Protocol for thesis submitted in

partial fulfilment for the

degree of M.S. in general surgery .

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Title : Chronic Duodenal Ulcer.

Aim of work : To study the new methods of diagnosis and management of chronic duodenal ulcer and doing a comparative study between the different methods of treatment.

Introduction.

Review of literature:

- Anatomy.
- Patho-physiology of duodenal ulcer.
- Aetiology of chronic duodenal ulcer.
- Clinical picture of chronic duodenal ulcer.
- Complications.
- Investigations.
- Management.

Discussion.

Conclusion and recommendation.

Summary.

References.

Arabic Summary.

Supervisor

Prof.Dr. M. Samoh Zaki.

Medicine

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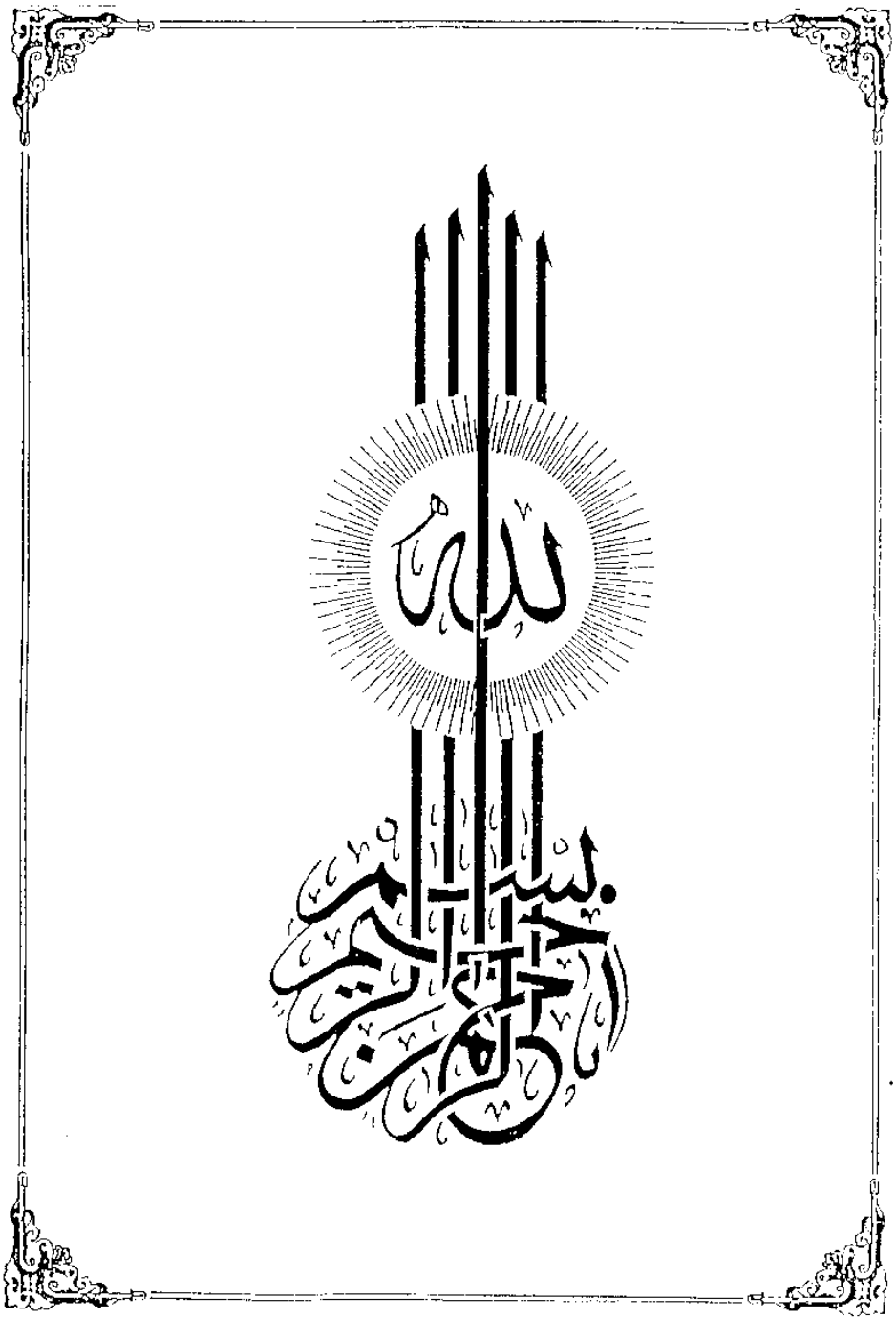
M. Gamal El Din

1985



C O N T E N T S

SURGICAL ANATOMY

OF THE VAGUS NERVES

So far as is known, the vagi nerves are the sole source of parasympathetic innervation of the foregut and midgut. The boundary between the midgut and hindgut is at the splenic flexure of the colon. At this boundary vagal innervation ends and sacral innervation begins.

Below each pulmonary hilus, the left and right vagi descend on either side of the esophagus, and in the lower thorax they branch and communicate with each other forming the esophageal plexus, which surrounds the esophagus. The branches of the esophageal plexus then unite to form two, and only two, vagal trunks-one anterior and the other posterior to the esophagus. The anterior vagal trunk divides into the anterior gastric and hepatic vagal divisions. The posterior trunk divides into the posterior gastric and celiac vagal divisions.

Prior to the normal emberyological gastric rotation, the vagal trunks lie on either side of the esophagus and their gastric divisions descend to the stomach along the lesser curvature. After rotation, the trunks and gastric divisions assume anterior and posterior positions(Fig. I).

The anterior and posterior gastric divisions reach the stomach at the cardia and descend along the lesser curvature beneath the anterior and posterior peritoneal surfaces of the lesser omentum. The stomach is innervated by terminal branches from the anterior and posterior gastric nerves (Fig. 2). Vagal innervation of the stomach is segmental. Each terminal branch from the gastric nerves innervates its own small segment of the stomach with a minimum of overlap (Griffith, 1962).

The hepatic vagi accompany the vestige of the left hepatic artery(or the aberrant left hepatic artery when it is present)

within the lesser omentum to the hepatic plexus, which is an autonomic plexus at the porta hepatis.

The celiac division of the vagi, largest of the four truncal divisions, descends within the gastropancreatic peritoneal fold to the celiac and superior mesenteric autonomic plexuses. Figure 3 shows the distribution of the hepatic and celiac branches of vagi.

The pyloric nerve from the anterior trunk or its gastric division classically runs in the lesser omentum midway between the lesser curvature and the hepatic vagi to the distal antrum and pylorus. When the pyloric nerve is present in this classic position, the anteriorgastric nerve ends proximal to the distal antrum, and descending fibers from the hepatic vagi to the pylorus may be absent. When the pyloric nerve is not in its classic position, the distal antrum and the pylorus receive innervation from one of the two following routs : I) the pyloric nerve parallels or a part of the greater anterior gastric nerve, which can be seen to go all the way to the pylorus, or 2) the pyloric nerve runs adjacent to the most inferior hepatic vagi and descends to the pylorus and distal antrum along with the gastric artery(Griffith,1978).

Surgical application :

There are three types of vagotomy (Fig. 4). Truncal vagotomy refers to transection of all abdominal vagi,that is, a complete gastric , complete hepatic, and complete celiac vagotomy. Selective gastric vagotomy transects all gastric vagi and preserves the hepatic and celiac vagi. Parietal cell vagotomy entails transection of the terminal gastric branches to the parietal cell mass, with preservation of the nerves of Latarjet and thier extramural end-branches to the antrum. Thus, although parietal vagotomy interrupts this intramural innervation of the antrum, the extramural innervation is sufficient to preserve motility of the antrum and pylorus.

The cause of incomplete vagotomy is usually not overlooking a small fiber closely applied to the esophagus within the esophageal fascia propria,instead, the usual cause of inadequate, incomplete vagotomy is failure to expose, and bring into the surgical field, a large fiber when the vagal system is initially encircled with esophgus. There are one or more posterior vagal branches at the cardiofundal level, which, because of the difficulty in locating them, may escape section and thus become instrumental in subsequent recurrent ulceration. Grassi named this nerve or nerves the "criminal" branch of the vagus, and he said that this branch originates from the posterior trunk of the vagus at a variable level(Grassi,1978).

The fallacy of the esophageal diaphragmatic hiatus as the landmark for truncal vagotomy :

The diaphragm has no emberyological or anatomical relationship with the vagal system. Therefore, we must recognize two points. First, as a result of the gastric rotation, the gastric vagal truncal divisions rotate with the stomach and assume constant anterior and posterior positions along the lesser curvature at the cardia. In contrast, at the higher level of the esophgus as it passes through the hiatus, the effect of gastric rotation on the position of the vagal trunks is not so pronounced. Therefore, at the hiatus the trunks lie in variable positions. Second, the vagal system at the hiatus exists as the esophageal plexus or the two trunks or the four truncal divisions.(Fig.5).

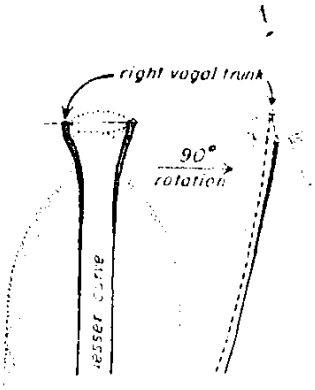


Fig. I
Fffect of stomach's rotation on the position of the vagal trunks.

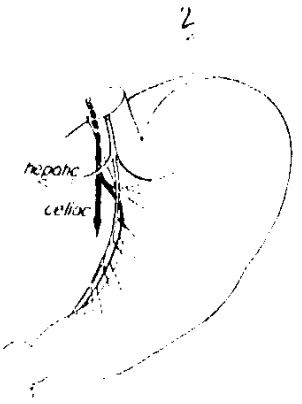


Fig. 2
Innervation of the stomach by the anterior and posterior gastric vagal divisions.

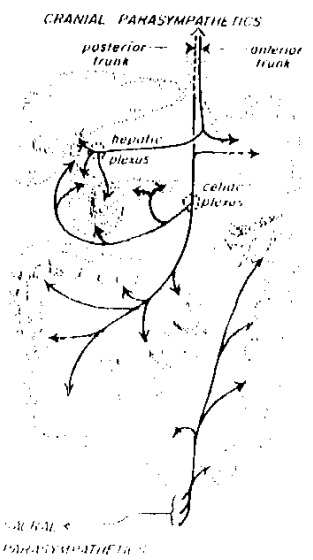


Fig. 3. Distribution of the hepatic and celiac vagi. Preganglionic celiac vagal fibers reach the midgut in company with the superior mesenteric artery and its branches.

Fig. 3

After, Griffith, I962.

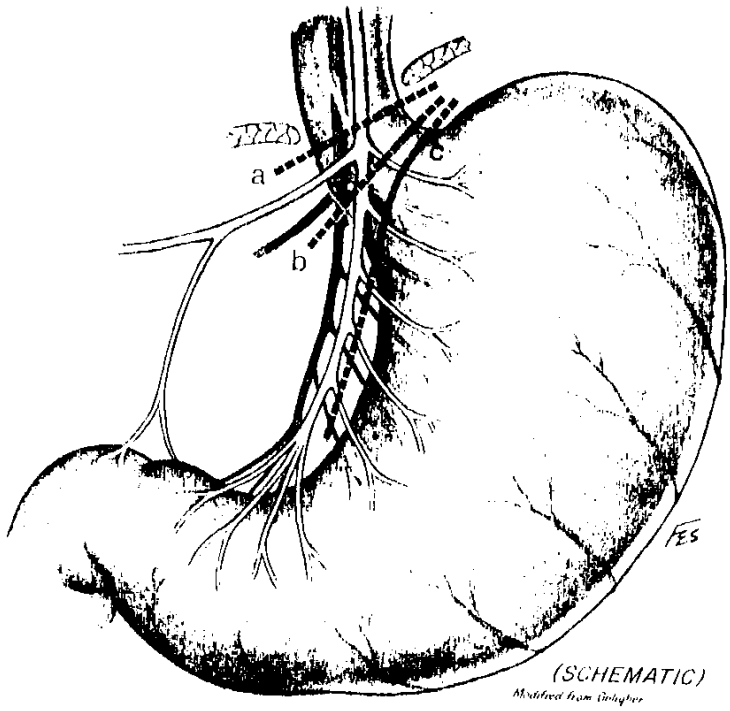


Figure 4. Types of vagotomy: *a*, truncal; *b*, selective; and *c*, highly selective parietal cell vagotomy.

Fig. 4

After, Rossi and Braasch, 1980.

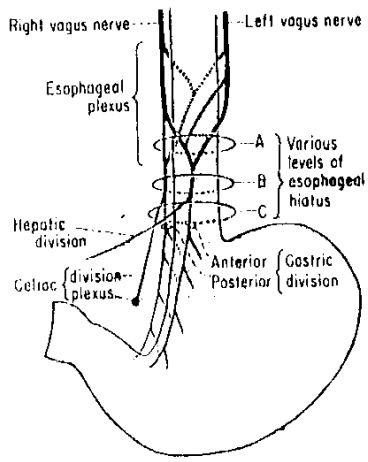


Fig. 2-9. The variable levels of the diaphragmatic esophageal hiatus around the vagal system. (A) The "multiple nerves" of the esophageal plexus. (B) The two vagal trunks. (C) "Multiple nerves" of the four truncal divisions.

Fig. 5

After, Griffith, 1978.

GASTRODUODENAL

PHYSIOLOGY AND PATHOPHYSIOLOGY

Gastric mucosa and its products:

The gastric mucosa consists of a surface layer of mucus-producing columnar cells and coiled, somtimes branched, glands. The glands contain mucus,parietal(oxyntic),chief(peptic), and endocrine cells. The glands of the cardiac area cotain few parietal and chief cells. In the corpus fundus region (oxyntic gland area) the oxyntic glands contain mucus cells interspersed with parietal cells in the midportion of the gland, chief cells at the base of the gland, and endocrine cells in the basal half of the glands. A transitional zone, 0.5-2cm wide, separates the oxyntic gland area from the pyloric gland area and contains mixed glands. The glands of the antrum contain mucus cells, very few parietal cells, and endocrine cells. The mucosa of the antrum is the richest source of gastrin in the body.

The electrolytes in the gastric juice are a mixture of secretions from parietal and non parietal cells. The non parietal cell secretion is a mucus-containing juice that is slightly alkaline due to a bicarbonate , and it contains sodium, potassium, and chlorid in about the same concentrations as found in the extra-cellular fluid. The parietal cells secrete hydrochloric acid and potassium chlorid solution in a ratio of about I5:1 . The maximal hydrogen ion concentration found in the gastric juice is about I50 mmol/liter (Olbe, I978).

During acid secretion the metabolism in the parietal cells is mainly aerobic. Stimulation and inhibition of secretion may be regulated intracellulary by cyclic AMP (adenosine monophosphate) and cyclic GMP (guanosine monophosphate). Both H^+ and CL^- are actively transported into the gastric lumen by separate pumps (Fig. 6). The H^+ may be thought of as a product of the dissociation of H_2O. Carbonic acid is formed from the hydration of CO_2 . The carbonic acid dissociates, and the resulting bicarbonates excreted

into the bloodstream. Water passively enters the gastric lumen following the actively transported ions, and the secretion is isotonic or nearly so.

The concentration of acid secreted by parietal cells is about I50 mmol/liter a million times greater than the hydrogen ion concentration in the blood. The ability of the stomach to secret and hold this highly concentrated solution within its lumen is intrinsic to the mucosa itself and not to any special anatomic structure (Lawrence, I979).

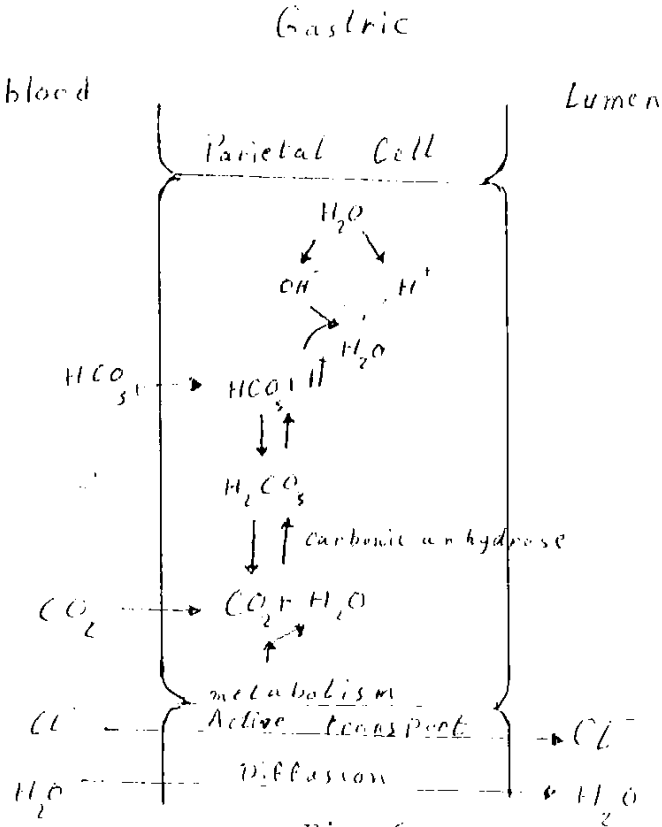


Fig. 6
intracellular processes in formation of gastric hydrochloric acid

The intrinsic factor is a mucoprotein which forms a coplex with vitamin B_{I2}. In presence of calcium ions the specific complex facilitates absorption of vitamin B_{I2} in the distal ileum. The site of origin of intrinsic factor has been localized to parietal cells.

Pepsinogens are synthesized in the chief cells of the oxyntic gland area and to a lesser extent in the pyloric area and stored as visible granules. Cholinergic stimuli, either vagal or intramural, are the most potant pepsinogogues, although gastrin and secretin are also effective. Below a PH of 5 the pepsinogens are converted to pepsins. The pepsins are responsible for the protease activity. Its optimal PH is about 2.0. The pepsinogens are separate into two immunochemically different groups, pepsinogens I and pepsinogens II. Gastric pepsinogen secretion for the most part parallels gastric acid secretion,

Seventy five percent of people secrete blood group antigens into gastric juice. The trait is genetically determined and is associated with a lower incidence of duodenal ulcer than in non secretors.

Mucus is a heterogeneous mixture of glycoproteins manufactured in the mucus cells of the oxyntic and the pyloric glands. Mucus provides a weak barrier to the diffusion of H^+ and protects the mucosa.

Endocrin cells in the pyloric gland area have been shown to contain gastrin (G cells). Other endocrine cells in the gastric mucosa are enterochromafin (EC) cells cotaining 5-hydroxytryptamine and enterochromaffin-like (ECL) cells that prodominate in the oxyntic gland area. The ECL cells do not contain visible amounts of histamine, however, histamine has been found in the mucosal mast cells (Lundell, 1975).

Resistance of the gastroduodenal mucosa :

Normal acid secretion in many patients with duodenal ulcer has led to speculation that there is reduced mucosal resistance to ulceration in these patients. It is a function of the normal mucosa to prevent reentry of hydrogen ions and the movement of sodium ions across the mucosa. Ivery (1971) used the term mucosal barrier to describe these functions.

Rubbing of the mucosa or local application of acid or hypertonic solutions increases the gastric mucus secretion. The main function of the gastric mucus is lubrication.

Of major importance in the protection of the mucosa is the alkaline state of the mucosa, since acid secretion must be accompanied by production of an equivalent amount of bicarbonate which diffuse into the small vessels of the mucosa. In addition, the

surface epithelial cells actively secrete bicarbonate into the surface. Brunner's glands which are localized in the submucosa of the proximal duodenum secrete an alkaline mucus containing pepsinogens. Duodenal ulcer patient produce a lower bicarbonate response to duodenal acidification than healthy subjects. Bicarbonate secretion is stimulated by prostaglandins which are synthesised in the gastric mucosa (Johansson et al, I980).

Phases of gastric secretion :

Gastric secretion is said to occur in three phases: a cephalic phase, a gastric phase, and an intestinal phase. However, these three phases in reality fuse together (Guyton, I977).

The cephalic phase :

The cephalic phase of gastric secretion occurs even before food enters the stomach. It results from the sight, smell, though, or taste of food. Neurogenic signals causing the cephalic phase of the secretion can originate in the cerebral cortex or in the appetite center of the hypothalamus. They are transmitted by the dorsal motor nuclei of the vagi to the stomach. This phase of secretion accounts for about one tenth of the gastric secretion normally associated with eating a meal.

The gastric phase :

Once the food enters the stomach, it excites the gastrin mechanism, which in turn causes secretion of gastric juice that continues throughout the several hours that the food remains in the stomach.

In addition, the presence of food in the stomach also causes (a) local reflexes in the myenteric plexus of the stomach and (b) vago-vagal reflexes that pass all the way to the brain stem and back to the stomach. Both of these reflexes cause