The Boston Medical and Surgical Journal

TABLE OF CONTENTS

December 15, 1921

NEW ENGLAND SURGICAL SOCIETY

THE PATHOLOGICAL AND PHYSIOLOGICAL BASIS FOR THE SURGICAL TREATMENT OF CHRONIC GASTRIC AND DUODENAL ULCER. By David Cheever, M.D., Boston.	707
GASTRO-JEJUNAL ULCERS. By Edward R. Lampson, M.D., Hartford, Conn.	712
DISCUSSION OF PAPERS OF DR. CHEEVER AND DR. LAMPSON.	715

ORIGINAL ARTICLE

HEMORRHAGIC OSTEOMYELITIS. By Ernest H. Arnold, M.D., New Haven, Conn.	717
--	-----

THE MASSACHUSETTS MEDICAL SOCIETY

ADDRESSES DELIVERED BEFORE THE SUFFOLK DISTRICT MEDICAL SOCIETY MEETING OF OCTOBER 26, 1921.	721
THE SIXTH JOINT MEETING OF THE DISTRICT SOCIETIES.	726

BOOK REVIEWS

Practical Tuberculosis. By Herbert F. Gammons, M.D., St. Louis, Mo.	727
Practical Chemical Analysis of the Blood. By Victor C. Myers, Ph.D., St. Louis.	727
The Practical Medical Series (Charles L. Mix, General Editor.) Vol. i. General Medicine. Edited by Frank Billings and Burrell O. Raulston.	727

EDITORIALS

THE PRACTICE OF MEDICINE BY NURSES.	728
THE COMMITTEE ON PUBLIC HEALTH OF THE MASSACHUSETTS MEDICAL SOCIETY.	729
JOURNAL AFFAIRS.	729
SMALLPOX.	730
CONTINUATION STUDY FOR PRACTITIONERS.	730
MEDICAL NOTES.	730
NEWS ITEMS.	731

OBITUARIES

WALTER CHANNING, M.D.	731
JOHN JOSEPH FLYNN, M.D.	732
CHARLES WALTER SWAN, M.D.	733

MISCELLANY

FOURTEENTH ANNUAL CHRISTMAS SEAL SALE.	733
STAFF CLINICAL MEETING OF THE MASSACHUSETTS GENERAL HOSPITAL, NOVEMBER 7, 1921.	733

CORRESPONDENCE

MATERNITY LEGISLATION. Benjamin Loring Young.	735
FEE TABLES. Walter L. Burrage.	737
A DIFFERENT SORT OF MEDICAL MEETING. Henry A. Christian.	738

The New England Surgical Society

THE PATHOLOGICAL AND PHYSIOLOGICAL BASIS FOR THE SURGICAL TREATMENT OF CHRONIC GASTRIC AND DUODENAL ULCER.

BY DAVID CHEEVER, M.D., BOSTON.

[From the Surgical Service of the Peter Bent Brigham Hospital, Boston.]

ALTHOUGH Chauvinists among internists and surgeons continue to differ widely in their views, it is clear from an examination of recent literature that the two groups as a whole find themselves much more in accord than formerly in the treatment of peptic ulcer. That the subject continues to excite great interest and some controversy is attested, however, by the flood of contributions which fill medical periodicals, and few gatherings like the present escape the presentation of the subject in some form or other. It seems probable that the increasing appreciation among clinicians of the normal anatomy and physiology of the gastro-intestinal tract, and of the changes wrought in it by disease, is responsible for this promising approach to an entente cordiale. Two groups of men cannot reason from identical premises and reach views at total variance with each other, and the fundamental sciences, anatomy and physiology, together with the morbid changes wrought by

disease, constitute the premises upon which the science of medicine is founded.

Examination of the pathology of peptic ulcer involves consideration of the most promising views as to its cause. Among these may be noted:

1. Mechanical, thermal or chemical trauma.
2. Infection of the mucous membrane by infarction emboli of bacteria from some chronic focus, such as the appendix, the gall-bladder, the teeth, tonsils, or sinuses, with continued reinfection.
3. Localized trophic disturbances, similar to Raynaud's disease.¹
4. Systemic disturbances produced by acute or chronic poisons of various types, including those associated with other diseases, and with extensive burns of the body surface.
5. Faulty pose and defective breathing movements impairing the nervous and circulatory tone in the abdominal viscera.²
6. Infection with an alleged specific fungus, *Oidium albicans*.³

As an indispensable associated factor there is usually invoked the digestive or corrosive action of the gastric juice, which is held by many to be the chief agent in developing and preventing the healing of the ulcer after its causation by one of the above named agents.

With regard to mechanical and thermal trauma, it appears that the gastric mucosa is not easily injured. Ivy,⁴ experimenting on dogs, exposed a pouch of the pyloric antrum to the outer world for a period of nearly a

year, without being able to note any ulceration, bleeding, or change in its character. Having established an experimental ulcer, he manipulated and rubbed it twice daily with bread crumbs (doubtless simulating peristalsis) until marked congestion and some bleeding occurred, which caused the ulcer to require three times the normal interval for healing and resulted in considerable scar formation. It is the common experience with surgeons that after the extensive traumatism caused by gastric resections, the wounds of the stomach wall, including the mucosa, heal kindly and promptly. The duodenal mucosa heals with almost equal readiness, both in experimental animals and man. It seems certain then that physical trauma is not an important element in causing ulcer.

The amazing feat of Rosenow in cultivating strains of bacteria which have an elective affinity for the gastric or duodenal mucosa and cause ulcers when injected into the blood stream of experimental animals, leaves little doubt that foci of infection elsewhere in the body may be responsible, in the form of bacterial embolic infarctions, for the tissue injury which results in ulcer. This is supported by the analogy of haematogenous infections of joint synovia and other serous membranes and of the endocardium. Clinical proof, however, is difficult to obtain, although some observers believe that they have been able to note significant lesions of the appendix or gallbladder in a large proportion of cases.

An interesting observation by Reeves⁵ calls attention to a peculiar arrangement of the arterial supply along the lesser curvature of the stomach and duodenum, predisposing to vascular stasis and to failure of compensatory circulation in case of injury, which may explain the great frequency of ulcer in this situation. To the other causes mentioned above, Smithies⁶ adds a number of suggestions based on the analysis of 522 proved cases of ulcer, to the effect that in 14.7%, the cause was arteriosclerosis, with or without vascular hypertension, in 13% visceral hypertonia, that is, vagus or splanchnic hyperfunction, in 5.2% visceral hypotonia, and in 3.4% metabolic dysfunction involving the thyroid, suprarenal or pituitary. Smithies is able to analyze the entire series and classify the cases into ten etiologic groups, and his paper is a plea for treatment directed toward these alleged causes of ulcer rather than toward the ulcer itself. At present these suggestions are almost purely speculative.

It may be granted then, that some form of injury in the broadest sense to the visceral wall is the first step in the establishment of ulcer. Doubtless small, transient, rapidly healing ulcers are very common, causing symptoms classified vaguely as "indigestion" or "gastritis," accompanied usually by pylorospasm and hypersecretion. They are usually

not diagnosed unless, as occasionally happens, they are characterized by a smart hemorrhage or even by perforation. They tend to spontaneous and complete recovery and probably have no especial tendency to recurrence.

But a small minority fail to heal, grow larger and become chronic. For years, in fact, according to Smithies, since the time of Celsus, the acid gastric juice has been held responsible. The "corrosion" or "autodigestion" of the injured tissue has been considered to be the essential cause of the failure of gastric ulcer to heal. The occurrence of ulcer only in the stomach, the portion of the duodenum proximal to the opening of the bile and pancreatic ducts and in the jejunum at or just beyond the stoma of a gastrojejunostomy, regions where alone the acid pepsin is active, is strong evidence of the part played by this agent. This is supported by the clinical association of hyperchlorhydria with ulcer, and by the relief and cure of ulcer following neutralization by alkalis. But contrary views are not lacking; and there is abundant evidence that ulcer and hyperacidity are not necessarily associated. Wilensky⁷ studied 63 cases before and after operation, and found that a very small group showed a hypo-acidity, another group showed no disturbance of acid secretion, while still another was normal except for a prolonged digestive period. He reaches the conclusion that ulcer is not necessarily accompanied by disturbances of acid secretion, but his statistics show that the majority of his cases have co-existent excess of acid. Smithies, pleading, as noted above, for the treatment of ulcer by indirect methods, says that the well-nigh universal belief that ulcer is due to the corrosive action of the acid juice is not warranted by experimental laboratory, or clinical facts. An unhealed ulcer has been noted in an acid-pepsin free stomach and without ulcer symptoms. Provided that the mucosa is intact and bile is not present, hydrochloric acid of ten times a normal concentration may be introduced into the stomach of the experimental animal without producing ulcer, and in cases of unhealed ulcer, acid of five times normal strength will not cause symptoms in the quiescent stage. An ulcer has been known to progress to a fatal termination in the total absence of acid. He refers justly to the fact that an acid medium is the normal habitat of the gastric mucosa, in which it may be supposed to thrive better than in a neutral or alkaline one, and states that prolonged alkalization will lead to a compensatory over-acidity of the acid cells, and may ultimately result in the degeneration of the mucosa. He calls attention also to the extraordinarily active healing of the stomach in spite of the acid medium, after surgical trauma.

These observations are adequately explained by the pathology of chronic ulcer. If a piece

of gastric mucosa be excised, and placed in a normal acid-pepsin medium at body temperature, it will be digested like any other bit of animal tissue. Evidently when no longer living it possesses no inherent attributes which prevent digestion. If, after surgical trauma, the tissues necrose, they undergo autodigestion and are thrown off. Ivy⁴ and others have noted the production of postoperative ulcers from pressure of the gastro-enterostomy forceps. If, then, autodigestion is inevitable after tissue death, it is certain that there must be a degree of devitalization short of actual death where the tissue cells fail to resist this process. In the last analysis, it must be through the blood and lymph stream that the maintenance of vitality is afforded, and it seems as though Nature had made especial provision for this in the case of the stomach by the magnitude of its arterial blood supply and the richness of the vascular anastomosis, especially in the submucous layer, from which the mucosa is supplied. Every surgeon has observed that the incised stomach bleeds furiously, and has wondered that so many wounds, overlappings, reconstructions, and sutures, may be made simultaneously without failing to secure healing.

The probable sequence of events in the formation of ulcer may be outlined thus: as a result of an infarction embolism with thrombosis, an area of mucosa is devitalized beyond the critical degree and surface "corrosion" takes place. In the vast majority of cases this heals quickly and spontaneously, having caused no symptoms at all, or only transient symptoms, or very rarely an acute hemorrhage. In the exceptional case, as a result of the extent of the original devitalization, or of failure of the ulcerated area to obtain rest, or of mechanical irritation by gastric contents, or of constant reinfection from the original focus, or of some obscure disturbance of neuro-trophic or endocrine control, or of altered gastric physiology (as will be noted later), the ulcer does not heal, penetrates deeper into the visceral wall and becomes chronic. Instead of the rapid proliferation of mucosal epithelium over a relatively intact submucosa and the regeneration of the secretory glands, we note the filling of the defect with granulation tissue over which the mucosa may spread, perhaps with little impairment of its vascular nutrition. On the other hand, this may not succeed, the granulation tissue becomes converted into fibrous scar tissue, both at the base and sides of the ulcer; this tissue becomes more dense and compact and becomes progressively devascularized by the contraction and thrombosis of its vessels. Thus the vascularity upon which the mucosa depends so strongly for the maintenance of its vitality is destroyed, and healing is imperfect, or cannot be made permanent, or fails altogether.

Thus is produced a pathological condition essentially similar to chronic ulcer elsewhere. It is not specious to compare it to chronic indurated varicose ulcer of the lower leg. Here the ulcer fails to heal first on account of infection, irritation associated with lack of rest, and circulatory impairment due to the varicose veins. Later if these conditions are not mended, the vascular granulation tissue base is replaced by a thick layer of dense fibrous tissue through which no vessels can penetrate adequately to nourish regenerating epithelium, and it is a common observation that rest, disinfection of the ulcer and operation on the varicose veins may alike fail to secure healing. Further, if the fibrous base is widely excised, giving free access of vigorous new vessels to the affected area, spontaneous healing, perhaps aided by grafting, will result.

In the leg ulcer, we have the indurated base, the local infection, and the stagnating venous circulation to reckon with; in the peptic ulcer we have the indurated base, perhaps local infection, and certainly the unfavorable action of the gastric juice on ill-nourished tissue cells.

The disturbed physiology is equally important to consider with the pathology. The nerve supply of the stomach and duodenum is through the vagus and sympathetic, whose afferent fibres carry impulses which are translated into motor or inhibitory reflexes transmitted through their efferent fibres. These afferent stimuli are not of the nature of tactile sensibility or of pain, for it is well known that severe trauma including cutting, pinching, cauterizing and the application of chemical agents can be inflicted on the stomach and intestines without causing any sensation. The plausible theory that the pain of ulcer is due to the irritant action of hydrochloric acid on the nerve endings exposed in the ulcer must be discarded. Most of the afferent nerve endings are found in the submucous and muscular coats, rather than in the mucous membrane, and when these are exposed in ulcerations to the irritant action of the acid gastric juice, an excessive reflex motor activity is engendered (Endelman⁸), which would increase greatly intra-gastric tension were it not relieved at first by the relaxation of the pylorus on the approach of a peristaltic wave, according to the "law of contrary innervation." But when the chyme, acid or hyperacid, passes into the duodenum, an excessive reflex inhibition of relaxation of the pyloric sphincter is caused, so that relief of the intra-gastric pressure is not afforded, and increased tension results, which, according to Hurst⁹, is the only adequate stimulus producing visceral pain. The pylorospasm causes delay in the emptying of the stomach, which in itself causes hypersecretion, which in its turn causes more ulcer irritation, from which results more vigorous peristalsis, and more obstinate inhibition of

sphincter relaxation, establishing thus a vicious circle of disordered physiology.

It is not intended in this paper to champion the cause of surgery in the treatment of peptic ulcer, but rather to examine the basis for treatment as revealed by present-day conceptions of the pathology and pathological physiology of the condition. Medical treatment as carried out by Sippy¹⁰ and others aims to improve the health as a whole and diminish general reflex nervous response by physical and mental rest, and to correct acidity, hypersecretion, and pylorospasm by alkaline neutralization and lavage. To this is added, as a matter of course, the treatment of any of the local or systemic conditions enumerated by Smithies⁹ and others as being possible etiologic factors. This treatment is the logical corrective of the deranged physiology of the viscera involved; it is certain to relieve symptoms and may be expected to cause healing of the ulcer unless the local pathological condition makes this impossible. That the clinical results of medical treatment are excellent does not admit of a doubt. Eggleston¹¹ says that among 156 cases treated medically 72% reported no return of symptoms after the lapse of at least 3 years, and 28% reported recurrences. These cases were "proved" by the usual clinical and laboratory tests. He states that patients are expected to keep up a modified diet for months or even years. Sippy¹⁰ gives an even higher percentage of medical cures. Not only is the cure symptomatic, but in many instances x-ray studies have shown the gradual and complete disappearance of a penetrating crateriform lesion. At postmortem examinations it is a not unusual occurrence to find completely healed scars whose histology makes it reasonably certain that the lesion was of the deeply penetrating chronic type.

Surgery offers a direct attack upon the pathological process and promises its cure by removing it, and at the same time tries to correct the physiological dysfunction by a permanent artificial expedient. The consensus of opinion is crystalizing more and more in favor of the excision of the ulcer where possible. Excision of ulcer at the pylorus, on all but the highest part of the lesser curvature, and almost anywhere on the anterior surface of the stomach is usually practicable except in the face of unusual complications, while trans-gastric resection of posterior penetrating ulcer, though more formidable, can usually be carried out. Resection removes the indurated, avascular scar tissue which forms the base and periphery of an ulcer and secures prompt healing of the sutured wound by insuring it an adequate blood supply. The observation that wedge-shaped excisions at the lesser curvature sometimes interfere seriously with the peristaltic wave and give imperfect clinical results, has led some authorities like Moynihan¹²

and Urrutia¹³ to advise sleeve resection, on the ground that the mortality is but little higher and the results more favorable. The widely held view that excision is more essential in gastric than in duodenal lesions on account of the frequency of malignant metamorphosis of the former has undergone considerable modification lately. Gross and Held¹⁴ do not agree with Wilson and McCarty¹⁵ and with Cole¹⁶ that cancer frequently develops on ulcer, and say that such cases are almost invariably ulcerated but primarily malignant lesions. Wilensky⁷ holds the same view. On the other hand, C. H. Mayo¹ believes that gastric ulcer is essentially malignant, while Deaver¹⁷ considers carcinomatous degeneration a decided menace. Whichever view shall be proved to be correct, at present the emphasis is placed on the desirability of excision. Most surgeons would agree also that at times it is necessary on account of the impossibility of distinguishing between the gross characteristics of ulcer and carcinoma.

In duodenal ulcer the question is modified by three considerations: first, the technical difficulty of excision on anatomical grounds; second, the possibility of effective exclusion of the ulcer by proximal closure of the viscus; and third, the exceeding rarity of carcinoma in this situation. While many favorably placed duodenal ulcers may be excised, it is justifiable not to insist on their removal since the complete rest and freedom from hydrochloric acid digestion afforded by exclusion make it unnecessary.

The surgical correction of pathologically altered gastro-duodenal physiology is obtained by two similar methods, each of which has its advocates,—gastrojejunostomy and some form of pyloroplasty or gastroduodenostomy. These expedients secure a more prompt emptying of the stomach, neutralize to some extent the acid gastric juice by admixture with duodeno-jejunal contents, cause relaxation of pylorospasm and prevent hypersecretion by preventing retention. It is undoubtedly true as shown long ago by Cannon and Blake¹⁸ and confirmed by other observers that in the presence of an open pylorus the gastro-jejuno-stoma may transmit but a part of the contents of the stomach and indeed may close completely, but experienced surgeons like Deaver¹⁷ are doubtless right in maintaining that a properly placed and made opening will endure in spite of it. The neutralization of gastric juice obtained varies within wide limits as to degree and time. Wilensky¹⁹ studied 63 cases, for the most part within three months of operation, and found usually an immediate lowering of total acid concentration after operation, with subsequent return to its former level or even exceeding it, which he attributes to a stimulus to the acid-secreting cells following the attempt at neutralization, in accordance with physio-

logical laws. Lemon²⁰ observed a reduction in free hydrochloric acid of 46% following gastroenterostomy. The weight of evidence is decidedly in favor of the view that a partial neutralization is obtained and persists long enough to favor the healing of any operative wound. The advocates of pyloroplasty, like Horsley,²¹ Davis,²² and Finney and Friedenwald,²³ maintain that this operation promotes the emptying of the stomach in a more physiological manner, avoids the possibility of gastro-jejunal ulcer, and minimizes the pain, vomiting, bleeding, and diarrhoea which are the occasional sequelae of gastro-jejunosomy.

Recent statistics as to the end-results of surgery prove to be divergent, as might be expected. C. H. Mayo¹ reports the operative mortality in 4,532 cases of duodenal ulcer as 1.76%, and in 1,191 cases of gastric ulcer 3.77%. Most of the deaths occurred in neglected or complicated cases of long standing. Among 438 cases of duodenal ulcer operated on and traced, 70% were found to be "well," 27% were "improved," and 3% were "unimproved." This authority justly remarks that all except emergency cases had been treated by many physicians for long periods before having the benefit of surgery, and makes the comment that more patients die from complications than from operations. Eggleston¹¹ mentions 41 patients operated on with a mortality of 7.4%, and an end result of 44% "cured," and 36% with recurrence. Coffey²⁴ reports 233 cases operated on by various methods with a mortality of 4.3%. Deaver²⁵ has operated on 55 cases of acute perforation with extravasation, with the remarkable mortality rate of 5.5%. Only two cases had subsequent symptoms, and in both closure was made without gastro-enterostomy. Menghetti²⁶ analyzes 700 cases, with a mortality after thermocauterization and simple gastro-enterostomy of 1% and after partial gastrectomy of 5%, and remarks that the latter operation is certain to be curative. Eusterman²⁷, in analyzing 82 cases of gastro-jejunal ulcer states that 8% of the cases at the Mayo Clinic had a recurrence of symptoms within one year after operation for ulcer. Smithies²⁸ examining 273 patients after operation (of whom 21% had been operated on within 6 weeks) found 49.8% well or with minor digestive upsets, 38% improved but still dyspeptic, 6.9% not improved, and 5.1% made worse by the operation. Davis²³ quotes Bastedo as saying that "in our own practice ulcer cases that have had one or more operations come for treatment almost as frequently as cases that have not been operated on," and gives it as his own opinion that from 15 to 40% of operations for gastric ulcers are failures. Perhaps the most striking figures are quoted by Balfour²⁹ from investigations of the Actuarial Society of America showing that of 2,323 patients operated on at the Mayo Clinic

between 1906 and 1915, the operative mortality from all causes in 545 cases of gastric ulcer was 4.5% and in 1,684 cases of duodenal ulcer was 2%. 17% of those operated on for gastric ulcer died within an average period of 3.6 years, and 5% of those operated on for duodenal ulcer have died in an average period of 3.4 years. Among the duodenal ulcers, the mortality was actually slightly less than in a similar general population group. The insurance investigator says: "While I cannot prove my statement, I believe that a much larger number of persons would have died but for the operation."

It is clear that there is a wide discrepancy in the statistics of the results of surgical treatment of ulcer from different sources, and it is to be hoped that additional data will be forthcoming until more reliable evidence can be deduced as to the value of various therapeutic measures. As a contribution in this field, the following figures from the clinic of the Peter Bent Brigham Hospital, Boston, are offered as a preliminary survey. It is intended that a more accurate analysis of the results of particular procedures may be presented later.

At this hospital from its foundation in 1913 to December 31, 1920, operations were performed for the relief of gastric and duodenal ulcer on 134 patients. There were six post-operative deaths, giving a mortality of 4.4%. Of the total number, 82 were duodenal ulcers, 51 were gastric, and 1 was not stated. This is a larger proportion of gastric ulcers than is usually found, and raises a question as to the interpretation of the location of the often indeterminate pyloric ulcer. Of the 82 duodenal ulcers, 48 have been traced, and of these 29, or 60%, are "well," 12, or 25%, have "slight symptoms," 7, or 14%, have "definite recurrence," and no case reports a worse condition than before operation.

Of the 51 gastric ulcers, 29 have been traced, and of these 22, or 75.8%, are "well," 5, or 17.2%, have "slight symptoms," 2, or 6.8%, have "definite recurrence," and no patient reports a worse condition than before operation. Looking at the matter more broadly, it is perhaps fair to say that at the expense of a mortality rate of 4.4% in the entire series, the 60.1% who could be traced yield a percentage of 88.3% cured or much improved, and a percentage of 11.7% with definite recurrences, some of whom indeed admit a certain degree of benefit. While the operative mortality figures run to December 31, 1920, but 8½ months previous to this inquiry, the end-results refer to patients from 18 months to 2 years after their discharge from the hospital.

When it is remembered that surgical statistics deal only with proved cases of ulcer and that it is undoubtedly true that the patients who are operated on present more serious con-

ditions and complications on the average than do those who are treated medically, is it not reasonable for surgery to claim an honorable and accepted rôle in the treatment of chronic ulcer?

REFERENCES.

- ¹ Mayo, C. H.: Gastric and Duodenal Ulcers. *Ann. Surg.*, lxxiii, 323, March, 1921.
- ² Bourcart, M.: Pathogenesis of Round Ulcer in the Stomach and Duodenum. *Abstr. in Jour. A. M. A.*, lxxiii, 871, Sept. 13, 1919.
- ³ Askanazy, M.: Pathogenesis of Round Gastric Ulcer. *Abstr. in Jour. A. M. A.*, lxxv, 1165, Oct. 23, 1920.
- ⁴ Ivy, A. C.: Studies on Gastric and Duodenal Ulcer. *Jour. A. M. A.*, lxxv, 1540, Dec. 4, 1920.
- ⁵ Reeves, T. B.: A Study of the Arteries Supplying the Stomach and Duodenum, and their Relation to Ulcer. *Surg., Gyn., and Obstet.*, xxx, 374, April, 1920.
- ⁶ Smithies, F.: Significance of Etiologic Factors in the Treatment of Peptic Ulcer. *Jour. A. M. A.*, lxxiv, 1555, June 5, 1920.
- ⁷ Wilensky, A. O.: Disturbance of Acid Secretion Accompanying Ulcer of the Duodenum and Stomach and Changes Following Operation. *Surg., Gyn., and Obstet.*, xxvi, 506, May, 1918.
- ⁸ Edelmann, quoted by Muller, G. P., and Ravdin, I. S.: Perforated Gastric and Duodenal Ulcer without Previous Pain. *Ann. of Surg.*, lxxiv, 223, August, 1921.
- ⁹ Hurst, A. F.: The Sensibility of the Alimentary Canal. London, 1911.
- ¹⁰ Sippy, B. W.: Ulcer of the Stomach and Duodenum. In *Oxford Loose-Leaf Medicine*, Vol. iii, p. 123, New York, Oxford Press.
- ¹¹ Eggleston, E. L.: Critical Review of 500 Cases of Gastric and Duodenal Ulcer. *Jour. A. M. A.*, lxxv, 1542, Dec. 4, 1920.
- ¹² Moynihan, B.: Surgical Treatment of Gastric Ulcer. *Jour. A. M. A.*, lxxv, 330, July 31, 1920.
- ¹³ Urrutia, L.: Gastric Ulcer. *Arch. Espan. Enf. Ap. Digestivo*, 1919, 2, 676. *Abstr. Jour. A. M. A.*, lxxv, 923, March 27, 1920.
- ¹⁴ Gross, M. H., and Held, I. W.: Indications for Surgical Intervention in Ulcus Ventriculi. *Surg., Gyn., and Obstet.*, xxvii, 567, December, 1918.
- ¹⁵ Wilson, L. B., and McCarty, W. C.: The Pathological Relationship of Gastric Ulcer and Gastric Carcinoma. *Am. Jour. Med. Sci.*, cxxxviii, 846, December, 1909.
- ¹⁶ Cole, L. G.: Roentgen Diagnosis of Indurated Gastric Ulcer. *Am. Jour. Roent.*, ii, 793, November, 1915.
- ¹⁷ Deaver, J. B.: Operative Removal of Peptic Ulcer, Removal of Ulcer with Gastro-enterostomy or Gastro-enterostomy alone? *Ann. of Surg.*, lxx, 526, November, 1919.
- ¹⁸ Cannon, W. B., and Blake, J. B.: Gastro-enterostomy and Pyloroplasty. *Ann. of Surg.*, xli, 686, May, 1905.
- ¹⁹ Wilenski, A. O.: Disturbances of Acid Secretion Accompanying Ulcer of the Duodenum and Stomach, and Changes Following Operation. *Surg., Gyn., and Obstet.*, xxvi, 506, May, 1918.
- ²⁰ Lemon: Quoted by Mayo, *loc. cit.*
- ²¹ Horslev, J. S.: Some Underlying Principles of Intestinal and Gastric Surgery. *Ann. of Surg.*, lxxiii, 109, February, 1921.
- ²² Davis, B. B.: Comparative Results of Pyloroplasty and Gastro-enterostomy in Stomach Surgery. *Ann. of Surg.*, lxxiii, 450, April, 1921.
- ²³ Finney, J. M. T., and Fridenwald, J.: Thirteen Years' Experience with Pyloroplasty. *Surg., Gyn., and Obstet.*, xviii, 273, March, 1914.
- ²⁴ Coffey, R. C.: Gastro-enterostomy Still the Treatment for Chronic Gastric and Duodenal Ulcer. *Ann. of Surg.*, lxxi, 303, March, 1920.
- ²⁵ Deaver, J. B., and Pfeiffer, D. B.: Gastro-enterostomy in Acute Perforated Ulcer of Stomach and Duodenum. *Ann. of Surg.*, lxxiii, 441, April, 1921.
- ²⁶ Menghetti, S.: Outcome in 700 Operative Cases of Gastric and Duodenal Ulcer. *Rif. med.*, xxxvii, 131, Feb. 5, 1921. *Abstr. Jour. A. M. A.*, lxxvi, 1050, April 9, 1921.
- ²⁷ Eusterman, G. B.: Gastro-jejunal Ulcer. *Minnesota Med.*, iii, 517, November, 1920. *Abstr. Jour. A. M. A.*, lxxv, 1595, Dec. 4, 1920.
- ²⁸ Smithies, F.: Analysis of Stomach Following Gastro-enterostomy. *Surg., Gyn., and Obstet.*, xxvi, 275, March, 1918.
- ²⁹ Balfour, D. C.: Life Expectation of Patients Following Operations for Gastric and Duodenal Ulcer. *Ann. of Surg.*, lxx, 522,

termine, if possible, the cause or causes, and then find the means by which such untoward results may be avoided.

If we are going to retain gastroenterostomy as a curative measure in peptic ulcers, we must minimize our disastrous consequences, such as jejunal ulcer. Brawm, in 1899, was the first surgeon to find a jejunal ulcer which had followed a gastroenterostomy, and not until 1902 was one reported from France by Quénu. About one year later one was discovered in England. In 1910, W. J. Mayo had only seen three cases.

Since then, however, numerous surgeons have reported cases, and in 1919, Garnet Wright of Manchester, England, wrote a most excellent article and studied minutely all of the cases up to that time, together with three of his own which numbered one hundred and fifty-five. Eusterman, in 1920, gave a clinical study of 83 cases operated upon at the Mayo Clinic, and Judd has recently cited 101 cases. The increased number is probably due to two causes. One, the greater number of gastroenterostomies being performed, and the other the fact that the diagnosis of gastro-jejunal ulcer is more frequently and more accurately made, because we are now aware that some of the unfortunate late results of the operation are caused by the gastro-jejunal ulcer rather than a recrudescence of the primary lesion for which the operation was performed.

The frequency of their occurrence is very difficult accurately to determine, due to causes that cannot be controlled, especially the lack of careful follow-up system of operative cases. An approximation, however, may be made. During the past five years, from January 1, 1915, to January 1, 1920, there have been performed at the Hartford Hospital, 147 gastroenterostomies. At the Hartford Hospital there have been three jejunal ulcers, an occurrence of two per cent. This is probably low, due to the fact that some have been overlooked. In 1028 gastroenterostomies, as reported from various clinics, there occurred seventeen gastro-jejunal ulcers, an incidence of about 1.7%. The percentage now is on the increase, due to the better diagnosis of the poor post-operative results and probably more closely approximates 3% than 2%.

By most authorities the chief and primary cause given is the use of the non-absorbable suture which ulcerates through even though it had only been used in approximating the serous surfaces. This suture is supposed to act as an irritant mechanically, and also more actively as a source of infection. In many instances such a suture has been found at the time of operation, either hanging free or else clearly exposed in the ulcerated area, but had such a suture been used, as was formerly always the case, would it not have been exposed had the ulcer been due to some other cause?

GASTRO-JEJUNAL ULCERS.

BY EDWARD R. LAMPSON, M.D., HARTFORD, CONN.
Surgeon to the Hartford Hospital.

It is exceedingly unfortunate that the usually happy outcome of the operation of gastroenterostomy for duodenal or gastric ulcer, accompanied by excision of the ulcer, where possible, should ever be dampened by the occasional occurrence of a jejunal or gastro-jejunal ulcer. It therefore should be our aim to de-