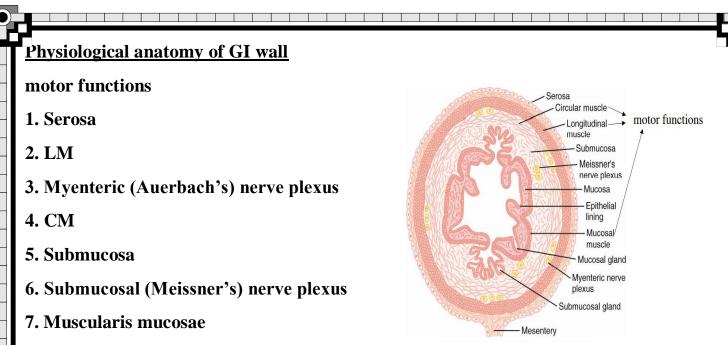


Function:

- Provides body with water, electrolytes, vitamins, nutrients.
- Functions through:
- (1) Movement of food through GIS
- (2) Secretion of digestive juices
- (3) Absorption of water, electrolytes, vitamins, & digestive products to circulation

Control of GI functions by local(by the GIT itself), nervous (sympathetic and parasympathetic system), and hormonal system



8. Mucosa

9.Epithelial lining

As we can see In the figure this is a cross section from a gut ,there is a Connective tissue covering outside(serosa),((so GIT is formed from 4 layers from inside to outside : mucosa, submucosa, muscular layer, serosa)) the muscle is the functional unit of GIT ((motility)) so we have 2 types of muscle: longitudinal muscle along the tract itself and circular muscle.

the 2 systems innervating the GIT only are : myenteric plexus((located between LM and CM))and submucosa nerve plexus ((within submucosa))

GI SM

Arranged in bundles of fibers separated by loose CT

• Electrically connected with GJ ((gap junctions in order to propagate AP)) (low-resistance movement of ions)

• Electrical signals initiating muscle contractions travel more rapidly along length of the bundle than sideways

• GI SM functions as a syncytium --- AP elicited anywhere within muscle travels in all

directions in muscle

• Due to connection between LM and CM layers, excitation of one of these layers often

excites the other

* syncytium أي كوحدة واحدة

*وأي نشاط كهربائي يتحرك distally and longitudinal اكثر من ان يتحرك على الاطراف

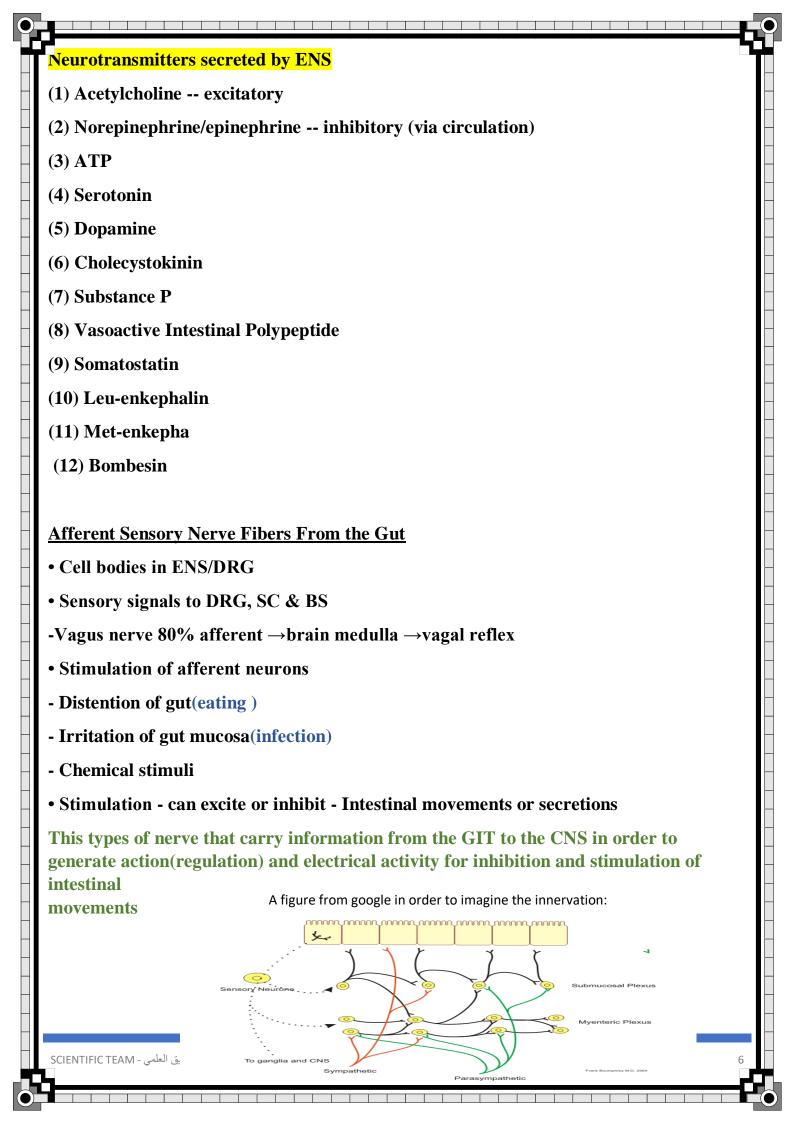
*So any excitation of one type of muscle ((LM)) will lead to excitation of another muscle

Electrical Activity of GI SM • SM is excited by continual slow, intrinsic electrical activity along membranes of muscle fibers. (has it's own electrical activity doesn't need any stimulation)) ((the nervous system is going to regulate such an activity)) • Normal RMP in SM of gut is - 50-60 mV (Avg -56 mV) Voltage of RMP of SM can change to different levels Spikes (millivolts 0 --10 -Depolarization **RMP** potential can change resulting in Slow -20-different activities ** Stimulation by 1. Norepinephrine 2. Sympathetics • Types of electrical waves: -60 -Resting Stimulation by 1. Stretch Hyperpolarization 2. Acetylcholine 3. Parasympathetics Slow waves 12 18 24 30 36 42 48 54 Seconds 6 -Rhythmical changes in MP, not AP Not AP: means that they can't generate actual contraction (so it is not depolarization), except in stomach they can cause some contractions -Slow changes in RMP. -5-15 mV intensity, 3-12/min freq. -Cause: interactions among the SM cells & interstitial cells of Cajal (electrical pacemakers for SM cells) ---cyclic changes in MP due to activity ion channels. Such waves are generated by interactions between smooth muscles and pacemaker results in movement of ions -Don't cause GI muscle contraction (except stomach) -- stimulates spike potentials -muscle contraction. So if the RMP reach threshold ((become less negative)) they will have another type of waves called spikes Note : these types of waves occur spontaneously • Spikes: • When slow waves reach threshold (-40 mV) -- spike P – depolarization- Ca2+ entery -contraction -True AP. So the spike waves are important in AP (actual contraction in the gut itself) and it is generated by slow waves الفريق العلمي - SCIENTIFIC TEAM

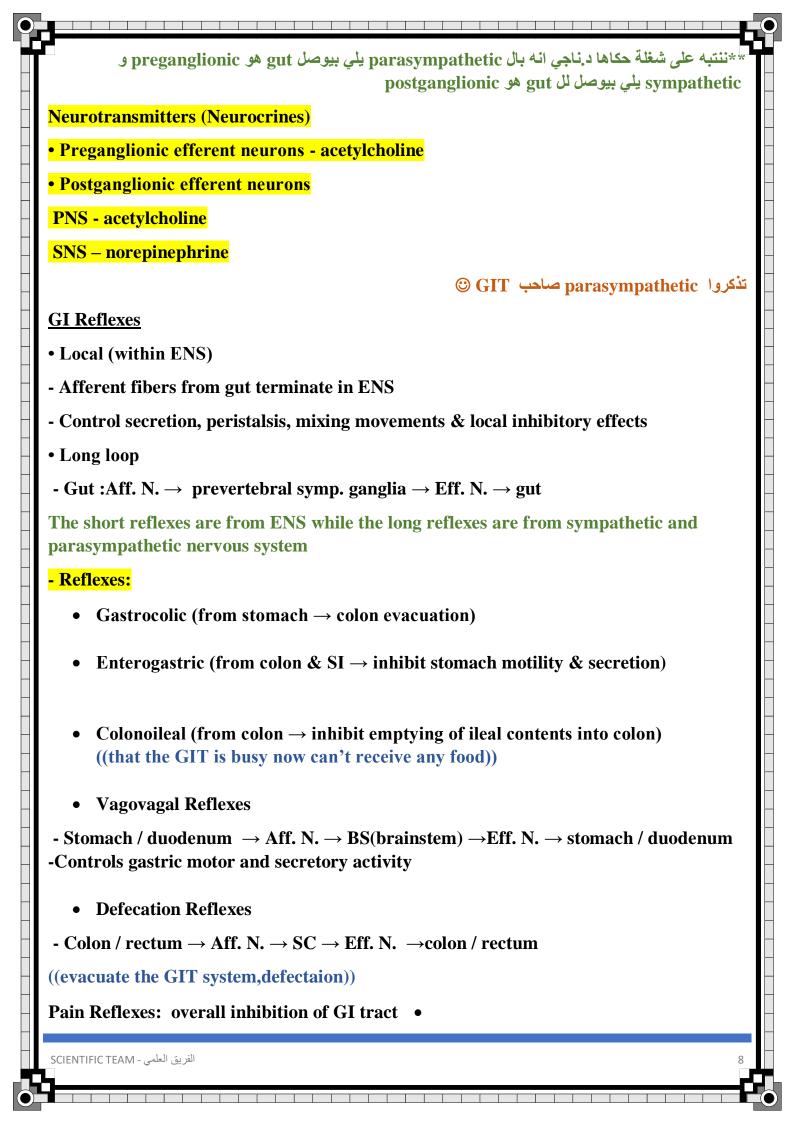
So they don't happen unless there is a stimulus for example : stretch of the gut (ingestion of the meal), Ach parasympathetic, GIT hormones. -- Slow wave P -- spike potential frequency (range 1-10 spikes/s, duration 10-20 ms) • AP in GI SM vs nerves: • Nerve: Na through Na channels (rapid) • GI SM Ca2+ (mainly)+ Na through Ca2+-Na+ channels (slow) lead to longer duration AP in GI SM • More negative RMP – hyperpolarization((by sympathetic nervous system)) **Tonic Contraction** • Continuous (no relaxation). • Usually observed in sphincters. • Not associated with basic electrical rhythm of the slow waves. • Caused by : -Continuous repetitive spike potentials -Hormones -Continuous entry of Ca2+ into cell in ways not associated with changes in MP Sphincters : where the food is stopped while moving from one part to another هلا هاى العضلة دائما منقبضة والسبب يعود حتى مايصير evacuation ويصير يرجع الاكل لورا But unless they receive relaxation signal to open and push the food but they are usually contracted and have their own electrical activity **Neural Control of GI Tract** The autonomic nervous system (ANS) of the GI tract comprises both extrinsic (sympathetic and parasympathetic) and intrinsic nervous systems (myenteric and submucosal plexus)

- Intrinsic Control Enteric nervous system
- Esophagus to anus
- Can function independently of extrinsic nerves
- Controls movements & secretion
- Myenteric (Auerbach's) plexus

- Submucosal (Meissner's) plexus
Both of these plexus are connected to each other so any stimulation of one of them will result to stimulate another one
external stimulation ما الله ENS جس هو ENS
ENS - Myenteric Plexus
 Location Between longitudinal and circular SM layers
• Function - <u>controls GI motility</u>
- Stimulatory influences -
• - tonic contraction (tone)
• - contraction frequency / intensity
• - velocity of conduction of excitatory waves (peristalsis)((moving food through GIT)
- Inhibitory- <u>vasoactive intestinal polypeptide</u> – inhibits sphincter muscles (pyloric & ileocecal valve)
Pyloric: the myenteric plexus relax the sphincter to push the food from the stomach to deudonom
Ileocecal valve: the myenteric plexus relax it to improve the evacuation from ileum to caecum
Ascending Heoceal Valve Cecum Appendix Appendix
ENS – Submucosal
Location - submucosa
• Function - Control secretion
- Absorption (local blood flow)
- Contraction of muscularis mucosa (infolding)((to increase the surface area for absorption))
Note :both plexuses uses local reflexes to relay information within the GI tract.
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 Extrinsic Control - Autonomic nervous system Parasympathetic - mainly stimulates (Ach) Sympathetic - mainly inhibits (NE) Parasympathetic Innervation Cranial Division - (mostly Vagus N.) - first half of gut Scaral Division (S2-4) - (Pelvic N.) - second half of gut Scaral Division (S2-4) - (Pelvic N.) - second half of gut Neurons - preganglionic - long postganglionic - short, entirely in ENS Symapse with ENS neurons (mainly) Stimulation - Excites ENS (in general) Parasympathetic nerves also contain afferent sensory fibers (80%) Defecation reflex(increase the defecation because the parasympathetic system innervates the lower part) Symapse in prevertebral ganglia Postganglionic Neurons (long) - Originate at T5-L2 (cell bodies). Synapse in prevertebral ganglia Postganglionic Neurons (long) - Originate in ganglia (cell bodies). Synapse in prevertebral ganglia Postganglionic Neurons (long) - Originate in ganglia (cell bodies). Nerve endings mainly secrete norepinephrine Inhibitory (a) Decreasing activity of ENS (mostly) (b) Inhibit SM (except mucosal SM)-Slight activity Sympathetic nerves also contain afferent sensory fibers(%50) 		
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Hormonal control of GI motility

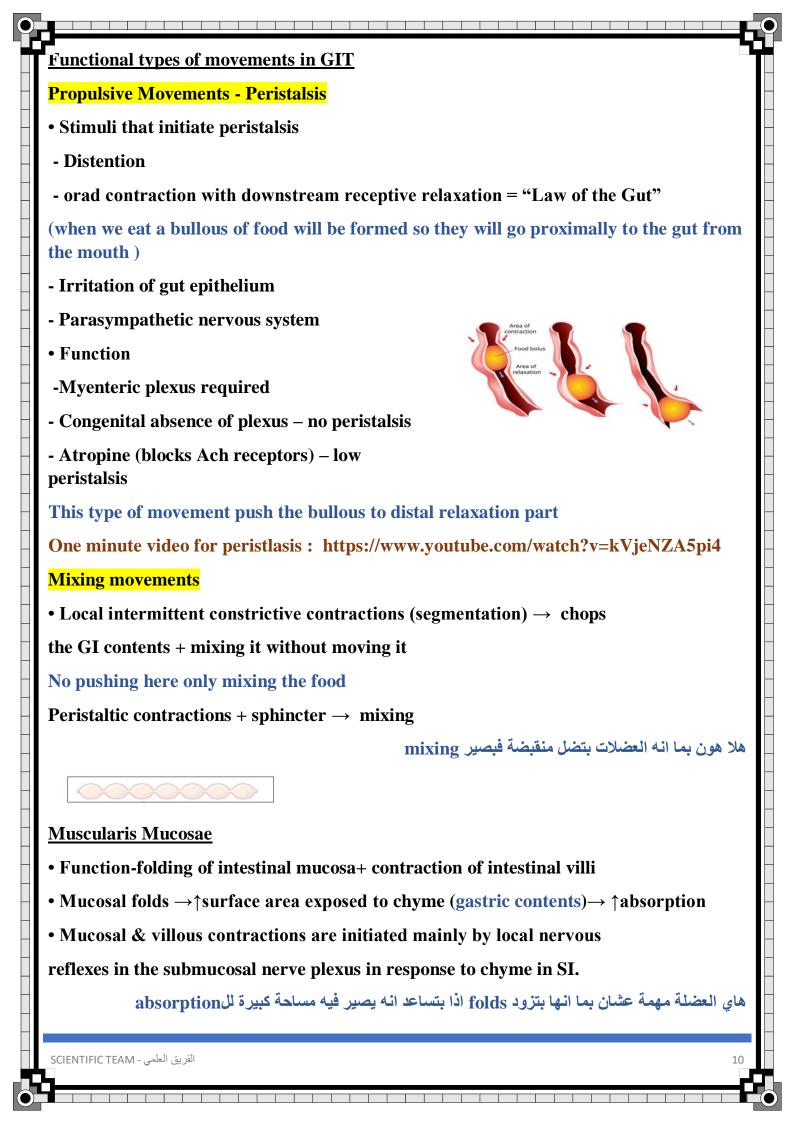
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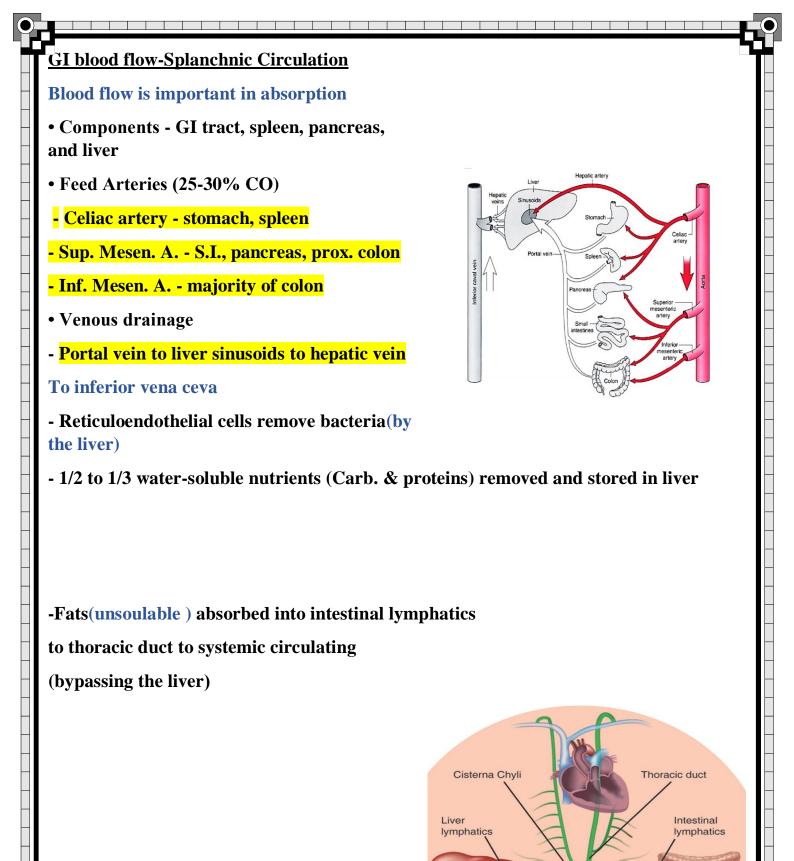
The GIT hormones will be secreted locally and they will reach the GIT in order to work distally

Hormone	Stimuli for Secretion	Site of Secretion	Actions
Gastrin	Protein Distention Nerve Vagal/ gastrin-releasing (Acid inhibits release)	G cells of the antrum, duodenum, and jejunum peptide	Stimulates Gastric acid secretion Mucosal growth
Cholecystokinin	Protein Fat Acid	I cells of the duodenum, jejunum, and ileum	Stimulates Pancreatic enzyme secretion Pancreatic bicarbonate secretion Gallbladder contraction Growth of exocrine pancreas Inhibits Gastric emptying Appetite-vagus
Secretin	Acid Fat	S cells of the duodenum, jejunum, and ileum	Stimulates Pepsin secretion Pancreatic bicarbonate secretion Biliary bicarbonate secretion Growth of exocrine pancreas Inhibits Gastric acid secretion
Gastric inhibitory peptide Glucose-dependent insulin	Protein Fat Carbohydrate otropic peptide	K cells of the duodenum and jejunum	Stimulates Insulin release Inhibits Gastric acid secretion Gastric emptying
Motilin	Fat Acid Nerve bited by food ingestion	M cells of the duodenum and jejunum during fasting	Stimulates interdigestive myoelectric complex Gastric motility Intestinal motility

Note about motilin :

That this hormone is increased during fasting , it increases the gastric motility and when we eat this hormone will be suppressed





	Blood flow through intestinal villus
	Intestinal villis: the main absorption unit of the small intestine
	 Countercurrent Blood Flow in the Villi
	- 80% oxygen is shunted from artery to vein
	- Not harmful
	- In disease conditions e.g Circulatory Shock \rightarrow
_	Splanchnic blood flow $\downarrow \rightarrow$ Villus tip or entire
	villus suffers ischemic death $\rightarrow \downarrow$ Absorptive capabilities
	- Lymph flows freely from the central lacteals of
	villi into lymphatic system Lymph to lymphatic system
	We can see in the figure that the oxygen can go to
	the veins side before reaching the top of the villi(normal condition)but if there is shock less oxygen will be reaching the top of the villi decreasing the absorption
_	Control of Gut Blood Flow
	 Blood flow proportional to local activity
	- Meal \rightarrow blood flow
	- high motor activity—high blood flow

- Causes of activity induced blood flow
- Vasodilator hormones
- CCK, VIP, gastrin, secretin.
- Vasodilator kinins-kallidin, bradykinin
- Low oxygen (high adenosine)
- Nervous control of blood flow
- PNS :gut activity \rightarrow high blood flow
- SNS, exercise, shock Directly decrease low blood flow- overcome> Autoregulatory escape (local metabolic vasodilator mechanisms)

Decrease blood flow(vasoconstriction)will be overcome by vasodilator mechanisms

-SNS – vasoconstriction of intestinal and mesenteric veins to <u>sustain (200-400 ml) the</u> <u>general circulation</u>

Good luck Hope 😊