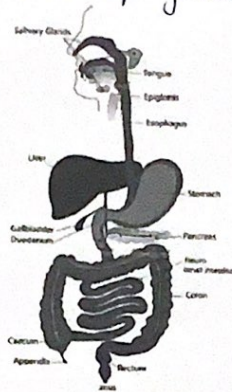




2. GASTRIC MOTILITY & VOMITING.

one of the imp. gastric motility is the Vomiting

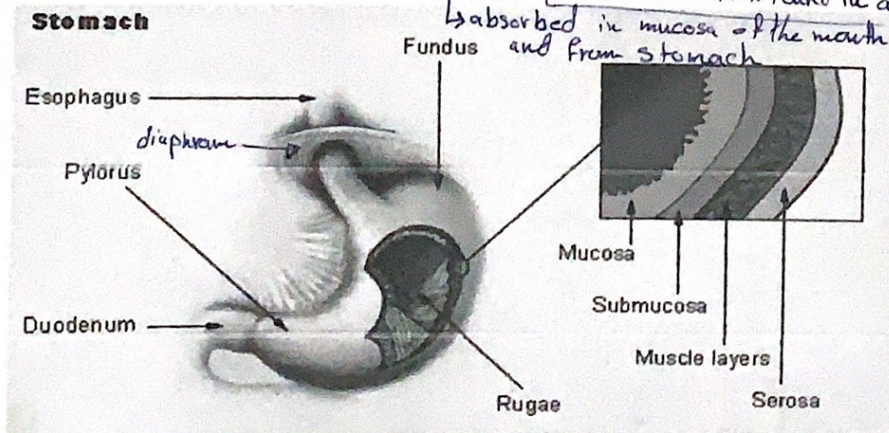


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The stomach

•Function of stomach:

- 1-Storage of food.
- 2-Slow evacuation of meal to allow good digestion and absorption. → it gives the food *enough time to be good digested*
- 3-Partial digestion of proteins and fats. First place to digest Fat and Protein in stomach (by gastrin and pepsin)
- 4-Sterilization of ingested food by high acidity (HCl)
- 5-Secretion of HCl, enzymes,....
- 6-Help defecation by gastro-colic reflex → example: after the baby takes their meal → the defecation will happen
- 7-Absorption of small amounts of water and alcohol. * also this reflex found in adults will happen



Gastric Motility

***Filling and Storage of food in the stomach:** *how? by the stress relaxation by smooth muscles
 The stomach accommodates up to one liter of food without increase of intra-gastric pressure

because :

- a. Plasticity of gastric wall.
- b. Receptive relaxation.

c. Law of Laplace: $P = T/r$ ($\uparrow P \rightarrow \uparrow$ radius with less \uparrow in tension \rightarrow press towards normal)
 $pressure = \frac{2 \text{ tension}}{\text{radius}}$

***Types of movements of the stomach:**

a-Tonic gastric waves : 1 wave / 20sec
 -Regular weak contractions (3 waves/min) which take place in empty stomach, mainly in the fundus to maintain the intra-gastric pressure & mix gastric secretion with food.

b. Receptive relaxation :

- It is a reflex relaxation of the fundus and body to receive the bolus of food.
- Initiated by vagal reflexes (conditioned and unconditioned).
- Also by plasticity of gastric muscles.

c. Peristaltic movement :

-Distension of stomach by food \rightarrow stimulate stretch receptors \rightarrow vago-vagal reflex peristalsis at the middle of stomach and proceeds toward the pyloric antrum with gradual increase in strength leading to:

- Grinding of food to fine particles.
- Emptying of fine particles into the duodenum (propulsive movements).
- Peristalsis in opposite direction from pyloric antrum to fundus (Anti-peristalsis) \rightarrow pyloric mill for mixing of food with gastric secretion.

الحركة الدودية
 بداية التقلص
 يتكون في ال fundus
 (Fundus is not part of it)
 start afferent vagal \rightarrow to the vagal nuclei in medulla \rightarrow efferent vagus
 حركة دفع
 * من عند ال middle of stomach
 يدفع وخالها بجزر ال grinding وال small particle يتزل لل duodenum والباتي
 يروح ال middle of stomach
 يتكسر و small particle وبتن ال عملية هيك

d. hunger contractions: (hunger pain)

-Fasting hypoglycemia → activation of the feeding center in hypothalamus →

•Sends impulse to limbic cortex → hunger sensation.

•Sends impulse to vagal nucleus → hunger strong painful contraction near the fundus

parasympatholike (Atropine injection or vagotomy abolish hunger contraction but not hunger sensation).

-They start slowly, then increase → tetanic contraction for 2 minutes then disappear and reappear in the next feeding time to reach

maximal intensity in 3-4 days then gradually disappear. (May due to ↓ sensitivity of feeding center to hypoglycemia).
after 1 week

-Basic electrical rhythm (gastric slow waves):

•3-5 cycles/min. due to partial depolarization of circular smooth muscle cells in the stomach wall.

•Some lead to spike potential → peristalsis.

•Start at midpoint of greater curvature (pace maker of the stomach).

mcq •Vagal and gastrin → ↑ spike pot. rate. (↑ contraction)

•Sympathetic & secretin → ↓ spike pot. rate.

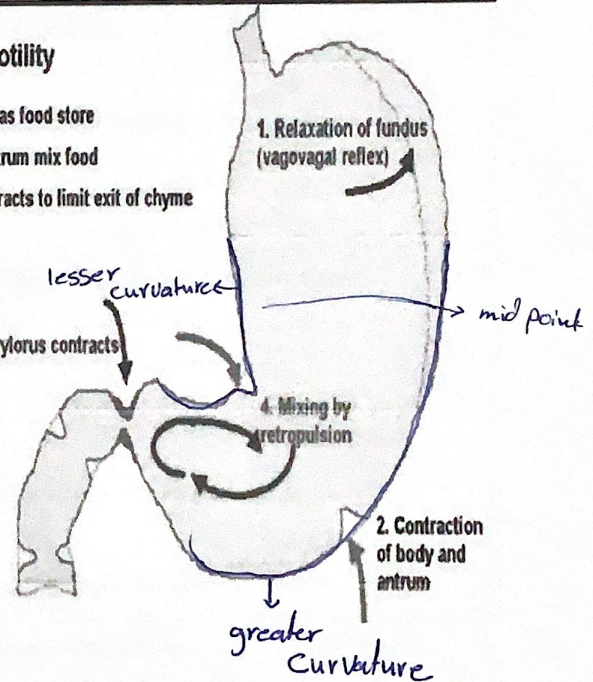
Gastric motility

Fundus acts as food store

Body and antrum mix food

Pylorus contracts to limit exit of chyme

hypoglycemia



• Nervous regulation of gastric motility:

a- Vagal (parasympathetic) : *in fundus*

- Inhibitory purinergic to proximal unit (not blocked by Atropine). → because the neurotransmitter is Purine not Acetylcholine
- Excitatory cholinergic to distal unit → from middle to the end of the stomach

b-Sympathetic: *ACh*

Inhibitory (nor adrenergic) to proximal unit.

c- Myenteric plexus: (as before) short & long reflexes.
↳ the ganglion is inside the wall of stomach

*Factors affecting gastric emptying :

With a mixed meal, the stomach usually empty in about 3 hours through the pyloric pump (50-70 cm. water) which regulate the rate of gastric emptying. The rate of emptying is controlled by:

A. Factors in the stomach:

1. Type of food: carbohydrate is the most rapid. Then proteins followed by fats.
2. Consistency of food: liquids more rapid which depends on type of food, degree of mastication and the strength of gastric peristalsis.
3. Volume of food:
 - Moderate volume of chyme → ↑ emptying via vago-vagal reflex and release of gastrin hormone.
 - Large volume → over distension → ↓ emptying.

↳ the pH is alkaline

B. Factors in the duodenum: the same role of the duodenum in the control of gastric secretion.

C. Emotional factors: *↳ the signals that released by duodenum to control the gastric secretion*

1. Pain: visceral and somatic pain → reflex inhibition of gastric emptying.
2. Depression & sudden fear → reflex sympathetic inhibition. → no digestion, no secretion ...
3. Anxiety & anger → reflex parasympathetic stimulation of emptying.

Vomiting

*Definition:

↳ some time from duodenum (appear as a yellow vomiting due to bile secretion)

-It is the expulsion of gastric contents through the esophagus, pharynx and mouth.

-It is a complex act controlled by vomiting center (in the medulla oblongata) and mediated by cranial nerves

important V, VII, IX, X & XII and spinal nerves to diaphragm and abdominal muscles.

-It is preceded by nausea, salivation and increase respiration.

*Centers:

a. Vomiting center : in the medulla oblongata.

b. Chemo receptor trigger zone (CTZ) : *zone*

-In close to vomiting center in M.O in the wall of fourth ventricle.

-Its stimulation by emetic drugs, motion sickness or metabolic causes → stimulation of vomiting center. (its lesion leads to loss of this reflex)

*Causes of vomiting: 1- Central vomiting:

Direct stimulation of CTZ by drugs as morphine, alcohol drinking, diabetic ketoacidosis, renal failure or early pregnancy.

the unexcreted toxin with stimulate CTZ → lead to excitation to CTZ → Vomiting

↳ inborn 2- Reflex vomiting:

Stimuli: Unconditioned:

- Irritation of back of tongue.
- Severe visceral pain (Renal colic, coronary thrombosis).

- Irritation of gastric mucosal center *↳ peptic ulcer*
- Irritation of semicircular canal *↳ when the CTZ is activated → stimulation of vomiting center*

Conditioned:

(cortical excitation of vomiting) Visual, olfactory and psychic (as morning sickness of pregnancy.)

Afferents : according to site of stimuli.

Center :

- Direct on vomiting center.
- Some to CTZ as semicircular canal irritation and psychic.

Efferents :

- Via cranial nerves V, VII, IX, X, XII.
- Phrenic nerve to diaphragm.
- Spinal nerves to abdominal muscles.

→ passive
Response : → vomiting.

Mechanism of vomiting :

1-Nausea: with salivation, ↑ H.R, sweating, stomach wall is relaxed, and antiperistalsis may occur in duodenum.

2-Retching: intermittent contraction of diaphragm and abdominal muscles against closed L.E.S, glottis, and diaphragmatic opening is also contracted.

3- Gastric evacuation :

- Strong contraction at the incisura separating the body from the pylorus.
- The cardiac sphincter relaxes and the stomach wall is completely relaxed (passive stomach).
- Powerful contraction of the diaphragm, abdominal muscle and pelvic floor muscle → ↑ intra abdominal pressure → squeezing the relaxed stomach and expulsion its contents to the mouth (anti peristalsis may occur in oesophagus).
- During vomiting the soft palate elevated, closure of glottis and inhibition of respiration to prevent the vomitus to pass to respiratory passages (as in swallowing).
- When the stomach is empty, antiperistalsis waves may drive the intestinal contents into the stomach (as bile juice).

N.B : in denervated stomach vomiting may occur by central stimulation of the CTZ or reflexly from oropharynx.

N.B : Effect and complications of vomiting :

a-Dehydration (loss of secretion).

b-Alkalaemia : due to loss acid and the resynthesis of acid is associated with ↑ alkaline tide in plasma.

c-Alkalaemia → ↓ ionized Ca^{+2} → tetany. Convulsions

d-Potassium loss.(hypokalaemia)

عمل HCL يتكونه المعدة بواسطة
اكتساب $NaHCO_3$ في الدم في Alkalinity في الدم