RECENT ADVANCES IN THE TREATMENT OF PEPTIC ULCER

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A survey of the literature dealing with recent advances in the treatment of peptic ulcer reveals a growing appreciation of the importance of the pathological physiology of this malady. In the various clinics and medical centers the gastro-enterologists are forsaking "rule of thumb" treatment and are attempting to individualize each case. The effect is that an ulcer patient is receiving more intelligent treatment than the mere prescribing of a diet and alkaline powders. Physicians are growing sensible to the fact that the most important therapeutic effort is not merely to assist in the *immediate healing* of the ulcer, but as far as possible, to prevent a *recurrence* of this lesion at a future date. It is well known that one of the most characteristic features of a peptic ulcer is its periodicity, its tendency to recur most commonly in the spring and fall of the year, often with almost symptomless remissions between these semi-yearly exacerbations of indigestion. Any treatment which fails to take into consideration these fairly rhythmic periods of reactivation will, in most instances, fail to be more than a palliative. With these facts in mind, it is the thesis of this paper to elaborate the following points: (1) the known etiological factors in the production of peptic ulcer; (2) the pathological physiology of the stomach and duodenum in ulcer cases; (3) the role of diet and drugs in the effort to restore normal function; and (4) procedures to adopt in the effort to prevent recurrence of an ulcer.

I. ETIOLOGY

Knowledge concerning the cause of peptic ulcer is far from conclusive. There are theories based upon suggestive evidence, but relatively few proven facts. This may be due to the fact that experimental duodenal ulcers can be produced so easily in laboratory animals and under conditions which do not always reproduce the probable causes in man.

When one considers the ulcer itself, it becomes apparent that the disease process is, in most instances, localized to the immediate vicinity of the ulcer. The sharply circumscribed lesion or lesions are characteristic of infarcted areas, as if a terminal or near terminal arteriole had become occluded. Thus as a result of infarction, a coin-shaped area of devitalized tissue is subject to auto-digestion by pepsin in the presence of free hydrochloric acid. This seems to me the only plausible explanation for the type of ulcer which is seen at autopsy.

The auto-digestion theory is substantiated by the fact that there has been no authentic report of the occurrence of acute peptic ulcer in the absence of free hydrochloric acid. No case of acute ulcer has been shown to develop, for instance, in a case of pernicious anemia. On the other hand Kapsinow and also Berg and Jobling have produced duodenal ulcers in dogs by diverting the flow from the common bile duct away from the duodenum. Thus the alkalinity of the duodenal contents is lowered, and the acid chyme, in passing through the pylorus, fails to be neutralized satisfactorily. This leads to the attractive theory that, in man, when there is a chronic cholangeitis, cholecystitis, or chronic pancreatitis, a lowered duodenal alkalinity plus duodenitis may prepare the mucosa for the rapid formation of a duodenal ulcer through chemical changes in the duodenal lumen.

If one of the steps necessary to the production of a gastric or duodenal ulcer is an infarction in the mucous membrane, it is obvious that this must be due to an arterial thrombus secondary to an embolus or a spasm, or to an ischemia produced by a localized lymphangitis.

Rosenow has carried out some of the most notable experiments on metastatic, embolic infection by streptococci. He has been able to produce ulcers in rabbits, by the intravenous injection of streptococci obtained from the infected teeth and tonsils of patients with peptic ulcer. Also, lesions of less severity have been produced by by injecting dead bacteria or filtrates of active cultures. The results of this work have been confirmed by Haden and Bohan.

Another avenue of transmission of infecting organisms to the gastric and duodenal mucosa has been suggested and advocated by Moynihan. He believed that in as many as 66 per cent of his patients who had had operations for ulcer, there had been an associated appendicitis. By injecting the lymphatics in cadavers he was able to trace the lymphatic channels from the appendix to the pancreatic area. As the result of his studies, he believed that there was a direct route for the extension of infection from the appendix to the duodenum and stomach. Contrary to this opinion of Sir Berkeley, however, Walton was able to find evidence for a preexisting appendicitis in only 4 per cent of his cases. In my own experience, I have found, not infrequently, the association of gall stones, or chronic appendicitis with acute or chronic peptic ulcer. That this may be more than a coincidence is attested by the fact that in two such cases the removal of an infected appendix caused a subsidence of ulcer symptoms for a period of more than two years. Yet, in many other instances, appendectomy has not cured the

ulcer, nor prevented its recurrence. I am quite convinced that the appendix and gall bladder must be regarded as possible foci of infection with but very little greater tendency to produce peptic ulcer than infection from the teeth, tonsils, prostate or cervix uteri.

There is much doubt in the minds of investigators, that swallowed pathogenic bacteria can produce peptic ulcer. Even though streptococci have been demonstrated in the crater and granulation tissue bordering the ulcers, as yet there is no proof that these come from the mouth. The most logical pathway would seem to be the blood stream or lymphatics.

It is well known, too, that severe toxaemia may be accompanied by the appearance of a peptic ulcer such as production of a duodenal ulcer after a severe burn.

Let me repeat my firm belief that a peptic ulcer will develop only if there has been a previous local devitalization of tissue by interference with the blood supply, and a secondary autodigestion by pepsin in the presence of free hydrochloric acid. I attach little importance to any principle of local trophic disturbance through a disorder of the gastric nerves save as this might influence the local blood supply. If there is any analogy between the formation of aphthous ulcers in the mouth and of peptic ulcers, it must operate through modifications in the vascular supply.

The second important factor in the development of ulcer lies, I believe, in the constitutional make-up of the patient. There appears to be a distinct type of individual who may develop a duodenal ulcer. The man with the *duodenal ulcer* diathesis is of the high-tension type, energetic, mentally alert, and nervous. He works hard, eats rapidly and relaxes little. He is short chested, rather stocky, athletic in tastes. His stomach lies in a high position, it is hypertonic and hypermotile. He is often spoken of as the human dynamo, the life of the party. The patient with *gastric ulcer*, however, falls more often into the enteroptotic group. He has a relaxed hypotonic stomach, he fatigues readily and is slowed in his reactions. There is no satisfactory explanation for these two different physiological panels. However, these two predisposing types do exist and the necessity of modifying the life of such persons according to their type must be taken into consideration when individual treatment is advised. I would not presume to imply that patients with enteroptosis may not have duodenal ulcers, or that the hyperkinetic individual who perhaps is the president of the Rotary Club, may not develop an ulcer on the lesser curvature of the stomach. However, the statements I have made concerning predisposition to the two types of ulcer, usually hold true.

From the foregoing brief remarks it can be realized that the living habits of the patient are closely linked with his constitutional make-up. Over-fatigue, when brought about repeatedly, acts by lowering general bodily resistance and thus a latent ulcer may become full-blown; excessive use of tobacco may act, as Langley has shown experimentally, by paralyzing the synapses of the sympathetic nervous system. Thus, the effect of excessive smoking may bring about over-activity of the vagi, accompanied by hypersecretion and hypermotility. A large number of patients who develop a duodenal ulcer are excessive users of tobacco.

The significance of this high-tension, dynamic, manic type of constitution in preparing the ground for the development of a peptic ulcer will be elaborated in the next section of this paper.

II. The Pathological Physiology of the Stomach and Duodenum in Cases of Peptic Ulcer

The two most important normal physiological functions of the stomach are secretion and motility. In ulcer patients these modalities are disturbed. The secretory imbalance is revealed by means of the gastric tube with the finding of hypersecretion and a high level of free hydrochloric acid. In roentgen-ray studies disturbances in motility are demonstrated, by hyperperistalsis and pylorospasm. These two factors appear to be the exciting causes of ulcer pain, as well as to interfere with the rapid healing of the damaged mucosa. I believe that I am in accord with most of the students of gastroenterology when I assert that the presence of free hydrochloric acid is necessary for the production of pain. A possible exception might occur in certain varieties of penetrating ulcer when the pain may arise from a local peritonitis or the involvement of adjacent structures. The common use of alkalies as well as the recent use of mucin are therapeutic efforts to neutralize the acid, or to lessen its contact with the ulcerated area.

For several years I have felt that much of the discomfort associated with peptic ulcer, and especially with duodenal ulcer, is due to pylorospasm. This works in two ways: first, by contributing, with the peristaltic waves, to increased intragastric tone; and second, by preventing the normal neutralization of acid chyme which occurs when the bile-stained duodenal contents are regurgitated through the pylorus into the stomach. A second effort in therapy, then, is to influence pyloric relaxation and diminish peristalsis by the use of atropin, and its derivatives, and possibly of other drugs. In my experience, the control of the gastric tone and of pylorospasm is of much greater significance in giving symptom-

atic relief than is the inhibition or neutralization of hydrochloric acid. However, the best results come with the use of both methods.

So far I have carefully refrained from using the terms vagotonia or sympatheticotonia. While medication used to depress the vagus activity gives most satisfactory results, I still believe that the best term to use in this connection is simply disturbance in the autonomic nervous system. In this way one remains noncommittal. One can easily conceive that hyperepinephrinemia produces arteriolar spasm through the sympathetics, and that an accompanying vagotonia can produce muscular unrest in the whole gastrointestinal system.

III. The Role of Diet and Drugs in the Effort to Restore Normal Function

The following outline of management includes some of the methods used in the treatment of ulcer cases. I have summarized the therapy in a graphic form so that I can simplify my discussion.

A — Medical Management (Simple Ulcers)

Rest in Bed	Modification of Living Habits		Ambulatory treatment
Diet			
Lenhartz	1. Dietary	Regularity	Diet
Sippey		Calmness	Smooth diet
Smithies		Vitamins	Extra Cream
Coleman			
Leube	2. Work	Tension at work	
Others		Dissatisfaction	
		Worry	
	3. Social and Habits	Loss of sleep	
		Alcohol	
		Tobacco	
		Sex	
	4. Relaxation	Vacations	
		Midday rest	
		Hobbies	

Medication
- Olive oil
- Alkalies
- Atropin
- Bromides
- Mucin
- Nitrites
- Non-specific proteins

Removal of foci of infection

B — Surgical Management (Refractory Ulcers; Complications)
- Gastro-enterostomy
- Partial Gastrectomy
- Plastic Operations

The first decision the physician must make is whether to treat his patient in bed or whether he may permit him to be up and go to work. In recent years, I have found that most of my patients, both laborers and brain workers, make more progress when allowed to be ambulatory, but with considerable modification in their normal living habits. I am thoroughly convinced that the important factor is relaxation of nerve tension and not a horizontal position of the body. A man whose children are hungry, or an executive who imagines that his business is going to the bad will not get much relaxation when treated in bed.

In looking over my records, selected at random, of ten cases of simple acute ulcer treated in this way, I find that all but two, or 80 per cent, were relieved of symptoms within ten days after treatment was begun. All of these patients, whose ages ranged between 26 and 57 years, and whose symptoms had existed from 5 months to 13 years, had simple acute ulcers, as manifested by a typical history of nervousness and repeated exacerbations of pain, high gastric acidity, and stomach lesions demonstrable by x-ray examination. I advised a simple bland diet with extra cream midway between meals and at bed time. The medication consisted in the use of olive oil, one tablespoonful before meals; an alkaline powder, three to six times a day; atropin, grains 1/100 three times a day; and the liberal use of chewing gum. With this regimen the symptomatic relief has been just as satisfactory and as lasting as in patients who have been treated by a modified Sippy routine. The ambulatory patients have adjusted themselves to a normal routine while the patients treated by means of a Sippy, or similar type of routine, still must adapt themselves to their work after the period of rest in bed. In short, I feel quite certain that the important features are not in the diet, whether it be the Sippy, Lenhartz, Smithies or what not, but rather in the living adjustments plus the simple medication used. Undoubtedly some patients will require a Sippy regimen, but I believe that these are few in number and possibly limited to those in whom the disease has been complicated by hemorrhage.

The purpose of medication in the treatment of an acute peptic ulcer is four-fold: (1) to inhibit the secretion of hydrochloric acid; (2) to partially neutralize the acid already present; (3) to relieve gastric hypermotility and pylorospasm; and (4) to protect the ulcer from contact with digestive juices.

I shall discuss these features in the order just given. The secretion of hydrochloric acid is the result of nervous or hormonal activity. It can be inhibited by the use of fats and by atropin.

With this in mind, it is my custom to administer one tablespoonful of olive oil before meals, three times a day, and to prescribe a small glass of one-half milk and one-half cream, midway between meals, and at bed time. The atropin is given in doses of grains 1/100 two or three times a day.

The second purpose of medication, partially to neutralize acid already formed, is best accomplished by antacids. The use of alkalies in the treatment of incipient, acute or chronic ulcers is therefore a logical therapeutic procedure. Let us now consider the neutralizing efficiency of some of the alkalies in common use.

Sodium Bicarbonate. This salt relieves ulcer pain almost immediately, because of its ability to neutralize hydrochloric acid very rapidly, and also because of the distension effect upon the stomach by the formation of carbon dioxide. Both of these effects relieve, for a time, pylorospasm and gastric hypermotility. However, the chief objection to this alkali is that sodium bicarbonate has the peculiar property of stimulating a great secretion of gastric juice after neutralization is completed. Then too, if ulcers are near the perforation point the distension caused by the carbon dioxide gas, may facilitate perforation. This alkali should only be used when other forms of treatment have failed to relieve pain. In such instances, it should be given very much diluted and at half hourly intervals, to neutralize the acid as rapidly as it is formed. However, the use of bicarbonate of soda in the treatment of peptic ulcer should be avoided if possible.

Magnesium Oxide. This is a more desirable alkali to use since it has nearly four times the neutralizing power of sodium bicarbonate and, furthermore, it does not form carbon dioxide. However, it also stimulates a secondary increase in acid secretion after the initial neutralization. It has a mild laxative effect.

Calcium Carbonate. This alkali is more desirable for use in ulcer than either sodium bicarbonate or magnesium oxide. It does not produce a secondary hypersecretion by the gastric mucosa nor is the excess of alkali absorbed into the body. Calcium carbonate passes unaltered in the feces, yet there is some increase in the alkalinity of the blood, due to the reconversion of the calcium chloride formed in the stomach to calcium carbonate in the intestine. However, this alkaline effect is considerably less than that of sodium bicarbonate. The powdered chalk, on the other hand, does form carbon dioxide in the stomach, which is an added disadvantage.

Tribasic Calcium and Magnesium Phosphates. In 1923 Greenwald pointed out that the tribasic phosphates of calcium and magnesium act as antacids in the stomach but not as systemic alkalies, since

they are excreted in the feces rather than in the urine. Other workers have found that these salts act as efficient alkalies in the treatment of ulcer, although, at times, they give less relief than sodium bicarbonate. These salts do not give rise to toxic symptoms nor any elevation in the blood urea. By regulating the relative proportion of the calcium and the magnesium salt, diarrhea can be prevented. Their neutralizing strength is about one-half that of an equal weight of sodium bicarbonate.

Sodium and Potassium Citrate are also efficient alkalies and can be added to milk without the loss of neutralizing power.

With regard to alkalosis, I should like to say a few words. This condition is most likely to develop in patients who have renal insufficiency as manifested by a high non-protein nitrogen of the blood, or who have pyloric obstruction with resultant vomiting. The use of *sodium bicarbonate* or of *calcium carbonate* which effect the pH of the blood, should be avoided in such patients.

Symptoms of alkalosis usually develop within the first two weeks of treatment. The early symptoms are loss of appetite, irritability, dry mouth, headaches, dry skin, muscular soreness and finally nausea and vomiting. In our experience at the Cleveland Clinic any such symptoms have been of a mild type and have cleared up with reduction in the amount of alkali taken. It is best, with such patients, to use only the tribasic phosphates.

The use of bismuth salts in the treatment of peptic ulcer has a long history. However, recent work has shown that it is valueless either to neutralize acid or to coat over the ulcer crater. It has the added disadvantage of producing a black stool which might be taken for melena.

To summarize the discussion on alkalies, it appears that the most satisfactory alkalies for use in the treatment of peptic ulcer in the order of their importance are the following: (1) tribasic phosphates of calcium and magnesium; (2) calcium carbonate; (3) sodium and potassium citrate; (4) oxide of magnesia; and (5) sodium bicarbonate.

One may inquire whether or not it may be possible to lessen gastric secretion before neutralization is attempted. Pavlov showed in 1914 that olive oil diminishes the secretion of gastric juice. This is most likely brought about by inhibiting the gastric secretory factor as well as by producing a delay in evacuation of the stomach. Thus, in the absence of pylorospasm or obstruction, there is a greater tendency for the alkaline duodenal contents to be regurgitated into the stomach. Cream and sweet butter, undoubtedly, act in a similar manner and have the added advantage of being food

rich in calories. I have already outlined the method of using olive oil before meals and cream midway between feedings. The third reason for the use of medication in the treatment of peptic ulcer is to prevent hyperperistalsis and pylorospasm. It is very important to overcome this physiological imbalance, or else the unfavorable symptoms may be prolonged or healing may be delayed. Pylorospasm increases discomfort or pain, it prevents the normal regurgitation of alkaline bile and pancreatic juice into the stomach and also prevents the gastric secretion from passing into the small intestine. With pylorospasm there is an increase in the number of peristaltic waves in the gastric wall, so that this organ is writhing and churning with unusual activity. This fact may be substantiated clinically, for I have been able to relieve pain in several cases of chronic duodenal ulcer by the use of atropin alone. I prefer to use atropin or its less toxic derivative, novatropin, rather than the tincture of belladonna, since the latter may have variable strength. Atropin inhibits gastric secretion in the same manner that it inhibits motility, by affecting the nerve endings of the vagi.

Thus it is that if the secretory or motor functions of the stomach can be controlled the symptoms in uncomplicated ulcer cases can be relieved.

Of the recent advances in ulcer therapy, perhaps the most promising is that advocated by S. J. Fogelson. Basing his treatment on the physiological observations of Heidenhain, of Pavlov, and of Ivy and Kim, he suggested that gastric mucus was an ideal antacid in that (a) it combines readily with free acid; (b) it is a natural substance which plays normally a protective, soothing and lubricating rule in the function of mucous membranes; and (c) its secretion or ingestion causes no chemical disturbances in the body and no unfavorable effect on gastro-intestinal secretory or motor activity."

Using powdered mucin, prepared by Armour & Company, he found that one gram combines with 1 c.c. of 0.5 per cent hydrochloric acid. In his chemical experiments, he used one ounce of powdered mucin with each meal and 20 to 40 grains in tablet form at intervals of one-half to one hour, when the patient had severe pain, or when the gastric contents after an Ewald test meal showed an unusually high level of free acid. He found that the symptoms disappeared after three days of treatment and there was continuous absence of pain. He believes that there are two factors involved in the relief, namely: (1) protective coating of the ulcer, and (2) neutralization of free hydrochloric acid and delaying the progress of pepsin through the layer of mucin. With this work in mind, one must agree with the chicle manufacturers, that to chew gum aids digestion.

Among the more recent forms of treatment for ulcer is that of protein shock. Meyer and Kartoon used the intravenous injection of foreign protein in the treatment of peptic ulcer. Their conclusions were as follows: (1) Relief from pain during treatment for peptic ulcer by injection of foreign protein is independent of changes in acidity. (2) Diminution in gastric tonus and contractions are important factors in the relief from pain. (3) Increase in vascularity in the capillary bed in and about the ulcer is an important factor. (4) Non-specific protein therapy is only an adjunct in the treatment of peptic ulcer.

Parathormone has been used in the treatment of peptic ulcer in doses of 2 to 3 units daily, on the basis of its ability to reduce muscle tone. However, the advocate of this treatment used also atropin sulphate in doses of 1/100 grain, which of course vitiated his results, for as stated above, atropin itself will relieve the pain.

Very recently Beams and Barlow have shown that the use of nitrites in the form of amyl nitrite, nitroglycerin, and sodium nitrite were very efficient antispasmodics. These drugs were used experimentally during fluoroscopic observation of the gastro-intestinal tract. The conclusions drawn from this study were as follows: (1) The effect of nitrites on abdominal pain arising from the gastro-intestinal tract was observed in sixty patients. All of the patients without organic lesions were relieved by the nitrites. (2) Evidence has been offered which indicates that relief from pain by the nitrites is dependent on cessation of peristalsis and diminution in tone. The failure to obtain relief is probably due to the inability of the muscles to relax. (3) Of 200 patients observed in the fluorscopic studies only ten failed to show cessation of peristalsis and diminution of tone in the stomach and intestines following the use of nitrites. (4) Nitrites have been found to be a great aid in differentiating organic deformities from functional ones. (5) The antispasmodic action of nitrites is to be preferred to that of atropin, but neither is wholly satisfactory.

This work by Beams and Barlow suggests the advisability of giving physiological doses of nitrites in those cases of painful ulcer in which atropin has failed to give relief or in which atropin is not well tolerated. I have not had an occasion, as yet, to use this drug.

The treatment of uncomplicated peptic ulcer has many features in common with diabetes. In both disease, the living habits of the patient have been faulty, they have eaten improperly; high nervous tension has been a common feature; both diseases are chronic, both tend to recurrences; both may develop serious complications, and both require dietary regulation. In peptic ulcer, as in diabetes,

the patient must be instructed in the main features of the disease so that he can cooperate intelligently with the physician. There is no reason why an ulcer clinic should not be as important in the establishing of proper treatment of peptic ulcer as similar clinics are in the treatment of hyperglycemia.

Therefore, our therapy must begin by a better regulation of the patient's living habits. He must be told that the use of tobacco, in any form, must be discontinued until the ulcer is healed, that the surreptitious use of tobacco is a greater sin than the stealing of his neighbor's purse. He must shun Bacchus to the possible sacrifice of conviviality. He must substitute milk for Madeira, water for wine. Then too, it is important for him to retire sufficiently early to get nine hours of sleep at night, and he must rest during the day, particularly after meals. All of this may require a complete "about face" in his habits of living. If his friends can not sympathize with this complete conversion in his life, then he should seek those who live at a more sane level. Let such a patient eat more slowly and more regularly, let him follow with fidelity the diet list and medication prescribed. Such virtue will be rewarded.

Treatment of an acutely bleeding ulcer. The patient should be put to bed at once. If there is vomiting of blood or melena, morphine sulphate, grains 1/6, with atropin sulphate grains 1/100 should be given by hypo. He should be starved until all bleeding has ceased for at least forty-eight hours. An empty stomach is less likely to bleed. If bleeding will not cease, blood clots can be evacuated from the stomach by the careful use of a stomach tube. The lavage fluid may consist of ice cold water, four to six ounces, and the washing is repeated until the water is free of blood. If this is insufficient, the washing fluid may consist of iced 1/1000 ferric chloride solution, followed by the instillation of drachms one of 1/1000 solution of epinephrine.

Blood transfusions are indicated if the hemoglobin falls to 40 or 50 per cent. Fluid can be given by Murphy drip — using 5 per cent glucose in physiological sodium chloride solution. A similar solution can be used intravenously. A surgeon should always see the patient in consultation, for, if all of these measures fail, operation will be required. Occasionally, in spite of all care and even though repeated transfusions have been used, the patient will continue to bleed and finally die. Hence, these cases are emergency problems and should be watched with great care and attention.

How is the physician to know when an acute ulcer is healed? This is a difficult matter to establish with certainty. The presumptive evidence of a healed ulcer is cessation of pain, tenderness or

rigidity; the absence of occult blood in the stools and x-ray evidence for healing. In *gastric ulcers*, Hurst and Stewart advocate x-ray examination at the end of two or three weeks of treatment and then at weekly intervals until the crater is reduced to a minimum or until the deformity has disappeared. Where the deformity remains unchanged and occult blood persists in the stools, operation is advocated on the basis that the ulcer may be malignant.

If all evidence indicates that the ulcer has healed it is still important to keep the patient on a limited diet and under regular periods of observation for the ulcer diathesis is still present.

I shall deal very briefly with surgical treatment in peptic ulcer. The well established indications for surgical intervention are as follows: (1) intractable ulcer, (2) carcinoma suspect, (3) pyloric obstruction, (4) recurrent hemorrhage, and (5) perforation.

I should like to emphasize that pyloric obstruction with retention should have the advantage of medical treatment before an operation is performed. I believe that roentgenologists will agree with me that they have no certain method of determining whether the obstruction is due to stricture, spasm or edema. The use of atropin as a differentiating method has proven disappointing. Even if a carcinoma is suspected, especially when free hydrochloric acid is present in the stomach, there is no particular harm to be done by giving the patient two weeks of medical treatment followed by a second x-ray study. To quote Frank H. Lahey, "with increasing experience with ulcer, I have learned that pyloric obstruction occurring with active ulcer symptoms is most commonly due to spasm, infection and edema and that a large majority of such patients can be relieved of their obstruction by non-operative measures, rest, diet and neutralization, and that surgery is much less frequently indicated."

I wish to emphasize the importance of having a surgeon see all patients in whom hemorrhage is present or in whom perforation or organic pyloric obstruction is suspected. It will be much easier for him to have a complete picture of the case, should emergency operation be required.

Summary

In the modern treatment of peptic ulcer we attempt to accomplish three things: (1) relief of the symptoms, (2) healing of the ulcer, and (3) prevention of recurrence. To attain these ends it must be realized first that there is such an entity as a constitution predisposed to ulcer. With this in mind, an attempt is made to modify the patient's living habits and to regulate his life even after

the acute ulcer has healed. Diets, alkalies, atropin and olive oil or other fats also are prescribed. In addition prompt surgical intervention must be afforded the patient when the proper indications are present. After the operation, the internist must again assume the responsibility of treatment.

I believe that our success will be directly proportional to the care with which patients are educated in a knowledge of their disease, and are thus influenced to give the physician their willing cooperation. The medical profession is as yet in a maze of uncertainty about ulcer. It is to be hoped that further knowledge of the factor of focal infection or the new attack of adrenal denervation, such as is being pioneered by Doctor Crile, may help to lead us out of the wilderness.

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