Smooth muscle contraction

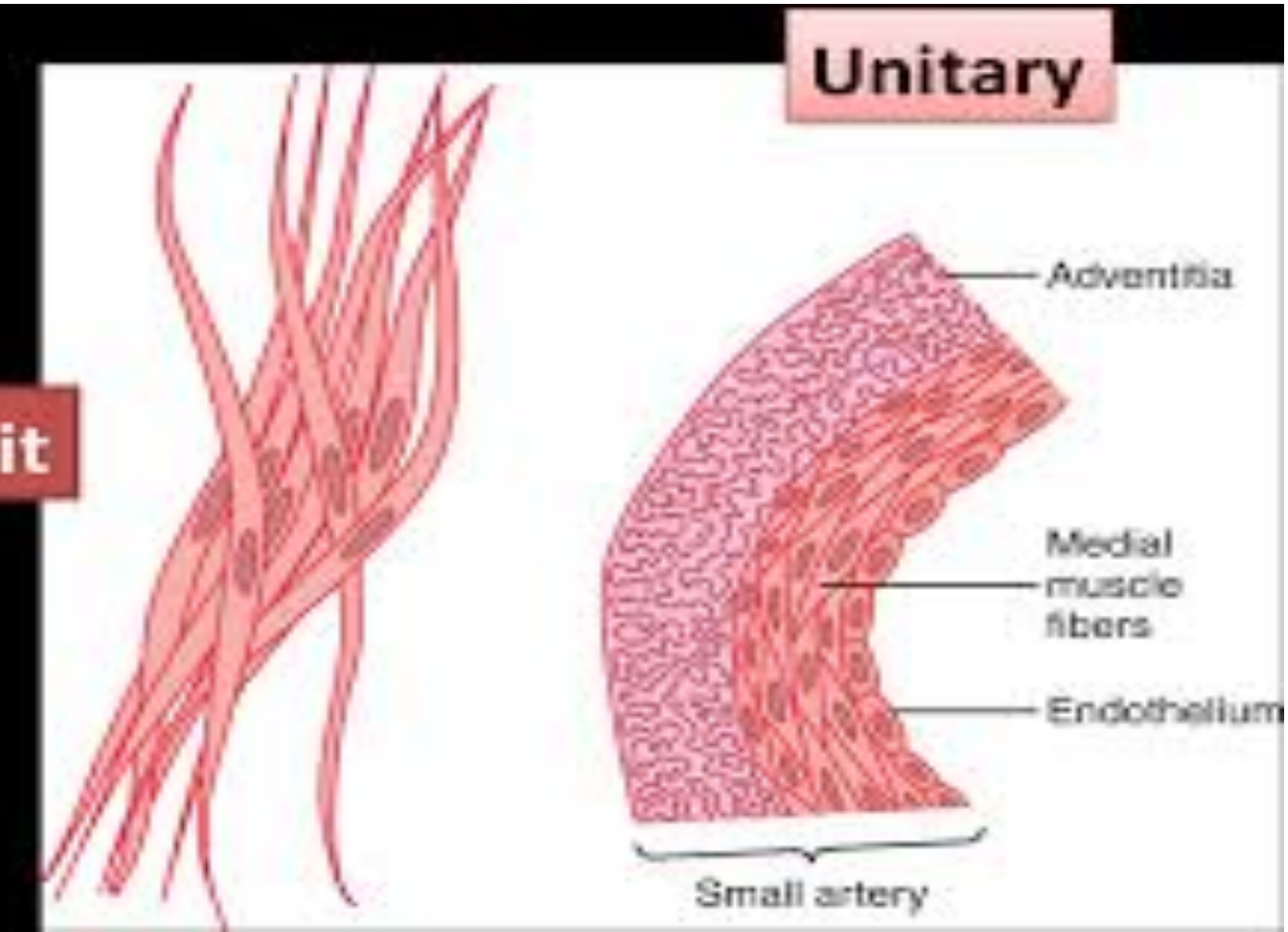
GI motility

All the figures in this ppt are taken from guyton, ganong, tortora and google images

Movements of GIT

- Movements
- reflexes

smooth muscle

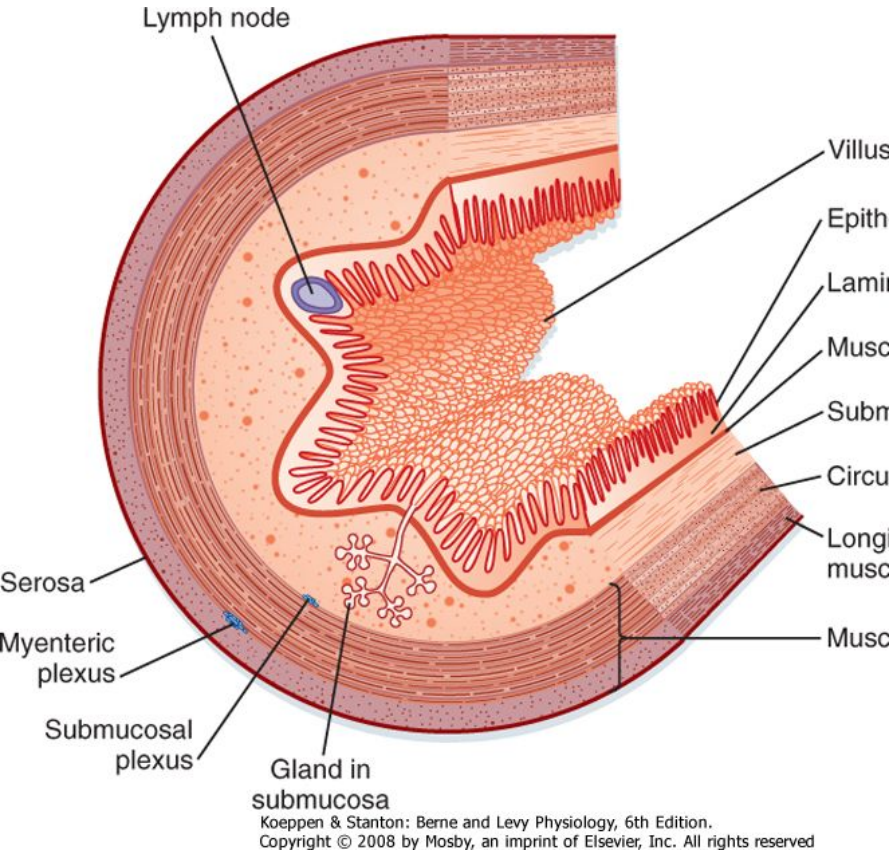


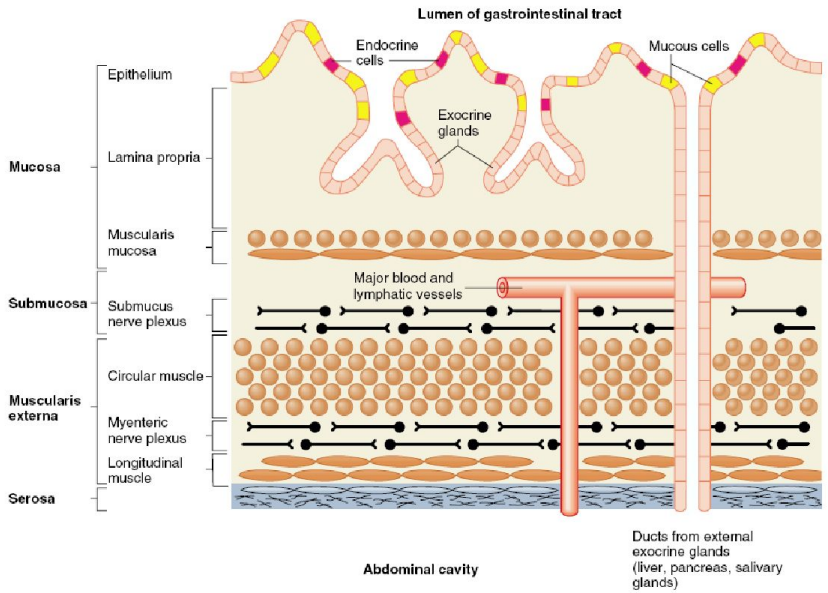
- Single unit or unitary smooth muscle

2 layers of smooth muscle in GIT

Muscularis externa
and

Muscularis interna or muscularis mucosae





Intensity- varies between 5-15mv

Frequency-(3-12/min)

Stomach - about 3

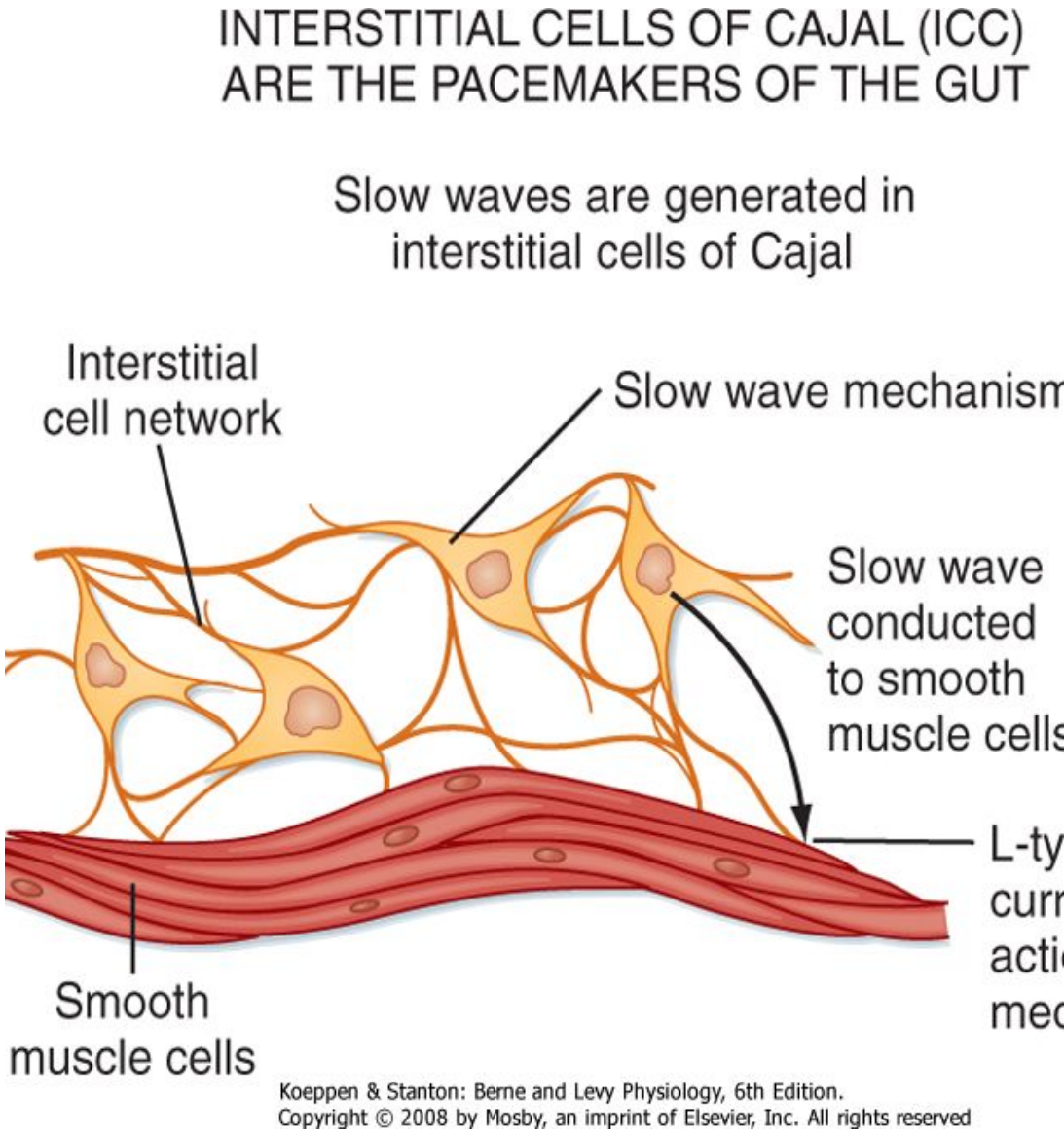
Duodenum- about 12

Terminal ileum -8-9

So rhythm of contraction is same.

ICC are specialised cells interposed between smooth muscle layers

Basic electrical rhythm (BER)



2 types of potentials BER or slow wave & spike potentials

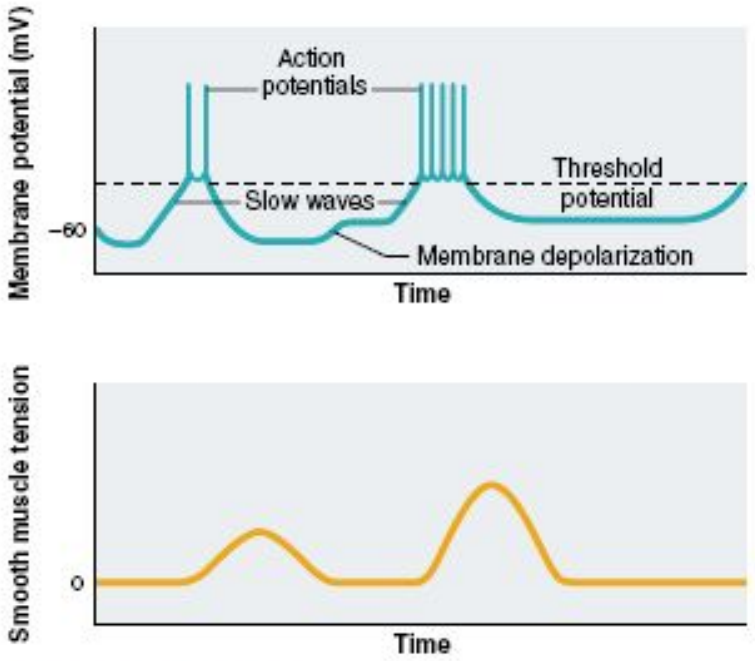
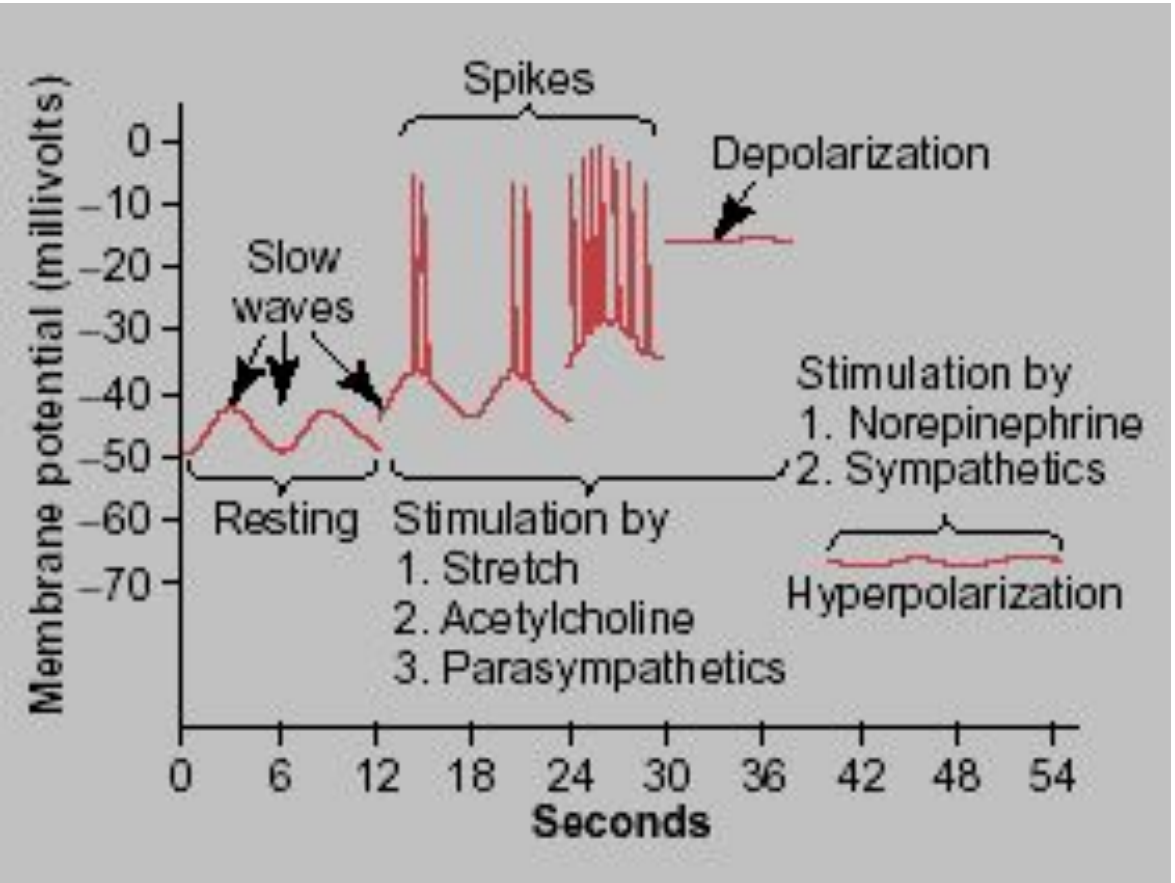


FIGURE 17–23
Slow wave oscillations in the membrane potential of gastric smooth-muscle fibers trigger bursts of action potentials when threshold potential is reached at the wave peak. Membrane depolarization brings the slow wave closer to threshold, increasing the action-potential frequency and thus the force of smooth-muscle contraction.

- ICC has unique ion channels that open periodically & so they undergo cyclical changes in membrane potential.but can not cause ,uscle contraction.
- They excite appearance of spike potentials which actually can cause muscle contraction

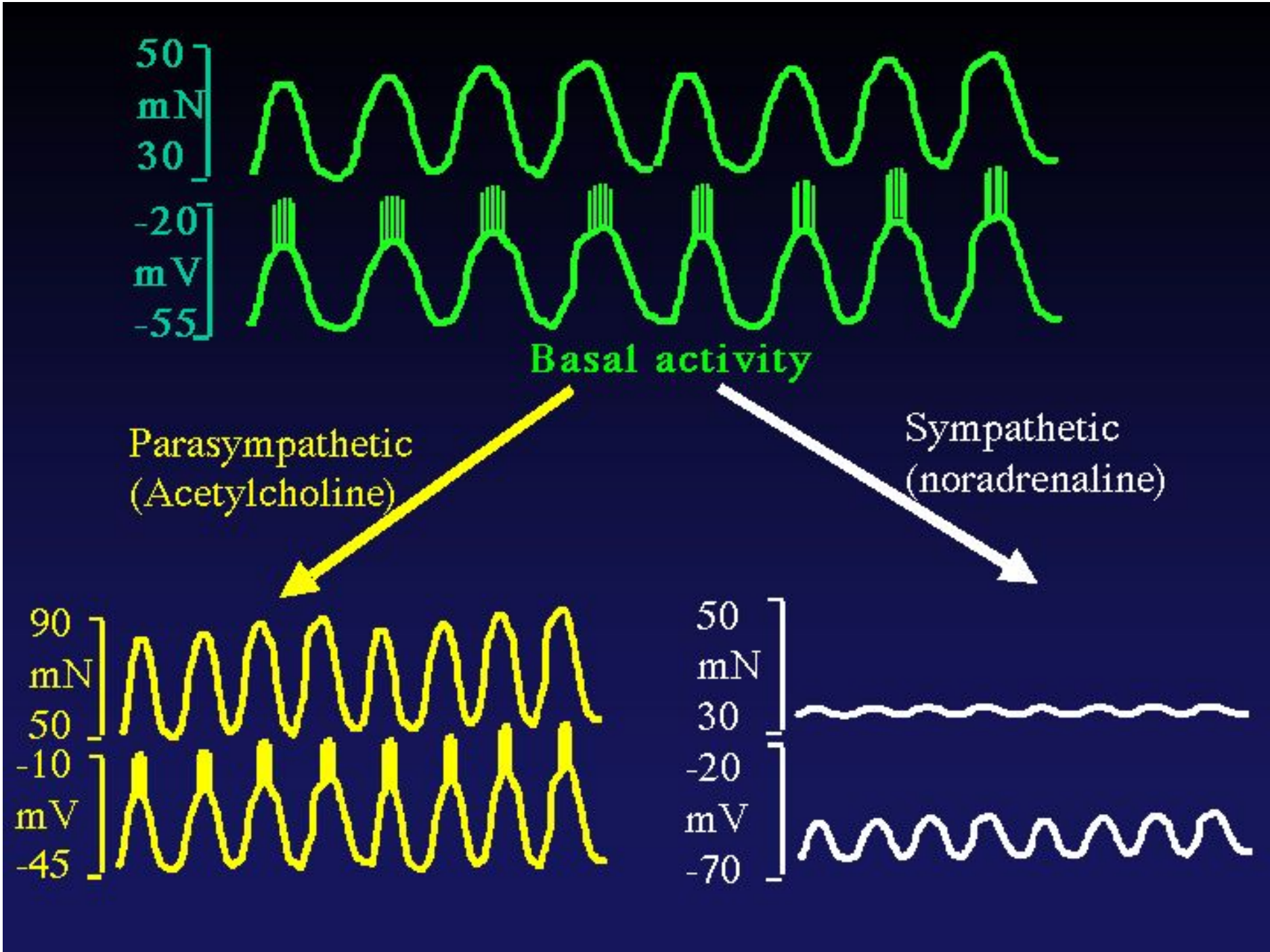
2 Variations in RMP(not fixed)

Base line raised and
Base line depressed



RMP – averages about-56 mv (undulating RMP) when depolarised (less negative) then it is more excitable &

when hyperpolarised the fibers become less excitable.


50
mN
30
-20
mV
-55
Basal activity
Parasympathetic
(Acetylcholine)
Sympathetic
(noradrenaline)
90
mN
50
-10
mV
-45
50
mN
30
-20
mV
-70

2 types of contractions

Phasic or rythmic

Intermittent contraction

Contractions last for seconds

At oesophagus, gastric antrum, small & large bowel

- **Tonic**

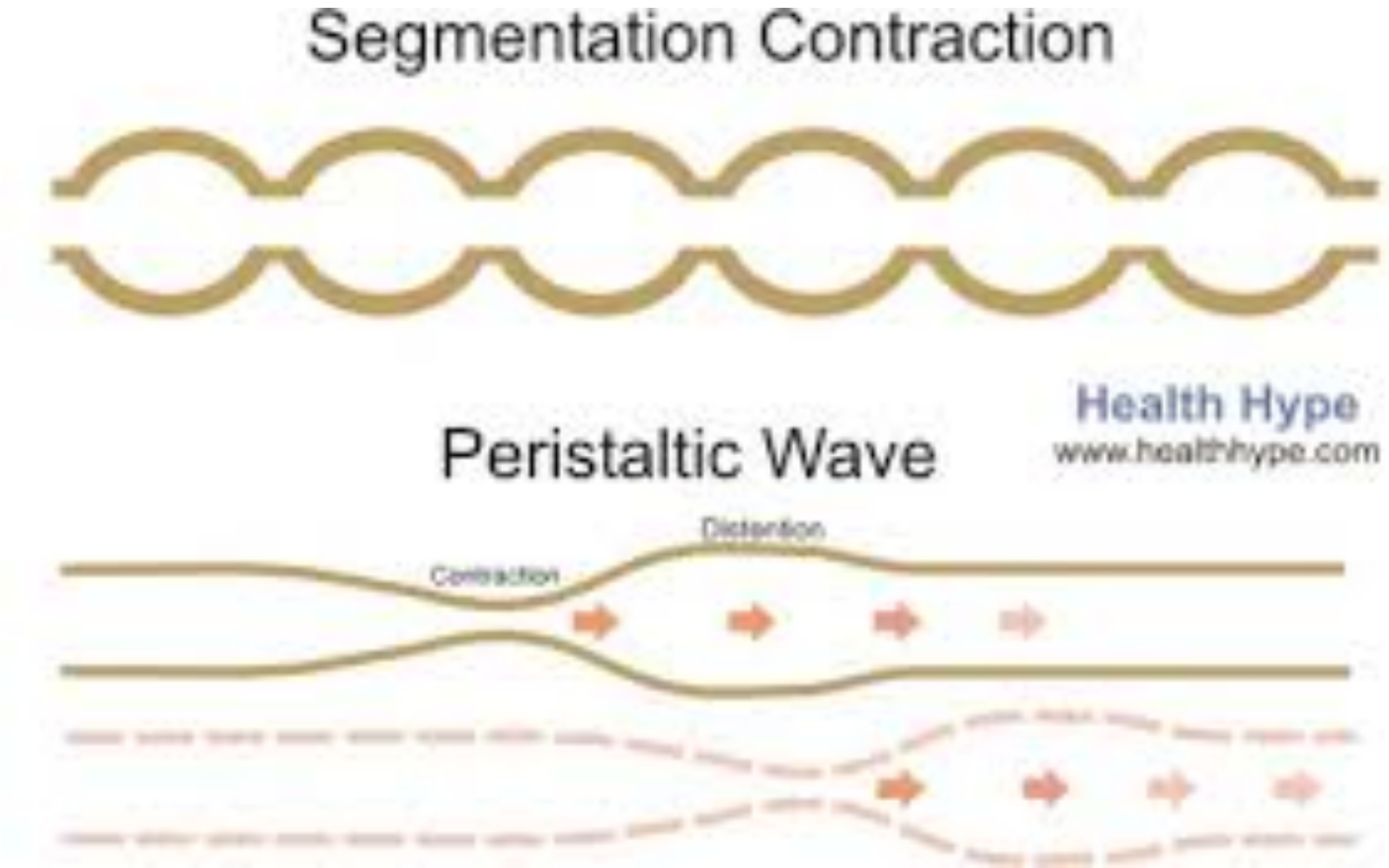
  Prolonged persistent

  Contractions last for mins to hours

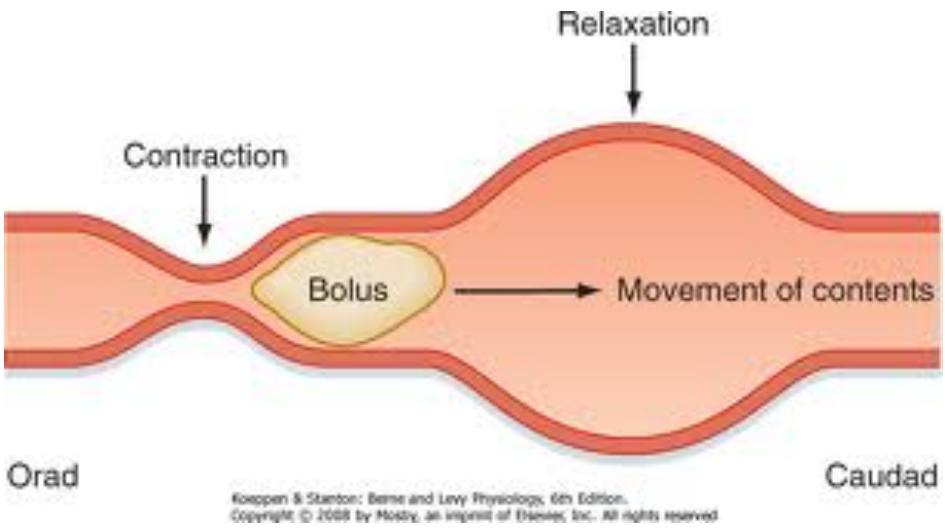
At orad stomach, LES, ileocaecal, internal anal sphincter

2 types of functional movement

Segmental or mixing **and** **Peristalsis or propulsive**



Law of gut



- Movement of food is from orad to caudad in normal circumstances.
- Myenteric reflex or peristaltic reflex.

2 types of regulation

Neural

Autonomic & somatic

ANS:-Parasympathetic & sympatheic

Parasymathetic-stimulatory neurotransmitter

Inhibitary neurotransmitter

Hormonal(enterochromaffin cells)

Gastrin

Secretin

CCK

Types of gastric motility

- Motility of the empty stomach, which includes:
- migrating motor complex
- hunger contractions
- Gastric motility related to meal:
- Receptive relaxation
- Mixing peristaltic waves
- Retropulsion
- Gastric emptying

Motility of empty stomach

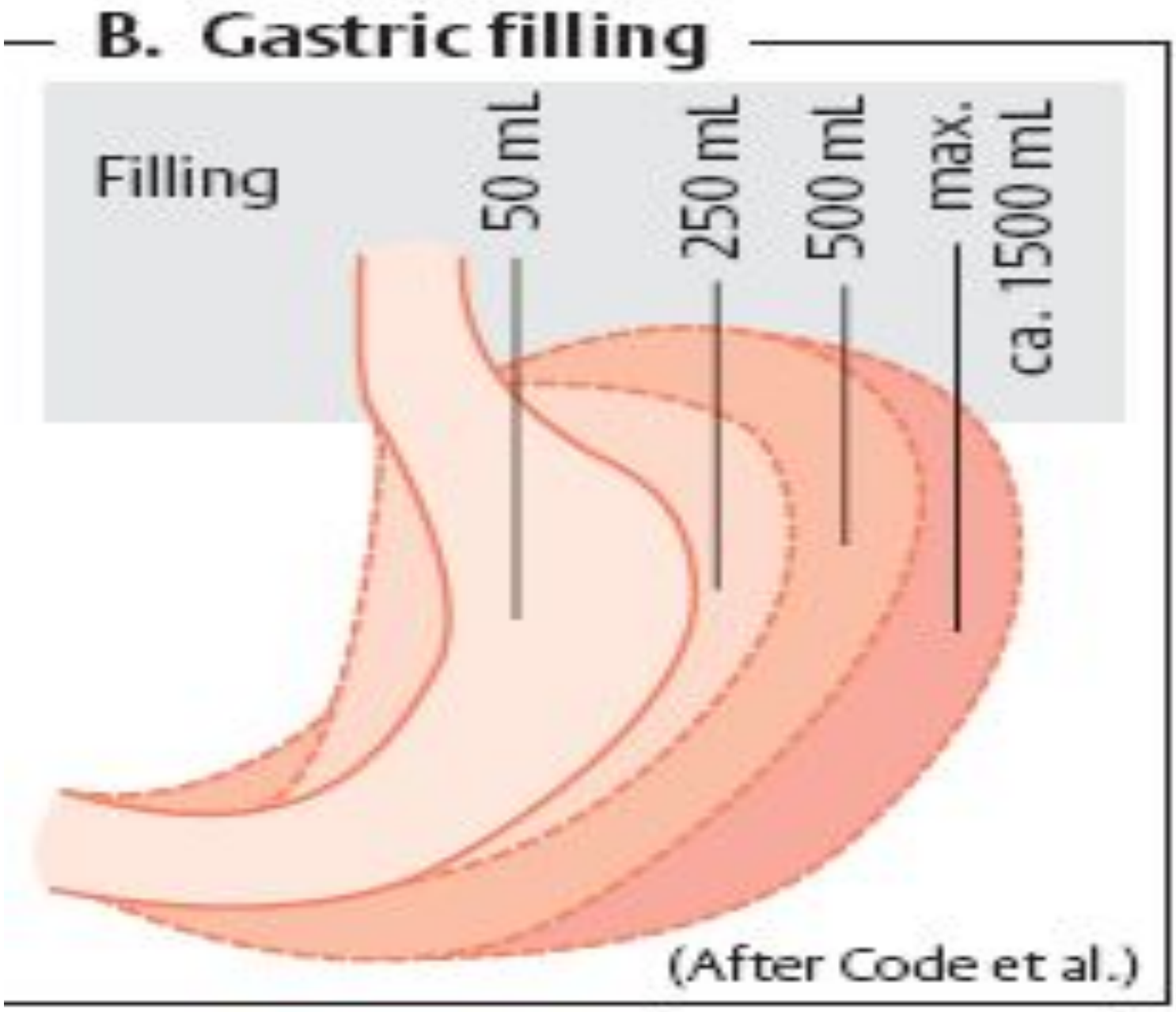
- Migrating motor complex:
- Peristaltic wave that begins in the stomach and travels through the entire GIT during Interdigestive phase.
- It removes any food remaining in the stomach and small intestine during Interdigestive period
- Hormone motilin- secreted in small intestine, increases the strength of MMC.
- The MMC are abolished immediately after the entry of the food in the stomach.

Hunger contractions:

- Mild peristaltic contractions occur in empty stomach(if for several hours), which over period of time increases in intensity and are called hunger contractions.
- Intense in young healthy person & if blood glucose less
- 12-24 hrs after last meal in starvation. greatest in 3-4 days & gradually weakens in succeeding days
- When succesive contractions fuse to cause tetanic contractions lasting for 2-3 min, produces mild pain(hunger pangs) in gastric pit. These are associated with sensation of hunger.
- Most intense in young healthy individuals (as GI tone is high).Increased by low blood glucose

Gastric motility related to meal

- Receptive relaxation and accommodation:
- The passage of each bolus of food stimulates the stretch receptors of the orad/cardia region and produces relaxation.
- As the organ distends, **lesser curvature** remains nearly constant in position and contour whereas the **greater curvature** of the body and fundus is increase markedly in length.
- So when the food enters the stomach and fills it, the smooth muscles increases in length so that between peristaltic contractions the intragastric pressure remains nearly constant.


B. Gastric filling
Filling
50 mL
250 mL
500 mL
max.
ca. 1500 mL
(After Code et al.)

Mixing movement

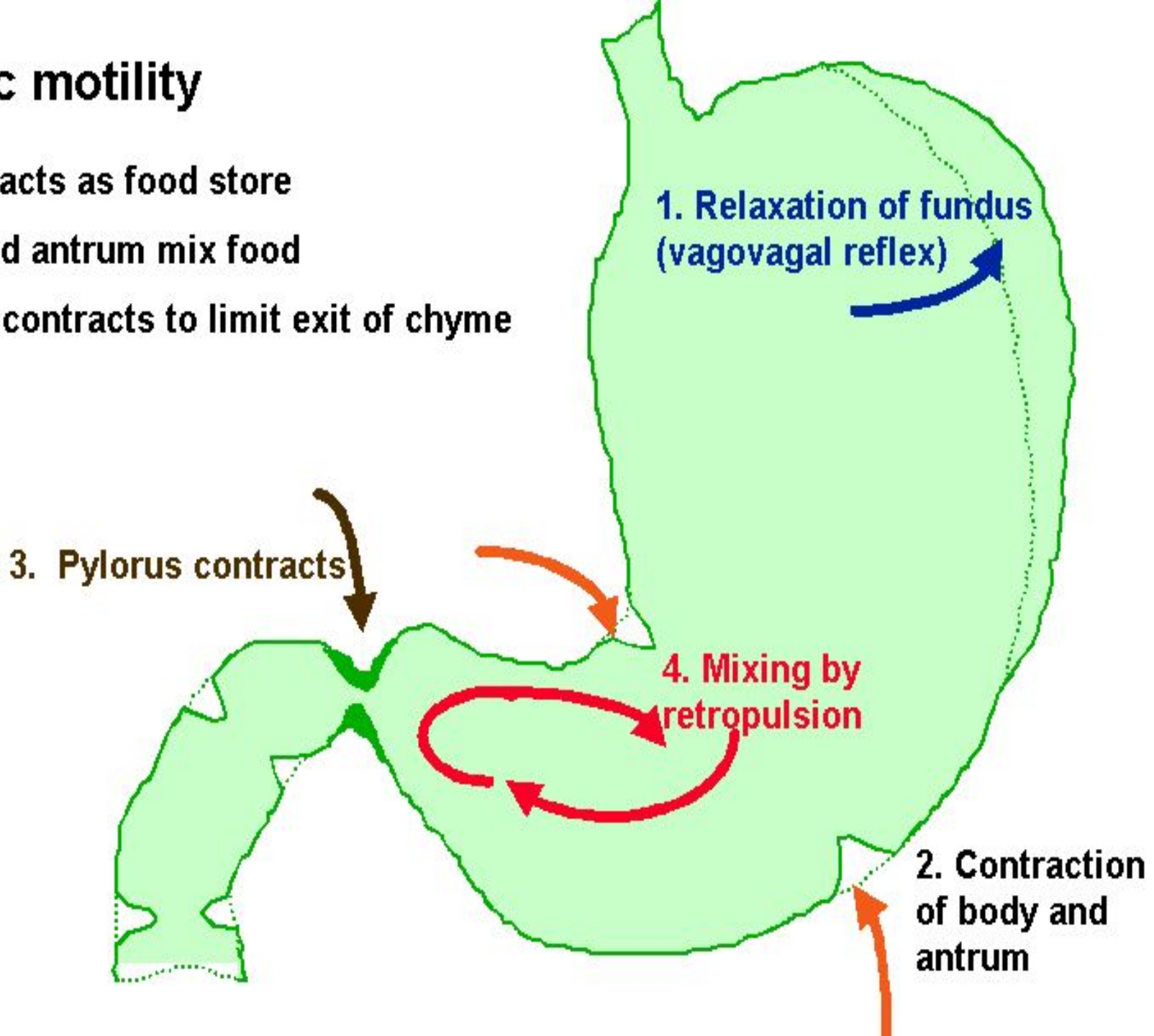
- Mixing waves or constrictor ring(peristalsis):-initiated every 15-20secs ,weak in body but becomes stronger towards pylorus
- More the stomach is emptied ,it starts from more orad part
- Moving constrictor ring, with upstream squeezing of chyme in center, due to closed pylorus is called retropulsion

Gastric motility

Fundus acts as food store

Body and antrum mix food

Pylorus contracts to limit exit of chyme



Gastric emptying

Regulation :-controlled by signals from stomach & duodenum (more stronger)

- **Gastric factors**:-secretion of highly acidic gastric juice
- Gastrin
- Promotes Gastric emptying

Food volume in stomach:-stretch increases activity of pyloric pump & inhibit pylorus

Gastrin:-digestive products of meat release gastrin

- **Type of food ingested**:

  Carbohydrate rich food- rapid emptying

  Protein rich food - slow emptying

  Fat rich food- slowest emptying

Gastric emptying

- **Duodenal factors**:-
- Inhibit gastric emptying(either slow or stop)
- Directly from the duodenum to the stomach through ENS
- Through extrinsic nerves:-prevertebral sympathetic ganglia & back through inhibitory sympathetic fibers to stomach
- Vagus -brain stem(NTS)-Vagus-stomach
- They inhibit pyloric pump propulsive contractions & increase tone of sphincter

Enterogastric reflex

- Activated in 30 secs
- Degree of distension of duodenum
- Presence of irritation of duodenal mucosa
- Degree of acidity of duodenal chyme
- Degree of osmolality of chyme
- Presence of certain breakdown products of proteins & also fat

GI motility controlled by both humoral and neural mechanisms

1. Extrinsic nervous system

Parasymp = acetylcholine release = increased contraction

2. Intrinsic nervous system

Receptors in GI tract/stretch = Ach, SP release = increased contraction

3. Hormones

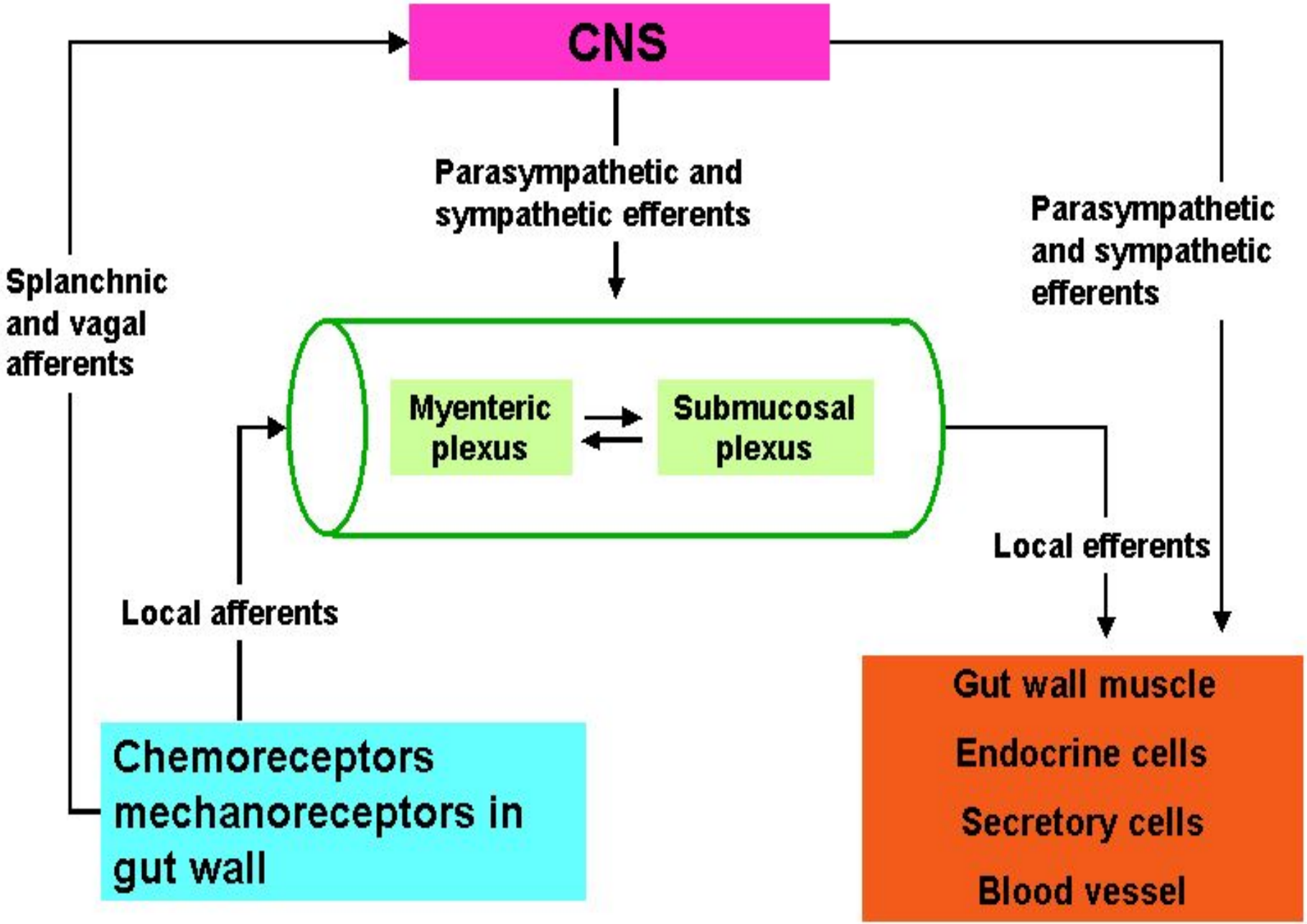
only hormones known to have physiological effects on motility are

motilin = increased gastric and intestinal motility
cholecystokinin = decreased gastric emptying

Control of gastric motility

Vagovagal reflex -	fundal relaxation
Myenteric plexus -	slow waves - contractions
Parasympathetic -	inc contraction force and freq
Sympathetic -	dec contraction force and freq

Reflex control of gut activity



Clinical problems with motility

Gastric emptying

too slow = gastric carcinoma or ulceration (vagotomy)

Results in nausea and vomiting, diarhoea, cramps.

Patients seek help for difficulty swallowing (oesophageal scarring) or dental erosion.

too fast = usually found in patients with duodenal ulcer

Don't know if cause or effect - overwhelms protective defences of duodenum.

Delayed emptying

- Delayed emptying -diabetes mellitus ,Disorders of vagus
- Surgical Vagotomy-Rapid emptying – liquids, delayed emptying- solids vagotomy -impairs adaptive relaxation and increases contractile tone in reservoir
- Loss of propulsive motility after Vagotomy **Gastroparesis.** **Pyloroplasty -** pylorus is enlarged surgically to compensate for Postvagotomy gastroparesis
- **Idiopathic gastric stasis- cause is not known**
- **Hypertrophic pyloric stenosis-** impedes gastric emptying

  Thickening of the muscle of the pyloric canal with loss of enteric neurons.
- Absence of inhibitory neurons and failure of relaxation of the circular muscles- results in obstructive stenosis

Rapid emptying

- Rapid emptying-occurs in patients with Vagotomy and gastric antrectomy for the treatment of peptic ulcer diseases. these individuals have rapid emptying of solids and liquids. these effects are referred to as dumping syndrome- dumping large osmotic loads into the proximal small intestine
- so the patient with stomach removed must eat small large meals.
- If he take large meal, In absence of controlled emptying by stomach large meal enters into the intestine producing hypertonic solution. this hypertonic solution can cause large amount of water to enter from blood to intestine ,lowering blood volume and produce circulatory complications
- Large distension trigger vomiting in these patients.- **dumping syndrome**

THANK YOU