MEDICINE
KING SAUD UNIVERSITY

تمت المراجعة
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Physiology Team 436

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- Important
- Numbers
- Doctor notes
- Notes and explanation

# GIT PHYSIOLOGY

Lecture No.1

"The Best Time To Start Was Yesterday, The Next Best Time Is Now"

# General principles of GIT physiology

**Objectives:**

1. Physiologic Anatomy of the Gastrointestinal Wall.
2. The General Characteristics of Smooth Muscle.
3. The specific characteristics of smooth muscle.
4. Control of gastrointestinal function (ENS).
5. Functional types of movements in the gastrointestinal tract.
6. Gastrointestinal blood flow (Splanchnic circulation).
7. Effects of gut activity and metabolic factors on GI blood flow.

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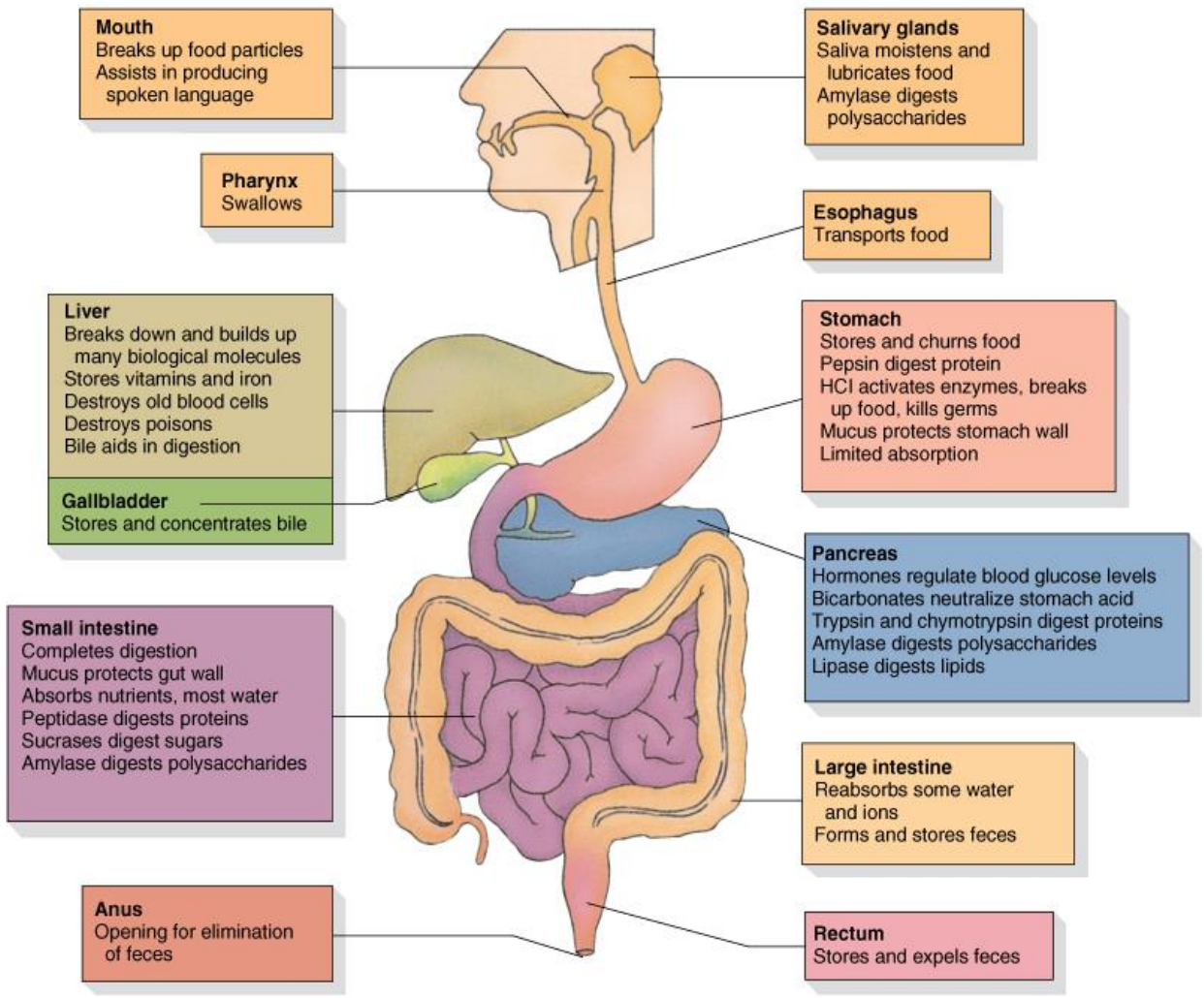
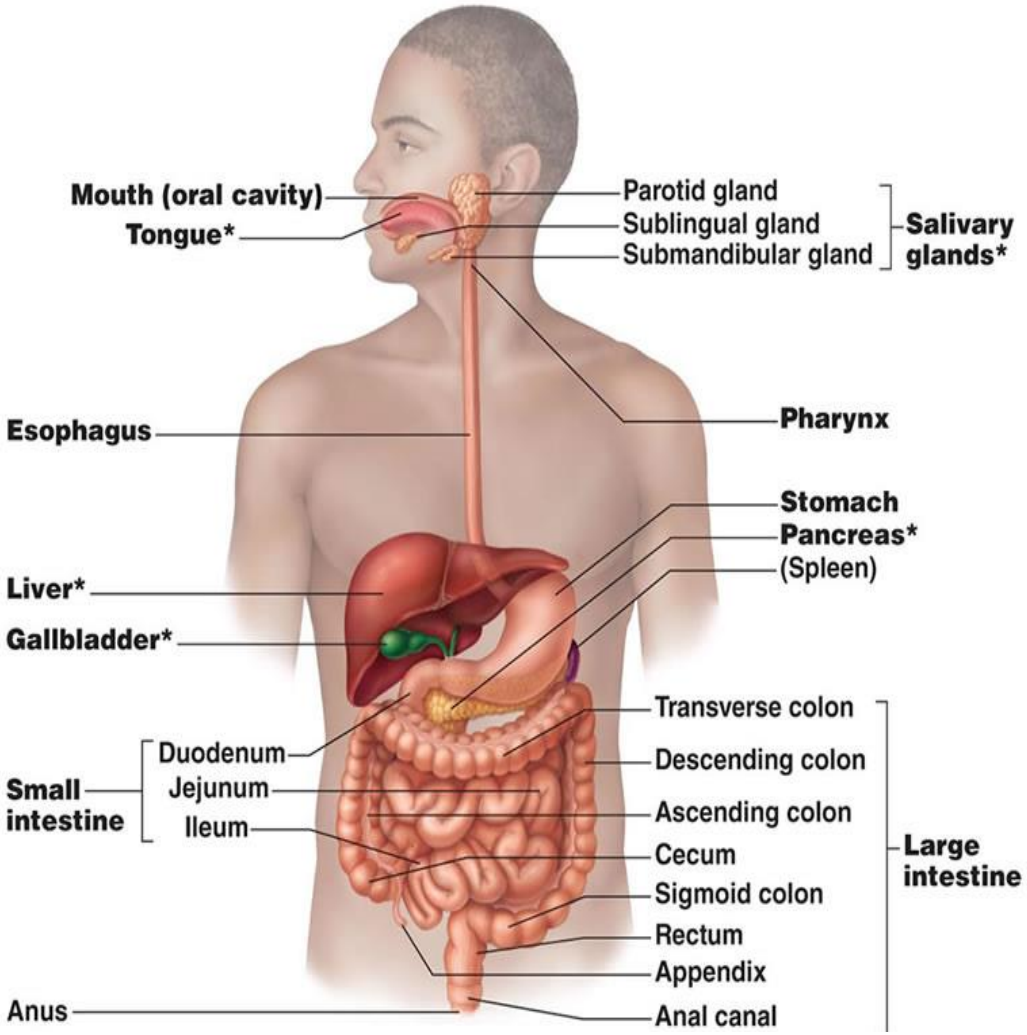
Just read it

# Gastrointestinal Tract



# General Principles of Gastrointestinal Function

GIT provides the body with a continual supply of water, electrolytes and nutrients. To achieve this requires:

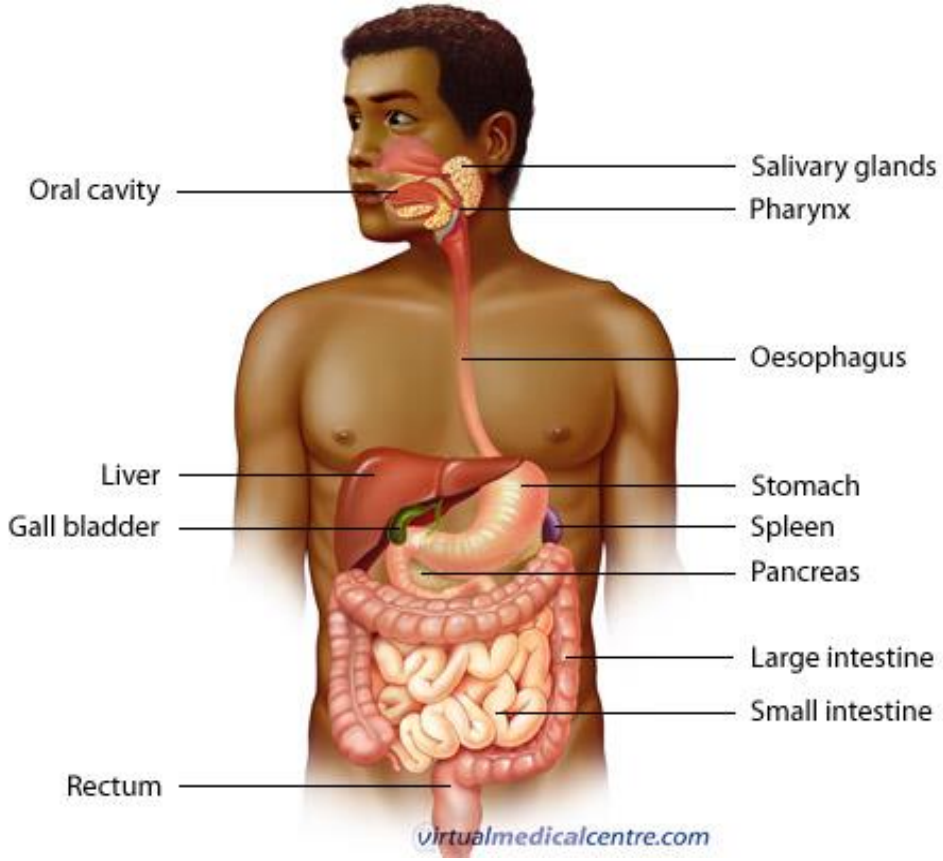
1. Movement of food through the alimentary tract.

2. Secretion of digestive juices and digestion of the food.

3. Absorption of water, various electrolytes, and digestive products.

4. Circulation of blood through the gastrointestinal organs to carry away the absorbed substances.

5. Control of all these functions by local, nervous, and hormonal systems.

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Smooth Muscle Function

0:54

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Important

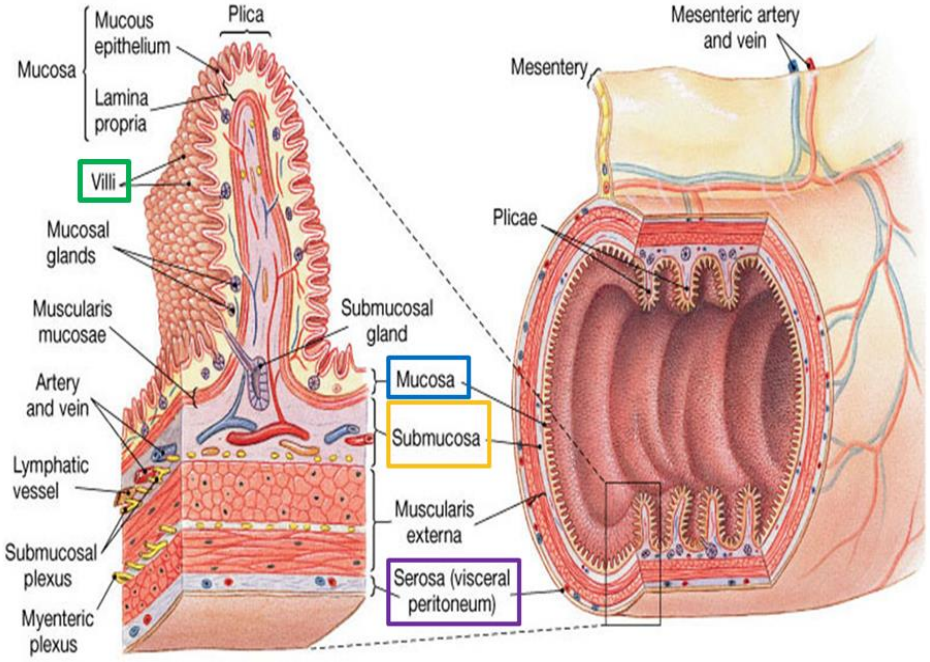
# General Principles of Gastrointestinal Motility

- Physiological anatomy of the gastrointestinal wall, the following layers structure the GI wall from outer surface inward:

1. The serosa (outer layer).
2. A longitudinal muscle layer.
3. Myenteric plexus.
4. A circular muscle layer.
5. The submucosa (has thin neuronal layer with submucosal plexus).
6. The mucosa (inner layer)

- In addition, sparse bundles of smooth muscle fibers, the mucosal muscle, lie in the deeper layers of the mucosa.

2 neuronal layers and 2 smooth muscle layer then submucosa layer (small and gray) has large blood vessels, large lymph vessels, glands and immune cells. Then small layer called muscularis mucosae then laminae propria (yellow area) has capillaries small blood vessels and small lymph vessels then velli (finger like layer) has villi and microvilli useful to increase surface area for absorption and secretion there is current secretion at the top of villi and absorption at pits, more villi in small intestine where real digestion happens not in the stomach. These layers are from the lower 1/3 of esophagus to the anus same layers but variations in type of cells and other minor changes.



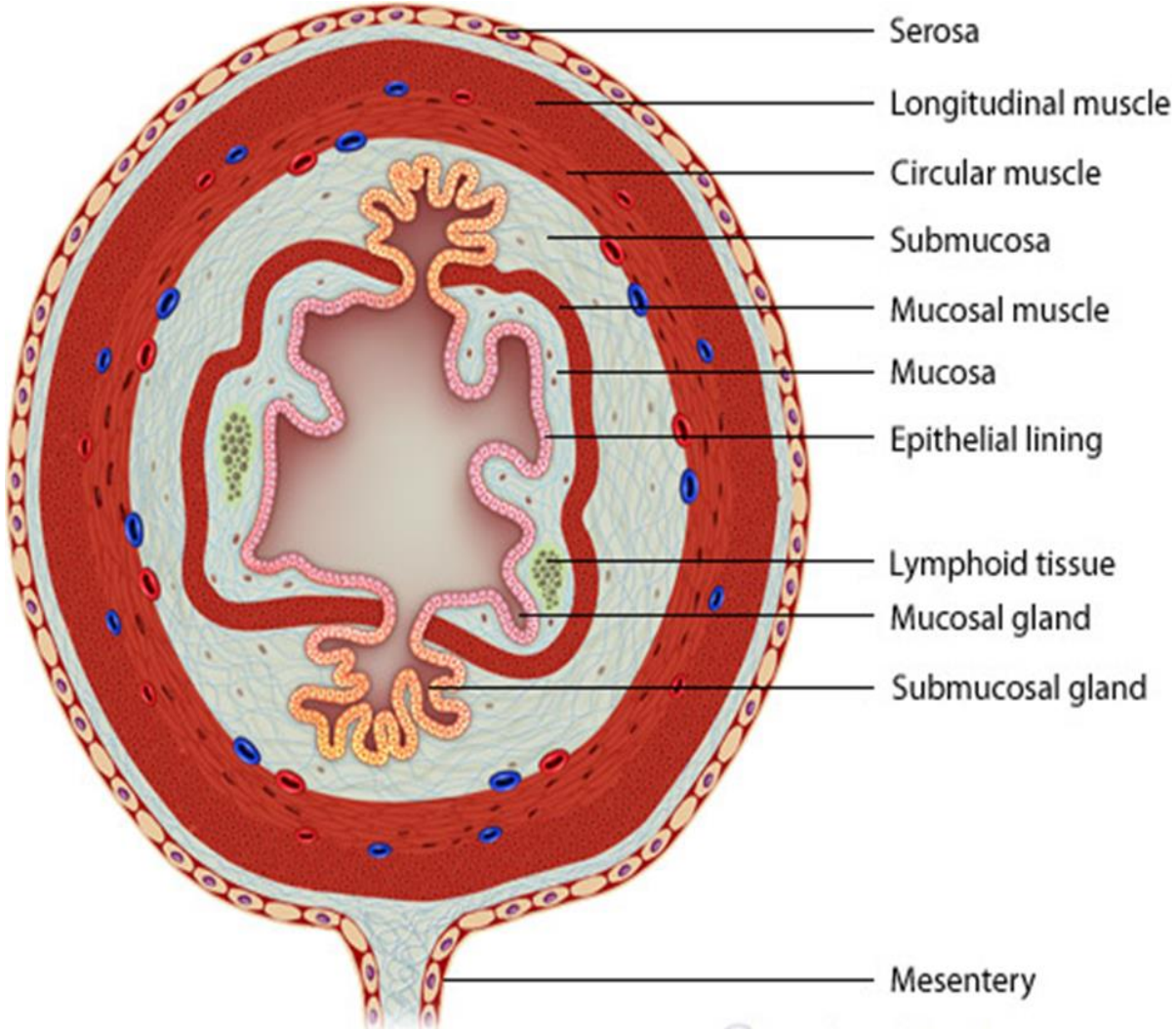
# Cont.

- **Mucosa** is the innermost, moist, epithelial membrane that lines the entire digestive tract.
- (1) It secretes mucus, digestive enzymes, and hormones;
- (2) absorbs digestive end products into the blood; and
- (3) protects against infectious disease.
- Consists of a lining epithelium, a lamina propria, and a Muscularis mucosa.
- Epithelium - simple columnar epithelium and goblet cells
- Lamina propria - areola C.T. with capillaries and lymphoid follicles
- Muscularis mucosa - thin layer, produces local movements of t mucosa

- **Sub mucosa** is a moderately dense connective tissue layer containing blood and lymphatic vessels, lymphoid follicles, and nerve fibers.

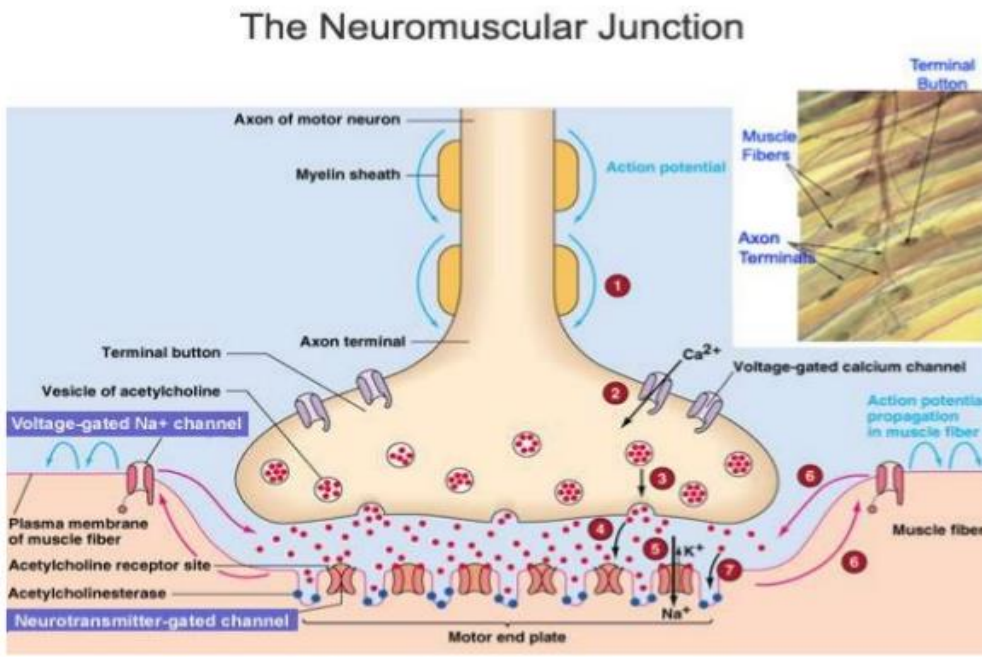
- **Muscularis externa** typically consists of smooth muscle and is responsible for peristalsis and segmentation.
- Contains the my enteric plexus of Auerbach, the other major intrinsic nerve plexus. Located between the two layers of smooth muscle, controls motility of the G.I. tract.

- **Serosa**, the protective outer layer of the intraperitoneal organs, is the visceral peritoneum

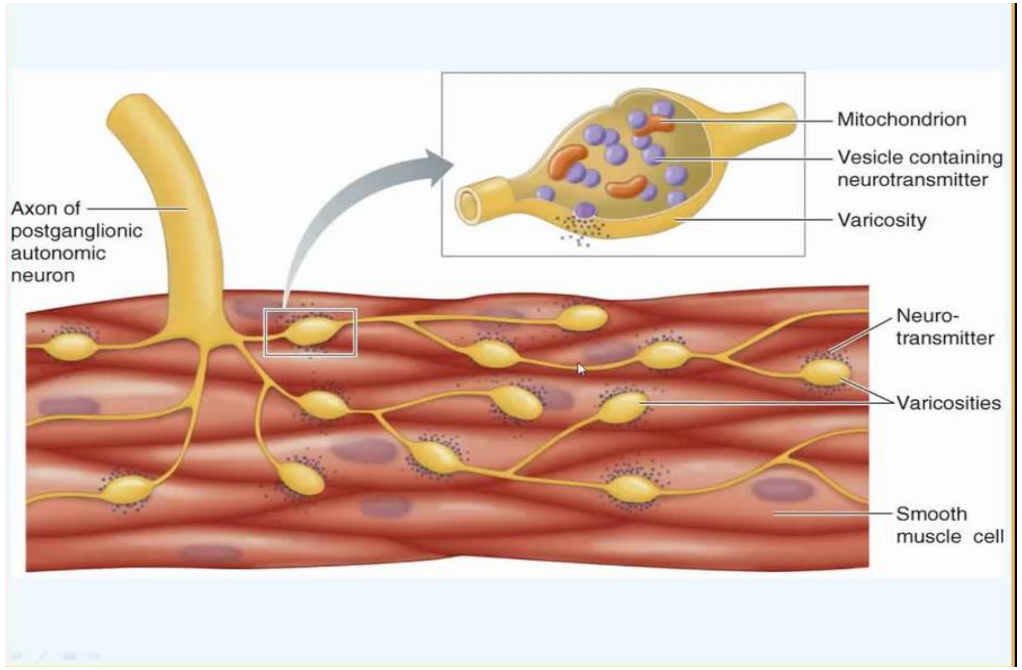


# Difference Between Skeletal And Smooth Muscles

Nervous control
Neuromuscular junctions




Smooth muscle

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Smooth Muscle vs Skeletal Muscle

4:19

skeletal muscles innervated by somatic nerve while smooth m are innervated by autonomic innervation.
There's dilatation around nerve ending away from the schwann cells so it's easier for neurotransmitters to diffuse.

# The General Characteristics of Smooth Muscle

## Smooth muscle classification

We only have one type of muscle in the GI system (smooth muscle only)

### Unitary type (Single unite)

- Most of the smooth muscle in the GI are unitary type.
- The difference between unitary type and multiunit type are in the functions.
- All of them maybe longitudinally or circularly.
- Can be activated by stretching, always in stomach and small intestine, Stomach always working without even food present.
- Contracts spontaneously in the absence of neural or hormonal influence but in response to stretch (such as in stomach and intestine).
- هذا النوع معتمد على نفسه! ما يحتاج هرمون او عصب يحفزه..
- طبيعي لما ناكل راح تتمدد المعدة ويصير لها stretching، مجرد أكل كمية بسيطة راح يسبب لنا stretching بالتالي راح يصير contraction لهذا النوع وتبدأ تهضم الطعام.
- Cells are electrically coupled via gap junction.
- Mass of 100-1000 smooth muscle fibers.
- Contract as a single unit, Arranged in sheets or bundles.
- Cell membranes are adherent at multiple points.
- Many gap junctions that allow ion movements.
- When action potential is generated it travel in all directions.
- Ex: GIT, bile ducts, stomach, intestine, ureters, uterus, blood vessels.

### Multiunit type

- Does not contract in response to stretch or without neural (such as in esophagus & gall bladder).
- Each fiber can contract independently.
- Controlled mainly by nerve signals.

Ex : Esophegus & gall bladder.

Other examples: ciliary & iris muscles in the eye and piloerector muscles of the hair.

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# Cont.

**Types of contractions**

سواء من أي نوع من أنواع العضلات تسوي لنا كنتراكشن، الكنتراكشن نوعين

- **Phasic (rhythmical) contraction**
  - Periodic contractions followed by relaxation; such as in gastric antrum, small intestine and esophagus.
    كنتراكشن يعقبه ريلاكسيشن
- **Tonic contraction***
  راحتها في انقباضتها، ماتبي تريّح
  - Maintained contraction without relaxation; such as in orad region of the stomach, lower esoghageal, ileocecal and internal anal sphincter.
    Only internal sphincter
  - Caused by:
    1- repetitive spike potentials. Spike potential = action potential
    2- hormones.
    3- continuous entry of ca ions (not associated with changes in membrane potentials).
  - Not associated with slow waves (often lasting several minutes or hours).

*Continues, can relax even when contracted, found in sphincters and erea of stomach upper and lower sphincters of esophagus, pyloric sphincter, iliosycal sphincter which is between small intestine and secum and in anal canal (2 sphincters, internal and external, internal always contracted, external voluntarily controlled consciously)

# The General Characteristics of Smooth Muscle and its Function

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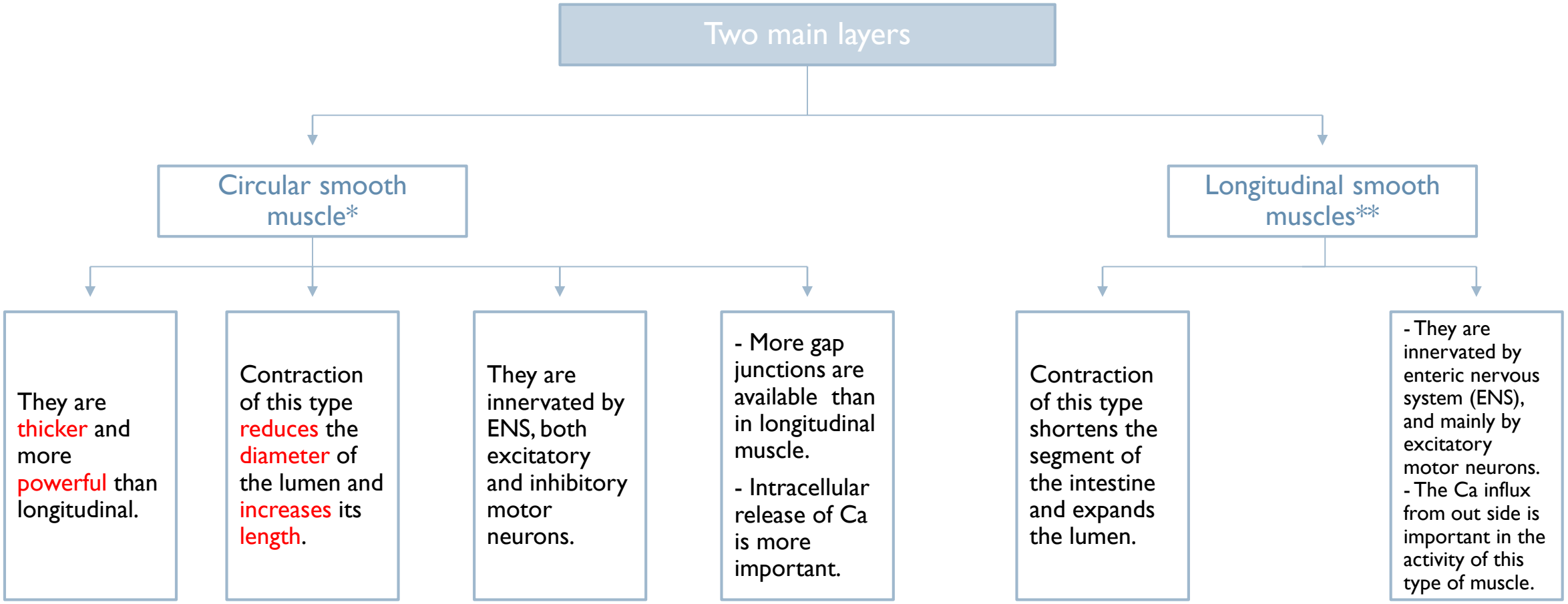


* For propulsion to move food forward they should relax in front of chyme.
** Shorten diameter and length.

# The Specific Characteristics of Smooth Muscle in the GIT

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## 1. Gastrointestinal Smooth Muscle Functions as a Syncytium (Once one cell is activated the whole are is activated because of gap junctions):

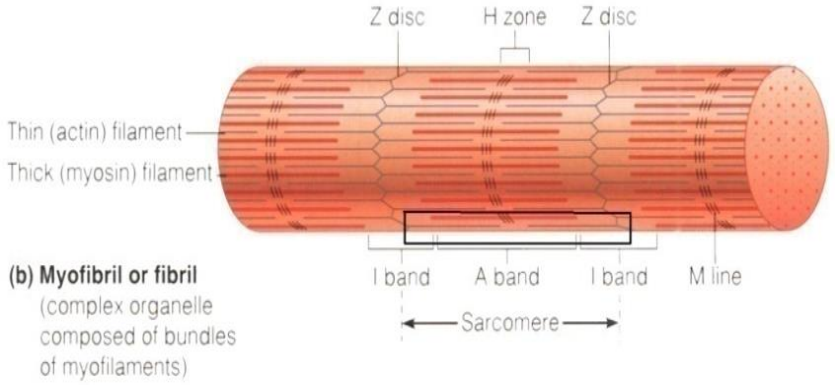
- The individual smooth muscle fibers are 200 to 500 µm in length and 2 to 10 µm in diameter, and they are arranged in bundles of as many as 1000 parallel fibers.

- Within each bundle, the muscle fibers are electrically connected with one another through large numbers of gap junctions.

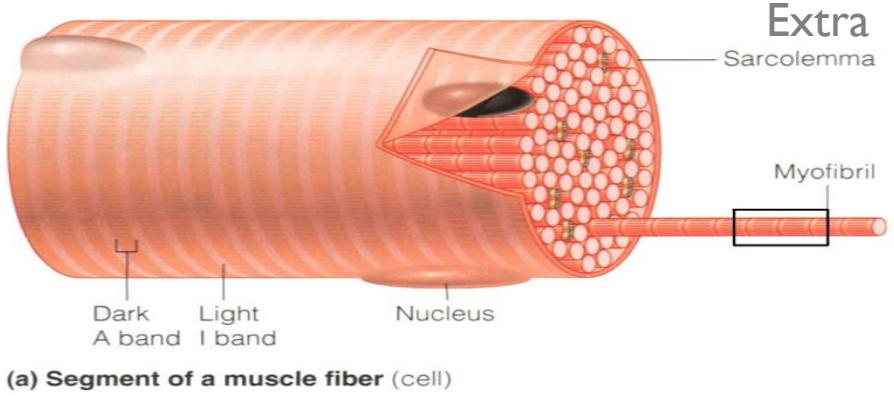
- Each bundle of smooth muscle fibers is partly separated from the next by loose connective tissue but they fuse with one another at many points, so each muscle layer represents a branching latticework of smooth muscle bundles. Therefore, each muscle layer functions as a syncytium; that is, when an action potential is elicited anywhere within the muscle mass, it generally travels in all directions in the muscle.

IMP:
Electrical activity in smooth muscle cells of GIT is very different by:
1-having slow waves and spikes
2-syncytium contraction

Extra




Extra



# Cont.

- Smooth muscle does not have the same striated arrangement as is found in skeletal muscle.
- There are large numbers of actin filaments attached to so-called **dense bodies.**
- Some of these bodies are attached to the cell membrane.
- Others are dispersed inside the cell.
- Some of the dense bodies of adjacent cells are bonded together by intercellular protein bridges. through these bonds that the force of contraction is transmitted from one cell to the next.

- Slow cycling of the myosin cross-bridges.
- Low energy requirement.
- Slowness of onset of contraction and relaxation.
- Greater max. force of contraction.
- Latch mechanism.
- Stress-Relaxation.
- Revers stress-relaxation.

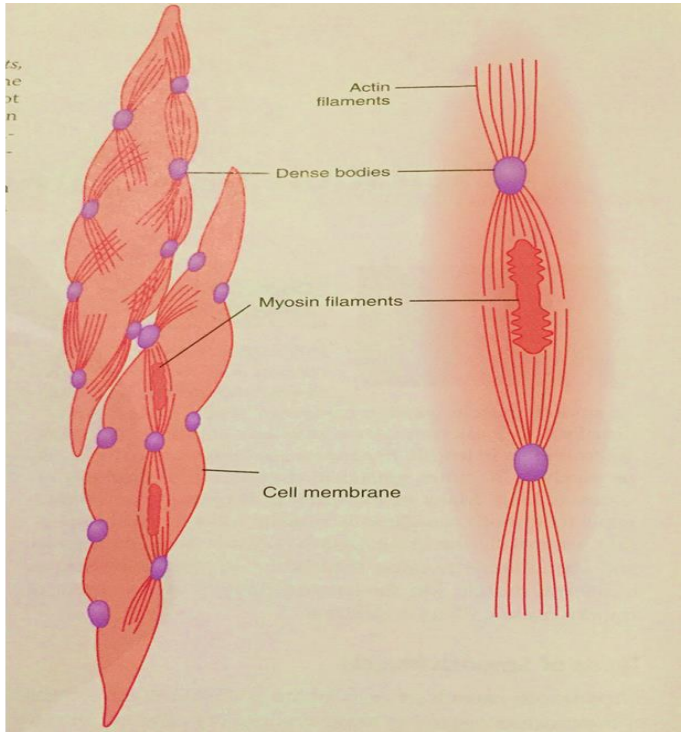



**Figure 8-2** Physical structure of smooth muscle. The upper left-hand fiber shows actin filaments radiating from dense bodies. The lower left-hand fiber and the right-hand diagram demonstrate the relation of myosin filaments to actin filaments.

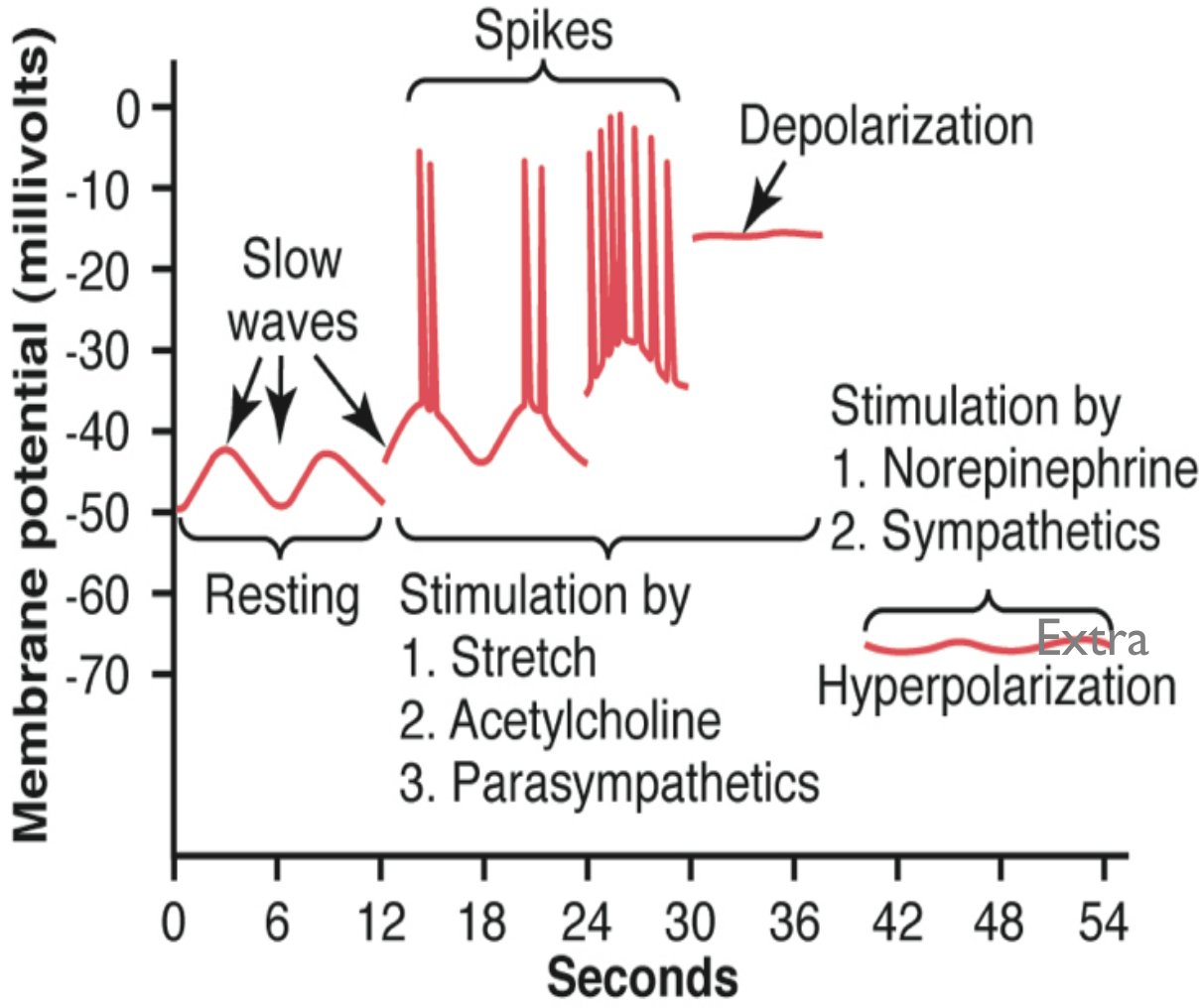
Important

# Electrical Activity of Gastrointestinal Smooth Muscle

## 2. Electrical Activity of Gastrointestinal Smooth Muscle:

- The smooth muscle of the gastrointestinal tract is excited by almost continual slow, intrinsic electrical activity along the membranes of the muscle fibers. This activity has two basic types of electrical waves:
- (a) slow waves.
- (b) spikes.

- Certain cells in these layers are different in resting membrane potentials is -50 these cells are closer to the threshold need small activation.
- Mynteric plexus and submucosal layers (neuronal layers in ) between circular smooth muscles.



Very Important

# Cont.

## Slow waves

- Most gastrointestinal contractions occur rhythmically. This rhythm is determined mainly by the frequency of so-called "slow waves" of smooth muscle membrane potential. These waves are not action potentials. Instead, they are oscillating depolarization and repolarization in the resting membrane potential with unknown cause.

- Their intensity usually varies between 5 and 15 mV, and their frequency ranges in different parts of the human gastrointestinal tract from 3 to 12 per minute:
  - about 3 in the body of the stomach.
  - as much as 12 in the duodenum.
  - about 8 or 9 in the terminal ileum (Jejunum ilium).
    - ليه ذكرنا بس هذه المناطق؟
  - Because they didn't record this in the esophagus and the large intestine

- Origin of slow waves: They may originate in the interstitial cells of Cajal (ICC, the GI pacemaker), which are abundant in the myenteric plexues.

- These ICCs form a network with each other and are interposed between the smooth muscle layers, with synaptic-like contacts to smooth muscle cells .Do not by themselves cause muscle contractions. Do not cause calcium ions to enter (only sodium ions). Mainly excite the appearance of intermittent spike potentials.
  - صحيح أن ال Slow waves قليلة، ولكن كل slow wave وحدة تعطينا 10 spike potential

## The spike potentials are true action potentials*

- They occur automatically when the resting membrane potential of the gut smooth muscle becomes more positive (<-40 mV) (the normal resting membrane potential is between -50 and -60 mV).

- The higher the slow wave potential rises > the greater the frequency of the spike potentials (usually ranging between 1 and 10 spikes per second.)

- The spike potentials last 10 to 40 times as long in gastrointestinal muscle as the action potentials in large nerve fibers, each gastrointestinal spike lasting as long as 10 to 20 msec.

- In gastrointestinal smooth muscle fibers, the channels responsible for the action potentials are somewhat different, they allow especially large numbers of calcium ions to enter along with smaller numbers of sodium ions and therefore are called calcium-sodium channels.

- These channels are much slower to open and close than the rapid Na channels of large nerve fibers.
- They cause muscle contraction.
- Spike duration up to 5seconds

*When we want to relax like in tonic contraction we must activate NE and sympathetic which will drag membrane potential.
NE is inhibitor, Sympathetic is inhibitor, Parasympathetic is excitatory

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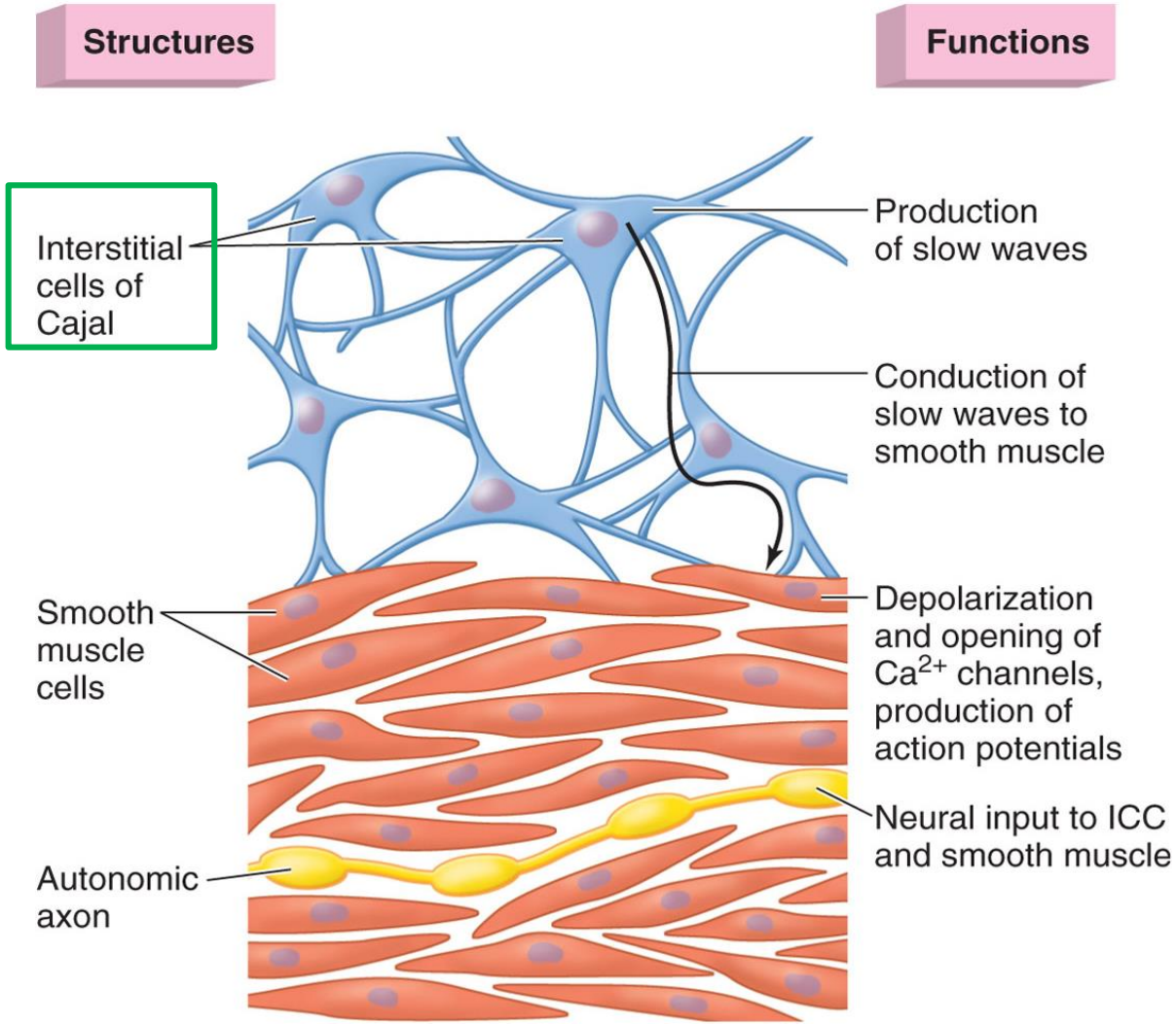
Very Important

# Cont.




ICC= interstitial cells of cajal, have different resting potentials, ICC controls movement of everything it is the pacemaker of GIT, لو وحدة كوّعت غيرها حتشتغل و كفاية

We don't need anything else from the outside to activate the GIT because we have ICC because ICC have:

1- less negative (-50)

2- their resting membrane potential is oscillating up and down it is not fixed. تذبذبknown as slow wave it is not action potential -50 to -30

When slow wave reach threshold we will have spikes

3- probability to reach threshold will increase by stimuli like stretching or parasympatheic stimulation.

Unitary cells are well innervated by ICC .

الزبدة:

نحتاج مضخة (Pacemaker ) وظيفتها تسوي activate وتحرّك باقي الخلايا، ما نحتاج شيء من برّا يساعد بالتحريك هذه الخلية Icc تكفي.

ما يعتبر هذا action potential

إنما هو slow waves اللي ينتج لي action potential.

We don't need to activate all these cells to generate the action potential, 1 or 2 are enough to generate it.

روعة ولا مش روعة؟

# Please read this before you complete the lecture

فلنفترض أن الجي أي سيستم عبارة عن تيم شغال..

ICC or the pacemaker

هم الممبرز اللي شغالين!

ولكن الممبرز ما يمشون من غير ليدرز يعتبرون ال Big boss

الليدرز هنا هم (Sympathetic+ Para Sympathetic).

فلنفترض ان الليدرز او واحد منهم ترك التيم فجأة (يعني انقطع عندي السبمباثتك او البارا سمبثتك بسبب حادث او جراحة او مشكلة)، طبيعي التيم بيواجه مشاكل أول يوم أو يومين.. لكن ما يتوقف شغل التيم (GI system) ليه؟

لأن فيه ليدرز ثانيين يجون وتمشي الأمور

الليدرز الجدد هم (Enteric nerve )..

Enteric nerve is specific for GI ststem

الليدرز ما يحتاجو الأكاديمك ليدرز بشكل دائم بالتيم لكن لو حصلت مشكلة او عندهم سؤال علطول يتوجهوا لهم لحلّها، وبالمقابل لازم يخبروهم بأي مستجدات بالتيم

الاكاديمك ليدرز هنا هم (brain) وهم المسؤولين عن النيرفز.

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# Smooth Muscles in the Gut

- Changes in voltage of the resting membrane potential:

- The resting membrane potential averages about -56 millivolts, but multiple factors can change this level.
- When resting membrane potential becomes less negative > called depolarization of the membrane > the muscle fibers become more excitable.
- When resting membrane potential becomes more negative > called hyperpolarization > the fibers become less excitable.

- Factors that depolarize the membrane that make it more excitable are:

1. Stretching of the muscle.
2. Stimulation by acetylcholine released from the endings of parasympathetic nerves.
3. Stimulation by several specific gastrointestinal hormones.

- Factors that make the membrane potential more negative-that is, hyperpolarize the membrane and make the muscle fibers less excitable-are:

1. The effect of norepinephrine or epinephrine on the fiber membrane.
2. Stimulation of the sympathetic nerves that secrete mainly norepinephrine at their endings.

# The Specific Characteristics of Smooth Muscle in the Gut

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- Calcium Ions and Muscle Contraction:

- Smooth muscle contraction occurs in response to entry of calcium ions into the muscle fiber.
- The slow waves do not cause calcium ions to enter the smooth muscle fiber (only sodium ions).
- Therefore, the slow waves by themselves usually cause no muscle contraction. Instead, it is during the spike potentials, generated at the peaks of the slow waves, that significant quantities of calcium ions do enter the fibers and cause most of the contraction.
- الكالسيوم هو الأساس هنا، ويأخذ شوي صوديوم معاه.

- Depolarization = spikes = AP. The cause: ( 80% Ca, 20% Na).
- Ca= funny current.

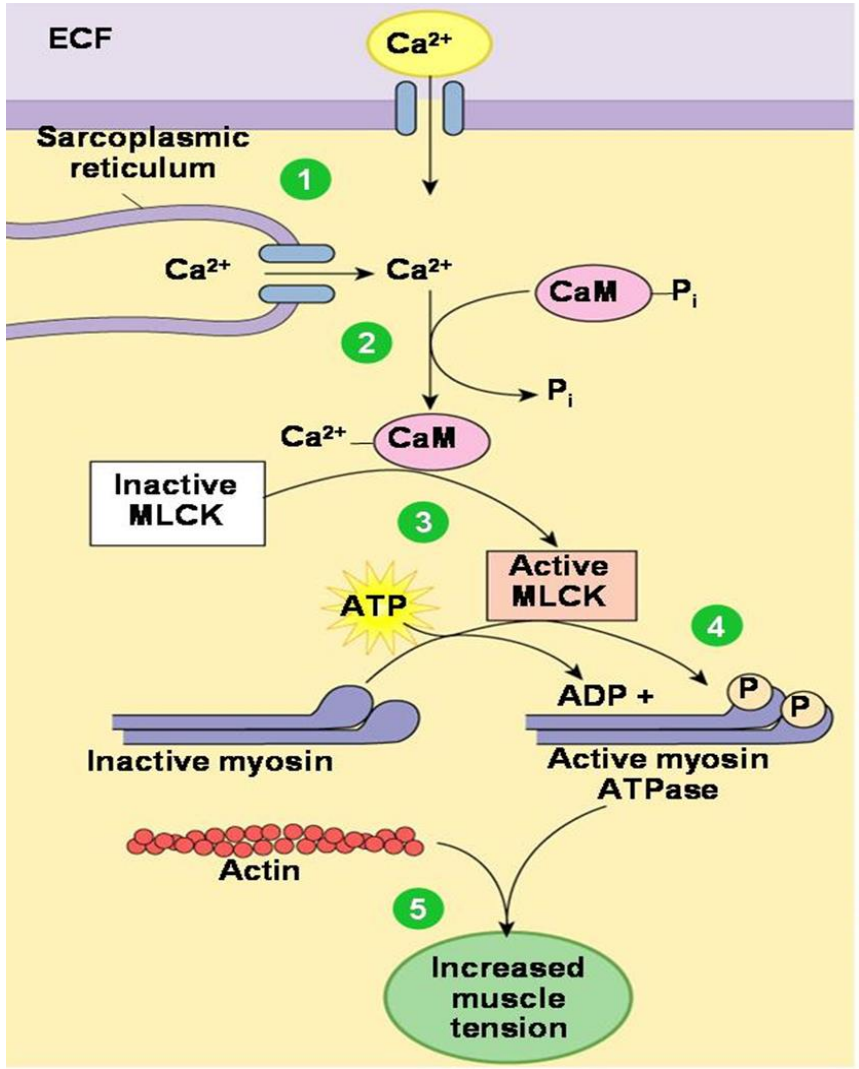
- Tonic Contraction of Some Gastrointestinal Smooth Muscle:

- Some smooth muscle of the GI exhibits tonic contraction as well as or instead of rhythmical contractions.
- Tonic contraction is continuous, not associated with the basic electrical rhythm of the slow waves but, often lasting several minutes or even hours.

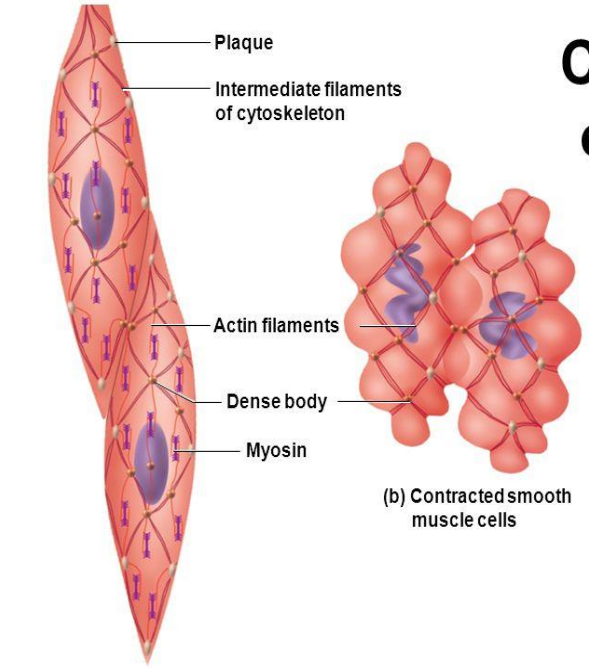
- Tonic contraction is sometimes caused by:

1. Continuous repetitive spike potentials.
2. Hormones.
3. Continuous entry of calcium ions into the interior of the cell brought about in ways not associated with changes in membrane potential (Not via voltage-gated Ca channels).

# Mechanism of smooth muscle contraction




1. Intracellular $Ca^{2+}$ concentrations increase when $Ca^{2+}$ enters cell and is released from sarcoplasmic reticulum.
2. $Ca^{2+}$ binds to calmodulin (CaM).
3. $Ca^{2+}$–calmodulin activates myosin light chain kinase (MLCK).
4. MLCK phosphorylates light chains in myosin heads and increases myosin ATPase activity.
5. Active myosin crossbridges slide along actin and create muscle tension.

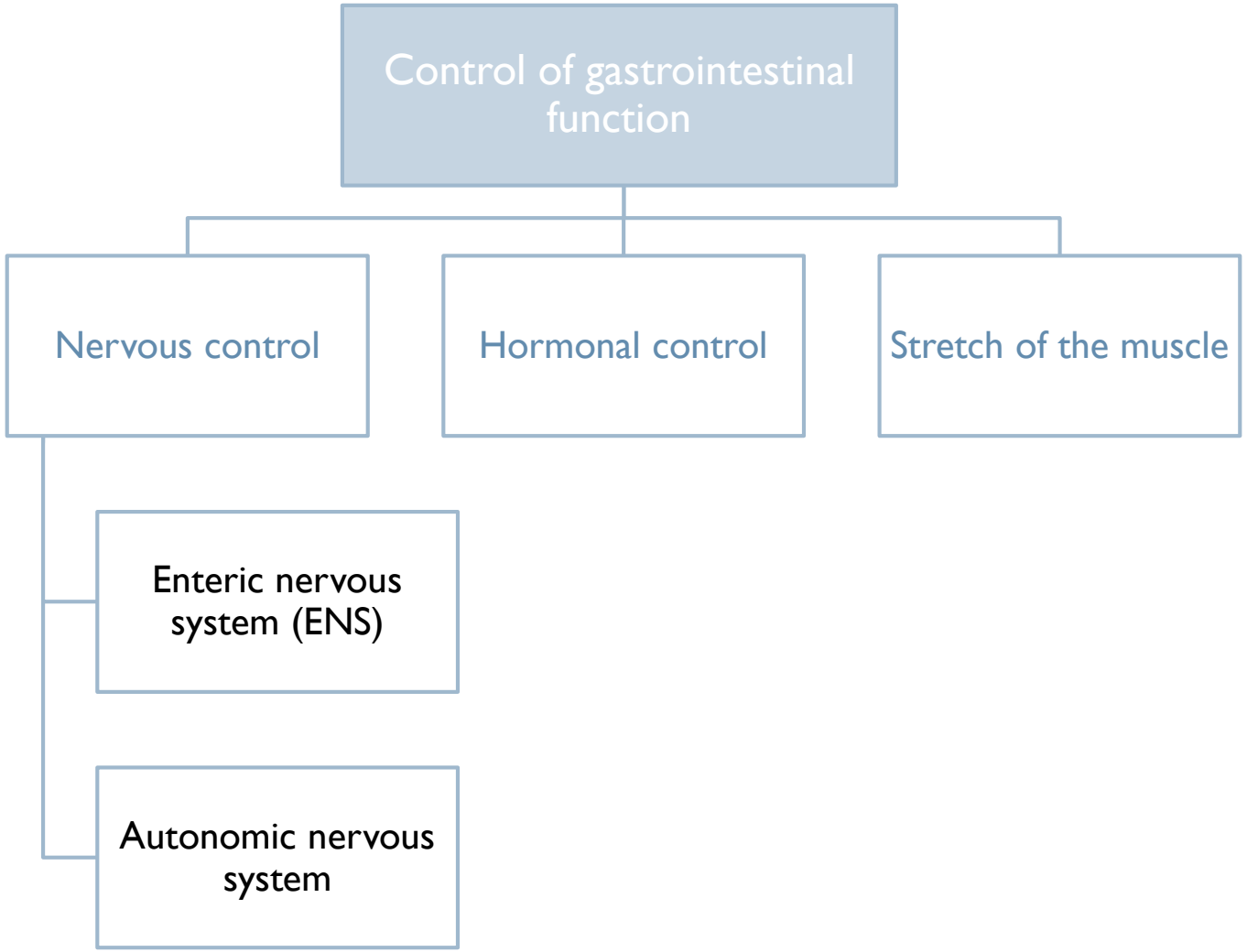



**Contraction of Smooth Muscle**

Figure 11.24

11-70

# Control of gastrointestinal function



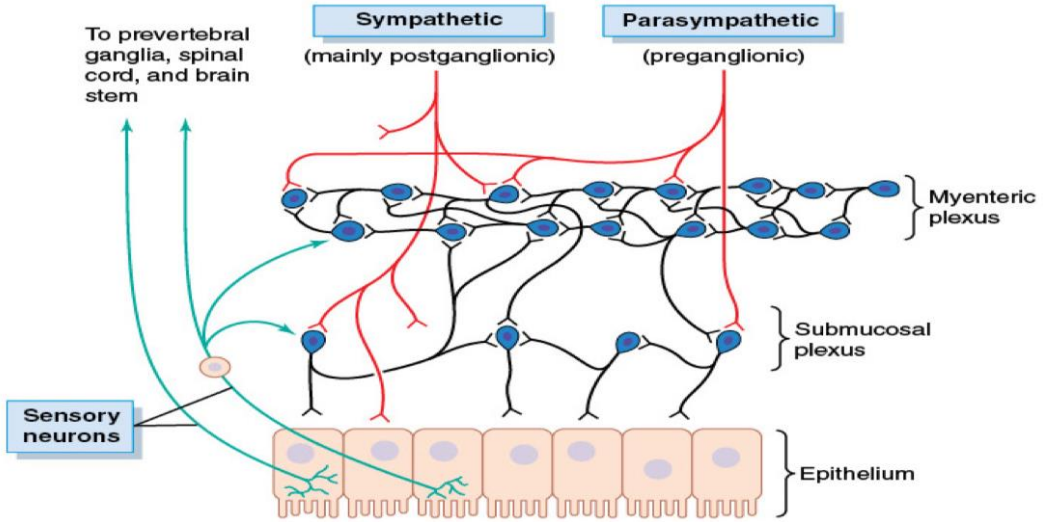
# Neural Control of Gastrointestinal Function-Enteric Nervous System (little brain)

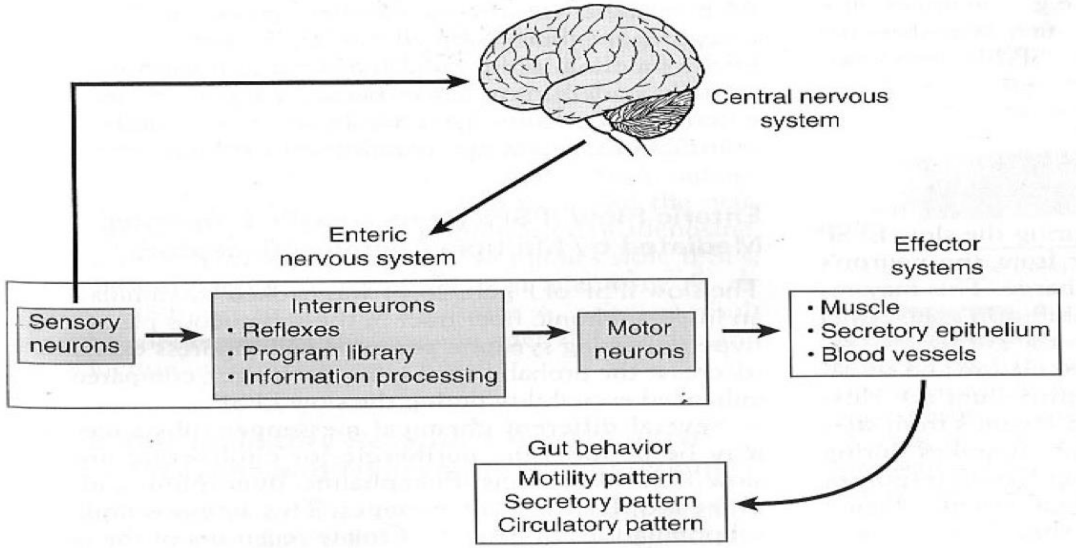
- Enteric nervous system is the nervous system of GI tract (Enteric NS is the 3rd division of ANS).
- It lies entirely in the wall of the gut, beginning in the esophagus and extending all the way to the anus. It has as many neurons as spinal cord (about 100 million).
- The enteric nervous system is composed mainly of two plexuses:

1. An outer plexus: lying between the longitudinal and circular muscle layers, called the **myenteric plexus or Auerbach's plexus**, a linear chain of interconnecting neurons, controls mainly the GI movements. Increase tone, rate, intensity and velocity of rhythmic contraction . some neurons are inhibitory

   (eg. Pyloric &ileocecal valves)

1. An inner plexus: called the submucosal plexus or **meissner's plexus or submucosal**, that lies in the submucosa, controls mainly gastrointestinal secretion and local blood flow.



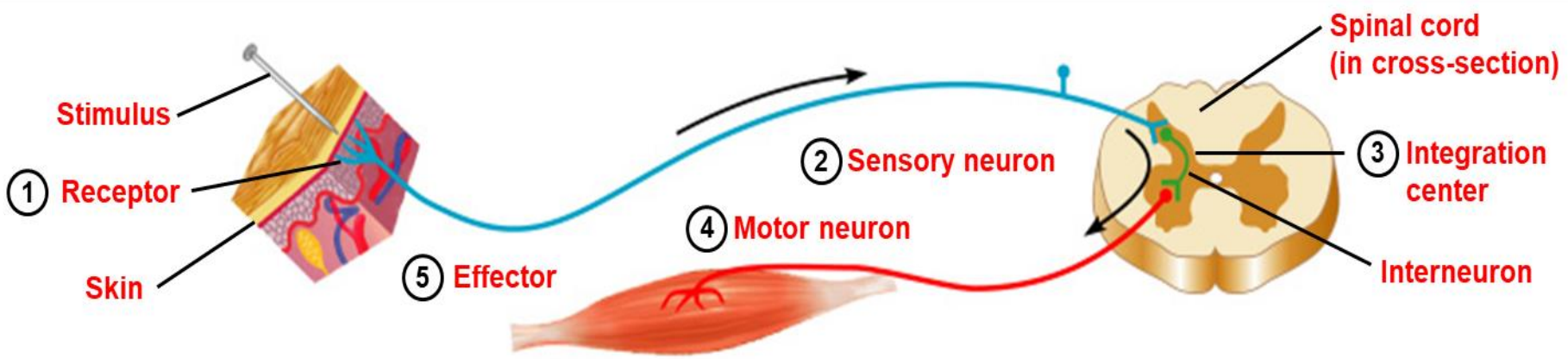
# Cont.

- Connected to CNS by sympathetic & parasympathetic fibers.
- The enteric nervous system can function on its own (autonomously), independently of the parasympathetic and sympathetic systems.
- However, these extrinsic nerves can greatly enhance or inhibit gastrointestinal functions.

- The sensory nerve endings send afferent fibers to both plexuses of the enteric system and then to:

1. the prevertebral ganglia of the sympathetic nervous system.
2. the spinal cord.
3. the vagus nerves all the way to the brain stem.

- These sensory nerves can elicit local reflexes within the gut wall.



# Differences Between the Myenteric and Submucosal Plexuses

| Differences Between the Myenteric and Submucosal Plexuses | |
|---|---|
| The myenteric plexus | The submucosal plexus |
| Consists mostly of a linear chain of many interconnecting neurons. | Controls local intestinal secretion, local absorption, and local contraction of the submucosal muscle that causes various degrees of enfolding of the gastrointestinal mucosa. |
| When it is stimulated, its principal effect is to:<br>1. increase tonic contraction.<br>2. increase intensity and rate of the rhythmical contractions.<br>3. increase velocity of conduction of excitatory waves along the gut wall. | |
| The myenteric plexus has excitatory and inhibitory motor neurons. | |

# Types of Neurotransmitters Secreted by Enteric Neurons

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Important

- The specific functions of many of GI neurotransmitters are not well known, but some research workers have discovered the effects of some of these substances as following:

| Types of Neurotransmitters Secreted by Enteric Neurons | |
|---|---|
| Excitatory Motor Neurons | Inhibitory Motor Neurons |
| Evokes muscle contraction and intestinal secretion | Suppress muscle contraction |
| Neurotransmitters of motor neurons:<br>- Substance P<br>- Ach | - NO (Nitric oxide)<br>- VIP (Vasoactive intestinal peptide)<br>- ATP (Adenosine triphosphate) |
| Neurotransmitters of secretomotor neurons (releasing of water, electrolytes and mucus from crypts of Lieberkuhn):<br>- Ach<br>- VIP (Vasoactive intestinal peptide)<br>- Histamine | Not needed in glands |

Types of neurotransmitters secreted by enteric neurons:
Acetylcholine, Norepinephrine, Substance P, Vasoactive intestinal peptide (VIP), Somatostatin, Serotonin, Dopamine, Cholecystokinin (CCK), Gama aminobutyrate (GABA).
The gases nitric oxide (NO) & carbon monoxide (CO).

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# Autonomic Control of the Gastrointestinal Tract

Autonomic Nervous System (ANS) is divided into :

1. Sympathetic Nervous System.
2. Parasympathetic Nervous System.
3. Enteric Nervous System (ENS).

## I. Parasymoathetic Innervation: (Parasympathetic more imp in GIT)

- The parasympathetic supply to the gut is divided into **cranial** and **sacral** divisions.
- The cranial parasympathetic nerve fibers are almost entirely in the vagus nerves.
- The esophagus, stomach, pancreas and the intestines down through the first half of the large intestine are innervated by vagus nerves except mouth pharynx.

- The distal half of the large intestine and the anus are innervated by the **sacral** parasympathetics which originate in 2nd, 3rd and 4th sacral segments of spinal cord & pass through the pelvic nerves (to execute the defecation reflexes).

- The postganglionic neurons of the gastrointestinal parasympathetic system are located mainly in the myenteric and submucosal plexuses. More extensive near the oral cavity and anus. Stimulation of these parasympathetic nerves causes general increase in activity of the entire enteric nervous system.

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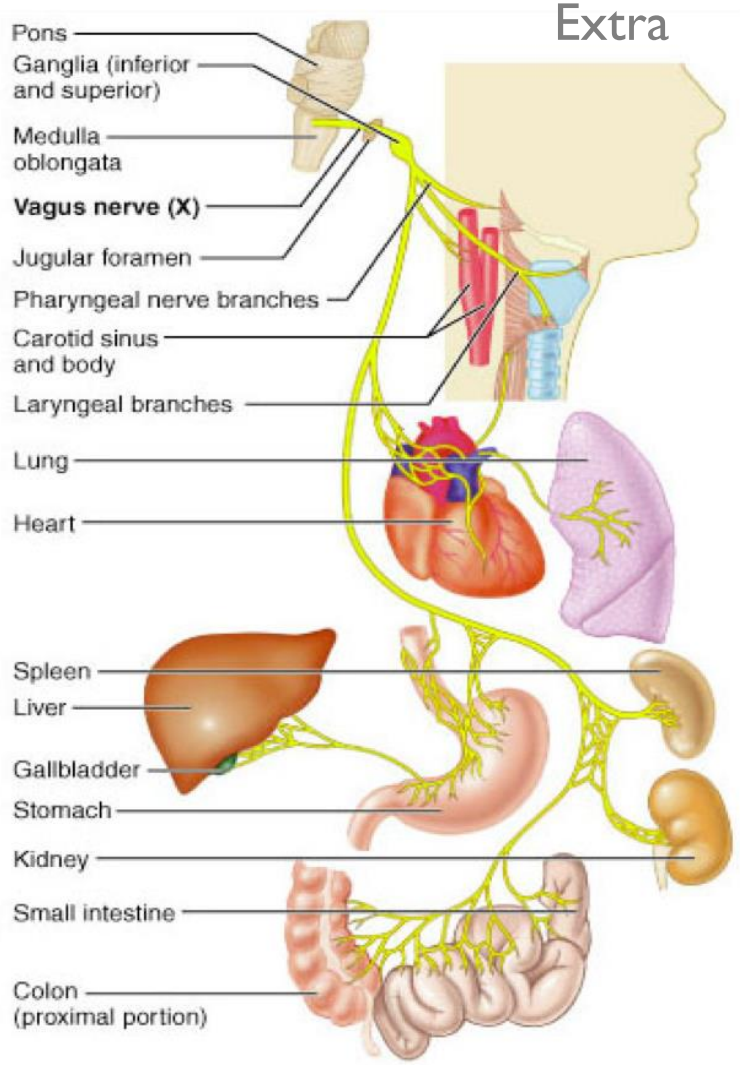

# Cont.

## II. Sympathetic Innervation:

- The sympathetic fibers to the gastrointestinal tract originate in the spinal cord between segments T-5 and L-2.
- Enter sympathetic chain then pass to ganglia: Celiac ganglia, Superior mesenteric ganglia, Inferior mesenteric ganglia.
- The sympathetics innervate essentially all of the GI tract, rather than being more extensive nearest the oral cavity and anus as is true of the parasympathetics.
- The sympathetic nerve endings secrete mainly norepinephrine.
- Stimulation of the sympathetic nervous system inhibits activity of the GI. Strong stimulation of the sympathetic system can inhibit motor movements of the gut so greatly that this literally can block movement of food through the gastrointestinal tract.
- Postganglionic fibers spread to all parts of the gut.
- Stimulation inhibit intestinal tract smooth muscle (except mucosal muscle)

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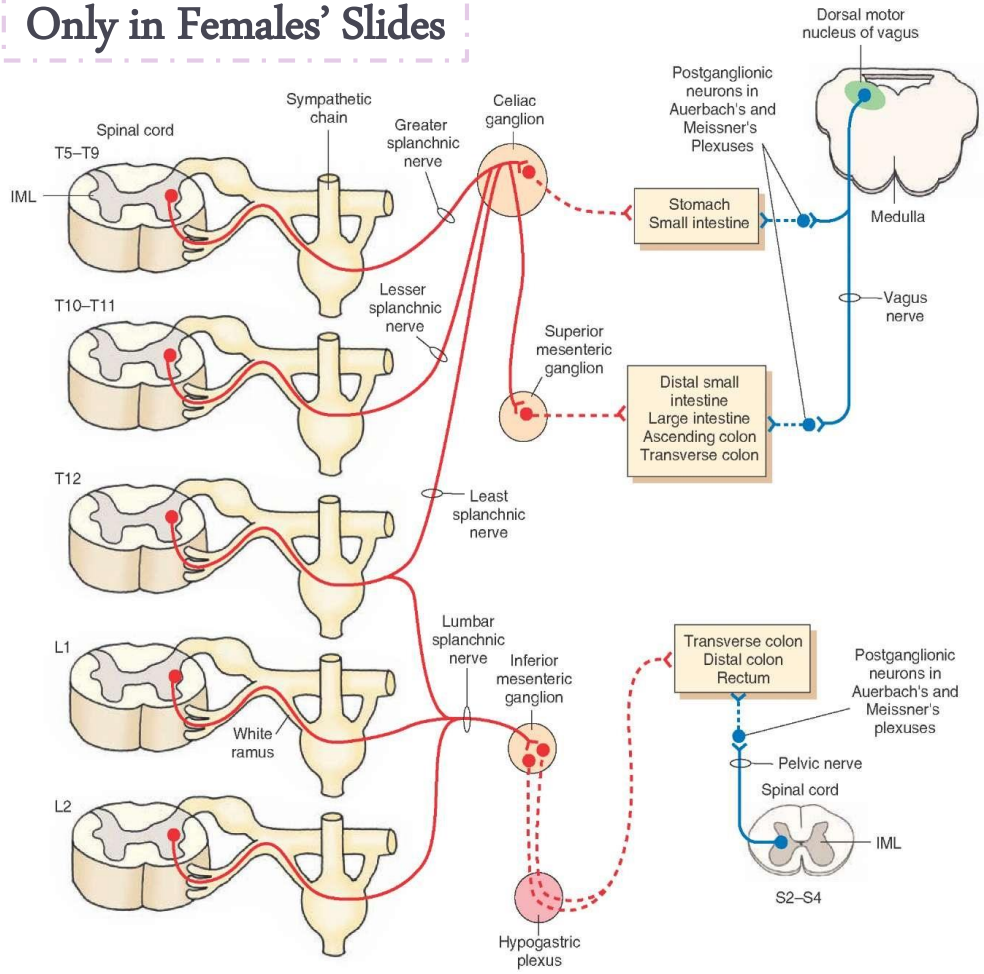

# Cont.

## III. Afferent Sensory Nerve Fibers from the Gut:

- Many afferent sensory nerve fibers innervate the gut. Some of them have their cell bodies in the enteric nervous system and some in the dorsal root ganglia of the spinal cord.

- These sensory nerves can be stimulated by:
- irritation of the gut mucosa,
- excessive distention of the gut
- presence of specific chemical substances in the gut.

- Signals transmitted through the fibers can then cause excitation or inhibition of intestinal movements or intestinal secretion.

- Other sensory signals from the gut go all the way to multiple areas of the spinal cord and even the brain stem. For example, 80% of the nerve fibers in the vagus nerves are afferent rather than efferent. These afferent fibers transmit sensory signals from the gastrointestinal tract into the brain medulla, which in turn initiates vagal reflex signals (vagovagal reflexes).

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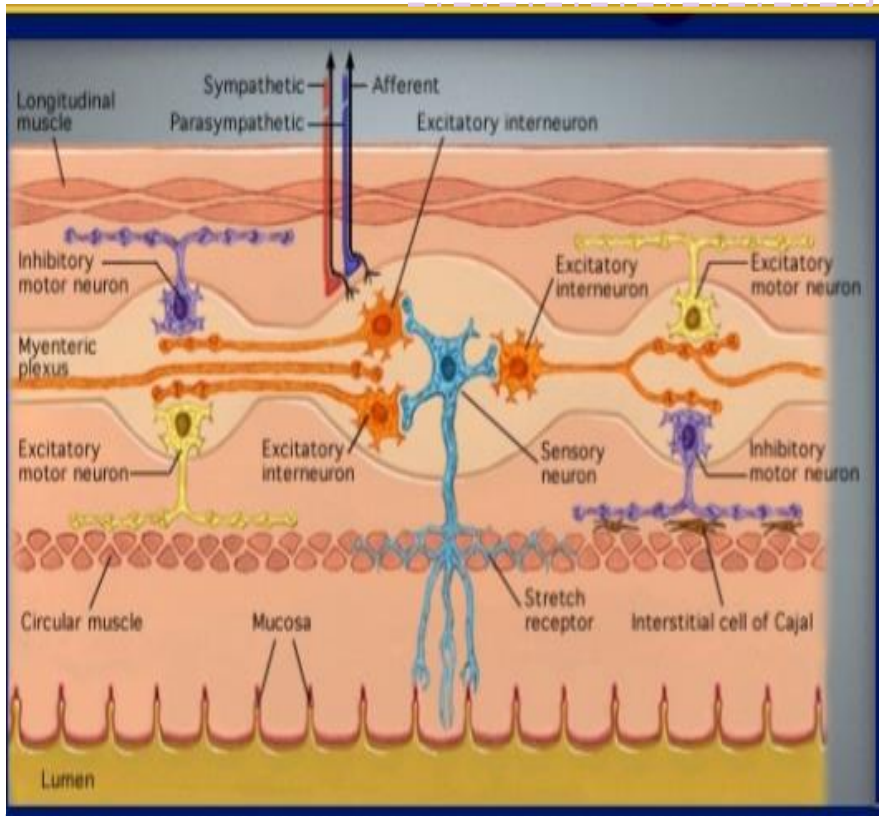

# Gastrointestinal Reflexes

*Beyond entericnervous system.
**Vagovagal both afferent and effernt by vagus.
All events in GIT are regulated by reflexes.

- The anatomical arrangement of the enteric nervous system and its connections with the sympathetic and parasympathetic systems support three types of gastrointestinal reflexes that are essential to gastrointestinal control. They are the following:

| 1. Reflexes that are integrated entirely within the gut wall enteric nervous system. | 2. Reflexes from the gut to the prevertebral sympathetic ganglia and then back* to the gastrointestinal tract. | 3. Reflexes from the gut to the spinal cord or brain stem and then back to the gastrointestinal tract. |
|---|---|---|
| Controls secretion, peristalsis, mixing contractions and local inhibition | These reflexes transmit signals long distances to other areas of the gastrointestinal tract.<br>1. the gastrocolic reflex: signals from the stomach to the colon= stimulate evacuation of the colon.<br>قاسترو يعني معدة، كولك يعني قولون- فالإسم المركب يعني أنها جاية سينسوري من المعدة ورايحة موتور للقولون وهكذا لباقي الأسماء.<br>2. the enterogastric reflexes: signals from the colon and small intestine to inhibit stomach motility and stomach secretion.<br>3. the colonoileal reflex: reflexes from the colon to inhibit emptying of ileal contents into the colon. | 1. **Reflexes from the stomach and duodenum to the brain stem and back to the stomach—by way of the vagus nerves—to control gastric motor and secretory activity<br>2. Pain reflexes that cause general inhibition of the entire gastrointestinal tract<br>3. Defecation reflexes that travel from the colon and rectum to the spinal cord and back again to produce the powerful colonic, rectal, and abdominal contractions required for defecation (the defecation reflexes). |

# Hormonal Control of Gastrointestinal Motility (GI Peptides)

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احفظوا الجدول زي اسمكم

| Hormone | Site of secretion | Stimuli for secretion | Actions |
|---|---|---|---|
| Gastrin | G cells of the antrum, duodenum and jejunum. | • Protein<br>• Distention of the stomach<br>• Vagal stimulation (GRP)<br>• Acid inhibits release | Stimulates: gastric $H^+$ secretion and growth of gastric mucosa. |
| Cholecystokinin (CCK) | I cells of the duodenum, jejunum, and ileum. | • Protein<br>• Fatty acids<br>• Acids | Stimulates: pancreatic enzyme secretion, pancreatic $HCO_3^-$ secretion, gallbladder contraction, growth of the exocrine pancreas, and relaxation of the sphincter of oddi.<br>Inhibits: gastric emptying. |
| Secretin | S cells of the duodenum, jejunum, and ileum | • Acids and fat in the duodenum. | Stimulates: pepsin secretion, pancreatic $HCO_3^-$ secretion,biliary $HCO_3^-$ secretion, and growth of the exocrine pancreas.<br>Inhibits: gastric $H^+$ secretion. |
| Glucose-dependent insulinotropic peptide (GIP) | K cells of the duodenum and jejunum. | • Protein<br>• Fatty acids<br>• Oral glucose | Stimulates: insulin secretion from pancreatic β cells.<br>Inhibits: gastric $H^+$ secretion. |
| Motilin | M cells of the duodenum and jejunum | • Fat<br>• Acid<br>• Nerve | Stimulates:<br>• Gastric motility<br>• Intestinal motility |

# Hormonal control

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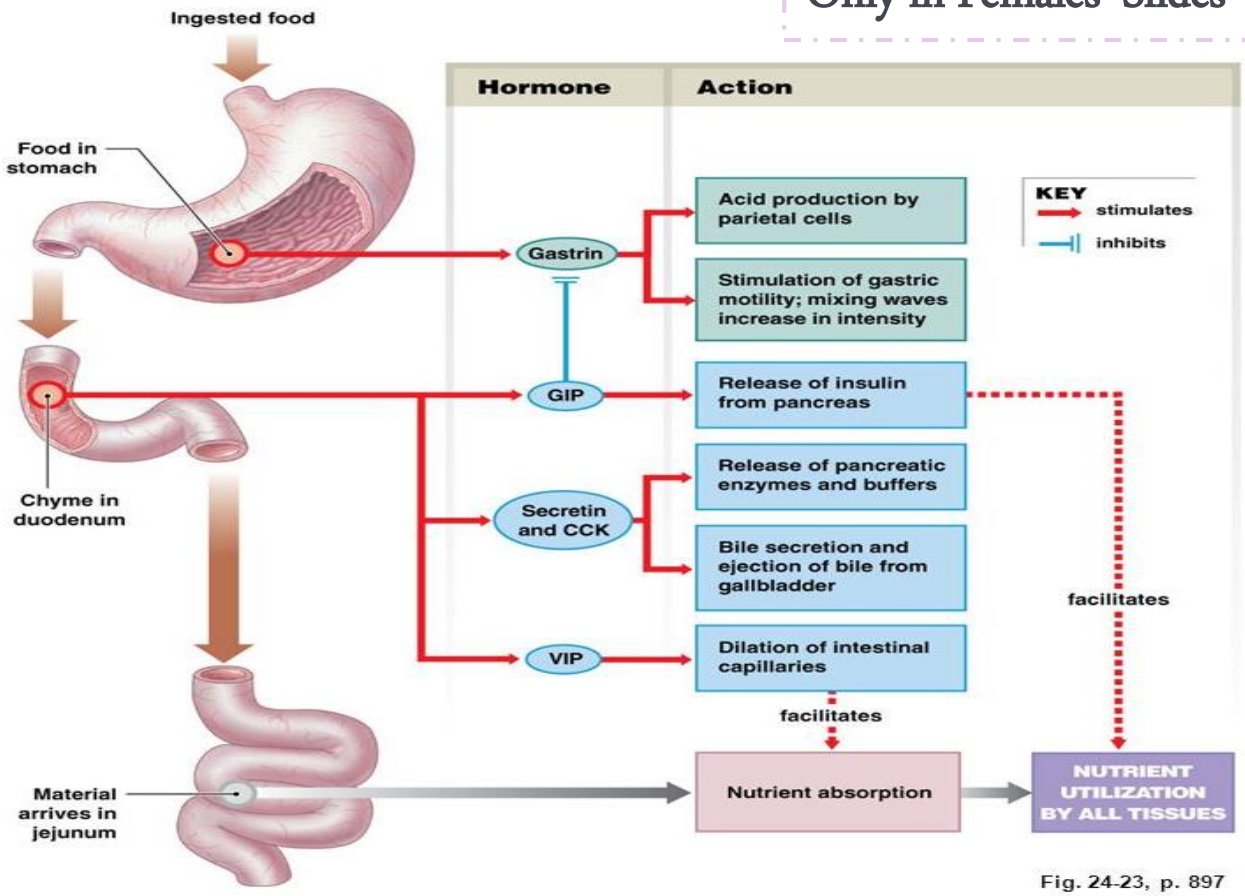


Fig. 24-23, p. 897

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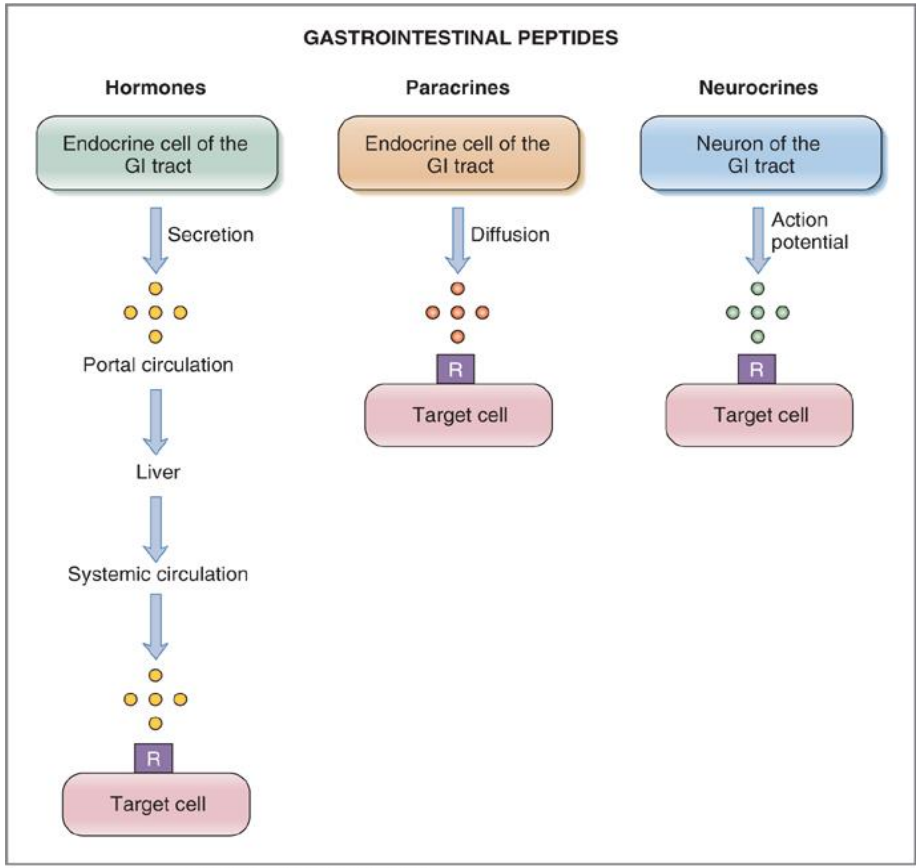

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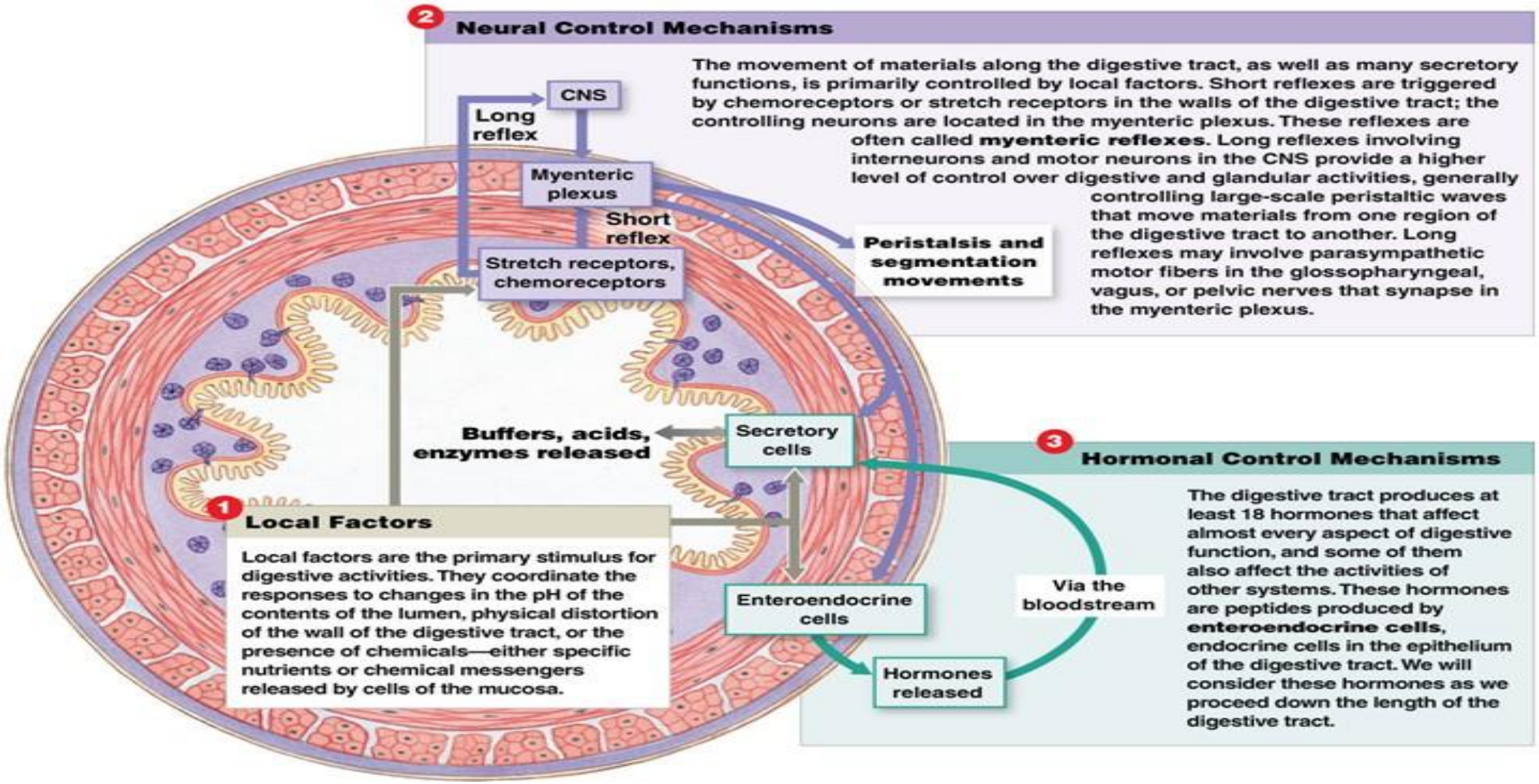

2 Neural Control Mechanisms
The movement of materials along the digestive tract, as well as many secretory functions, is primarily controlled by local factors. Short reflexes are triggered by chemoreceptors or stretch receptors in the walls of the digestive tract; the controlling neurons are located in the myenteric plexus. These reflexes are often called myenteric reflexes. Long reflexes involving interneurons and motor neurons in the CNS provide a higher level of control over digestive and glandular activities, generally controlling large-scale peristaltic waves that move materials from one region of the digestive tract to another. Long reflexes may involve parasympathetic motor fibers in the glossopharyngeal, vagus, or pelvic nerves that synapse in the myenteric plexus.
CNS
Long reflex
Myenteric plexus
Short reflex
Stretch receptors, chemoreceptors
Peristalsis and segmentation movements
Buffers, acids, enzymes released
Secretory cells
3 Hormonal Control Mechanisms
The digestive tract produces at least 18 hormones that affect almost every aspect of digestive function, and some of them also affect the activities of other systems. These hormones are peptides produced by enteroendocrine cells, endocrine cells in the epithelium of the digestive tract. We will consider these hormones as we proceed down the length of the digestive tract.
Via the bloodstream
Enteroendocrine cells
Hormones released
1 Local Factors
Local factors are the primary stimulus for digestive activities. They coordinate the responses to changes in the pH of the contents of the lumen, physical distortion of the wall of the digestive tract, or the presence of chemicals—either specific nutrients or chemical messengers released by cells of the mucosa.

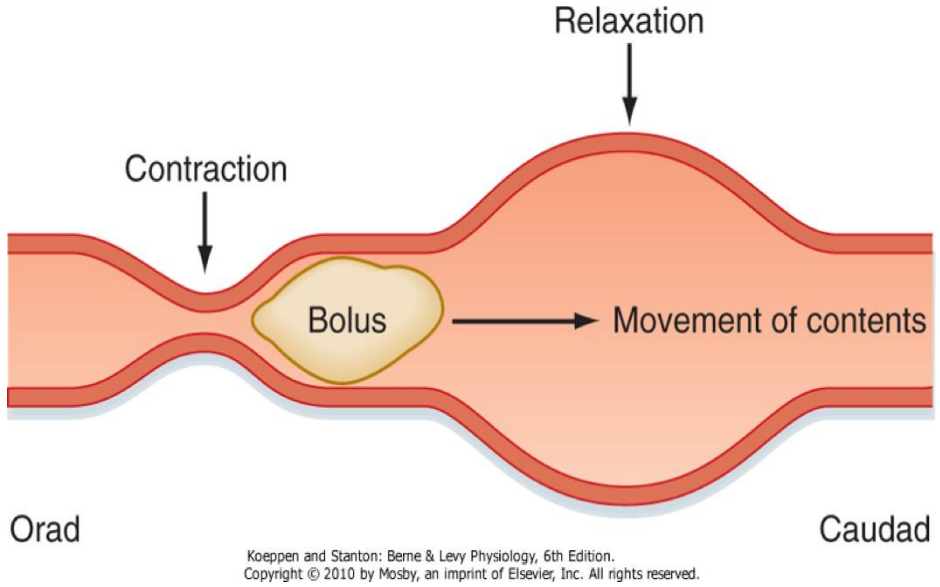
# Functional Types of Movements in the GIT

- Two types of movements occur in the GI tract:

Animation

1. Propulsive Movements – Peristalsis:(Doesn't care about absorption only cares about moving the food)

- Peristalsis which is inherent property of syncytial smooth muscle tube.
- Organizes propulsion of material over variable distances within the GI lumen.
- Usual stimulus is **distention**. Other stimuli that can initiate peristalsis include chemical or physical irritation of the epithelial lining in the gut. And Stimulation of enteric nervous system
- Myenteric plexus is important !! ( can not occur without it).
- Atropine (cholinergic blocker) depresses propulsion.
- Contractile ring moves forward.
- Contractile ring 2-3 Cm behind stimulus.
- Receiving segment : 1. contraction (longitudinal M) 2. relaxation (circular M).
- Propulsive segment: 1. contraction (circular M). 2. relaxation (longitudinal M).





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Peristaltic Reflex and the "Law of the Gut".

When a segment of the intestinal tract is excited by distention and thereby initiates peristalsis, the contractile ring causing the peristalsis, which normally begins on the Orad side of the distended segment and moves toward the distended segment, pushing the intestinal contents in the anal direction (Caudad direction) for 5 to 10 cm before dying out.

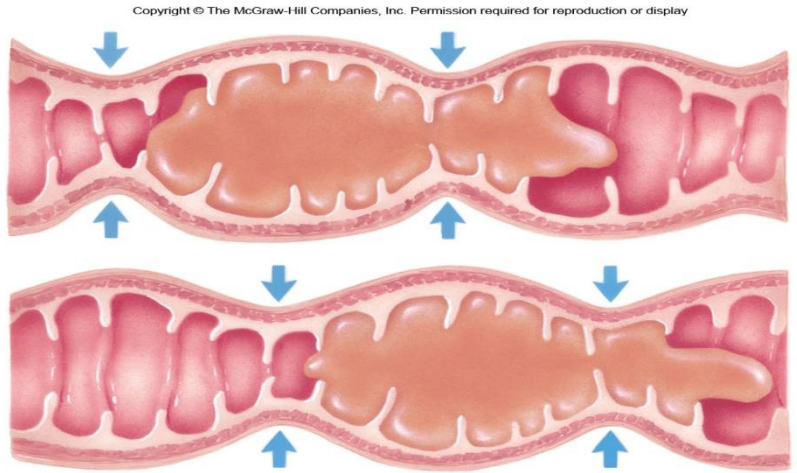
# Cont.

2. Mixing movements (segmentation):

- Provides mixing of intestinal contents (know as chime) with digestive juices (Blend different juices with the chime).
- Bring products of digestion in contact with absorptive surfaces.
- Segment of bowel contracts at both ends.
- A second contraction occurs in the center of the segment.
- Chime is forced forward and backward.
- Can occur independent of central input.
- Cares about absorptions.

Animation





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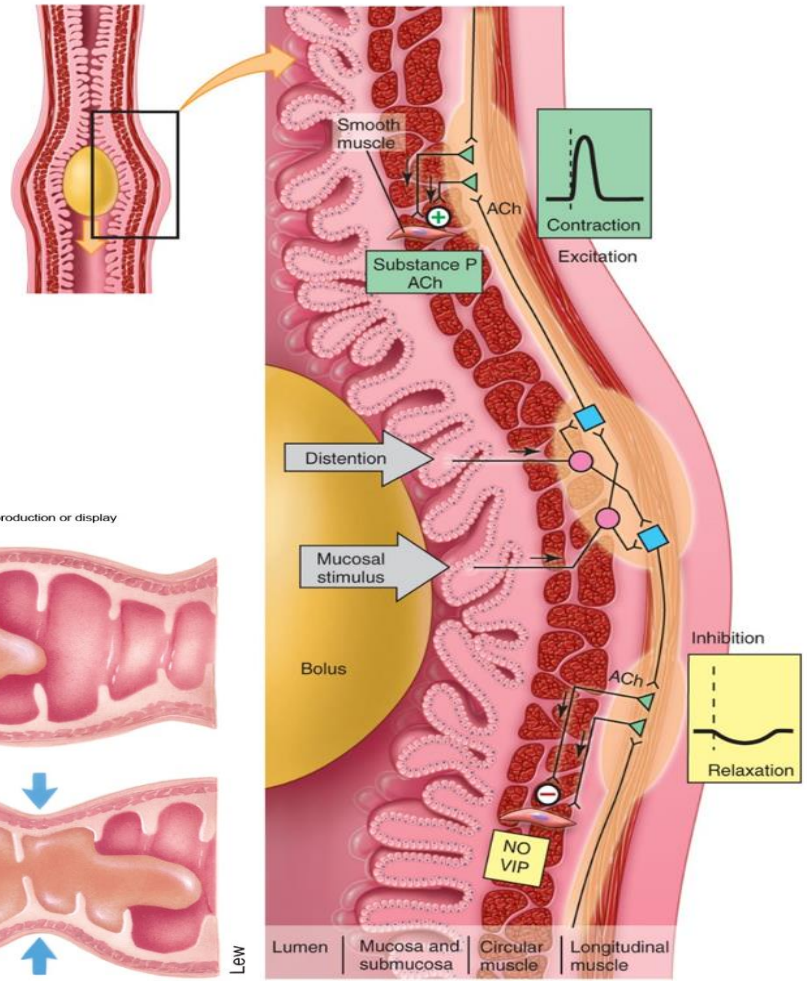
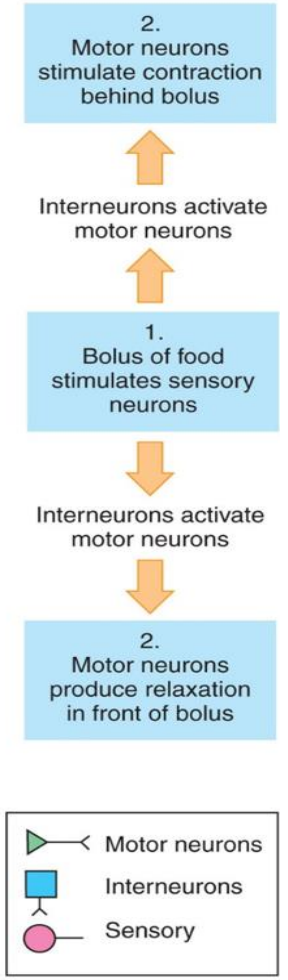
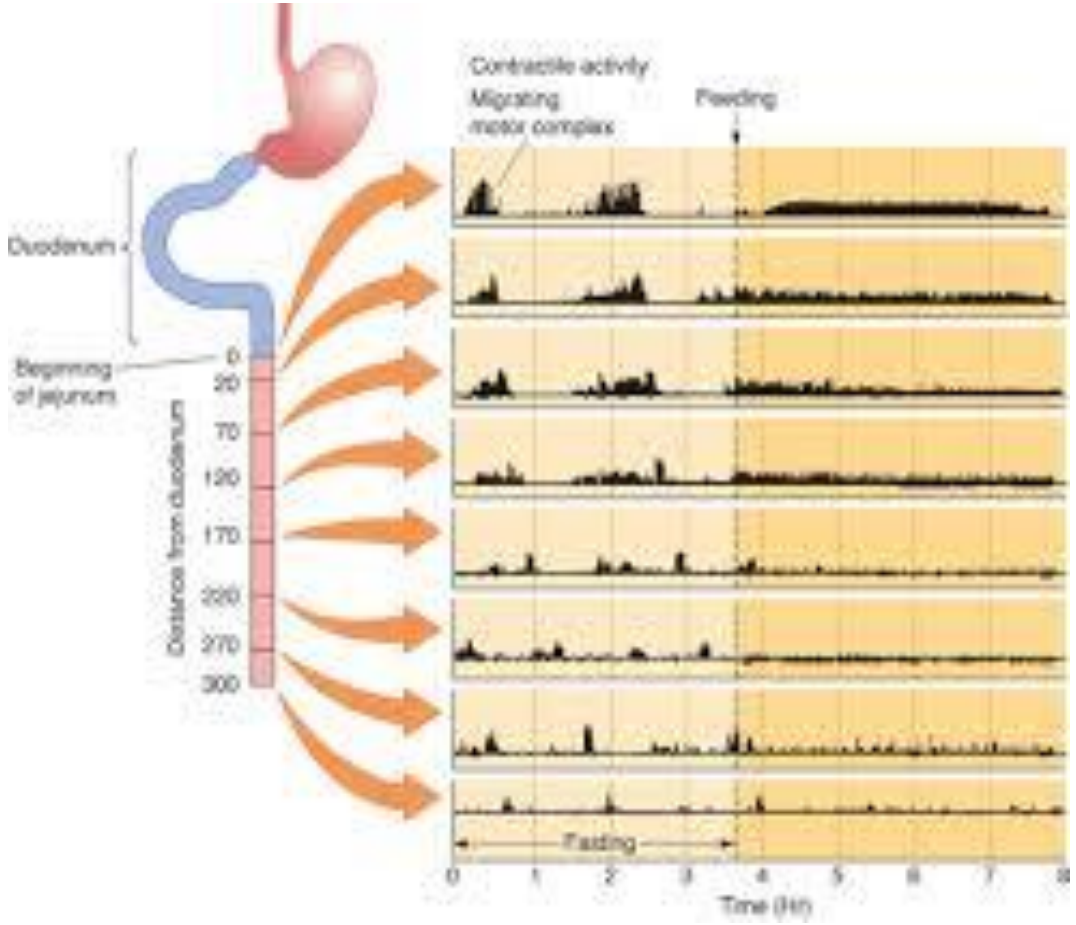


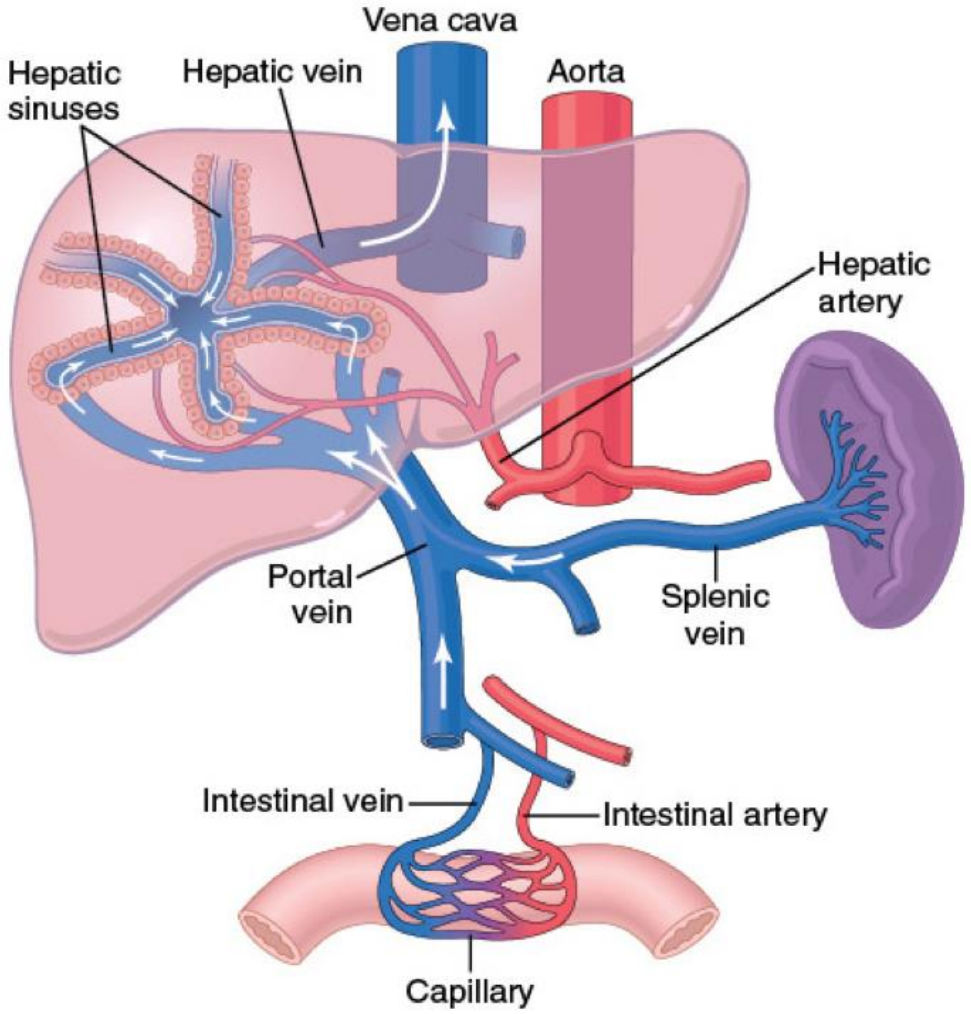
# Cont.

- Migrating motor complex (MMC):
- Cycles of motor activity migrate from the stomach to the distal ileum.
- Have 3 phases:
  - Phase1 quiescence.
  - Phase2 irregular electrical & mechanical activity.
  - Phase3 burst of regular activity.
- Occurs during fasting.
- Initiated by motilin.
- Migrate at a rate of 5cm/min.
- Occurs at intervals of ~ 90 min.
- Accompanied by increase gastric, bile and pancreatic secretion.
- Serve to clear the stomach and small intestine of luminal contents.



# Gastrointestinal Blood Flow "Splanchnic Circulation"

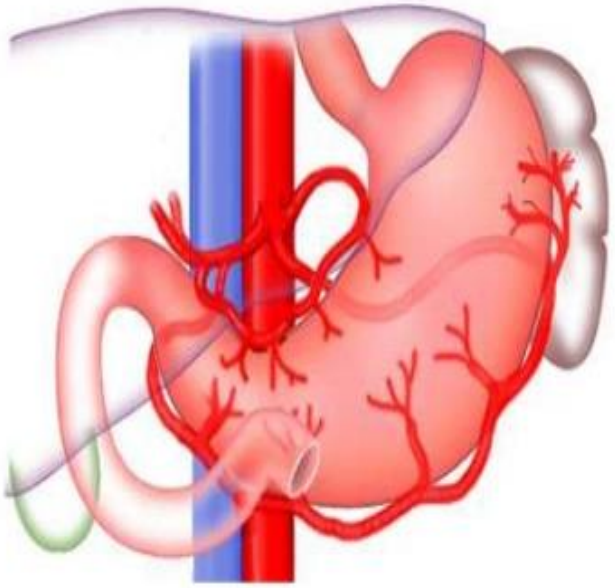
- Splanchnic circulation includes the blood flow through the gut itself plus blood flows through the spleen, pancreas, and liver.
- The design of this system is such that all the blood that courses through the gut, spleen, and pancreas then flows immediately into the liver by way of the portal vein.
- In the liver, the blood passes through millions of minute liver sinusoids and finally leaves the liver by way of hepatic veins that empty into the vena cava of the general circulation.

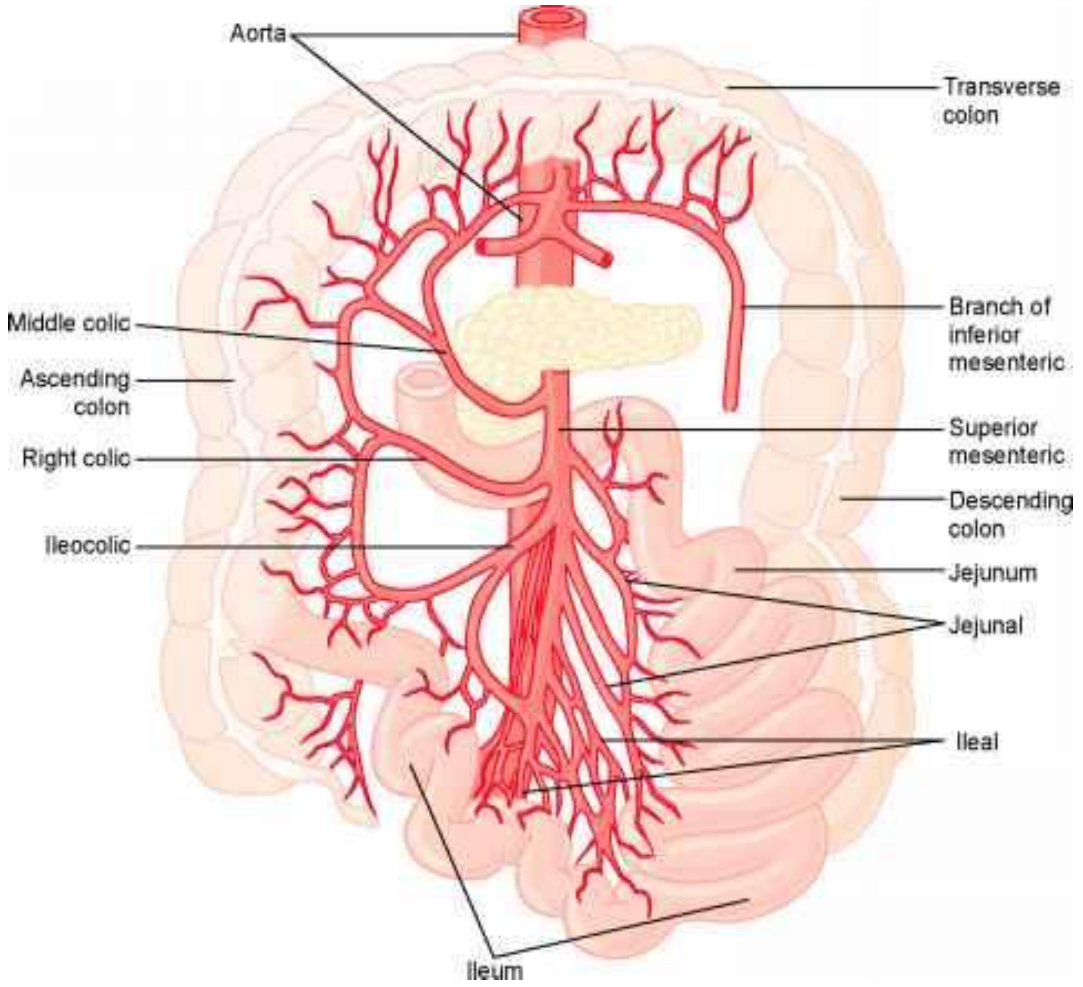


# Cont.

## Celiac Trunk

- It arises from the abdominal aorta immediately below the aortic hiatus of the diaphragm anterior to the upper part of vertebra LI.
- It divides into the:
  - ***left gastric artery,***
  - ***splenic artery,***
  - ***common hepatic artery***.





# Effect of Gut Activity and Metabolic Factors on Gastrointestinal Blood Flow

**Possible Causes of the Increased Blood Flow During Gastrointestinal Activity**

Most of vasodilators such as the peptide hormones, including cholecystokinin (CCK), vasoactive intestinal peptide (VIP), gastrin, and secretin.

Some of the GI glands release into the gut wall two kinins, kallidin and bradykinin (vasodilators).

- Decreased oxygen concentration in the gut wall can increase intestinal blood flow at least 50 to 100 percent.
- Four folds increase in adenosine (vasodilator) secretion due to decrease oxygen.

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- Blood flow in GIT is directly related to local activity
- Blood flow in villi during active absorption increased up to 8 folds
- After a meal blood flow increases greatly then return to resting level over 2-4 hrs

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# Nervous Control of Gastrointestinal Blood Flow

- Stimulation of the parasympathetic nerves going to the stomach and lower colon increases local blood flow at the same time that it increases glandular secretion.

- Sympathetic stimulation, by contrast, has a direct effect on essentially all the gastrointestinal tract to cause intense vasoconstriction of the arterioles with greatly decreased blood flow. But the local metabolic vasodilator mechanisms override the sympathetic vasoconstriction effects, returning the normal blood flow to GI muscle and glands.

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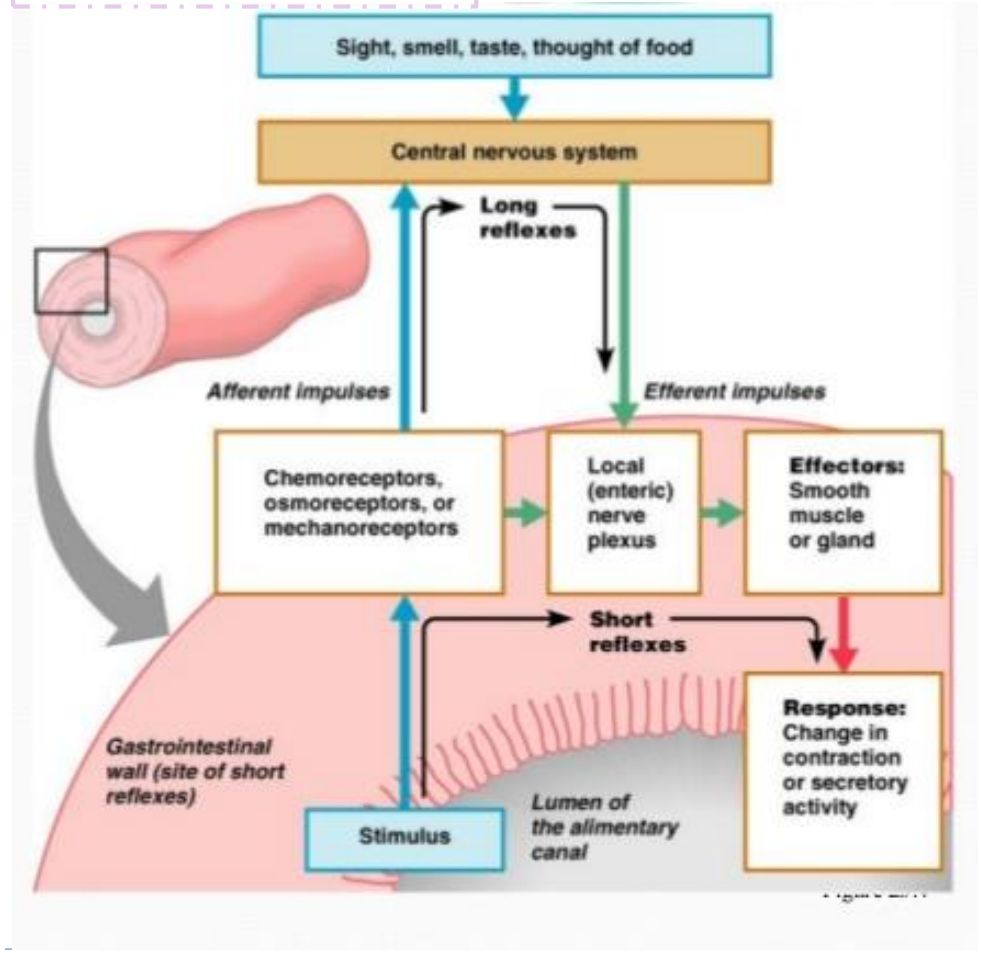

# Thank you!

اعمل لترسم بسمة، اعمل لتمسح دمعة، اعمل و أنت تعلم أن الله لا يضيع أجر من أحسن عملا.

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QUIZ

اقتراحات وشكاوي


**References:**

- 2017-2018 Dr. Hana Alzamel's Lecture.
- 2017-2018 Dr. Mohammed Al Zoghaibi's Lecture.
- Guyton and Hall Textbook of Medical Physiology (Thirteenth Edition.)




اللهم اني استودعتك ما حفظت وما قرأت وما فهمت، فرده لي وقت حاجتي إليه يا من لا تضيع عنده الودائع.