showed a loss of 60.2 gm. Cod liver oil showed a gain of 45.5 gm. over the normal, while with the same rats Vinol showed a net loss of 43.5 gm. Cod liver oil showed a gain of 19.5 gm. over the normal, while with the same rats Wampole's Preparation showed a loss of 31.6 gm. Cod liver oil showed a gain of 45.4 gm. over the normal, while with the same rats Waterbury's Compound showed a net loss of 31.7 gm.

Not only did cod liver oil show a marked superiority as a source of nutriment over Hagee's Cordial, Vinol, Wampole's Preparation and Waterbury's Compound, but it also showed a remarkable reconstructive and recuperative power in its ability to enable rats to gain weight rapidly and steadily after having suffered from a deficiency in nutriment when fed the four preparations named above.

I wish to express my appreciation of the courtesies extended to me by my colleagues, Dr. T. B. Osborne, Prof. L. B. Mendel, Dr. A. J. Wakeman and Miss Edna L. Ferry in conducting these experiments. To Mr. Hugo Lange my thanks are also due for his efficient assistance in the care, feeding and weighing of the animals.

SYMPTOMS AND SIGNS OF GASTRIC CANCER: AN ANALYSIS OF 712 CONSECUTIVE OPERATIVELY AND PATHOLOGICALLY PROVED CASES *

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The material forming the basis of this report was obtained from my records of cases at the Mayo Clinic and my service at Augustana Hospital, Chicago. Of the 712 cases studied, all had been operatively and pathologically proved to be gastric cancer. Doubtful cases were excluded.

Sex.—The sex ratio was approximately that of gastric ulcer; namely, 483 males and 229 females, or 2.1 to 1.

Age.—While instances were recorded at as low as 20 years, the age of more than three-fourths of the patients ranged between the fifth and the eighth decades.

Etiologic Factors.—A family or blood-relationship history was proved in 9.4 per cent. of cases. A history of trauma was demonstrated in 3.1 per cent. of instances. In 2.2 per cent. the trauma appeared to precipitate symptoms.

With respect to *definite symptomatology*, it is well to admit here that 1.9 per cent. of cases gave absolutely no symptoms pointing to our clinical conception of gastric cancer. In such instances, exploratory operation or post-mortem examination gave the diagnosis.

Another appropriate admission is that we have no proved clinical procedure, other than history taking, which enables us to make early diagnosis of gastric cancer. Our so-called "early diagnoses" are usually lucky guesses, or are incidents in gastric exploration. The only dependable early diagnosis is made after laparotomy by a capable surgical pathologist by means of sections of tissue stained and examined under high power.

Information obtained by means of the Roentgen ray, by the estimation of the formol index, by the Wolff-

Junghans' test or by the edestin digestion test, sometimes is of aid in *excluding cancer* in its early stage, but is of relatively little value in determining for us the presence of the earliest type of cancer—the cancer which we can honestly say is surgically curable.

It is to be more generally realized as men make tabulations of cases and then study them with liberal minds, that in proved cancer cases we have clinically at least three types of happenings leading up to a diagnosis of cancer. My study indicates that the following is the order of their relative incidence: 1. A symptom-complex of two very *definite* and *different* stages: (a) gastric disturbance, non-malignant and generally chronic; (b) gastric malfunction of malignant type. 2. A symptom-complex generally of a distinctly apparent, pernicious nature. 3. A group of cases in which there is usually evidence of years of gastric disturbance of irregular form, clinically, on which is superimposed a type of affection easily recognized as malignant.

Group 1.—Symptom-Complex Wherein Two Definite and Different Stages of Disease are Present.—This group furnishes 61 per cent. (434 cases) of my series. The average duration of all symptoms in the first stage was 11.7 years. The average duration of all symptoms in the second period is nearly 6 months. The two periods differ markedly. The anamnesis brings out very well the characteristic manifestations of each period. It is only by disengaging the patient from his present state and endeavoring definitely to locate his first departure from gastric health that one gets the key to the situation.

(a) In 61 per cent. of my proved cancer cases the total time of gastric complaint was somewhat over 12 years. For practically 11½ years the symptomatology was that which, clinically, we associate with chronic gastric ulcer. This first period was characterized by a long series of *attacks of "indigestion."* These attacks were associated with *epigastric distress or pain* in 88 per cent. of instances. In 85 per cent. the distress had some relation to the taking of food, the interval being longer or shorter according to the character of food taken. After a solid, mixed meal, the pain relief was longer, but when it recurred it was unmistakably more severe than when lighter forms of nourishment had been indulged in. Other forms of pain relief were alkalies, vomiting and diet. In 54 per cent. of instances the pain was referred to some part of the back. The appetite was usually limited by the fear of taking food and thus causing pain, or by the dread of bringing on bleeding. The food desire in this first stage was generally strong. Weight was lost in the attacks but gained between them. The average *weight loss* at this period was 17.4 pounds. *Constipation* was noted in 52 per cent. of instances. In 8 per cent. there was *diarrhea*. *Vomiting* occurred in 60 per cent. Rather more than 70 per cent. vomited in attacks. The time of vomiting was generally when distress was most marked. *Bleeding* (hematemesis or melena) was noted in 17 per cent. of instances during this stage. Of the whole number bleeding, 49 per cent. had bled at least two years prior to coming under observation. Of those who bled within the two year period over 65 per cent. were of the group with long previous history. In this group *anemia* was rarely marked, the average hemoglobin being toward 80 per cent. (Tallquist or Dare).

(b) If this class of cases pursues the above outlined history, the following may ensue: (1) Complete

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healing with stoppage of all symptoms; (2) typical chronic, *benign* gastric ulcer may result, or (3) gastric cancer may form, whereas previously there had been but a simple process. And no one can prognose in a given case presenting the clinical picture of gastric ulcer just *which kind of ulcer will heal or continue its benign course or which will ultimately form the suitable ground for cancer*. When we recall the pathology in these cases we can hardly blame ourselves for not catching the ulcer clinically in transition to cancer. All ulcers have about and in them hyperplastic tissue—tissue of defense and of healing. No one can tell in a given case when this hyperplasia will change over from the simple type to the malignant form. *Clinically* we are only warned by the appearance in a patient of a more or less continuous gastric disorder, where attacks had before been the rule. This may occur with or without the onset of pyloric obstruction. Where previously there was trouble in spells, with food relief, we note food aggravation, night distress, delayed vomiting, continuous presence of occult blood in the stools, anemia, cachexia, weight loss steadily maintained, weakness, and that peculiar, hopeless apathy exhibited by the fascias. Pain is rarely marked. Vague distress, "heavy feeling" or soreness in the epigastrium are the rule. Severe pain was observed in but 19 per cent. of our cases. It was of "colicky" nature in 14.2 per cent. Gassy and bloated sensations are generally constant and annoying.

In the second stage vomiting becomes more constant and is frequently in greater amount. The odor of the vomitus changes from distinctly sour or yeasty to cheesy or rancid. The color of the vomitus may exhibit little if any alteration. Inasmuch as more than 96 per cent. of my cases of gastric cancer were of the medullary type, oozing of blood was not uncommon. This pathologic state usually imparts a tan to deep brown color to vomitus or stomach extracts. In a little more than 17 per cent. of my 712 cases of gastric cancer was the vomitus of the classic "coffee ground" color. When it presented these characteristics the disease was far advanced in 84.2 per cent. of instances, and inoperable (so far as resection was concerned) in 77 per cent. The vomitus generally shows excess of thick, tenacious mucus, intermixed with sizable food remnants, frequently poorly chymified.

The stools rarely show macroscopic changes unless hemorrhage complicates, or liver, pancreas or adjacent hollow viscera become involved. On a milk diet, chemical tests for blood were positive in 83.4 per cent. of cases tested.

Group 2.—Symptom-Complex in Cases Clinically Malignant from the Inception of Gastric Malfunction.

Of the 712 cases of gastric cancer studied, 31.6 per cent. (225 cases) fell in this group. Careful history taking failed to elicit any clinical type of gastric complaint in these patients previous to an average time of 7.4 months before they came under observation. It should be emphasized that because no gastric disturbance had been previously noted, the conclusion should not be forced that such patients had had no gastric pathology previously. On the contrary, our post-mortem or laparotomy findings of healed ulcers, and even of gastric new growths in individuals who had had no clinical pointings referable to gastric disease, should lead us to exercise caution in insisting when or how malignant processes in the stomach arise. The longest history of gastric symptoms in this group

extended over nearly three and one-fourth years; the shortest barely three weeks.

In this group, which I have, from the pathologic view of the disease, styled "primary" gastric cancer, often it is extremely difficult to state from clinical presentings just when cancer began. Many so-called "primary" cancers have histories extending over weeks of time, where the symptom-complex closely approximates that which our present-day vogue associates with gastric ulcer of the continuously active and progressive form. The clinical picture is different, however, in one important respect. To the symptom-complex is added the manifestations of a malignant systemic poisoning. Instead of fairly definite "attacks" of ulcer type of dyspepsia, and that alone, the process is continuous and associated with progressive weakness, anemia, weight loss with cachexia, vomiting, changing rapidly to the rancid kind. Epigastric pain may be severe and prostrating at first, but later moderates in severity, is not relieved, but is aggravated, by food. It ultimately becomes continuous. I have frequently had strikingly impressed on me the "ulcer-features" clinically in primary gastric cancer. The hurried progression to definite malignancy has seemed to emphasize the necessity for separate recognition clinically of the term "chronicity" in its pathologic sense and in its time-factor sense, meaning weeks, months or years of duration of disease. In many of these primary gastric cancers the clinical gamut of "gastritis," "ulcer" and "cancer" may be run in a few months or even weeks. The whole course of the ailment is compressed into a sort of "tabloid," with regard to time, from which parts are readily segregated that closely approximate clinically those stretched over years exhibited by the great group of cancers forming my Group 1. With this state of affairs it is not to be wondered at that patients present themselves to the specialist or the surgeon only when tumor appears in the epigastrium or when hopeless cachexia warns of impending death.

While the above type of onset of primary gastric cancer is not infrequent, fully two-thirds of this group have more or less vague recollections of how they were affected gastrically before loss of appetite, weakness, mental inertia, abdominal distress, weight loss or epigastric "hardness," "knob" or "lump" warned them that something graver than "chronic indigestion" impended. Rarely is the transition from perfect gastric health to the present state a rapid one. Aversion to certain forms of food (as meat, milk, sweets or legumes) is an early manifestation. While the appetite for some viands may be keen, the aversion to others may be marked and, for the individual, radical. Eructations, often of extremely disagreeable taste, generally become pronounced early. Gassy distention is troublesome. Unexplained nausea, with or without general weakness, manifests itself. In 17 per cent. of my cases of this group diarrhetic stools appeared without apparent cause. Vomiting may be the first symptom and its copiousness pronounced and alarming. It is generally bitter, sour, cheesy, or may be "rotten" in taste. Vomiting was observed in 79 per cent. of this class. In 64 per cent. of those vomiting it was a daily occurrence. In more than 80 per cent. "delayed" vomiting was exhibited.

Consideration of the types of cases making up my grouping of "primary" gastric cancer would be incomplete without mention of that class which I have styled "fulminant" cancer. In my series, there were thirteen instances (5.7 per cent.) in which the whole

history of the affection up to the time of laparotomy covered less than seven weeks. The average age of these patients was 49 years.

In this class, profound cancer intoxication was marked from the time of the first departure from gastric health. In nine instances, fever rose as high as 102 F. The leukocytes averaged 14,800. Anemia was of lower grade than is usual in gastric cancer. The hemoglobin averaged 62 per cent. Weakness, weight loss, mental dulness, foul eructations and copious vomiting, gassy distention of the abdomen, epigastric soreness and the appearance of a rapidly enlarging supra-navel mass occurred with astonishing speed. At operation the malignant process was always inoperable and perigastric lymphatic gland invasion extensive. The pathologic examinations of tissue removed showed cancer of the medullary type. The very rapid progression in this class of cases is not explained on the basis of the character of the tumor. I have shown that more than 96 per cent. of the cancers in my series were encephaloid, and yet rapid extension, such as I have outlined in this "fulminant" class, is not generally seen. The explanation for this in the varying resistance of the tissues of the individual patient, is not wholly satisfactory.

Group 3.—Gastric Malignancy Following Long History of Clinically Atypical Dyspepsia or Dyspepsia of Extragastric Origin.—This division comprises thirty-nine cases (5.5 per cent.). Two markedly different types of gastric malfunction are brought out by careful history taking. The clinical manifestations preceding those evidently malignant, extended over an average period of 9.4 years. The time covered by the supervening malignant stage averaged 5.9 months.

The clinical symptomatology of the affections when they were apparently of benign nature usually pointed to disease of the gall-bladder, appendix, large bowel or the pancreas. Seven patients of this class had had previous operations performed for anomaly of the appendix or gall-bladder or both. In four instances, gall-stones were found when laparotomy was made for the gastric cancer.

The clinical symptomatology when the malignant period supervened was in every way similar to that of the second period of cases making up Group 1 or of some portion of that exhibited by those in Group 2. The point to be emphasized is that after years of gastric departure from health, of atypic form, but evidently benign, so far as our clinical knowledge extends, the picture changed apparently abruptly to that of definite malignancy. It is only in those patients who had recently submitted to laparotomy for extragastric disease, however, that we can positively state that gastric ulcer or gastric cancer with atypic clinical manifestations had not existed for some time previously.

CONCLUSIONS

There is at present no known medical cure for gastric cancer. We have, as yet, no dependable means at our command for the recognition of the disease at a stage when we can positively state that the affection is surgically curable.

The cases of gastric cancer early diagnosed are those in which the clinical symptomatology is that which we associate with chronic gastric ulcer, and in which laparotomy has been urged on the suspicion that cancer might be present.

Where microscopists disagree regarding the diagnosis of sections of extirpated tissue, clinicians can

scarcely be expected to make early diagnosis of gastric cancer before laparotomy.

The question for clinicians and pathologists to settle is not how many cancers had previous ulcer pointings, nor how frequently gastric ulcer changes from simple to malignant form, but just how one is to tell, clinically, in a given case of chronic gastric ulcer, what future course it is destined to pursue. This is the crux of the situation. At present we have no means of prognosing the future course of any gastric ulcer, acute or chronic. The life history of the affection seems to depend on certain unknown factors that are apparently highly individual.

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TORTICOLLIS *

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The following paper is a critical analysis of one hundred cases of distortion of the neck which were entered in the records of the Boston Children's Hospital as torticollis. This analysis has been made in comparison with the findings as reported in the literature of the subject. The cases have been taken from the records of the Children's Hospital in the order in which they applied for treatment.

Any deforming relation between the head and the neck in which the sternocleidomastoid muscle was chiefly involved has been considered as torticollis, whether of congenital or acquired origin.

The sternomastoid muscle has its origin from the sternal notch and the sternal end of the clavicle and is inserted into the mastoid process of the temporal bone. The sternal and the middle portion of the muscle receives its blood supply from the sternomastoid branch of the superior thyroid artery. Unilateral contraction of this muscle pulls the head toward the shoulder, but elevates the chin and turns it away from the side of the contracting muscle. The other muscles about the neck which are most frequently involved in conjunction with the sternomastoid muscle are the scalenus anticus, the trapezius and the splenius capitis. The trapezius causes a more backward inclination and rotation of the head. The splenius capitis bends the head backward and toward the contracting muscle, but does not turn the chin in the opposite direction. The nerve supply to the sternocleidomastoid muscle is from the spinal accessory. The external portion of this nerve leaves the skull by the jugular foramen and descends obliquely outward to the sternocleidomastoid muscle and disappears under the muscle at a distance of about 2 inches from the apex of the mastoid process.

The pathologic work done on torticollis has not been extensive. The most interesting and most valuable has given rise to certain theories, advanced to explain the causation of congenital torticollis.

Lüning and Schulthess¹ have published the details of an autopsy on a child 5 months old, delivered by forceps, who showed at the time of birth a swelling of the middle part of the sternomastoid muscle and this muscle was two-thirds of an inch shorter than the corresponding muscle. Häusinger² reports a case in

* From the Orthopedic Clinic of the Boston Children's Hospital.

1. Lüning and Schulthess: Ztschr. f. orthop. Chir., 1891, i, Part. 1.

2. Häusinger: Joachimsal's Handbuch, p. 432.