1963-1964 COLLECTED PAPERS IN MEDICINE FROM THE MAYO CLINIC AND THE MAYO FOUNDATION VOLUME 55

W. B. Saunders Company · Philadelphia and London

Made in the United States of America

Press of W. B Saunders Company

LIBRARY OF CONGRESS CATALOG CARD NUMBER: 62–15093

EDITORIAL STAFF

Carl M. Gambill, A.B., M.D., M.P.H.
James R. Eckman, A.B., M.A., PH.D.
M. Katharine Smith, B.A.
Florence L. Schmidt, B.S.E.
Guy Whitehead, B.A., M.A., PH.D.
Werner Heidel, A.B., M.D.
Robert D. Knapp, Jr., A.B., M.D.
Bernard K. Forscher, A.B., PH.D.
Marc A. Shampo, B.S., M.S., PH.D.

Compiled by
Werner Heidel, A.B., M.D.

FOREWORD

For the fifty-fifth volume of the series, *Collected Papers* is being presented again, as it has been for the last two years, in two separate parts, medical and surgical. This change in format provided an increased number of pages as well as a convenient division of the subject matter. The expansion has made it possible to offer a broader representation of the work of this clinic, including that in the various specialties and at least a sampling from the basic sciences. However, the material continues to be selected largely with the general practitioner, the general surgeon, and the diagnostician in mind.

The material represented herein consists of papers submitted to the various journals in the period December 1, 1962, through November 30, 1963. Articles that have been published in the *Mayo Clinic Proceedings* and in the Mayo Clinic number of *Surgical Clinics of North America* are referred to only by title. The form of a number of the articles corresponds to the copy submitted to the respective journals; therefore, some differences in style may be noted between the article as it appears in this volume and as it appears in the journal.

Of the total of 708 articles represented in the fifty-fifth volume of *Collected Papers,* 85 appear in full, 72 in abridgment, and 73 in abstract form; the remaining 478, including 82 from the *Proceedings,* are listed by title only.

The names of the editors appear on the title page. Essential work was done also by the following: Margaret S. Thompson, executive assistant, and her assistants, Ruth Fiegel, Betty Calkins, Othalene Yates, and Margaret Riley; Eleonore Clappier, managing editor of the *Proceedings,* and her assistant, Rosemary Perry; and Leola McNish, Donabeth Postier, Glenell Colwell, Anita Bartel, and Catherine Siemback, who handled the proof.

SECTION OF PUBLICATIONS
MAYO CLINIC
AND
MAYO FOUNDATION

Rochester, Minnesota

CONTENTS

ALIMENTARY TRACT

GENITOURINARY TRACT

DUCTLESS GLANDS

BLOOD AND CIRCULATORY ORGANS

DERMATOLOGY

HEAD, TRUNK AND EXTREMITIES

THORAX

BRAIN, SPINAL CORD AND NERVES

RADIOLOGY

MISCELLANEOUS

ALIMENTARY TRACT

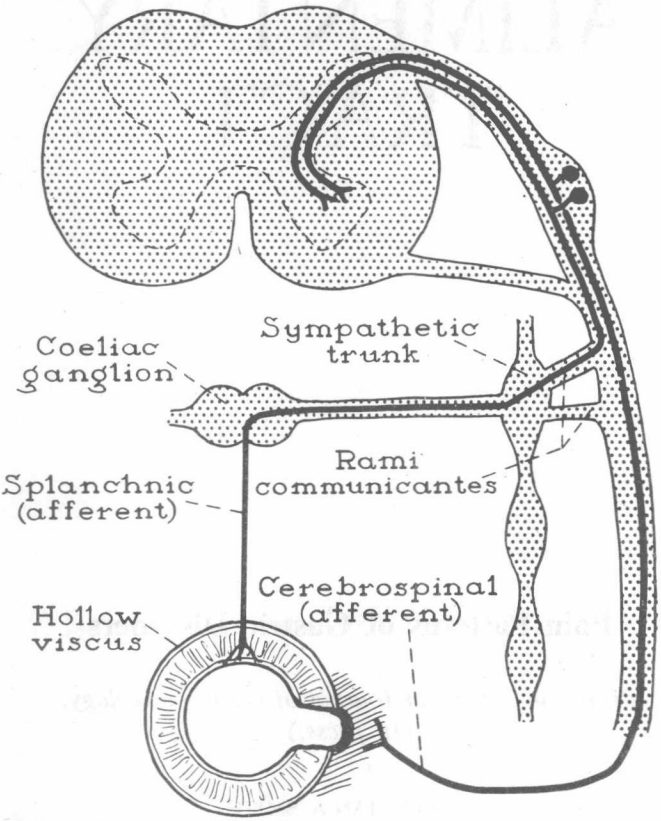
Pain Patterns of Gastric Disorders

From the American Journal of Gastroenterology.
(In press.)

By LUCIAN A. SMITH

In an era in which jet planes, nuclear bombs, and electronic computers are commonplace, the clinical analysis of pain patterns may seem old fashioned. The examiner may long for a more dramatic approach to the problem, but he should never fail to take advantage of the information available to him in the analysis of the pain described by the patient.

Pain or distress caused by a gastric disorder brings the patient to the physician more commonly than bleeding does, even though bleeding is more impressive. The patient seeks relief of pain, but without a correct interpretation of the pain pattern several errors can be made. A lesion that is difficult to demonstrate may be missed because the examiner does not have sufficient faith in the pain pattern to persist in his search. Early documentation of a lesion which is asymptomatic may divert attention from the continued search for another lesion which is actually causing the presenting symptoms. In some instances the pain is in a location so remote from the stomach that even the possibility that a gastric lesion is the source of the pain does not occur to the examiner. Finally, when there is inadequate understanding of pain patterns, misplaced therapy, at times heroic and doomed to failure, may be used.



Intrinsic visceral pain passes from receptors by way of splanchnic nerves to cord. When an ulcerating lesion perforates sufficiently to stimulate cerebrospinal receptors in adjacent peritoneum, somatic pain reaches cord by way of cerebrospinal pathway. Cell bodies of both pathways lie side by side in dorsal root ganglia.

In some ways it is unfortunate that the patient's description of the pain of uncomplicated peptic ulcer has been learned so thoroughly by students and physicians. I refer to the tendency to call any variation from the average account an "atypical" feature of the pain, when actually the variation may point accurately to the development of a complication of the ulcer or the presence of another lesion.

The pain patterns of gastric disorders have two pathways by which they reach conscious level in the patient (figure). The splanchnic nerve pathway transmits the pain of intrinsic lesions, and the cerebrospinal nerve pathway is usually involved when the causative lesion extends sufficiently to involve the serosa and adjacent tissues. Pain transmitted by the splanchnic pathway is initiated by an adequate stimulus, either spasm or distention; and because the stomach has bilateral innervation, this pain of intrinsic origin is perceived by the patient as if it were in the midline of the trunk. When ulceration is present, the physiologic peaks of secretion of free acid may trigger the spasm, and neutralization or combination of the free acid

will relieve the pain. Although the level of acidity is an important part of the induction of ulcer pain, such visceral pain is one of the earliest symptoms of ulcer and may occur as soon as the lesion has breached the mucosa. The fact that this occurs long before free acid is in contact with adjacent cerebrospinal nerve endings is presumptive evidence that spasm must be the intermediary mechanism.

When ulceration progresses through the gastric wall sufficiently to reach the serosal surface, the advance is preceded by inflammatory change in the tissues beyond the base of the ulcer. It is desirable to remember at all times that the stomach is a large organ and that its rather snug anatomic relationship to surrounding tissues and organs makes it possible for localized perforation to involve a variety of additional tissues, some with nerve endings (either splanchnic or cerebrospinal) and some with no nerve endings. When the additional nerves are stimulated, the resultant pain may not resemble ulcer pain in quality, location, or time sequence. If standard ulcer pain continues in conjunction with the new pain, now of extrinsic origin, the analysis of the new pain will lead to correct diagnosis of a complication, and perhaps even of the location of the ulcer.

Before the pain patterns of specific gastric lesions are considered, mention should be made of the anatomic relationships which are so important to the interpretation of pain. The crura of the esophageal hiatus and the adjacent dome of the left hemidiaphragm are innervated by phrenic fibers. The lesser omentum has a liberal supply of cerebrospinal nerve endings, while the greater omentum has none. There is no sensory nerve supply to the spleen, but ease of splenic infiltration may permit subsequent stimulation of the parietal peritoneum, which at that level is supplied by the eleventh and twelfth thoracic nerves. Anteriorly the pancreas is covered by parietal peritoneum, but the lower edge of this extends to form part of the transverse mesocolon, which is intimately related to the greater curvature of the stomach. Most of the pancreas lies behind the stomach, and gastric lesions of any part of the posterior wall and to a lesser extent of lesser and greater curvatures can penetrate to the pancreas, which has bilateral splanchnic innervation.

Whether it is large or small, the esophageal hiatal hernia may lead to symptoms that are the result of regurgitant esophagitis, incarceration of the fundus, or ulceration of the cardia or of the herniated fundus. When dysphagia is present, it is localized well by the patient. Positional distress may occur when the patient is in bed, before sleep occurs, and relief may be prompt when he sits up, stands, or belches. Such distress is interpreted as heartburn, and ulcer may be suspected. At times an associated diffuse spasm of the esophagus may simulate a cardiac catastrophe. Pain secondary to ulceration of the fundus or the cardia characteristically is high, and it may shift into the left side of the chest and is often referred to the interscapular zone and less frequently to the left shoulder cap and arm. At times a hiatal hernia may be found by accident; it is then important to be certain that the symptoms are due to the hiatal hernia and not to asssociated gallbladder disease or to epigastric angina of coronary disease.

The visceral pain of a shallow gastric ulcer unaccompanied by active penetration or perforation may be indistinguishable from that of an un-

complicated duodenal ulcer. Although it is customary to lay stress on the time sequence of the acid-cycle pain in gastric ulcer as contrasted with that in duodenal ulcer, this feature usually has little value in diagnosis. Gastrospasm is prominent in some patients, and reflex pylorospasm with vomiting of food just eaten may occur in a patient who has an ulcer in the distal half of the stomach. Pain at night is infrequent unless pyloric obstruction is present, because hyperacidity and nocturnal hypersecretion are not common. If nocturnal pain and retention vomiting occur, one should remember that simultaneous occurrence of gastric and duodenal ulcers may be confusing the history. Even though the visceral pain has bilateral transmission and usually is felt in the midline of the epigastrium in a symmetrical location, an early shift of the pain toward the left and upward will often occur when there is progressive deepening of the ulcer, prior to perforation. Approximately three fourths of benign gastric ulcers occur along the posterior edge of the lesser curvature. The remainder will be found in the prepyloric segment or on the posterior wall or the greater curvature. When the ulcer is in its common location on the lesser curvature, perforation to the lesser omentum, the lesser omental sac, the undersurface of the liver, the pancreas, or the crura often will give left thoracic and interscapular pain. The addition of left shoulder cap pain with or without arm pain should suggest that the ulcer is high in the stomach. Perforation posteriorly to the head or body of the pancreas at times duplicates the intractable pain of an expanding intrinsic lesion of the pancreas, being perceived at the twelfth thoracic to the second lumbar level in the back. This is the pain that leads the patient to assume certain postures, often bizarre, in the effort to obtain relief. It is not commonly realized that the corresponding anterior pain is as low as the umbilicus, or the tenth thoracic dermatome. When such severe pain of pancreatic origin occurs as another phase of the gastric ulcer history, the examiner may be reasonably safe in assuming extrinsic rather than intrinsic origin, but the possibility of associated carcinoma of the pancreas must not be forgotten. The ulcerating lesion on the greater curvature of the stomach may produce a varied pain pattern which can be illustrative of both upward and downward referral. If such a lesion is high it may involve the dome of the left hemidiaphragm and give rise to left shoulder cap pain with or without left arm pain. If the extension causes perisplenitis the irritation of the parietal peritoneum of the eleventh and twelfth thoracic segments may give pain in the left portion of the trunk even as low as the left lower quadrant. Lower abdominal pain also is seen when an ulcer of the greater curvature involves the left transverse mesocolon. This development may be suggested if frank pancreatic pain occurs concurrently with the left lower quadrant pain. Perforation of a gastric ulcer on the posterior wall into the lesser omental sac may occur with sufficient rapidity to permit the extension of irritating metabolites upward to the dome of the diaphragm, and this will give rise to phrenic referral as well as to pancreatic pain.

Carcinoma of the stomach may not cause any pain, or it may produce sufficient symptoms to direct attention to the stomach. The pattern created will depend on the size, the location, the ulcerative or invasive character of the lesion, and the absence or presence of acid in the gastric contents.