should not be construed as evidence of hypertrophic gastritis.

I do not agree with Dr. Schindler about the dogs. The gastric mucosa of our dogs was very much the same as that of man.

Pigment spots are frequently observed. Their true significance is poorly understood. They may be related in some way to ulcer, but that has yet to be proven.

As to the relationship between gastroscopic and proctoscopic findings, we have studied that frequently and have found no correlation whatever.

It has been asked how many bulbs full of air are required to produce this effect which I have described. This question I cannot answer. However, one cannot measure intra-gastric pressure in that way, as the patient may eructate or after a period of time the stomach, itself, may relax and, therefore, lower the pressure. The only method of measuring intragastric pressure is an apparatus such as we have used in this experiment.

The X-ray evidence of hypertrophic folds disappearing after atropine is in line with what I have said. The X-ray diagnosis of hypertrophic gastritis probably represents nothing but the contraction of the gastric musculature which throws the mucosa into folds. This subsides when the patient relaxes or is given atropine.

I wish to go on record as saying that gastroscopic examinations are extremely important. They are indeed fascinating, but so far as chronic gastritis is concerned it seems to me that we have gone off on a wide tangent and would do wisely to pull in our sails and start again.

Gastritis Simulating Peptic Ulcer*

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MANY physicians still hesitate to accept gastritis as a legitimate member of the clinical group of diseases of the stomach. Observations by a number of competent workers correlating symptoms and gastroscopically demonstrable appearance of tissue are indicating the importance and formidableness of gastritis as a symptom-producing entity. Furthermore, some definite impressions regarding the character of the symptoms which gastritis may produce are being formulated.

Moynihan and his school have made the syndrome which spells peptic ulcer so well known that any complex of symptoms suggesting this disease, if such a lesion cannot be demonstrated by roentgenologic examination or exploration, is likely to be classified and often mistreated as a "functional stomach." Further experience with the gastroscope, no doubt will relegate to the limbo of diagnostic subterfuge many terms, such as "Hale-White hemorrhages," "gastrotoxic hemorrhages" and "pseudo-ulcer."

From a review of data available to us for study we have been unable to discover a consistently recurring syndrome for certain types of gastritis. For instance we cannot say that hypertrophic gastritis will regularly cause a certain group of symptoms. Indeed, it may not give rise to any complaints at all. Conversely, however, a syndrome definitely suggestive of peptic, or more particularly gastric, ulcer which includes in many instances the complication of hemorrhage is quite capable of being produced by gastritis. When bleeding is present in these cases, it usually is found to be associated with the superficial dissolution of tissue. Furthermore the development of such a syndrome in the presence of gastritis in a stomach in which the secretory rates are adequate should not be unexpected. We see no reason to deny to gastritis the same pain producing mechanisms and pathways which are utilized by ulcer in indicating its presence.

MATERIAL STUDIED AND RESULTS

This discussion results from a review of sixty cases of gastritis, most of which were under our personal observation in the hospital. In many instances it was possible to make parallel follow-up observations on the clinical and gastroscopic condition. Usually the gastroscopically demonstrable improvement, disappearance or reappearance of the intragastric lesion was mirrored in a corresponding mutation in the pattern of the clinical picture.

Cases were accepted for inclusion in this series only if we were convinced that the symptoms which clinically suggested the presence of peptic ulcer were caused by gastritis as demonstrated at surgical exploration or by the gastroscope. In many instances roentgenographic investigations of the gall bladder (Graham-Cole method) were undertaken but results were always negative. No case of gastric anacidity was included. The great majority (75 per cent) of these patients were of a highly nervous, persistent, intensive type, similar in fact to the individuals who are likely to have duodenal ulcer. One of every two patients included in this series was an inveterate tobacco smoker; those who smoked cigarettes were most numerous. Fifty per cent of the patients used alcohol and about a third of these drank immoderately.

It is not within the scope of this paper to attempt a classification of the various types of gastritis and discuss the symptoms caused by each group. In 84 per cent of the cases included in this series chronic erosive or hypertrophic types of lesions were considered to be present; in 40 per cent of these there was evidence of mucosal dissolution. The remaining 16 per cent of the cases in the series were classified as instances of the

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acute catarrhal types of gastritis from the gastric evidence.

A composite picture of the symptoms presented by these patients would cause the presence of a peptic ulcer to be suspected. As in cases of peptic ulcer symptoms were often intermittent. In twenty-three cases symptoms were intermittent at first but later distress occurred almost daily. Twenty-one patients had always had intermittency of bouts but twelve had had distress practically every day since the onset. Four had had attention called to the gastric disorder by symptoms other than distress. The duration of symptoms varied from two weeks to forty years; the average was about eight years.

There was a fairly consistent recurrence of distress at certain definite periods following the meal. In some instances there was epigastric discomfort, a feeling of fullness, sometimes with slight nausea, shortly following the meal. Often distress occurred immediately on eating or a few minutes afterward, especially when coarse, fibrous, fatty or irritating foods had been ingested. This type of distress was most likely to be described as a gassy, filled-up sensation of pressure in the epigastrium with associated bloating, belching and regurgitation. More often, a burning type of distress developed with or without this preliminary disturbance, but with a distinct interval of postprandial comfort. This distress was spoken of as sourness, hunger distress, rawness, heartburn, or just burning pain and was frequently severe. It was often situated high in the epigastrium, was frequently substernal, and at times was in the left side of the epigastrium. The interval of comfort preceding such distress varied from fifteen minutes to three hours, and the type of distress suggested a chemical factor. This symptom caused more than 57 per cent of these patients to wake up at night, usually between 12 m. and 2 a. m. Some of the patients complained of distress which came on before their morning meal. This was especially likely to happen if they smoked a cigarette or two before breakfast. In many instances the pain was very severe. At times it was colicky. One of the most important diagnostic features in these cases was the location of the distress of which these patients complained. In 95 per cent of instances this was indicated as being high in the epigastrium, infrasternal or substernal, often slightly to the left of the midline. In more than 60 per cent of instances the distress seemed to extend upward, under, or along the sternum, or into the throat. Less frequently there was projection of distress to the left side of the thorax or back or even to the shoulder and left arm. This occurred usually during periods when the pain was at maximal intensity.

The ingestion of bland food usually brought relief. An alkali helped, particularly if it caused belching. There was sometimes an atypical response to the use of the stronger alkalis. These might produce complete relief of symptoms for a time, only to be superseded by no relief or even an aggravation of distress following their use.

Systemic manifestations were not marked as a rule. Occasionally, there was anorexia; loss of weight was not marked and weakness was not unusual. In rare cases there was a complaint of dizziness. Many of these patients complained of vomiting. More often this was actually regurgitation of sour material at the height of the distress.

In 33 per cent of the cases included in this series hematemesis or melena were present. Increased gastric secretory rates were noted in 35 per cent. The average for the group was 56 (Töpfer's method) for total acid and 44 for free hydrochloric acid. Blood tinged gastric contents were observed frequently. Many of these patients commented that the pressure of their clothes hurt the upper part of their abdomens. Epigastric tenderness was usually demonstrable.

Roentgenologic investigations failed to show the presence of peptic ulcer in any of these cases. In many instances two and three investigations were made without demonstrating any ulceration. In 40 per cent of these cases roentgenographic investigation of the gall bladder (Graham-Cole method) was made, always with negative results. In 21 per cent roentgenologic investigation revealed the presence of mucosal irregularities suggested by the roentgenologists as being evidence of gastritis. It is not within the scope of this paper to indicate the method and the results of the treatment of gastritis. Further observations are necessary on the results of treatment, which have been rather encouraging up to the present.

SUMMARY AND CONCLUSIONS

Our experience with sixty cases of definitely demonstrable gastritis, usually of the hypertrophic or granular type, scrupulously investigated to rule out all other possible organic or reflex causes for indigestion has led us, after prolonged skepticism, to these convictions:

1. Gastritis should be accepted as a legitimate member of the family of stomach diseases. The task of correlating more accurately the visual, clinical and pathologic findings in such cases however, still remains.

2. The patient who does not give roentgenologic evidence of disease in the upper part of the gastrointestinal tract, but who has symptoms referable to the epigastrium or left hypochondrium, which recur at fairly definite intervals following meals, often recur at night and are relieved by food or soda, even though these symptoms lack the clock-like precision of many peptic ulcers, should be considered to have gastritis, be given treatment for gastritis, and whenever possible gastroscopic examination should be made.

DISCUSSION

DR. HENRY A. RAFSKY (New York City): I would like to present two slides to show, how in two different types of cases, the clinical syndrome, which was due to a hypertrophic gastritis, definitely simulated a peptic ulcer.

(Slide) This patient, a 37 year old male, was operated upon for a large duodenal ulcer three years ago. A gastroenterostomy was performed. The patient remained well for about nine months. Since that time he has had periodic recurrences of the ulcer symptoms such as abdominal pain, nausea and vomiting. In addition the patient had three massive hemorrhages during this interval. After the last attack of bleeding he was re-admitted to our service at the Lenox Hill Hospital. All that the X-ray examination showed, at this time, was the large rugae which can be readily seen in the film. I gastroscoped the patient and at the site of the enlarged rugae evidence of a definite hypertrophic gastritis was visualized. No ulcer could be

seen. The patient was again operated upon. At this time no evidence of the previous ulcer could be found, but a thickened indurated area was palpated at the site of the hypertrophic gastritis. This area of hypertrophic gastritis was resected and was reported by the pathologist as "chronic gastritis." It is about fourteen months since this operation was performed and the patient has been well ever since.

(Slide) The next case is a different type of case. This patient was operated upon about ten years ago for what was regarded as an inoperable carcinoma of the pancreas. A cholecystogastrostomy was done to relieve the jaundice. The patient came back two years ago complaining of typical ulcer symptoms such as periodic attacks of pain two hours after eating and relieved by food. The X-ray, as you can see in the slide, showed a good functioning cholecystogastrostomy. No X-ray evidence of ulcer could be seen. When I gastroscoped this patient I found area of typical hypertrophic gastritis about one inch in diameter just where the bile kept emptying into the stomach. Small deposits of bile could be seen in various fields between the folds. This area of hypertrophic gastritis was confined to the region around this part of the stoma where you see the barium tract. On the basis of this finding, we treated the patient on an ulcer régime and ulcer diet. During the past two years he remained symptom free unless he went off his diet, at which time he experienced a recurrence of his ulcer symptoms.

I would like to show this third slide to illustrate the secretory response in this individual. Notwithstanding the fact that bile keeps pouring into the stomach all the time, you see a typical hyperchlorhydria curve, which is very frequently encountered in peptic ulcer.

DR. ASHER WINKELSTEIN (New York, N. Y.): The method used by Schindler in studying gastritis was to gastroscope a large number of patients, and, finding a normal appearance or a superficial, atrophic, or hypertrophic gastritis, go back to the clinical symptomatology and attempt to correlate it with the gastroscopic picture. This morning we have seen a tendency, which I think is a very good one, viz., to select very carefully certain clinical groups, gastroscope them, and then attempt a correlation.

In agreement with Dr. Rivers, I wish to state that we have had at our hospital recently a similar experience. We selected fifteen cases with typical ulcer symptoms, hyperchlorhydria, and negative X-ray findings for gastroscopy. Seven showed gastritis, four hypertrophic and three superficial, and the other eight were normal. We may conclude that about half of the cases with typical ulcer syndrome with negative X-ray findings are functional cases, and the other half are cases of gastritis. Whether the functional cases go into gastritis is conjectural.

We then took fourteen patients with true achlorhydria, determined by the fact that test meals plus histamine and neutral red showed no secretion of acid or neutral red. Nine of these showed an atrophic gastritis; four were a mixture of atrophic and hypertrophic gastritis.

It therefore seems possible to correlate certain definite clinical pictures with gastroscopic findings.

DR. DAVID H. ROSENBERG (Chicago, Ill.): Mr. President, Members and Guests: I have had the privilege of observing several patients who clinically manifested the peptic ulcer syndrome but in whom the roentgenographic and fluoroscopic findings were entirely negative. Some of these patients had been observed in various clinics, both in this country and abroad, and their symptoms were regarded by some physicians as functional, and by others as organic in nature, that is, due to peptic ulcer. Gastroscopic examinations, however, repeatedly showed chronic hypertrophic gastritis.

I should like to raise the question, Dr. Rivers, whether in some of these patients we may be dealing with an associated duodenitis (with or without superficial ulceration) which may be responsible for the clinical symptoms. One knows full well that in many patients with ulcer symptoms who roentgenographically reveal a negative stomach and duodenum, a superficial ulcer may be found in the duodenum at operation.

I should further like to ask Dr. Rivers his method of therapy in these cases. In my own experience I have found that the usual ulcer regime was all that was necessary.

DR. ANDREW B. RIVERS (Rochester, Minn.): I think that duodenitis is quite capable of presenting symptoms which are similar to those caused by gastritis. It is unfortunate that we cannot explore the duodenum as we do the stomach by means of the gastroscope. This would aid in diagnosing more cases of duodenitis. I am sure, however, that both gastritis and duodenitis are capable of producing symptoms similar to those caused by duodenal ulcer.

Regarding the treatment for gastritis: The time allotted for discussion is too brief to say much about this. I agree with Dr. Rosenberg that the type of therapy to be employed is much like that which is effective in treating ulcer. Each case has to be studied individually and suitable treatment employed. Bland diets, antacids, and antispasmodics are important.

Then, too, the use of vitamins in concentrated form is useful therapy. Occasionally, we use gastric lavage of weak silver solutions, metaphen, hydrogen peroxide, or salt solution.

Curtailment of the use of tobacco or alcohol and improvement of the general health are important in the care of this disease.