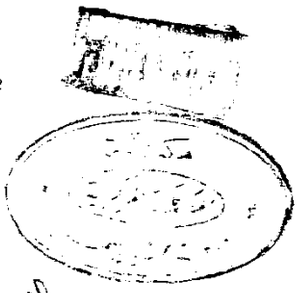
UPPER GASTROINTESTINAL HAEMORRHAGE DUE TO STRESS ULCERATION

An essay submitted in partial fulfilment for the

Master Degree in General Surgery



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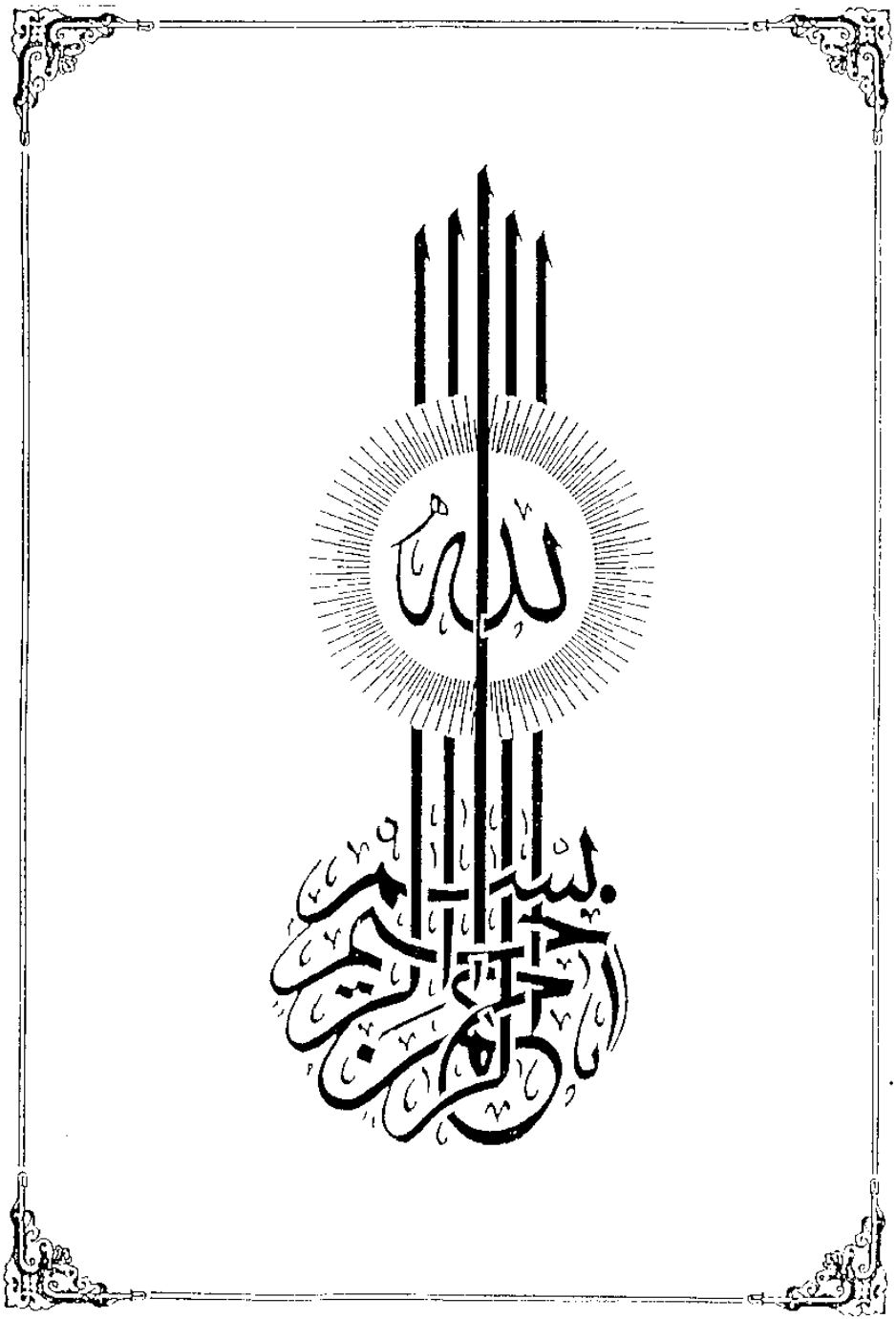

INTRODUCTION

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Stress ulcer is a serious complication that poses a further threat to patients already desperately ill. The mortality rates among those patients are high. The cause of death is usually due to haemorrhage.

The objective of this study is to describe the stress syndrome and the possible mechanisms by which stress leads to disturbance of gastric secretion and barrier and hence initiating the ulcer formation.

Also the diagnosis and different methods advocated for prevention and treatment are discussed.

PHYSIOLOGY OF GASTRIC SECRETION

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The stomach mucosa has two types of tubular glands, the gastric or oxyntic glands and the pyloric glands.

* The gastric glands : are located in the mucosa of the fundus and body of the stomach except along the lesser curvature and are composed of three different types of cells which are :

(1) The mucus neck cells : these type of cells secrete mucus mainly and some pepsinogen.

(2) The peptic (or chief) cells : these type of cells secrete large quantities of pepsinogen.

(3) The oxyntic (or parietal) cells : these cells secrete hydrochloric acid and the intrinsic factor.

* The pyloric glands : are located in the antral portion of the stomach. They are structurelly similar to the oxyntic glands, but contain few peptic and oxyntic cells. Instead, they contain almost entirely mucus cells that are identical with the mucus neck cells of the gastric

glands.

These cells secrete a large amount of thin mucus which protects the stomach wall from digestion by the gastric enzymes and small amount of pepsinogen.

Also the pyloric glands contain gastric cells (or G cells) which secrete the human gastrin.

In addition to the gastric and pyloric glands, the surface of the stomach mucosa between the glands has a continous layer of mucus cells that secrete large quantities of a far more viscid alkaline mucus that coats the mucosa with a mucus gel layer often more than 1 mm thick, thus providing a major shell of protection for the stomach wall as well as contributing to lubrication of food transport. Slight irritation to the mucosa directly stimulates the mucus cells to secrete copious quantities of this thick viscid mucus.

Also, a few mucus secreting cardiac glands, which are almost identical to the pyloric glands, are located

about 1 cm immediatly surrounding the entry point of the oesophagus.

Hydrochloric Acid Secretion :

The oxyntic (parietal) cell contain a system of intracellular canaliculi. The hydrochloric acid is formed at the membrane of these canaliculi and then conducted through openings to the exterior.

The postulated mechanism for the secretion of hydrochloric acid consists of the following steps :

1-Chloride ion is actively transported from the cytoplasm of the oxyntic cell into the lumen of the canaliculus. This creates a negative potential in the canaliculus, which inturn causes passive diffusion of positively charged potassium ions from the cell cytoplasm also into the canaliculus. Thus, in effect, potassium chloride enters the canaliculus.

2-Water is dissociated into hydrogen ions and hydroxyl ions in the cell cytoplasm. The hydrogen ion is then

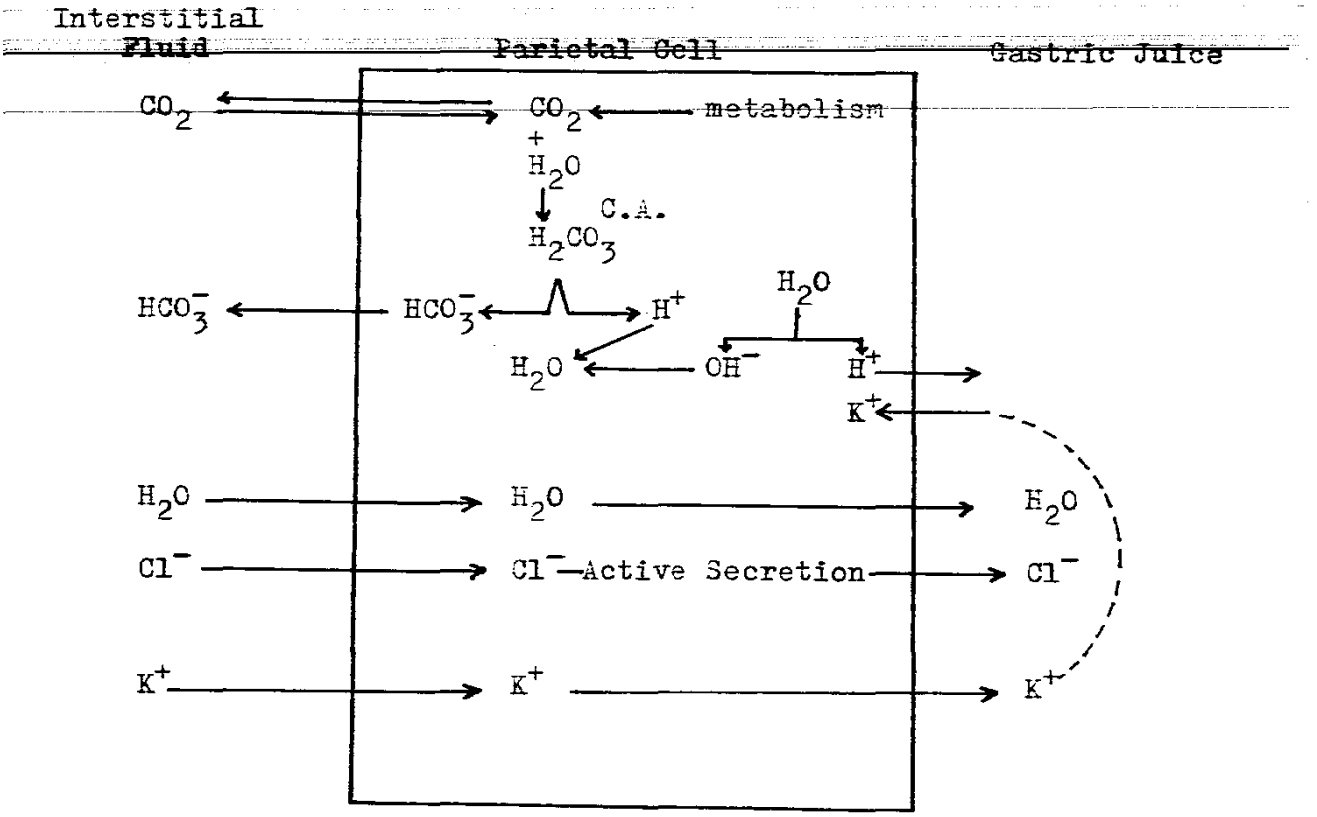

Fig.1: Postulated mechanism for the secretion of hydrochloric acid.
(Taken after Guyton,A.C. 1981)

actively secreted into the canaliculus in exchange for potassium ions. This active exchange process being catalyzed by H^+-K^+ ATP ase. Thus, most of the potassium ions that had been secreted along with the chloride ions are reabsorbed, and hydrogen ions take their place in the canaliculus.

3-Water passes through the cell and into the canaliculus by osmosis. Thus the final secretion from the canaliculus

is a solution containing hydrochloric acid in a concentration of 160 millimoles per liter and potassium chloride in a concentration of 17 millimoles per liter.

4-Finally , carbon dioxide, either formed during metabolism in the cell or entering the cell from the blood, combines with water under the influence of carbonic anhydrase to form carbonic acid. This , inturn, dissociates into bicarbonate ion and hydrogen ion. The hydrogen ion combines with the hydroxyl ion released in step (2) to form water. The bicarbonate ion , inturn, diffuses out of the cell into the blood. The importance of carbon dioxide in the chemical reactions for formation of hydrochloric acid is illustrated by the fact that carbonic anhydrase inhibition by the drug acetazolamide almost completely blocks the formation of hydrochloric acid.

Pepsinogen Secretion :

The peptic (chief) cells contain zymogen granules which contain the pepsin precursors and is activated in the intestinal tract.

The pepsin precursors are called pepsinogen and are activated by the gastric hydrochloric acid.

Human gastric mucosa contains 3 chromatographically distinct pepsinogen, which produce 3 pepsins with slightly different properties (pepsins 1,11& 111)(Ganong,W.F.1983), but perform essentially the same function.

Pepsin is an active proteolytic enzyme in a highly acid medium (optimum pH is 2.0) but above the pH of about 5 it has little proteolytic activity and soon becomes completely inactivated.

Therefore, hydrochloric acid secretion is equally as necessary as pepsin secretion for protien digestion in the stomach.

Regulation Of Gastric Secretion :

Gastric secretion is regulated by both nervous and hormonal mechanisms. Nervous regulation being affected through the parasympathetic fibers of the vagus nerves

as well as through local intrinsic nerve plexus reflexes, and the hormonal regulation taking place by means of the hormone gastrin.

Vagal Stimulation Of Gastric Secretion :

Nervous signals to cause gastric secretion originate in the dorsal motor nuclei of the vagi and pass via the vagus nerves to the intrinsic nerve plexus of the stomach and thence to the oxyntic gland. In response, these glands secrete vast quantities of both pepsin and acid, but with a higher proportion of pepsin than in gastric juice elicited in other way. Also, vagal signals to all the mucus secreting glands and epithelial lining cells cause increased mucus secretion as well.

Still another effect of vagal stimulation is to cause the antral part of the stomach mucosa to secrete the hormone gastrin, this hormone then acts on the gastric glands to cause additional flow of highly acidic gastric juice. Thus, vagal stimulation excites stomach secretion both directly

by stimulation of the gastric glands and indirectly through the gastrin mechanism.

Stimulation Of Gastric Secretion By Gastrin :

When food enters the stomach, it causes the antral portion of the stomach mucosa to secrete the hormone gastrin.

Gastrin is a large peptide secreted in two forms; a large form called G34, containing 34 amino acids, and a smaller form ,G17, containing 17 amino acids, though both of them are important, the smaller form is more abundant.

The food causes release of this hormone in 2 ways;

(1) The actual bulk of the food distends the stomach, and this causes the hormone gastrin to be released from the antral mucosa.

(2) Certain substances called secretagogues- such as food extracts, partially digested protien, alcohol (in low con-

centration), caffeine- also cause gastrin to be liberated from the antral mucosa.

Both of these stimuli- the distension and the chemical action of the secretagogues- elicit gastrin release by means of a local nerve reflex. That is, they stimulate sensory nerve fibers in the stomach epithelium which inturn synapse with the intrinsic nerve plexus. This then transmitts efferent signals to the gastrin cells, causing them to secrete the gastrin.

Therefore, any factor that blocks this reflex will also block the formation of gastrin. For instance, anesthetization of the gastric mucosa to block the sensory stimuli will prevent gastrin release, adminstration of atropine, which blocks the action on the gastrin cells of the acetylcholine released by the intrinsic nerves and also by the vagus nerves, will also prevent gastrin release.

Gastrin is absorbed into the blood and carried to the gastric glands where it stimulates mainly the oxyntic cells