Gastric Secretion: Phases and Factors (With Diagram)

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In this article we will discuss about:- 1. Methods to Study Gastric Secretion 2. Phases of Gastric Secretion and their Regulation 3. Factors 4. Depressants 5. Other Hormones 6. Tests for Gastric Secretory Function in Man.

Methods to Study Gastric Secretion:

There are several methods available in experimental animals and in man to collect gastric juice, enabling us to study the various aspects of gastric secretion and their regulation.

a. In Experimental Animals (usually in dogs):

1. Pavlov's pouch:

This pouch is made out of the stomach in the dog in such a way that the pouch opens through the skin to the exterior and is still connected to the main body of the stomach by a bridge.

The pouch helps in the collection of pure gastric juice uncontaminated by food. Being a part of the main stomach, Pavlov's pouch retains both parasympathetic and sympathetic nerve supply. Pavlov's pouch is very useful in the study of various phases of gastric secretion and their regulation.

2. Heidenhain's pouch:

This pouch is constructed in a way similar to Pavlov's pouch. The only difference is that Heidenhan's pouch does not have vagal supply. It has got sympathetic nerve supply and blood supply. It is also very useful in study of gastric secretion. Especially to study the effect of absence of vagal influence on gastric secretion. Secretion will be influenced by only the hormones.

3. Bickel's pouch:

It is nothing but Heidenhain's pouch without sympathetic supply. Totally denervated pouch.

4. Pavlov's sham feeding:

In this experiment, esophagus of the dog is exposed in the neck. A complete cut is made transversely and the two cut ends of esophagus are made to open one below the other through the skin of the neck. Therefore, any food placed inside the mouth once swallowed, does not enter the stomach. It comes out through the upper cut end of esophagus.

The animal gets all the pleasure of eating (sensation but the food does not reach the stomach. That is why this procedure is called SHAM or mock feeding). To make the animal survive, it is fed through the lower cut end of esophagus. This is very useful to study the cephalic phase of gastric secretion. The lower end is also useful to collect gastric juice from stomach with the help of a tube.

b. In Man:

a. Through Ryle's (nasogastric) tube:

A thin flexible rubber tube is passed through the nose or mouth and gastric juice can be collected from stomach by aspiration.

b. Gastric fistula:

Fistula is a communication between one hollow organ and another or a communication between a hollow organ and the exterior.

c. Gastroscopy:

Gastroscope is an instrument through which the interior of the stomach can be easily visualized. This is particularly done to determine ulcers, cancerous growths, etc.

Phases of Gastric Secretion and their Regulation:

There are four phases of gastric secretion:

1. Cephalic phase

2. Gastric phase

3. Intestinal phase

4. Inter-digestive phase

Cephalic Phase of Gastric Secretion:

Presence of food in the mouth stimulates gastric secretion through the taste pathways. This is an inherent reflex. Sight, smell and thought of food also stimulate gastric secretions through conditioned reflexes acquired during childhood. For all these stimuli to evoke gastric secretion, efferent vagus nerve should be intact. Since all the above stimuli act through higher centers in CNS, this is known as cephalic phase (Fig. 5.13).

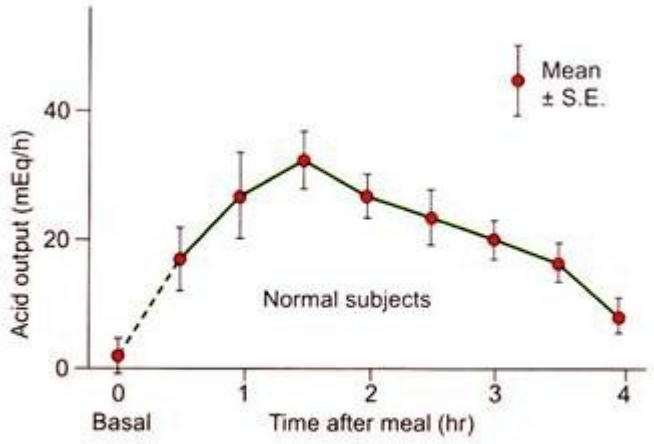

Fig. 5.13: Time required for maximal acid output after ingestion of food

Within 3-5 minutes of stimulation, the gastric secretion starts. In about 30 minutes, it reaches the peak, the volume of secretion being 30-150 ml in 20 min.

This phase can be very well demonstrated in sham fed dogs.

In sham fed animals, if both the efferent vagi supplying the stomach are cut, the cephalic phase of gastric secretion is abolished. The gastric juice produced by sight, smell and thought of food, i.e. food not being in stomach or mouth at all, is called appetite or psychic juice. Good music, pleasant surroundings, neatly dressed servers can influence the volume of juice secreted during this phase.

Appetite is anticipation of pleasure of eating. This significance of cephalic phase is that the stomach prepares itself to receive the food.

Emotional factor also acts through cerebral cortex. Feelings of anger and aggression increase gastric secretion. Chronic fear, depression, grief or sorrow all diminish gastric secretion. Most of the stimuli of cephalic phase act through different areas of cerebral cortex one of which is the limbic lobe including the hypothalamus.

From here, the stimuli reach the dorsal motor nucleus of vagus from where the preganglionic fibers take origin and reach stomach.

The postganglionic fibers from the plexus stimulate the chief and parietal cells in different ways:

(a) through acetylcholine which directly acts in gastric glands, (b) stimulates G cells of pyloric antrum through Bombesin. G cells in turn produce gastrin which acts on gastric glands (Fig. 5.14).

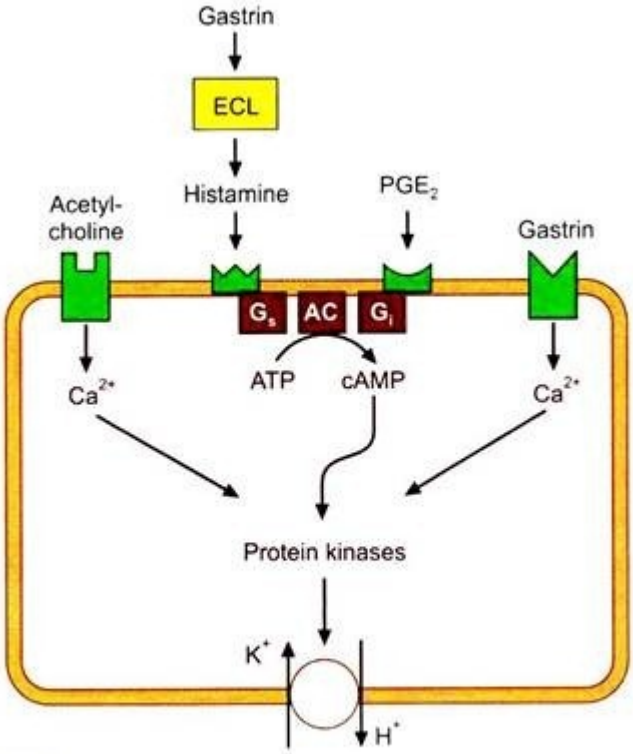

Fig. 5.14: Chemical substances acting on the parietal cells and intracellular reactions involved during the process of secretion of hydogen ion which later on reacts with chloride ion to form hydrochloric acid

Parietal and chief cells have got separate receptors for acetylcholine, gastrin and histamine.

Gastric Phase:

It is also called chemical or hormonal phase. Presence of food in the stomach stimulates gastric secretion. Secretion starts within 15 minutes of arrival of food in the stomach. The volume of secretion is 225 to 350 ml in 5 hrs.

Specific stimuli responsible for this phase are:

a. Mechanical distension of stomach by the food

b. Proteins and derivatives in the food

These stimuli act locally within the stomach. Their receptors are in the gastric mucosa and the efferent are local vagal fibers which stimulate the ganglion cells in submucosal plexus from where the postganglionic fibers stimulate the gastric glands.

Although vagus and gastrin can act independently on the gastric glands (parietal and chief cells), they potentiate the action of each other or have synergistic action.

Gastrin:

It is a very important GI hormone. It is a polypeptide.

Site of production:

G cells or APUD cells (Amine precursor uptake and decarboxylation cells) located in the pyloric antrum. Similar cells are found in duodenum also.

The hormone secreted by these cells in response to specific stimuli, enters the systemic circulation and reaches gastric glands.

Types of gastrin:

There are different types of gastrin.

The most important ones are:

i. G-17 LITTLE gastrin.

ii. G-14 MINI gastrin

iii. G-34 BIG gastrin

Gastrin is in use for studying gastric secretion. Pentagastrin has got last 4 amino acids of G-17 and one more amino acid. Pentagastrin has all the actions of G-17. All the gastrins are inactivated in liver and kidney.

Regulation of gastrin secretion:

Stimuli that release gastrin:

a. Mechanical distension of stomach

b. Partially digested food proteins

c. Vagal stimulation releasing Bombesin

d. Blood-borne factor, like epinephrine

e. Ca^{++} ions.

f. Alcohol

Factors that inhibit gastrin release are:

a. HCl in pyloric antrum or in duodenum (anytime when the pH of gastric contents falls below 2).

b. Hormones from duodenum, e.g.:

i. GIP (gastric inhibitory polypeptide)

ii. VIP

iii. Secretin

iv. Enterogastrone

v. Glucagon

Actions of gastrin:

Physiological actions:

i. Stimulates secretion of gastric juice rich in HCl and pepsin

ii. Stimulates gastric motility

iii. Promotes the growth of gastric mucosa.

iv. Increasing the tone of esophageal sphincter.

Intestinal Phase of Gastric Secretion:

Presence of food in the small intestine stimulates gastric secretion.

Specific stimuli:

Partially digested proteins and polypeptides stimuli are supposed to act through release of gastrin in duodenum. Duodenal gastrin can also be released by alcohol. Duodenum in response to presence of HCl, partly digested fat, hyperosmolar solutions in its lumen, inhibits gastrin release from pyloric antrum and thereby diminishes gastric secretion.

This inhibitory action is believed to be brought about through certain hormones examples of which are:

i. Enterogastrone (hypothetical hormone)

ii. GIP

iii. VIP

iv. Secretin

v. Glucagon from duodenum

Intestinal phase takes about 2 hrs to start and volume of secretion is 200-300 ml.

Interdigestive Phase:

Small quantity of gastric juice is continuously produced and is known as interdigestive phase secretion. The mechanism of this phase is not well understood. Emotional conditions and conditioned reflexes may be responsible for this phase. Rate of secretion is 30-60 ml/hr.

Other gastric stimulants:

Secretogogues

Secretogogue is any substance which increases the gastric secretion.

Histamine is a very powerful stimulant of gastric secretion, especially HCl. It is as powerful as gastrin and vagal nerve stimulation. It binds to specific receptors on the parietal cells known as H_2 receptors. Cyclic AMP is also involved in the action.

Combination of histamine with H_2 receptors can be blocked by drugs, e.g. cimetidine which is very useful in treatment of peptic ulcers.

Although histamine is produced in large quantity by stomach wall from mast cells, its physiological role is not very clear.

Factors Stimulating Gastric Secretion:

i. Condiments (flavoring agents)

ii. Vegetable extracts like soup

iii. Alcohol (ethyl alcohol) very powerful stimulant of gastric secretion. Probably acts by liberation of histamine and gastrin in stomach.

iv. Caffeine (coffee extract) very good stimulant of gastric secretion

v. Parasympathetic agents: Such as acetylcholine, etc. are stimulants.

vi. Cigarette smoking: Nicotine of tobacco in small doses stimulates autonomic ganglia. Vagal stimulation increases gastric juice secretion rich in acid and enzymes.

Depressants of Gastric Secretion:

i. Drugs that act against acetylcholine, e.g. atropine

ii. Anti-H_2 drugs, e.g. cimetidine

iii. Antacids, e.g. $Mg(OH)_2$

a. Aluminium hydroxide

b. Sodium bicarbonate ($NaHCO_3$)

c. Calcium carbonate All these neutralize HCl.

Other Hormones that Influence Gastric Secretion:

i. ACTH and glucocorticoids:

They stimulate gastric secretion. The hormone secretion is increased during stress. It is well documented that people undergoing chronic stress develop peptic ulcer.

ii. Insulin:

By causing hypoglycemia. Stimulates hypothalamus which in turn stimulates vagus leading to gastric secretion. For this to be effective, blood sugar level should fall to 50 mg% of the normal. This principle is made use of in a gastric function test (insulin test or Hollander's test).

iii. Somatostatin:

It is a hormone secreted by the neurons in the hypothalamus. Though its primary action is to inhibit GH release from anterior pituitary, it also inhibits gastric secretion by inhibiting gastrin release.

Tests for Gastric Secretory Function in Man:

Total and free acidity: Gastric juice is collected using Ryle's tube. HCl in this juice is in two forms: free H^+ ions called free acidity (20-60 mEq/liter) and a part of the acid is in combination with proteins.

This can be determined by titration of gastric juice against N/10 NaOH. Titrate to pH 3.5 using Topfer's indicator. It gives free acidity. On continuing titration to pH 7.4 using phenolphthalein will give total acidity.

Absence of acid in gastric juice is achlorhydria:

Decrease in acid content below normal is hypo- chlorhydria and increase above normal is called hyperchlorhydria.

Achlorhydria can be true or false. In true achlorhydria, HCl is absent from gastric juice even after injection of histamine or pentagastrin. This occurs in pernicious anemia and gastric cancer.

Hypochlorhydria can be produced in gastritis.

Total and free acidity is usually estimated either from gastric juice collected overnight or basal secretion (early in the morning).

Over the years, it has become a practice to study the effect of specific stimulants on gastric secretion. Initially, a carbohydrate meal in the form of porridge or toast and coffee is given, Gastric juice is collected before and after the meal and analyzed for HCl, pepsin activity, etc. Such a test is known as fractional test meal (FTM). This will also facilitate the person performing the test to understand the gastric motility and emptying time.

This was replaced by augmented histamine test. In this, instead of giving a meal, histamine is injected 0.04.mg/kg body weight to stimulate gastric secretion. To make histamine act only on the H_2 receptors of stomach, the other unwanted side effects of histamine are blocked by antihistamine drugs.

Because of the side effects, this test is also given up. Now pentagastrin is used. Pentagastrin is injected 5 μg/kg body weight. Gastric juice is collected before injection and every 15 min after the injection. In a normal response, the HCl secretion reaches a peak of 25-40 mEq/hr within 15 min; the peak is maintained for another 15 min and acidity comes back to normal value 30 min later.

The peak acid output after pentagastrin may be raised in duodenal ulcer. The peak may be lowered in gastritis or other diseases where gastric mucosa is damaged.

Hollander's test (insulin test):

Insulin test is effective only in the presence of vagus. This test is very useful to confirm the success of vagotomy, a common operation done for peptic ulcer.

Peptic ulcer:

Refers to the chronic type of ulcer caused due to acid peptic digestion of gastric or intestinal mucosa.

The classical sites of peptic ulcer are:

a. On the lesser curvature just proximal to pyloric region. This is known as gastric ulcer.

b. When the very beginning part of duodenum is involved, it is known as duodenal ulcer.

The factors that may be involved in the production of ulcers are:

a. Breakdown of gastroduodenal mucous barrier.

b. Excess of secretion of HCl.

c. In gastrinoma which is tumor of D cells, there is very high level of gastrin leading to increased HCl and pepsin secretion leading to peptic ulcer. This clinical condition is known as Zollinger-Ellison syndrome and there is hyperplasia of D cells of pancreas in this condition.

Other factors which may predispose the development of peptic ulcer are:

a. Chronic stress

b. Helicobacter pylori infection

c. Dietary factors, like spicy food

d. Alcohol

e. Smoking

f. Steroid administration

g. Administration of aspirin group of drugs.

Treatment of peptic ulcer:

i. Antacids

ii. Cimetidine, ranitidine, famotidine, etc. which are H_2 blockers

iii. Omeprazole which is a proton pump blocker

iv. Proglumide drug which inhibits the action of gastrin on G receptors

v. Bland diet

vi. Meditation

vii. Vagotomy

viii. One of the surgical procedures is gastrojejunostomy (by passing duodenum).

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