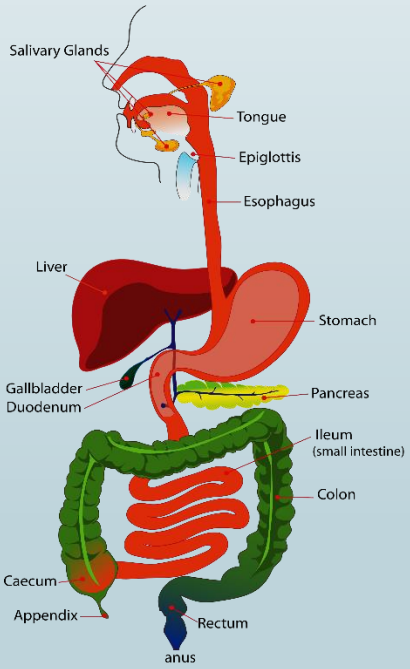
2. GASTRIC MOTILITY & VOMITING.



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The stomach

•**Function of stomach:**

1-**Storage** of food.

2-Slow evacuation of meal to allow good digestion and absorption.

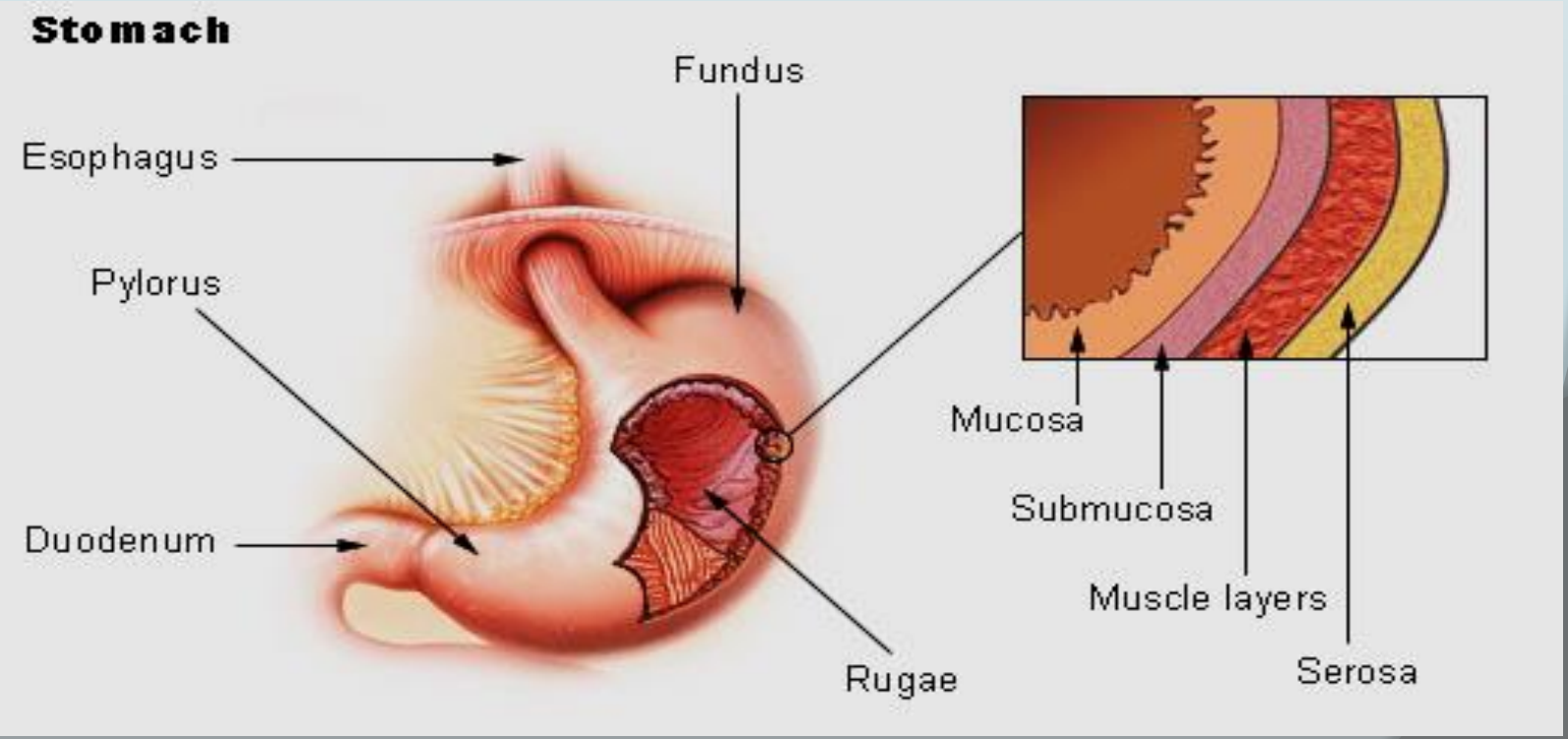
3-Partial digestion of proteins and fats.

4-**Sterilization** of ingested food by high acidity.

5-Secreation of **Hcl, enzymes**,….

6-Help **defecation** by gastro-colic reflex.

7-**Absorption** of small amounts of water and alcohol.



Gastric Motility

***Filling and Storage of food in the stomach:**

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 (↑ P →↑ radius with less ↑ in tension →↓ pressure towards normal).

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

A-Tonic gastric waves :

-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 → stimulate **stretch receptors** → 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-peristalisis**) → **pyloric mill** for mixing of food with gastric secretion.

D. hunger contractions :

-**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 (**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).

-Basic electrical rhythm (gastric slow waves) :

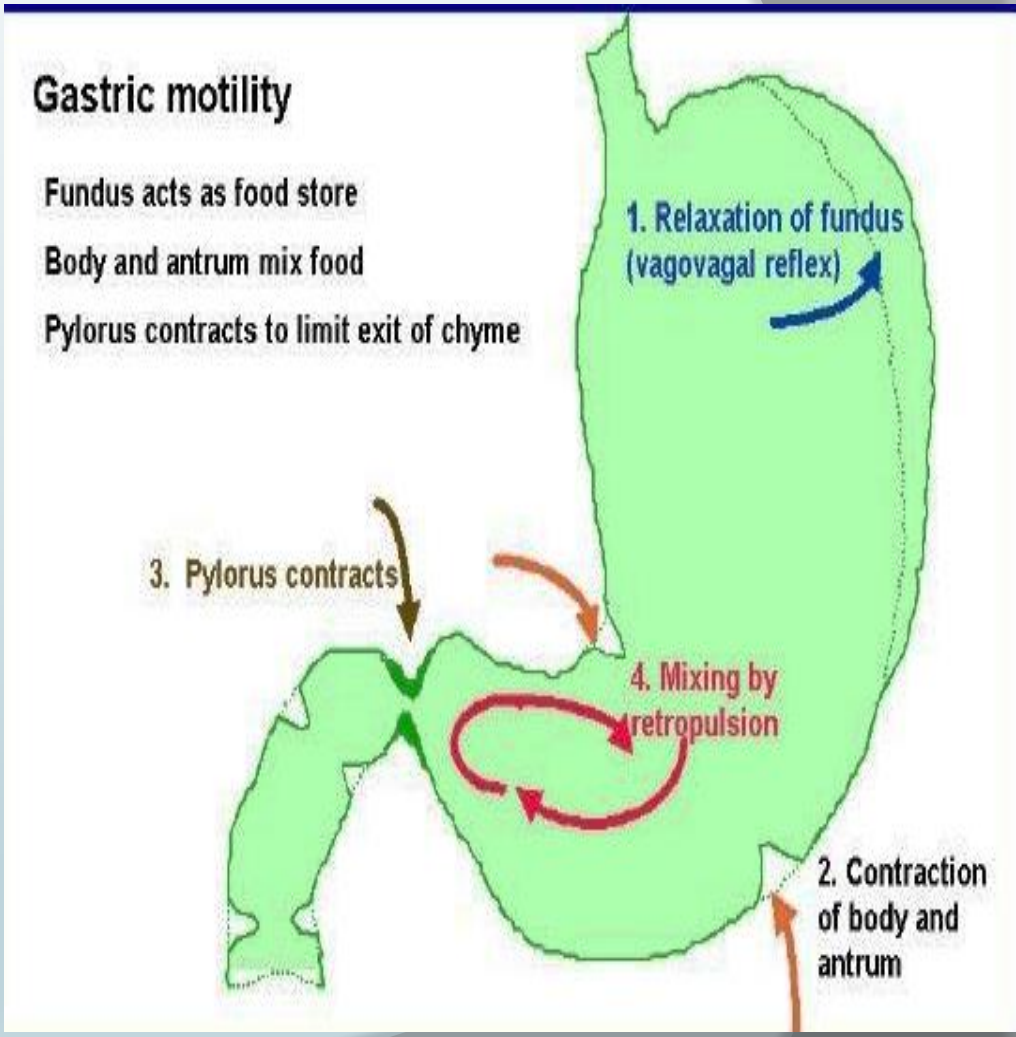
•**3-5 cycles/min**. due to partial depolarization of circular smooth muscle cells in the stomach wall.

•Some lead to spike potential → peristalsis.

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

•Vagal and gastrin H. →↑ spike pot. rate.

•Sympathetic & secretin H. →↓ spike pot. rate.



- **Nervous regulation of gastric motility:**

a- Vagal (parasympthetic) :
- Inhibitory purinergic to proximal unit (not blocked by Atropine).
- Excitatory cholinergic to distal unit.

b-Sympathetic:
Inhibitory (nor adrenergic) to proximal unit.

c- Myenteric plexus: short & long reflexes.

***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.

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

C. Emotional factors:

1.Pain: visceral and somatic pain→ reflex inhibition of gastric emptying.

2.Depression & sudden fear → reflex sympathetic inhibition.

3.Anxiety & anger → reflex parasympathetic stimulation of emptying.

Vomiting

***Definition:**

-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 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 tone (CTZ)** :

-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.

2- Reflex vomiting:

Stimuli: **Unconditioned:**

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

Conditioned:

(Cortical excitation of vomiting) Visual, olfactory and psychic .)

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, V11, 1X, X, X11.
- Phrenic nerve to diaphragm.
- Spinal nerves to abdominal muscles.

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 **reflexely** from oropharynx.

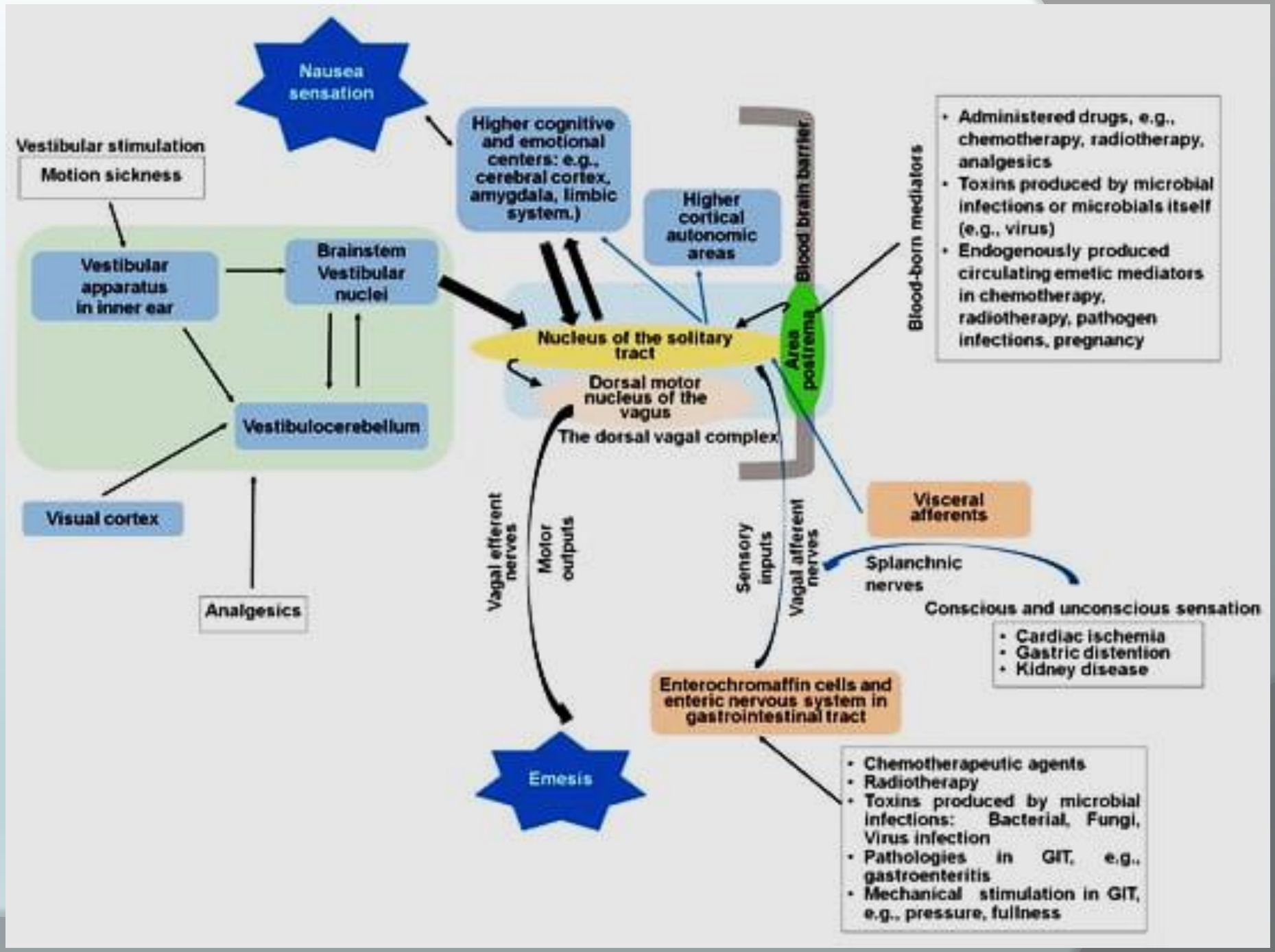
N.B : Effect and complications of vomiting :

a-**Dehydration** (loss of secretion).

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

c-Alkalaemia →↓ ionized Ca+2 → **tetany**.

d-Potassium loss**.(hypokalaemia**)


Nausea sensation
Vestibular stimulation
Motion sickness
Higher cognitive and emotional centers: e.g., cerebral cortex, amygdala, limbic system.)
Higher cortical autonomic areas
Blood brain barrier
Blood-born mediators
Administered drugs, e.g., chemotherapy, radiotherapy, analgesics
Toxins produced by microbial infections or microbials itself (e.g., virus)
Endogenously produced circulating emetic mediators in chemotherapy, radiotherapy, pathogen infections, pregnancy
Vestibular apparatus in inner ear
Brainstem Vestibular nuclei
Nucleus of the solitary tract
Area postrema
Dorsal motor nucleus of the vagus
The dorsal vagal complex
Vestibulocerebellum
Visual cortex
Analgesics
Vagal efferent nerves
Motor outputs
Sensory inputs
Vagal afferent nerves
Visceral afferents
Splanchnic nerves
Conscious and unconscious sensation
Cardiac ischemia
Gastric distention
Kidney disease
Enterochromaffin cells and enteric nervous system in gastrointestinal tract
Emesis
Chemotherapeutic agents
Radiotherapy
Toxins produced by microbial infections: Bacterial, Fungi, Virus infection
Pathologies in GIT, e.g., gastroenteritis
Mechanical stimulation in GIT, e.g., pressure, fullness

Thank You