THE THIRTY-FOURTH HAHNEMANN SYMPOSIUM

Gastrointestinal Emergencies

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Editors

The Thirty-Fourth Hahnemann Symposium

Gastrointestinal Emergencies

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Preface

The diagnosis and treatment of gastrointestinal emergencies often require diverse and sometimes technologically advanced procedures. However, appropriate selection and application of these procedures require a combination of basic clinical skills and a knowledge of the disease mechanisms. The 34th Hahnemann Symposium, "Gastrointestinal Emergencies," assembled participants from various disciplines throughout the country and abroad to review progress in the treatment of a number of diseases, to outline a practical approach, and to indicate current directions of research. This volume is based on the symposium presentations, but includes contributions from several authorities not represented on the program.

Although the emphasis of the book is on diagnosis and therapy, each part also includes a chapter dealing with the pathophysiology of that particular disease entity. To understand the complexities of such disorders as hemorrhagic gastritis, pancreatitis and diverticulitis, the mechanisms that may lead to the clinical picture are reviewed.

Several of the most common gastrointestinal emergencies, such as appendicitis, acute cholecystitis, and perforated peptic ulcer, have not been included in this book since we believe that the medical and surgical approaches to these problems have not changed appreciably in recent years, and that adequate information can be obtained from other sources.

The emphasis on radiology reflects the increasing role that arteriography plays in the diagnosis and treatment of gastrointestinal emergencies. Although the internist and the surgeon will not ordinarily be called upon to interpret such studies, it is essential that the physician understand what kind of information can be derived, how arteriography relates to other diagnostic procedures, and how this approach can be used for therapeutic as well as diagnostic purposes.

It is not surprising that controversy was encountered at the symposium, and this is also reflected in the text. Many of the therapeutic regimens advised for gastrointestinal emergencies have not been subjected to rigorous appraisal through controlled double-blind clinical trials. Until such information is available, the practitioner will have to select a course of therapy based upon his own experience, the opinions of others, and the available facilities.

Although it is commonplace to stress the team approach to medical emergencies, there is no more effective application of this concept than to gastrointestinal emergencies. The surgeon, the radiologist and the internist, working together, can deal more efficiently with the changing clinical picture. It is hoped that the diverse clinical skills of the various contributors bring this need into proper focus.

We specifically thank Edith Schwager, Executive Editor of the Department of Medicine, for her expert preparation of this text, and Robert Schaefer and his staff in the Division of Continuing Education for coordinating the symposium.

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PART I

Upper Gastrointestinal Hemorrhage

David A. Brodie, Ph.D. and Bahia D. Mitchell, Ph.D.

1
Pathophysiology of Stress Ulcer

Massive gastric bleeding as a sequela to stress is a medical emergency that has only recently been recognized as an individual entity. Although stress-induced gastric hemorrhage, both clinically and experimentally, has been known for over 40 years, most clinical cases have gone undetected and the true incidence is only now becoming clear. Experimentally, stress-induced gastric hemorrhage has been known since the early 1930s, when it was clearly demonstrated by Hans Selye. Research in the field remained dormant for several decades, but there was a resurgence in the late 1950s. At that time it was felt that stress-induced ulceration in animals was a model of peptic ulcer disease in humans; only within the last 10 years has it become plain that stress-induced gastric hemorrhage is indeed a model of the clinical condition of stress-induced gastric bleeding.

Although there has been a great deal of clinical and experimental research on this condition, the mechanism of this syndrome is still not completely understood. It seems apparent that it has multiple causes rather than one produced by a single physiologic derangement. Recent clinical work has demonstrated that this condition can be prevented if the clinician recognizes this probability in certain cases of severe stress. It has also been shown that stress-induced gastric hemorrhage may require a synergism of several stresses before it becomes manifest. Regardless of the cause, this disease is now coming into much sharper focus as a medical emergency, which is more easily prevented than treated, and the mortality rate will decrease as preventive and therapeutic measures are improved.

The term *stress* tends to be a catchword, with no true biochemical or physiologic measurement to indicate how much "normal stress" animals or humans can take, before the gastric mucosa literally erupts and bleeds. As yet there is no indication of why animals or humans can undergo stress of similar intensities, with some reacting with hemorrhage and others showing no change in the gastrointestinal tract whatsoever. As life on this planet becomes more "civilized," and the level of stress caused by daily living conditions increases, acute stress periods caused by physical or mental trauma will precipitate gastric bleeding. It is predictable that the incidence of this condition will climb steadily, and there is a reasonable possibility that stress-induced gastric bleeding may one day be as important a condition as peptic ulcer is now.

From Abbott Laboratories, North Chicago, Illinois.

HISTORICAL OVERVIEW

Almost 20 centuries ago, the Roman physician Celsus noted symptoms of gastrointestinal stress ulcer in men suffering the tensions and traumas of war.[1] Swan was the first to describe gastric hemorrhage in a burned subject, in 1823,[2,3] and Beaumont, in 1833, observed an acute gastric lesion in an alcoholic patient.[4] The term *Curling's ulcer* was given to "any acute upper gastrointestinal ulceration developing after burns,"[4] in accord with Curling's description of such an acute duodenal ulceration postmortem.[5] Later, *Cushing's ulcer* was used to describe neurogenic ulcers, after Cushing observed acute gastric lesions in patients with head trauma or brain tumors and after neurosurgery.[2,4] Acute gastric lesions are also associated with infectious diseases such as typhoid fever and pneumonia. With the development of gastroscopy during the past 20 years, advanced endoscopic techniques shed more light on the nature of these acute mucosal lesions, and a common term, *stress*, has served as a focal point to link together a symptom complex that occurs after diverse medical conditions.

EXPERIMENTAL MODELS

Stress ulcers can be easily produced in laboratory animals. Rats subjected to various ulcerogenic stresses such as restraint and forced exertion, ligature of the intestines, starvation, cold and electric shock develop stress ulcers in 3 to 24 hours[6,7] (Fig. 1).

Although their pathogenesis is not completely understood, the ulcers appear to result from a combination of gastric mucosal vascular stasis and the presence of acid in the stomach.[7] Erosions of the stomach and duodenum due to social stress have been produced in wild rat species. *Rattus norvegicus*, which coexists peacefully in single-sex groups, gets stress ulcers when placed in a mixed-sex colony.[8]

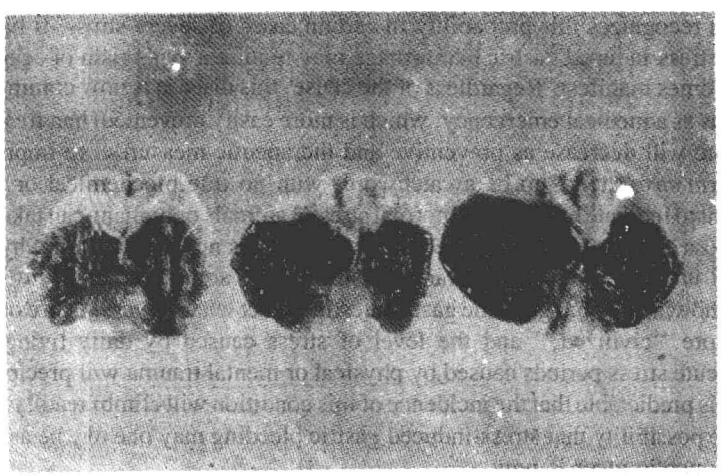

Fig. 1. Restraint ulcers in rats. *Left*. Control stomach. *Middle*. Gastric (corpus) ulcers after 24 hours of restraint at room temperature. *Right*. Gastric (corpus) ulcers after 3 hours of restraint at 3 C. Reproduced through the courtesy of Andre Robert, M.D., Ph.D., Kalamazoo, Michigan.

In guinea pigs subjected to hemorrhagic shock, lesions have been produced identical to the superficial linear erosions associated with shock or sepsis in humans.[9] Perforations have also been produced in guinea pigs by administration of subulcerogenic doses of histamine in beeswax combined with hydrocortisone.[10]

DEFINITION OF STRESS ULCER

The term "stress" is difficult to define succinctly; Selye devotes an entire chapter to its definition.[11] In this chapter, the term *stress ulcers* will be used to indicate the physiologic and biochemical changes that occur following exposure to noxious or painful stimuli, which may be either emotional or physical or both. These changes, through mechanisms still unclear, alter the gastric mucosa and may cause gastric hemorrhage.

Clinically, stress ulcers are acute erosions of the gastric and duodenal mucosa, which occur in patients after severe medical illness or soon after operations, neurosurgical procedures, episodes of trauma or hypotension, injuries, severe burns, frostbite, jaundice and renal or respiratory failure.[9, 12] The terms *stress ulcer, stress-induced gastric bleeding,* and *acute gastric mucosal hemorrhage,* therefore, suggest any form of stressful situation leading to these lesions.

The wide variety of causes and circumstances resulting in stress ulcerations and the predisposing factors, which vary from one patient to another, has led some workers to suggest that the term *stress ulcer* should either be abandoned[13] or referred to as *acute peptic ulcers associated with stress,* since stress ulcers are a form of peptic ulcerations and the acuteness of the condition is the main distinguishing factor between the two.[14] However, the term *stress ulcer* appears to be acceptable as a "shorthand" term for the condition.

DIAGNOSIS

Stress ulcers occur without warning symptoms, and their development is uniformly painless. Clinical recognition is therefore not possible at the outset. The first clinical indication is usually gastrointestinal bleeding and, in rare cases, perforation and obstruction.[15] The lesions may develop within hours of serious injury or sepsis, and the bleeding usually occurs between the second and the twelfth day.[15]

The first sign or symptom related to gastric hemorrhage is hematemesis, vomiting of the contents of the stomach accompanied by bright-red clotted blood. In cases of continued bleeding, the vomiting recurs, and "coffee ground" vomitus may be observed. In addition, there may be a sudden loss of strength, profuse perspiration, weakness, thirst, and difficulty in breathing. Gastric distention, shock, and falling hematocrit level are other symptoms which should be watched for in conjunction with hematemesis.[4, 16]

Diagnosis should be made by endoscopy, the only technique that can successfully identify multiple sources of bleeding. Endoscopy should also be performed in patients with upper gastrointestinal bleeding but negative roentgenographic results. Radiology is virtually useless, since the ulcers are shallow and superficial. Identification at surgery is reserved for gastroscopically inaccessible areas.[4] Angiography has been gaining acceptance as a valuable tool for identification of gastrointestinal bleeding; however, a bleeding rate of 0.5 ml/minute or more is a prerequisite for such a diagnosis.[14, 17]

PATHOPHYSIOLOGY AND ETIOLOGY

Source and Site of Bleeding

In a patient population, acute erosions are probably the largest single cause of gastrointestinal bleeding. The erosions are usually multiple and most commonly found in the body and the fundus of the stomach: the esophagus, gastric antrum and duodenum are usually spared.[15] The lesions are characteristically shallow, and range from a few millimeters to more than a centimeter in diameter, usually without surrounding induration.[4, 16, 18] The small, discrete erosions with shallow bleeding or clotted blackened bases are easily recognized both at surgery and by gastroscopy, but diffuse erosions are difficult to characterize and diagnose. Acute gastric bleeding has been described as the gastric mucosa "weeping" blood.

Upon microscopic examination of the lesions, a pinpoint loss of surface epithelium is apparent at first. Bleeding is encountered when the petechiae develop into ulcers; 5 percent of the ulcers may enlarge or penetrate the muscularis. The ulcers cause rapid dissolution of the stomach wall.[19]

Mechanism

The development of the acute stress ulcer may be considered as a result of an imbalance between aggressive (acid and pepsin) and defensive (mucus, mucosal cell renewal, barrier to acid diffusion, etc.) factors within the stomach.[14]

In general, the various stressful situations and diseases causing these ulcers are thought to act by (1) increasing acid secretion; (2) altering the normal surface protective devices, i.e., mucus and surface epithelial repair; and (3) reducing blood supply by opening arteriovenous shunts or occlusion with thrombi.[13]

The known interrelation between the hypothalamus and control of gastric physiology suggests a causative role of this important brain area in stress ulcer. It has been suggested that stress causes a chain reaction involving the hypothalamus, the anterior pituitary, and the adrenal cortex. Older theories implicate the vagus nerve, which may be stimulated directly or through humoral involvement.[19] Although not a major factor, the presence of acid and pepsin is probably necessary for the development of ulceration.

Restraint stress had no effect on acid output in rats with chronic gastric fistulas, while stimulation of the hypothalamus through electrodes or following brain lesions increased acid output in fasting cats but not in dogs.[13] Stimulation of the anterior hypothalamus in cats reportedly increased the number of parietal cells in the stomach.[13]

A current hypothesis is that a variety of stresses can produce local mucosal microvascular adjustments unrelated to blood flow. This will result in pronounced engorgement of the blood vessels in the superficial mucosa, which will reduce the ability of the mucosa to withstand acid-pepsin digestion, so that ulceration or erosion will develop.[14]

In experimental studies leading to acute mucosal lesions, a decrease in mucus formation has been observed. For example, when cortisone is given to animals, there are: a marked decrease in the rate of mucus secretion, alteration in the viscosity and chemical properties of the mucus, and loss of its protective function.[14] There may also be an alteration in the local circulation to the wall of the viscus; this is still debatable since the mucosal layer itself does not normally prevent acid from bathing the surface of epithelial cells.