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TOTAL GASTRIC EMPTYING CHANGES AFTER TRUNCAL AND HIGHLY SELECTIVE VAGOTOMY FOR DUODENAL ULCERS

Thesis Submitted for Partial Fulfillment of the M.D. Degree in General Surgery

BY

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بسم الله الرحمن الرحيم

قالوا سبحانك لا علم لنا إلا ما علمتنا إنك أنت العليم الحكيم

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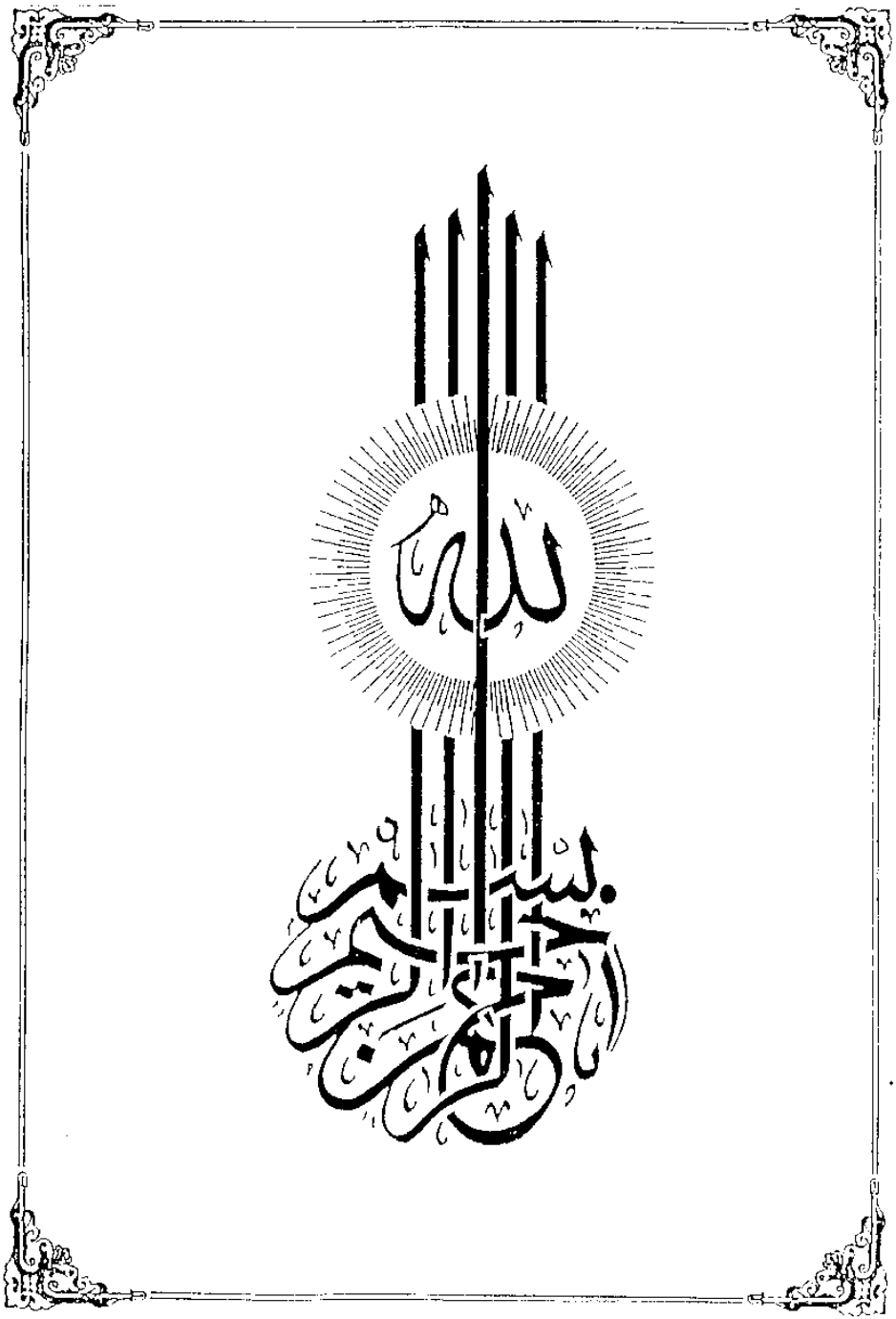

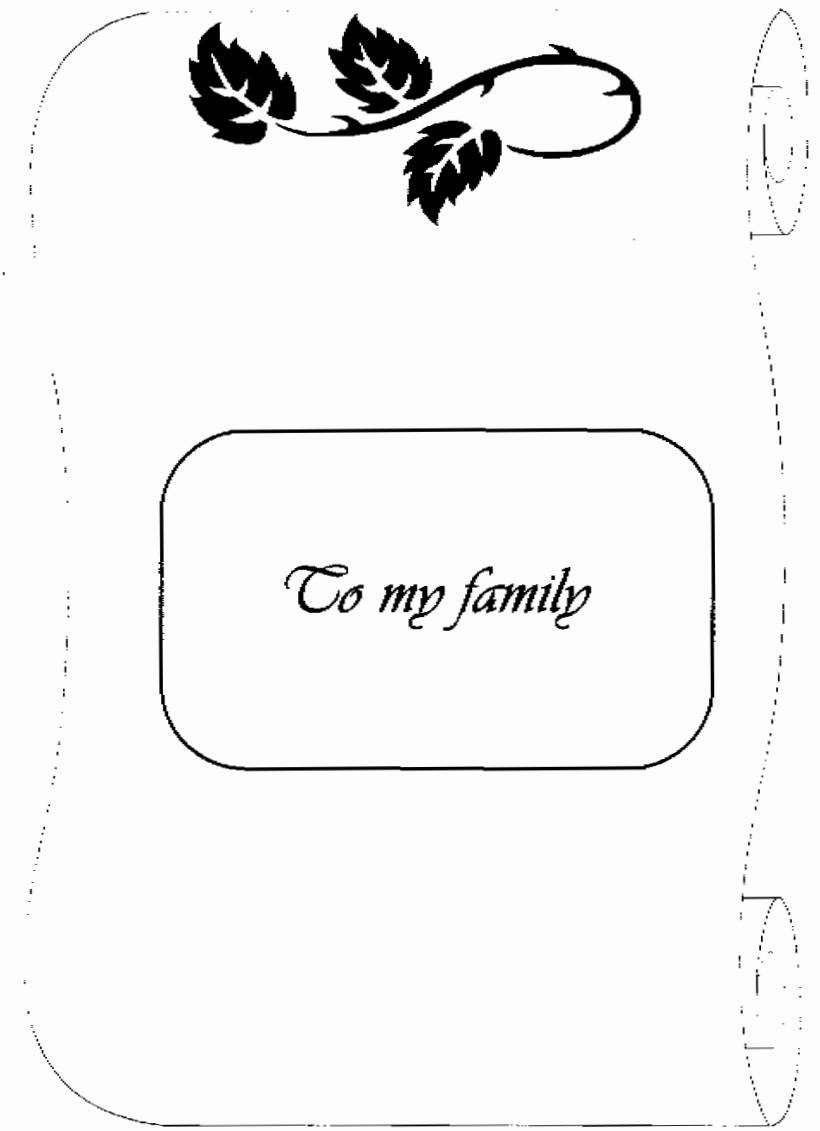
To my family

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Introduction

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Nearly a century has passed since *Bayliss and Starling* performed their pioneering studies on gastrointestinal motility ***(Bayliss and Starling, 1899)***. Nevertheless, our understanding of this topic remains rudimentary. Improved technology and an increased index of suspicion have allowed us to recognize altered gastric motility in a variety of clinical settings in which it had previously been unsuspected. However, clear definition of these motility alterations and of their precise contribution to the pathophysiology of the clinical disorders with which they are associated have been lacking. Surgeons must keep abreast of advances in the diagnosis and treatment of motility disorders so that operative intervention is applied appropriately. Similarly, surgeons will be challenged to identify subgroups of patients who may be at increased risk to develop complications after gastric surgery because of underlying motility disturbances and adjust their approach to minimize that risk. Although no clear cause and effect relation has been demonstrated, altered motility has been found in association with a variety of nonsurgical and postoperative settings. As this relation is better defined, perhaps patients who are at risk to develop complications of surgery can be better identified so that treatment can be tailored toward their specific defect. Technological advances can also be expected to provide new and more effective interventions in this expanding field.

Aim of the Work

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Vagotomy is known to abolish gastric acid secretion and cures ulcer disease. However, paralysis of the stomach follows truncal vagotomy and although highly selective vagotomy maintains antral motility, it is not to be expected that denervation of proximal stomach is not without ill effects. At least there is loss of receptive relaxation and early satiety is expected after highly selective vagotomy. On the other hand, dumping constitutes a not uncommon complication following vagotomy operations. It is obvious that vagotomy operations inflict a variety of motility changes.

The present study aims at the observation of possible gastric motility disorders that follow truncal and highly selective vagotomy operations with the intention of better understanding of the symptoms that may follow surgery for duodenal ulcers.

A comparative analysis of the results obtained after different vagotomy operations will be conducted and compared with normal control subjects.

Review of Literature

Normal Gastric Motility

The stomach performs a number of tasks requiring close coordination of motility and secretion, including mechanical breakdown and preliminary digestion of a meal and controlled emptying of the partially digested meal (chyme) into the duodenum ***(Hocking and Vogel, 1991)***. Gastric motility is controlled by a complex interaction of neural and hormonal influences superimposed on the unique physiologic characteristics of the gastric smooth muscle cells.

The muscle layers of the stomach are composed of sheets of cells that are closely coupled, allowing them to function as an electrical syncytium. The membrane potential of these cells varies spontaneously, generating three types of electrical potentials: (1) the resting potential or maximum membrane potential; (2) the slow wave or pace setter potential (also known as electrical control activity), representing a slow, oscillating partial depolarization of the cell membrane that does not reach the threshold for cellular contraction; and (3) the action potential or spike potential (also known as electrical response activity), a calcium dependent rapid fluctuation in membrane potential that triggers a contraction ***(Hocking and Vogel, 1991)***. Spike potentials, and thus contractions, can occur only during certain phase of the slow-wave cycle. Therefore, the slow-wave frequency of the stomach is set by a "pacemaker" region along the greater curvature which, like the sinoatrial node of the heart, contains cells with an intrinsically higher frequency than the remainder of the organ. Slow waves are synchronized circumferentially and propagate

distally with increasing velocity toward the pylorus so that the distal antrum contracts simultaneously in the so-called terminal antral contractions ***(Kelly, 1981 and Szurewski, 1984)***.

The activity of the stomach varies with time and in response to a meal. The stomach and remainder of the upper gastrointestinal tract do not "rest" between meals. Rather, they undergo cycles of activity known as the interdigestive migrating motility complex (IDMMC), arising in the esophagus and stomach and migrating down the small bowel ***(Carlson et al., 1966; and Soper and Sarr, 1988)***. The cycle, lasting approximately 90 minutes in man, consists of a quiescent phase (I), a phase of increasing activity (II), a short phase of intense contractile activity (III), and a brief transition phase (IV) back to phase I ***(Code and Marlett, 1975; and Soper and Sarr, 1988)***. The high amplitude contractions associated with phase III activity clear indigestible material from the gastrointestinal tract, leading to the term "interdigestive housekeeper" ***(Code and Schlegel, 1974)***. Interdigestive cycling continues until ingestion of a meal initiates a complex cascade of neural and humoral events leading to a less well-defined pattern known as the "fed" pattern ***(Spencer et al., 1990)***.

Although the electrophysiologic characteristics of the gastric smooth-muscle cells provide the frame work for gastric contractions, gastric motility is primarily modulated by neural input ***(Hocking and Vogel, 1991)***. There are three levels of neural control: (1) the CNS, represented by the vagus nerve (parasympathetic); (2) the prevertebral ganglia (sympathetic); and (3) the enteric nervous system. The latter is composed of the myenteric and submucosal neural plexus and represents a primitive nervous system complete with glial cells and capable of independent function ***(Hocking et al., 1988)***. The enteric nervous system is modulated by vagal and