

A New 'Physiology in Medicine' Series: Gastrointestinal Disorders

Editorial This issue of HOSPITAL PRACTICE contains the first of a series of seven articles that will appear monthly and deal with the physiology and pathophysiology of gastrointestinal disorders. These articles continue the "Physiology in Medicine" series jointly presented by this journal and the American Physiological Society.

It comes in the wake of a six-article series on dynamic imaging of the coronary circulation in health and disease. It was with that series that the American Physiological Society revived its "Physiology in Medicine" program specifically designed to provide practicing physicians with an ongoing correlation between basic physiologic concepts and developments and the problems confronted in clinical diagnosis and therapy. That program had previously been conducted as a joint effort with *The New England Journal of Medicine*.

In a sense the second series will have a more ambitious objective than the cardiac imaging unit, which focused almost exclusively on ischemic heart disease—a protean problem, to be sure. On the other hand, the gastrointestinal series will address a wide range of common and often perplexing clinical entities, focusing particularly on certain aspects of hepatic dysfunction, on the metabolic derangements leading to gallstone formation, on the pathophysiology of the secretory diarrheas, on the physiology of gastric acid secretion and the pathogenesis of peptic ulcer, and

on bilirubin metabolism and congenital jaundice.

It is an ambitious undertaking, and we feel most fortunate that our collaborators include some of the most outstanding contributors to and teachers of current physiologic and pathophysiologic concepts. A rundown of the series attests to this, as well as to the breadth of the content.

The first article in the series, written by Mortimer Levy and Marvin J. Wexler of McGill University, appears in this issue of HOSPITAL PRACTICE. Its subject is the physiology and pathophysiology of salt and water balance in liver disease. Levy and Wexler review currently proposed pathogenic mechanisms for inordinate renal salt and water avidity in liver disease. Specifically, they focus on two mechanisms currently invoked to account for salt and water accumulation in hepatic disease: the "overflowing" hypothesis, which proposes that a primary signal from the liver provides the stimulus for salt and water retention in hepatic disease; and the "underfilling" hypothesis, which proposes that abnormalities in hepatic perfusion referable to the cirrhotic process produce underfilling of the arterial tree, a reduction in effective circulating volume, and secondary salt and water retention by the kidney.

The second article in the series, by Steven Schenker and Anastacio M. Hoyumpa, Jr., of the University of Texas, San Antonio, reviews the physiology and pathophysiology of hepatic coma. Schenker and Hoyumpa will provide a summary of the means used to produce hepatic coma experimentally, of the contribution of the liver to hepatic encephalopathy, of brain pathology in hepatic encephalopathy, and of the roles of various toxins—for example, ammonia,

This editorial is by Thomas E. Andreoli, Professor and Chairman of the Department of Internal Medicine at the University of Texas Health Science Center, Houston. Dr. Andreoli serves as the editor of "Physiology in Medicine" for HOSPITAL PRACTICE and the American Physiological Society.

Editorial

mercaptans, and fatty acids—in the pathogenesis of hepatic encephalopathy. The article will also discuss the changes in brain neurotransmitters in hepatic encephalopathy and the ways in which these changes derange cerebral function.

In the third article of the series, David H. Van Thiel of the University of Pittsburgh will review the effects of ethyl alcohol upon gonadal function. It is, of course, well recognized that chronic alcoholism in men is accompanied by hypogonadism. Van Thiel will present an argument consistent with the view that it is the alcohol abuse per se, rather than the associated alcoholic liver disease, that is primarily responsible for hypogonadism. Van Thiel will also propose that the feminization seen in alcoholic men—for example, gynecomastia—requires the presence of both liver disease (most notably portosystemic shunting) and alcohol abuse.

The fourth article in the series, by Henry J. Binder of Yale University, will deal with the pathophysiology of the secretory diarrheas. Binder's article will review the derangements in ion secretion and reduced ion absorption that result in diarrhea. The article will also provide a summary of the normal mechanisms for salt and water absorption by the small intestine and describe the role of various secretagogues in the pathogenesis of diarrheas induced by infection and by various laxatives.

In the fifth article of the series, Bernard F. Smith and J. Thomas Lamont of Boston University will review the pathogenesis of gallstones. The article will provide a careful analysis of the role of bile acids in minimizing cholesterol deposition in the biliary tract, the abnormalities in lipid metabolism in individuals with gallstones, and therapeutic strategies that follow from

those pathophysiologic considerations.

The sixth article, by Jon I. Isenberg of the University of California, San Diego, will consider the physiology of gastric acid secretion and the pathogenesis of peptic ulcer disease. Isenberg will provide a frame of reference for understanding the mechanisms of hydrochloric acid secretion in the gut. He will also assess the role of normal protective factors that prevent autodigestion and the derangements of those factors in peptic ulcer disease. Contributions of various agents, both provocative and protective, in the pathogenesis and treatment of peptic ulcer disease will be reviewed.

Finally, the gastrointestinal series will conclude in January 1985 with an article by John L. Gollan of Brigham and Women's Hospital, Boston, on bilirubin metabolism and congenital jaundice. This presentation will summarize our knowledge of the processing of bilirubin by hepatocytes and will discuss in detail various congenital abnormalities of liver function that result in jaundice, among them Gilbert's disease, the Dubin-Johnson syndrome, and Rotor's syndrome.

The diversity that will be represented in this series is reflective of the diversity of the processes required for humans to fulfill one of the most basic of the requirements of all living organisms: the ingestion, transport, and metabolism of nutrients. As with the cardiac imaging articles, the goal of this series will be to shed light on such basic processes, in this instance the light of the dramatic achievements in understanding that have been made by physiologists and their closely allied scientific colleagues.

THOMAS E. ANDREOLI, M.D.