GASTRITIS IN THEORY AND PRACTICE*

BY

STANLEY J. HARTFALL, B.Sc., M.D., M.R.C.P.

HONORARY PHYSICIAN, LEEDS PUBLIC DISPENSARY AND HOSPITAL;
ASSISTANT PHYSICIAN, THE GENERAL INFIRMARY AND ST. JAMES'S
HOSPITAL, LEEDS; LATE LEVERHULME RESEARCH SCHOLAR,
ROYAL COLLEGE OF PHYSICIANS, LONDON

More than one-quarter of all patients who consult the medical profession suffer from gastro-intestinal disorders, and the incidence of these affections is increasing under the stress of modern life. Often these disorders are minimal in their symptomatology, being regarded as minor maladies and correspondingly unimportant. Gastritis is a typical example, but its association with many serious and even lethal diseases, as revealed by modern clinical and experimental research, calls for a review of its position in medicine and for some attempt to assess its importance. In my review I shall stress certain aspects of the gastritis problem in which I have been especially interested due to my association with Professor M. J. Stewart, Dr. A. F. Hurst, and Dr. E. Meulengracht, to whom I shall ever remain indebted for inspiration and encouragement.

The clinicians of two or three generations ago used the term "gastritis" in a somewhat indefinite way to indicate a low-grade catarrhal inflammation of the mucous membrane of the stomach. A certain clinical symptomatology was described, and while there was much speculation no certain views existed as to pathology or aetiology. Wild and unsubstantiated speculations of Broussais and the French school had brought the subject into disrepute, and we find Trousseau,¹ although recognizing the disease, emphasizing its extreme rarity and doubting whether his students had ever heard it mentioned in their hospital practice. He says "to pronounce the word 'gastritis' is equivalent to a desire to renew a useless controversy raised by a defunct school."

Towards the end of the nineteenth century, however, the clinical diagnosis of gastric catarrh became more popular, especially as the "Magenkatarrh" of the Germans, and it was associated with a post-mortem condition of the stomach—the not uncommon, discoloured, autodigested, and gelatinous state of the mucous membrane. This was only later fully appreciated as being an artificial post-mortem appearance. So again gastritis and gastric catarrh fell into disrepute, for while pathology was making enormous strides in some directions, this condition had no certain pathology; the diagnosis, in fact, almost disappeared from current medicine, and was replaced to a large extent by "nervous dyspepsia." With this diagnosis presumably it was unnecessary to provide a pathological basis for the disturbance in the stomach.

As students we were discouraged from using the term "gastritis," and the chief reason for this was that the so-called inflammatory or atrophic lesions of the mucous membrane could not be demonstrated clearly owing to the rapid post-mortem changes. But already attempts had been made to overcome this difficulty and to obtain stomachs in such a state of preservation that it would be possible to distinguish between the changes due to gastritis and those due to autolytic digestion and hypostasis. Owing to the cloud under which gastritis seemed to lie, it was a long time before much attention was paid to these important observations in this country.

Clinical Associations of Achlorhydric Gastritis

The first outstanding work was that of Hayem,² who introduced bichromate solution into the stomach shortly

after death and so preserved the mucous membrane. Hayem gave a careful description of the various changes in the mucous membrane that accompany gastritis, and attempted to correlate them with changes in gastric secretion, which, with the introduction of the stomach tube, were then attracting attention. The important work of Faber^{3, 4} and the Scandinavian school followed his discovery that the injection of almost half a litre of 10 per cent. formalin solution through the abdominal wall into the peritoneal cavity and stomach preserved the mucous membrane in a very satisfactory manner for histological examination. Again attempts were made to correlate the histological appearances and the changes in gastric secretion, especially with those conditions where acid and pepsin secretion was diminished or abolished. The view gradually gained ground that there was always a definite underlying gastric pathology—chronic gastritis—responsible for achlorhydria.

This view—achlorhydria ex gastritis—must be contrasted with the view which was more widely held in this country, mainly owing to the influence of Hurst,⁵ that achlorhydria results from an inborn error of gastric secretion. This constitutional gastric secretory defect may express itself very rarely as a congenital achlorhydria, but much more frequently as an inherent liability of the mucous membrane to fail to secrete acid if exposed to deleterious influences. Such influences would have little or no effect on, and would easily be resisted by, individuals not possessing this constitution, which has been called by Hurst the hyposthenic gastric diathesis. It will be seen that the two theories are not mutually exclusive. We shall discuss later how this theory of the constitutional gastric diathesis has been ingeniously extended to embrace certain other gastric secretory changes, but before doing so it is convenient to recall the important clinical associations of achlorhydric gastritis. They are:

1. Addisonian pernicious anaemia; subacute combined degeneration of the cord.
2. Idiopathic hypochromic anaemia of middle-aged women (the majority); simple achlorhydric anaemia of Witts.
3. Carcinoma of the stomach of the type following gastritis.
4. Ill-defined group: asthenic individuals the subjects of gastro-intestinal upsets, attacks of dyspepsia, diarrhoea, debility, and soreness of the mouth and tongue; also in a proportion of cases of rheumatoid arthritis, asthma, and allergic diseases, and Graves's disease, where its significance is doubtful.
5. After anastomotic gastric operations in a proportion of cases but by no means in all.
6. In individuals apparently in normal health but probably the subjects of symptomless chronic gastritis.

Within the last few years renewed attention has been given to the gastritis problem following the intensive studies of the gastric histopathology of fresh operation specimens of the stomach (Konjetzney).⁶ This work has not only confirmed the histological descriptions of the mucous membrane of the stomach the seat of chronic gastritis, as described by Faber and his co-workers,⁴ but has disclosed the fact that such lesions might be associated with peptic ulcer and carcinoma. In the case of peptic ulcer, where gastric acidity is certainly not abolished and is often increased, it was realized that here was gastritis *not* associated with depression of gastric secretion but frequently with the reverse condition—hyperchlorhydria. Another feature frequently noted was the presence of numerous tiny gastric erosions—particularly of the pyloric region. It had long been held that the genesis of an acute, and ultimately a chronic, ulcer was through the failure of healing and extension of one such erosion. In some of the operation specimens this was apparently happening, and all stages between an acute gastritis, acute haemorrhagic gastritis with erosions, acute, subacute, and more or less chronic ulcer, could be traced. Thus ulcerative gastritis and acid gastritis were

* A lecture delivered to the York Medical Society, February 9th, 1936.

described, accompanied by hypersecretion, and gastritis was given a new significance in the genesis of peptic ulcer. Instead of, as heretofore, the gastritis being secondary to the ulcer, the ulceration was secondary to the gastritis.

Histological Changes in the Gastric Mucosa

The various histological changes met with in the gastric mucous membrane are manifold, and can be summarized only in a very brief way. The normal distribution of the various secreting gastric glands composing the normal mucous membrane must be briefly mentioned.

The digestive juice, by which one implies the acids and ferments, is secreted by the oxyntic and chief cells, which form tubular glands situated in the body and cardiac end of the stomach. In this region, therefore, the gastric mucosa consists of long tubular glands of closely packed cells with narrow septa and little interstitial tissue. Of the two well-defined types of cell, the chief cells are aggregated towards the base and the oxyntic towards the neck of the glands. These peptic glands are mainly responsible for the secretion of hydrochloric acid and ferments—pepsin and renin. They are gradually replaced towards the pyloric region by the so-called pyloric glands composed of an alveolar tubular arrangement of clear epithelial cells, very like those composing Brunner's glands in the duodenum. The pyloric region elaborates the specific intrinsic anti-anaemic factor, and it is believed that a pyloric secretion serves as a further stimulus to the acid secretion of the fundus glands (Meulengracht).^{7 8}

So far as gastritis is concerned, the naked-eye appearance of the mucous membrane of the stomach may show acute or subacute inflammatory changes which give it a reddish appearance. The mucosa is often overlaid with a thick layer of mucus. According to naked-eye and microscopical inspection these changes may be local or general. The inflamed and reddened mucous membrane of the acute and subacute varieties may be confined to the pyloric region or spread over the whole corpus. Few or many scattered erosions may be seen, with tiny pin-head sloughs. The mucous folds may be thickened and hypertrophied, and occasionally mammillated. With the more chronic atrophic changes the mucous membrane is flattened, thin, and pale.

A variety of microscopic changes affecting the secreting cells are visible on examination, but for purposes of comparison we may first study the microscopical appearance of the normal healthy stomach. This shows no atrophy of the mucosa, no large mucus-carrying cells (beaker cells), only sparsely distributed and small lymphoid follicles, and little or no round-celled infiltration. The interstitial connecting tissue is small in amount, and the vascularity of the submucosa is not increased. These negative features of the normal stomach may be contrasted directly with the changes in gastritis. Microscopically the mucous membrane may show a variety of widely differing appearances ranging through inflammatory oedema, hypertrophy, to atrophy. Both hypertrophic and atrophic changes may coexist in the same stomach. The character of the individual cells is altered in the direction of cloudy swelling and oedema in the more acute varieties of gastritis, but in the chronic forms there is a separation, a thinning out, and a reduction of the number of glands composing the mucous membrane (atrophy). There is also an increase in the number of large mucus-carrying cells and regions of metaplasia, areas in which the gastric epithelium takes on an intestinal pattern.

All these changes occur, of course, at the expense of the normal gastric epithelium. In addition, there is an overgrowth of the interstitial connective tissue, with marked increase in the round-celled infiltration by lympho-

cytes, eosinophils, and plasma cells. The submucosa is thickened by round-celled infiltration and increased vascularity. A variety of names have been used to describe the forms of gastritis associated with these microscopical findings, and the following classification is based mainly upon the morphological appearance (Simpson).

Acute gastritis.....	{ Simple (parenchymatous) Haemorrhagic } Erosive Membranous }
Subacute gastritis	{ Simple (parenchymatous) Hypertrophic (mammillary)
Chronic gastritis	Atrophic

Gastritis and Gastric Secretion

The correlation of the newer discoveries concerning acid gastritis and peptic ulcer on the one hand, and achlorhydric gastritis and its clinical associations on the other, is incomplete. Porges⁹ has put forward the view that gastritis localized to the pyloric end of the stomach may act as a stimulant to the acid secretion of the body and cardiac end. In this way pyloric gastritis becomes the precursor, if not the actual cause, of hyperchlorhydria. Faber⁴ also emphasizes the difference between a local gastritis of this type—the aetiology of which, he suggests, may be chiefly mechanical, resulting in hyperchlorhydria and the peptic ulcer syndrome—and the generalized gastritis of toxic haematogenous origin affecting the whole gastric mucous membrane, and so resulting in achlorhydria.

Hurst,⁵ on the other hand, has extended the theory of gastric diathesis to cover hyperchlorhydric and hypochlorhydric or achlorhydric secretory anomalies. This may be summarized as follows. Approximately 10 per cent. of the population inherit the hypersthenic gastric diathesis which is often obviously familial. They have short, high stomachs, rapidly emptying; are hypersecretory, but capable of forming little mucus in response to irritants; and when exposed to unfavourable influences, especially gastric traumata, tend to develop gastric and duodenal ulcer. Another 10 per cent. have the hyposthenic diathesis, usually with a long, low, slowly emptying stomach, hyposecretion, a tendency to secrete mucus if irritated, and, under unfavourable circumstances, tend to develop achlorhydria and those diseases associated with it.

This theory does not exclude the possibility of gastritis and its causes operating in either diathesis, but explains the particular end-result which gastritis is likely to achieve: that is, acid gastritis—hyperchlorhydria—peptic ulcer; achlorhydric gastritis—pernicious and other anaemias—carcinoma ventriculi, etc. It does mean, however, that acid gastritis is not the precursor of achlorhydric gastritis, a fact which I think is borne out by clinical experience. It explains, too, why in some cases of peptic ulcer a low normal or even hypo-chlorhydria is sometimes encountered. This must be due to the associated gastritis and mucus formation partially reducing the acidity. Further, it explains why, after medical treatment, the acidity may be higher than at the commencement, a not infrequently observed phenomenon, which is the result of the improvement in the associated gastritis.

Simpson¹⁰ has shown experimentally in guinea-pigs and rabbits the importance of the acid factor, when there is accompanying gastritis, in producing gastric erosions and ulcer. Whereas a concentration of 2.5 per cent. HCl was required to produce gastric erosions alone, when an innocuous gastritis was induced a concentration of 0.182 per cent. (=50 c.cm. NaOH, which is only just at the high normal level) produced gastric erosions. Thus it would appear that, given hyperchlorhydria, any factor which precipitates gastritis—mechanical, thermal, toxic, or infective—may cause gastric erosions and their sequela—ulcer.

It is impossible to do justice in this paper to the other methods of investigating gastritis, and I can no more than mention that important contributions have been made following the adoption of special *x*-ray technique designed to reveal the outline of the mucous membrane of the alimentary tract (Forssell). A variety of pathological changes affecting the folds of gastric mucous membrane have been described, and the diagnosis of hypertrophic and atrophic gastritis may, in some cases, be made on *x*-ray diagnosis alone.

Equally it is impossible more than briefly to mention the important advances in diagnosis of gastritis which have followed the introduction of the flexible gastroscope. Definite gastrosopic pictures are seen corresponding to hypertrophic gastritis, ulcerative or erosive gastritis, and atrophic gastritis, and a conclusive diagnosis can be made on these appearances by those sufficiently familiar with them.

Changes in Gastric Secretion

We shall next consider the alterations in gastric secretion which accompany gastritis. It has long been known that gastritis and hypochlorhydria or achlorhydria were associated, and the inference was that the effects of chronic inflammation of the gastric mucous membrane would be to depress its various secretory powers, this effect increasing with age. The fact that gastritis might be associated with hyperchlorhydria was something new, and we have mentioned the various theories of localized or pyloric gastritis (Porges,⁹ Faber⁴) and gastritis in the constitutional hyperchlorhydric (Hurst²) which have been put forward to explain it.

I have long been interested in this problem from the point of view of gastric secretion, particularly since the introduction of the histamine test.^{11 12 13 14} Some previously published work of mine suggested that good evidence of depression of gastric secretion could be obtained by the histamine method. Witts and I¹² had shown that a progressive depression of secretory capacity of the stomach was present in the various achlorhydric anaemias, and this was largely independent of the degree of anaemia existing. It could be demonstrated by physostigmine and histamine stimulation of secretion. The volume of gastric juice and its content of acid and pepsin appeared to be roughly progressively diminished through the hypochromic anaemias to the achlorhydria of pernicious anaemia.

It seemed of interest to investigate by this method a group of more or less symptomless achlorhydric cases (free from anaemia or cancer of the stomach), possessing in addition, upon *x*-ray evidence, the hyposthenic stomach, and a group at the opposite extreme possessing hypersecretory and hypersthenic stomachs. It was of particular interest to make quantitative observations on the amount of mucus which could be obtained in the two respective groups, in view of the extension of Hurst's hypothesis that the hypersthenic stomach was incapable of forming much mucus while the hyposthenic stomach produced mucus freely.

Our knowledge of the functions of gastric mucus has always been somewhat obscure. It has usually been accepted that an increased secretion of mucus is the normal response of the stomach to irritation of all types. Its presence in visible, naked-eye excess has been accepted as evidence of gastritis. Thus among 336 cases of achlorhydria investigated at New Lodge Clinic¹⁶ a gross excess of mucus was noted in 85 per cent. Only rarely is mucus persistently lacking in such cases, and complete atrophy of the mucous membrane is thought to account for this absence. There are two varieties of gastric mucin: (1) the superficial secretion which is the ordinary, visible, test-meal mucus seen as a stringy, gelatinous material, usually floating on the top of the test-meal fraction; and (2) the

deep soluble muco-protein.¹⁵ The former can be precipitated by acetic acid; the latter cannot, and can only be estimated through its nitrogen or carbohydrate content.

The selection of group representatives of the hypersthenic diathesis was from cases of duodenal ulcer, twelve in all, nine men and three women, whose ages ranged from 27 to 60. Nine of the patients were symptom-free after medical treatment. Three had occasional discomfort of mild duodenal ulcer type but had been much improved by treatment, and their condition did not suggest active ulceration in the duodenum. They all possessed the high-lying cow-horn type of stomach with rapid emptying on *x*-ray examination, and all had hyperchlorhydria at some stage of the gruel test meal.

The patients representative of the hyposthenic diathesis were all achlorhydric in the gruel test meal, and had elongated J-shaped stomachs with less vigorous peristalsis on *x*-ray examination. Among them were four men and eight women, whose ages varied between 21 and 57. In four the condition was symptomless. Three of the women had mild hypochromic anaemia, which had responded well to treatment, and their blood was now normal, while five had indefinite dyspeptic symptoms, three with transient diarrhoea, and one had early chronic rheumatoid arthritis. The average differences in the gastric secretion following histamine stimulation are shown in the following table and diagrammatically in the figures. (Figs. 1 and 2.)

FIG. 1.

FIG. 2.

FIG. 1.—Diagrammatic illustration of the difference in volume, free acid, and pepsin secretion of the stomach after histamine stimulation in a group of patients possessing the hypersthenic and hyposthenic stomachs respectively.

FIG. 2.—Diagrammatic illustration of the difference in concentration of insoluble mucus and insoluble muco-protein in twelve patients possessing the hypersthenic and hyposthenic stomachs respectively.

	Resting Juice		Histamine Juice						Dose of Histamine (mg. Ergamine Acid Phos.)	
	Volume	Mucus	Volume	Acidity N/10 NaOH	Pepsin Gross Units	Mucus in mg. Nitrogen				
						Insoluble		Soluble		
						%	Total	%		Total
Hypersthenic Group (12 cases)	c.cm. 61.7	—	c.cm. 343 ^a	101.5	98	1.34	4.32	1.3	35.71	0.9 to 1.0
Hyposthenic Group (12 cases)	16.4	+	57.3	—*	12	1.8	11.26	6.1	20.5	0.75 to 1.8

* One case produced a trace of free acid at the height of secretion, but not in titratable quantities.

It will be seen that histamine stimulation produced a very large volume of highly acid gastric juice of high pepsin content in the hypersthenic group, but only a small volume with no free acid and a low pepsin content in the hyposthenic group.

Support for Hurst's Theory

With regard to the mucus findings, which were the chief point investigated, the method is possibly open to some criticism. It is obvious that the withdrawal of gastric juice through a Rehfuß tube, even by continuous

aspiration (as was used in these cases), fails to obtain the entire secretion. A certain amount is bound to be lost through the pylorus, and this will especially apply to the rapidly emptying active stomach in the hypersthenic group. More important in this investigation is the fact that it is impossible to withdraw all the thick, slimy, insoluble mucus which is secreted, probably over most of the surface of the stomach, although a fair sample of the soluble muco-protein is likely to be obtained. In interpreting the results we must remember that the insoluble mucus figures for the total output are likely to be low, especially in the hyposthenic group.

Taking the figures as they stand for mucus, it will be seen that the concentration of insoluble mucus in the hyposthenic group is sixteen times that in the hypersthenic group, while the total amount of insoluble mucus put out, calculated from the total volume obtained, is nearly three times as much (and may be more for experimental reasons mentioned) in the hyposthenic group as it is in the hypersthenic group, despite the enormous difference in the volume of juice secreted, which is nearly six times smaller in the hyposthenic stomach. With regard to the soluble muco-protein, the concentration is three times greater in the hyposthenic group, but the total output calculated from the total volume is one and three-quarters greater in the hypersthenic stomach. Since the insoluble mucus is that which is produced following irritation, these findings therefore support Hurst's thesis that the hypersthenic stomach is incapable of producing much mucus, while the hyposthenic stomach produces it in great excess.⁵

Clinical Aspects

The clinical picture of acute or subacute gastritis brought on by some dietetic indiscretion—alcoholism, food poisoning, etc.—is fairly similar. Such attacks, even in individuals of normal gastric secretion, are accompanied by diminution or abolition of acid and pepsin secretion. In many of these cases the changes are only temporary, and if a histamine test is repeated some weeks later a return of secretion is found. I have personally observed this in four cases of acute alcoholic gastritis, in two of post-influenzal gastritis, and in one of post-pneumonic gastritis. The patient who is the subject of recurrent attacks of chronic gastritis may not infrequently masquerade as a middle-aged dyspeptic, and the attacks of dyspepsia which recur from time to time are characterized by nausea, lack of appetite, epigastric fullness, and flatulence. They may be called "gastric chill," "gastric catarrh," and "cold on the stomach," or even "liverishness."

In relation to the aetiology of this condition I should like to emphasize the importance of the gastric lesions that accompany influenza. I have now collected considerable histological evidence that severe grades of gastritis accompany the influenzal attack, especially where the gastric symptoms are marked. The diagnosis of "gastric 'flu'" has for many years been derided professionally, but there is no doubt that the condition has as its basis a true gastritis. In this type of patient we perhaps do not recognize the frequency with which gastritis is associated with chronic infective conditions of the mouth, pharynx, upper respiratory tract, and accessory sinuses, nor perhaps do we appreciate fully the frequency with which gastritis accompanies general infectious states. In these conditions, especially, it is often latent and only discovered by routine examination. The occasional syndrome of achlorhydric diarrhoea in a proportion of cases of achlorhydria is now fairly well known. It is, however, important to stress the fact that achlorhydric gastritis may be clinically symptomless.

On the other hand, we have all met patients who suffer from dyspepsia of the ulcer type, the classically

recurrent regular bouts of pain at a definite time after food, and we have perhaps realized that the individual attacks here have often been precipitated by an indiscretion of diet. We know, too, that not infrequently, even in the most typical "ulcer" cases, radiological evidence of an actual ulcer is not dependable, and even at operation sometimes no ulcer is found. These are the cases of acid gastritis and duodenitis.

With regard to the whole subject of gastritis, as recognized clinically, it is perhaps insufficiently realized that in spite of the fact that the dyspeptic manifestations appear to be of relatively minor importance, and are perhaps fairly easily symptomatically treated, a whole train of serious sequelae are apt to follow, ranging from anaemic states to carcinoma and peptic ulcer. It is therefore the prevention of gastritis, particularly in patients of the hypersthenic and the hyposthenic diathesis, which must primarily occupy our attention if these more serious diseases are to be avoided.

Prophylactic Measures

It is a counsel of perfection to expect our patients to avoid all the possible causes of gastritis, and this is especially true of the toxic and haematogenous sources that are so common in general infections, but certain prophylactic measures can be adopted, especially if we have any evidence or suspicion that our patients belong to the hypersthenic or the hyposthenic group. These measures aim especially at reducing the irritation to which the inflamed gastric mucous membrane may be subjected, and they apply equally to either group of patients. Following a known attack of acute gastritis, the mucous membrane is especially vulnerable, and it is necessary that all factors tending to prolong the condition should be removed.

In the acute irritative stages the patient is completely rested, and no food is given for twelve hours other than fruit juice and soda-water. Vomiting, in so far as it helps in eliminating irritating material, is not at first suppressed by medical measures, but later, gastric sedatives may be required. Subsequently a bland and non-irritating diet must be selected which makes little demand upon the mechanical or digestive function of the stomach: non-irritating carbohydrates such as vegetable purées, fine gruel, citrated milk, rice, sago, arrowroot, cornflour, with cream and sugar. Abundant butter, cream, honey, lightly boiled eggs, and fish can be employed. The omission of irritant elements is very important. Alcohol, strong tea, and coffee should be prohibited; only very moderate smoking, and none at all in the acid gastritis types, should be permitted, and no raw or green vegetables, salads, uncooked fruits with skins and pips, pickles, chutney, curry, new bread, pastry, muffins, hot buttered toast, and the like should be taken. Faulty habits of mastication and ingestion must be corrected. Meals should be small in bulk and taken in an unhurried fashion. Long intervals between food should be avoided: a glass of milk in the middle of the morning, in the afternoon, and in the evening is advised.

It will be seen that the mechanical ingestion and dietetic factors which have to be corrected apply equally to either group, but in the hypersthenic types the importance of the acid factor in addition has to be recognized and treated. This, of course, follows the principles well known in the alkaline medication normally employed when the ulcer has declared itself. On the other hand, in the hyposthenic group medical measures must be adopted to restore or substitute the diminished secretions.

All active forms of focal and open sepsis must be searched for and eradicated, especially in the mouth, pharynx, accessory sinuses, and respiratory tract. From such sources as these swallowed infected material comes in direct contact with an already damaged mucous mem-

brane, and in the reduction or absence of free hydrochloric acid infection may gain a permanent foothold, which, in the acid gastritis types, renders the development of a subacute ulcer from an erosion more probable.

When depressed gastric function is partially or completely established recourse may be had to local treatment by lavage in an attempt to restore to health those areas of gastric mucous membrane which are not totally atrophied. At the same time substitution therapy in the form of hydrochloric acid and pepsin by the mouth can be instituted. The effect of lavage is to remove sticky viscid mucus, retained food debris, and abnormal bacterial products, by which means the stomach is more completely rested, healing is augmented, and restoration of the normal secretion is helped. The efficacy of this treatment can be judged by the fact that out of thirty-four cases of achlorhydria in which this treatment was employed free acid returned in twenty-eight, or 82 per cent.¹⁶ The best solution for gastric lavage is hydrogen peroxide, 1 drachm to a pint of warm water. The stomach is washed out every morning before any food is taken. After the first few treatments given by a physician many patients can be taught, and are quite able, to carry out gastric lavage alone.

A more simple form of lavage can be instituted by allowing the patient to drink about twelve ounces of warm water containing 1 drachm of sodium bicarbonate to the pint, the patient lying alternately for ten minutes on the left and right sides for about half an hour. Hydrochloric acid and pepsin can be administered in mild cases as the well-known mixture:

Rx	Acid. hydrochlor. dil.	...	...	...	...	℥ x-xv
	Tinct. nuc. vom.	...	...	...	...	℥ v
	Glyc. pepsin.	...	...	...	...	5 j
	Inf. gent. co.	...	...	...	ad.	3 j
	3 j t.d.s., a.c., ex aq.					

or, in more severe cases, as a flavoured drink to be taken just before or with meals, according to the following (Hurst):

Rx	Acid. hydrochlor. dil.	...	...	...	...	3 j
	Pepsin	...	...	...	...	gr. v
	Aq.	...	...	...	ad.	3 j

Take 3 j and add a pint of water and the juice of two oranges.

The patient is ordered to take six ounces of this drink as stated before food. An alkaline mouthwash may be required in some cases to prevent any unpleasant action of the acid upon the teeth, or the mixture can be sucked through a straw. Individual patients may show different susceptibilities to this mixture, and some may require even greater dilution before they can tolerate it.

Summary

The evolution of our knowledge of gastritis has been briefly traced, with special attention to the secretory changes with which it is accompanied, and we have seen that gastritis is important in modern medicine because it is a very common affection, having a multitude of aetiological factors and associated with a number of serious diseases, with which it stands in causal relationship.

That gastritis causes depression of secretion and eventually achlorhydric gastritis has been long known, but "acid gastritis" is of more recent discovery. Achlorhydric gastritis and acid gastritis tend to occur especially in individuals possessing the hyposthenic and the hypersthenic gastritis respectively.

Examination of the gastric juice secreted after histamine stimulation in a group representative of each constitutional type shows important secretory differences, which supports Hurst's theory of a constitutional gastric diathesis.

In both groups serious diseases are likely to supervene in the presence of gastritis: anaemia and cancer, etc., in the hyposthenics; and ulcer in the hypersthenics.

Gastritis affecting individuals having a normal gastric constitution is less likely to be followed by these serious sequelae.

To prevent the sequelae of gastritis it is important to treat the disease in the earliest stages. The preventive treatment is largely dietetic, with the omission of all gastronomic iniquities; elimination of focal sepsis is important. Appreciation of the type of gastritis leads to recognition of the acid factor and its control by alkalis, or the achlorhydria and the consequent treatment by substitution with hydrochloric acid and pepsin, if lavage fails to restore secretion.

REFERENCES

- ¹ Trousseau: *Clinical Medicine*, 1872.
- ² Hayem, quoted by M. E. Rehfuß: *Diseases of the Stomach*, Philadelphia, 1927.
- ³ Faber, K.: *Lancet*, 1927, i, 901.
- ⁴ Idem: *Gastritis and its Consequences*, 1935.
- ⁵ Hurst, A. F.: *British Medical Journal*, 1933, ii, 89.
- ⁶ Konjetzney, G. E.: *Die entzündliche Grundlage der typischen Geschwursbildung im Magen u. Duodenum*, Berlin, 1930.
- ⁷ Meulengracht, E.: *Acta Med. Scand.*, 1934, lxxxii, 352.
- ⁸ Idem: *Proc. Roy. Soc. Med.*, 1935, xxviii, 841.
- ⁹ Porges, O.: *Magenkrankheiten*, Vienna, 1929.
- ¹⁰ Simpson, C. K.: *Guy's Hosp. Reports*, 1935, lxxxv, 102.
- ¹¹ Burger, C. N., Hartfall, S. J., and Witts, L. J.: *Ibid.*, 1933, lxxxiii, 497.
- ¹² Hartfall, S. J., and Witts, L. J.: *Ibid.*, 1933, lxxxiii, 3.
- ¹³ Hartfall, S. J.: *Ibid.*, 1933, lxxxiii, 37.
- ¹⁴ Idem: *British Medical Journal*, 1934, i, 136.
- ¹⁵ Webster, D. R., and Komarov, S. A.: *Journ. Biol. Chem.*, 1932, xcvi, 133.
- ¹⁶ Hartfall, S. J.: *Guy's Hosp. Reports*, 1932, lxxxii, 13.

OBSERVATIONS ON THE INTUBATION TREATMENT OF PEPTIC ULCERS

BY

W. C. D. MAILE, M.D.CAMB.

HONORARY PHYSICIAN, WOKING VICTORIA HOSPITAL

The treatment of peptic ulcers has always been a very difficult problem and a source of great controversy. An opportunity arose last year which enabled me to try the intubation treatment as suggested by Young.¹ This, in short, consists of the introduction of a small tube through the mouth into the duodenum to the distal side of the ulcer, and passing the "feed" through it. In a comparatively short space of time I had under my care twenty-five cases of gastric and duodenal ulcers.

In 1934 Bourne² published the results of the treatment of gastric and duodenal ulcers by duodenal feeding in twenty cases. He used as "feeds" milk, 8 ounces, and one egg, alternately with Benger's food (cooked for forty-five minutes) and half an ounce of cream, seven "feeds" being given in the twenty-four hours. In cases of undue loss of weight he added 4 ounces of sugar and 4 ounces of cream, distributed over the seven "feeds." Seventeen were cases of gastric ulcer and three of duodenal ulcer. If the pain was severe he gave alkaline powders by the mouth.

In the twenty-five cases which came under my care the intubation and general technique was carried out in the manner described by Bourne. Of these, fourteen were cases of gastric ulcer and eleven of duodenal ulcer. The sex distribution was: females twelve and males thirteen; and the age incidence 31 to 64 years.

X-Ray Examinations and Haematemesis

Definite ulcers or incisuras were seen in fifteen cases—gastric six, duodenal nine. Tenderness or irregularity of the caput, but no definite ulcer or incisura, was observed in eight cases—gastric six, duodenal two.

Two patients had severe haematemesis and melaena; coagulin (ciba) was given until the haemorrhage ceased, which occurred in each case in about thirty-six hours.