevated edges. Microscopical : Extremely intense inflammation of the entire resected area and small breaks in the continuity of the mucosa with

strongly infiltrated surroundings. In other words, gastritis with erosions. The disease had lasted 10 years and had very definite classical symptoms. The erosions cannot be considered chronic; they are more transitory and changing manifestations of the inflammation. The fundamental (primary) lesion is a marked chronic gastritis, localized to the pyloric portion of the stomach."

Comment : Faber considers the symptoms a result of acute exacerbations of the small erosions rather than any true ulcers at any time. The case is of interest because it shows that, although a severe chronic gastritis may be present, it may be so localized that a normal curve or even a hyper-chlorhydria may be produced by the remainder and thus shadow the diagnosis.

Conclusions

1. The etiologic scope is as massive and confusing as ever; however, the tendency of those in the 10-16% class with constitutional achlorhydria or achylia to be more prone to the development of chronic gastritis is being given greater consideration.
2. The most reliable diagnostic measures for stomach disease are still x-ray and gastric analysis. In the latter hydrochloric acid estimation still remains the criterion, but further investigations for an acceptable enzyme determination are being encouraged.
3. The attitude that chronic gastritis is one of today's most important medical problems is increasing. Although there is not a uniformity in belief that chronic gastritis is or is not the most important precursor of cancer of the stomach, most appreciate the seemingly close relationship of gastric cancer, Addison's disease, and chronic gastritis.

4. The treatment of chronic gastritis is not one of administration of hydrochloric acid alone but must include gastric lavage, proper diet, exercise, and removal of causative factor.
5. Some diseases referrible to the gastro-intestinal tract, such as pernicious anemia, may be cured by the treatment of the gastritis alone. In all cases where a gastritis exists concurrently with some other pathology, the gastritis must also be treated to cure the patient.

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