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Regulation of Stomach Secretion

Approximately 2–3 L of gastric secretions (gastric juice) is produced each day. The amount and type of food entering the stomach dramatically affects the secretion amount, but up to 700 mL is secreted as a result of a typical meal. Both nervous and hormonal mechanisms regulate gastric secretions. The neural mechanisms involve reflexes integrated within the medulla oblongata and local reflexes integrated within the enteric plexus of the GI tract. In 876 Part 4 Regulations and Maintenance addition, higher brain centers influence the reflexes. Chemical signals that regulate stomach secretions include the hormones gastrin, secretin, gastric-inhibitory polypeptide, and cholecystokinin, as well as the paracrine chemical signal histamine. Regulation of stomach secretion is divided into three phases: cephalic, gastric, and intestinal.

1. **Cephalic phase.** In the cephalic phase of gastric regulation, the sensations of the taste and smell of food, stimulation of tactile receptors during the process of chewing and swallowing, and pleasant thoughts of food stimulate centers within the medulla oblongata that influence gastric secretions. Action potentials are sent from the medulla along parasympathetic neurons within the vagus (X) nerves to the stomach.Within the stomach wall; the preganglionic neurons stimulate postganglionic neurons in the enteric plexus. The postganglionic neurons, which are primarily cholinergic, stimulate secretory activity in the cells of the stomach mucosa. Parasympathetic stimulation of the stomach mucosa results in the release of the neurotransmitter acetylcholine, which increases the secretory activity of both the parietal and chief cells and stimulates the secretion of **gastrin** (gas_trin) and histamine from endocrine cells. Gastrin is released into the circulation and travels to the parietal cells, where it stimulates additional hydrochloric acid and pepsinogen secretion. In addition, gastrin stimulates endocrine cells to release histamine, which stimulates parietal cells to secrete hydrochloric acid. The histamine receptors on the parietal cells are called H2 receptors, and are different from the H1 receptors involved in allergic reactions. Drugs that block allergic reactions do not affect histamine-mediated stomach acid secretion and vice versa. Acetylcholine, histamine, andgastrin working together cause a greater secretion of

hydrochloric acid than any of them does separately of the three, histamine has the greatest stimulatory effect.

2. **Gastric phase.** The greatest volume of gastric secretions is produced during the gastric phase of gastric regulation. The presence of food in the stomach initiates the gastric phase. The primary stimuli are distention of the stomach and the presence of amino acids and peptides in the stomach. Distention of the stomach wall, especially in the body or fundus, results in the stimulation of mechanoreceptors. Action potentials generated by these receptors initiate reflexes that involve both the CNS and enteric reflexes, resulting in secretion of mucus, hydrochloric acid, pepsinogen, intrinsic factor, and gastrin. The presence of partially digested proteins or moderate amounts of alcohol or caffeine in the stomach also stimulates gastrin secretion. When the pH of the stomach contents falls below 2, increased gastric secretion produced by distention of the stomach is blocked. This negative-feedback mechanism limits the secretion of gastric juice. Amino acids and peptides released by the digestive action of pepsin on proteins directly stimulate parietal cells of the stomach to secrete hydrochloric acid. The mechanism by which this response is mediated is not clearly understood. It doesn't involve known neurotransmitters, and, when the pH drops below 2, the response is inhibited. Histamine also stimulates the secretory activity of parietal cells.

3. **Intestinal phase.**

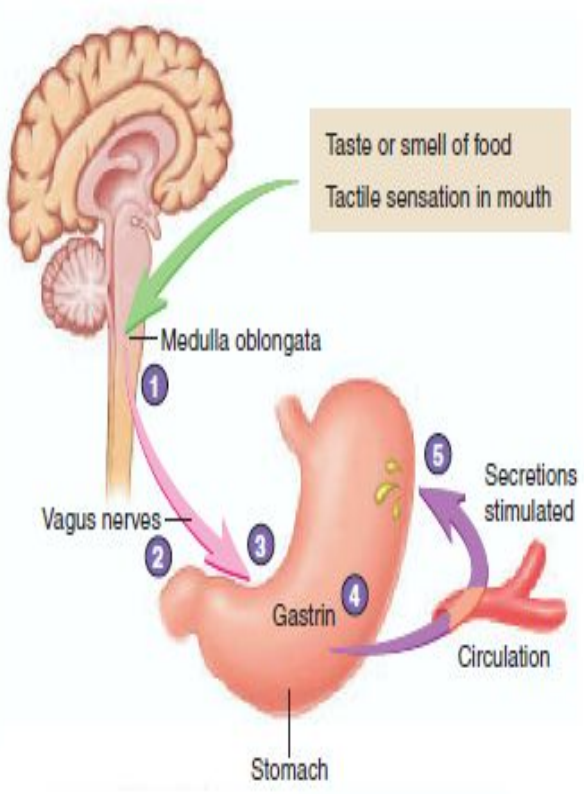
The entrance of acidic stomach contents into the duodenum of the small intestine controls the intestinal phase of gastric regulation. The presence of chyme in the duodenum activates both neural and hormonal mechanisms. When the pH of the chyme entering the duodenum drops to 2 or below, or if the chyme contains fat digestion products, gastric secretions are inhibited. Acidic solutions in the duodenum cause the release of the hormone **secretin** (se-kre ⎣tin) into the circulatory system. Secretin inhibits gastric secretion by inhibiting both parietal and chief cells. Acidic solutions also initiate a local enteric reflex, which inhibits gastric secretions. Fatty acids and certain other lipids in the duodenum and the proximal jejunum initiate the release of two hormones: **gastric inhibitory polypeptide** and **cholecystokinin** (ko ⎣le ⎢-sis-to ⎢-kı ⎣nin). Gastric inhibitory polypeptide strongly inhibits gastric secretion, and cholecystokinin inhibits gastric secretions to a lesser degree. Hypertonic solutions in the duodenum and jejunum also inhibit gastric secretions. The mechanism

appears to involve the secretion of a hormone referred to as **enterogastrone** (en_ter-o ⊦-gas_tro ⊦n), but the actual existence of this hormone has never been established. Inhibition of gastric secretions is also under nervous control. Distention of the duodenal wall, the presence of irritating substances in the duodenum, reduced pH, and hypertonic or hypotonic solutions in the duodenum activate the enterogastric reflex. The **enterogastric reflex** consists of a local reflex and a reflex integrated within the medulla oblongata. It reduces gastric secretions.

Cephalic Phase

1. The taste or smell of food, tactile sensations of food in the mouth, or even thoughts of food stimulate the medulla oblongata (*green arrow*).
2. Parasympathetic action potentials are carried by the vagus nerves to the stomach (*pink arrow*).
3. Preganglionic parasympathetic vagus nerve fibers stimulate postganglionic neurons in the enteric plexus of the stomach.
4. Postganglionic neurons stimulate secretion by parietal and chief cells and stimulate gastrin secretion by endocrine cells.
5. Gastrin is carried through the circulation back to the stomach (*purple arrow*), where it stimulates secretion by parietal and chief cells.

(a)



Gastric Phase

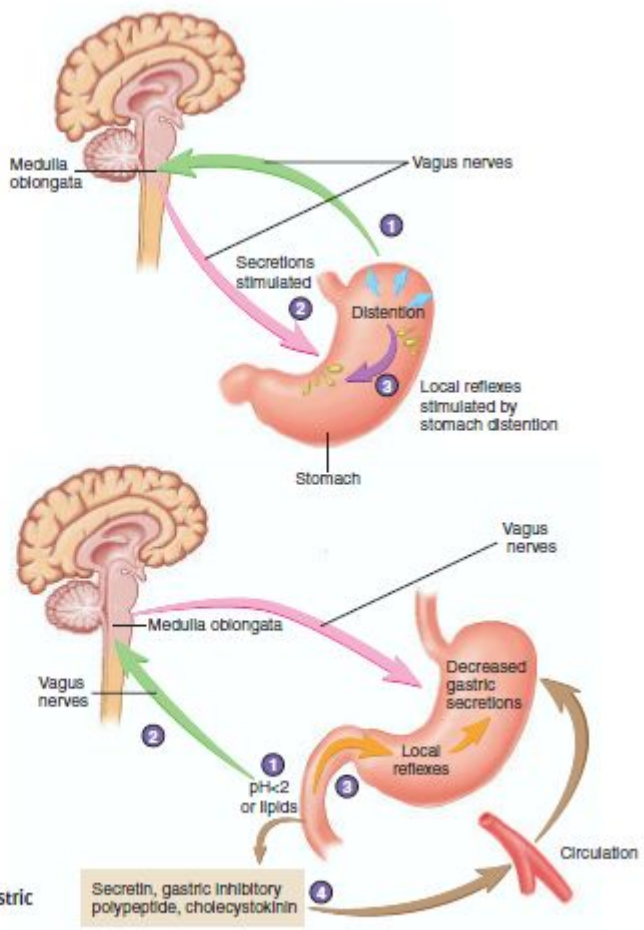
1. Distention of the stomach activates a parasympathetic reflex. Action potentials are carried by the vagus nerves to the medulla oblongata (*green arrow*).
2. The medulla oblongata stimulates stomach secretions (*pink arrow*).
3. Distention of the stomach also activates local reflexes that increase stomach secretions (*purple arrow*).

(b)

Intestinal Phase

1. Chyme in the duodenum with a pH less than 2 or containing fat digestion products (lipids) inhibits gastric secretions by three mechanisms (2–4).
2. Sensory vagal action potentials to the medulla oblongata (*green arrow*) inhibit motor action potentials from the medulla oblongata (*pink arrow*).
3. Local reflexes inhibit gastric secretion (*orange arrows*).
4. Secretin, gastric inhibitory polypeptide, and cholecystokinin produced by the duodenum (*brown arrows*) inhibit gastric secretions in the stomach.

(c)



Process Figure **The Three Phases of Gastric Secretion**

(*a*) Cephalic phase. (*b*) Gastric phase. (*c*) Intestinal phase.