

# REGULATING DIGESTION: CNS AND ENTERIC NERVOUS SYSTEM (ENS)

← جهاز عصبي خاص في GI (تقسيم عدد الاغصان الموجود في spinal cord)

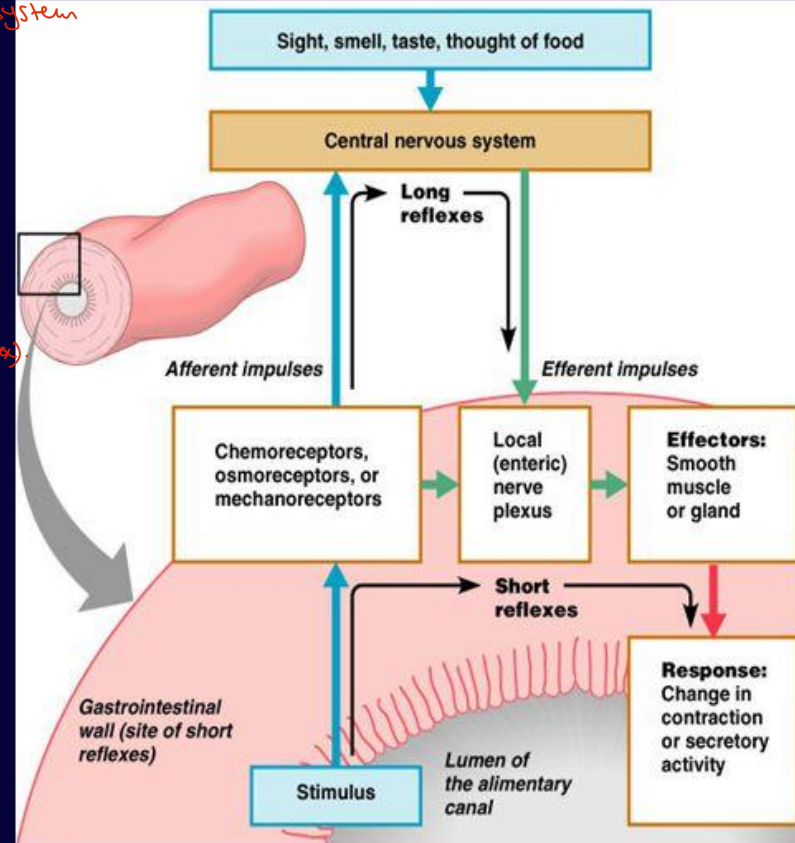
# Nervous Control of the GI Tract

- Intrinsic controls → enteric nervous system

- Nerve plexuses near the GI tract initiate short reflexes
- Short reflexes are mediated by local enteric plexuses (gut brain)

- **Extrinsic controls** → Brain (long reflex)

- Long reflexes arising within or outside the GI tract
- Involve CNS centers and extrinsic autonomic nerves
- Parasympathetic reflexes



# Regulating Digestion: CNS and Enteric Nervous System (ENS)

- **Extrinsic** → راجع الى الـ Brain  
والجهاز العصبي المركزي  
↓

- a. **Autonomic:**

1. parasympathetic: enhance GI function → mainly
2. sympathetic: inhibit GI function (لا نه نعيم الـ adrenaline)  
↓  
بجهاز هاضمة عصبية

- b. **Voluntary:**

Voluntary to skeletal muscles in oral cavity

Voluntary to external anal sphincter → somatic

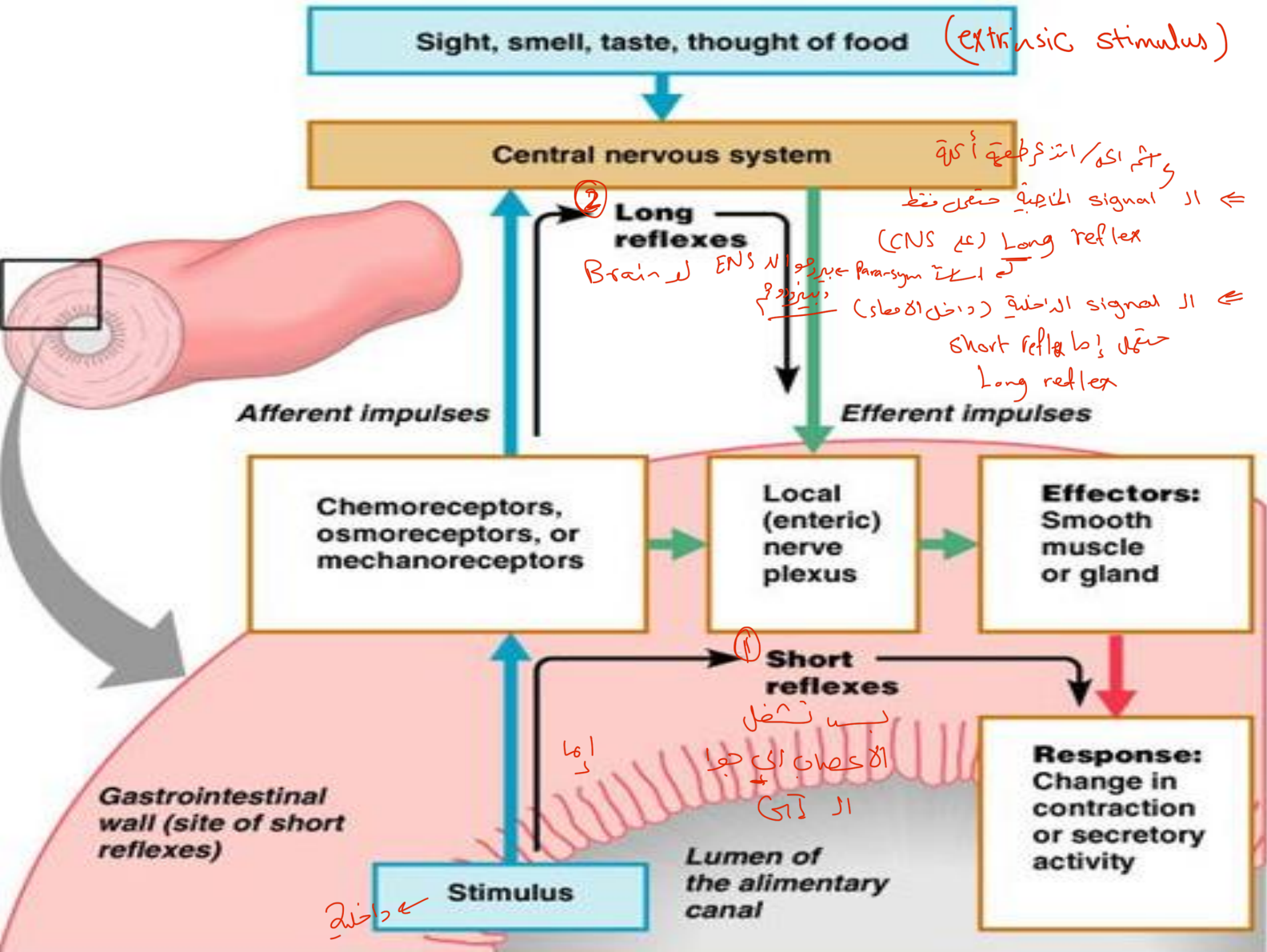
صايفترافق  
فوق غير  
في منطقة

- **Intrinsic (Enteric)**

- a. **Myenteric plexus**: control mainly GI movement

- b. **Submucosal plexus**: control mainly GI secretion and blood flow

له تفرقة بين الـ B. V. و الـ S. V.  
والجهاز العصبي المركزي



# Regulating Digestion: CNS and Enteric Nervous System (ENS)

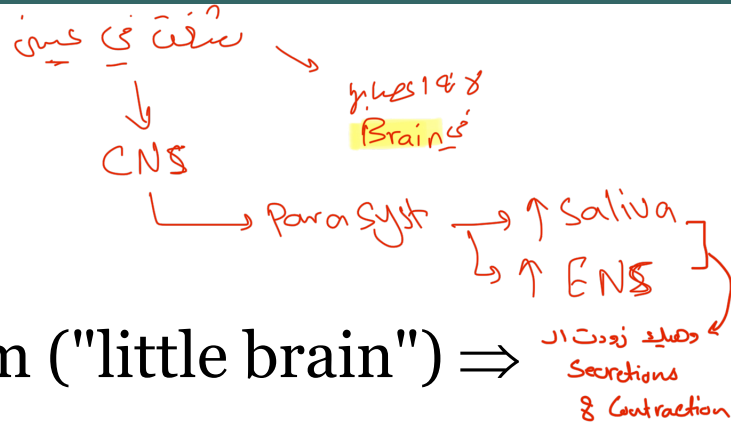
- Long (Cephalic) Reflexes:

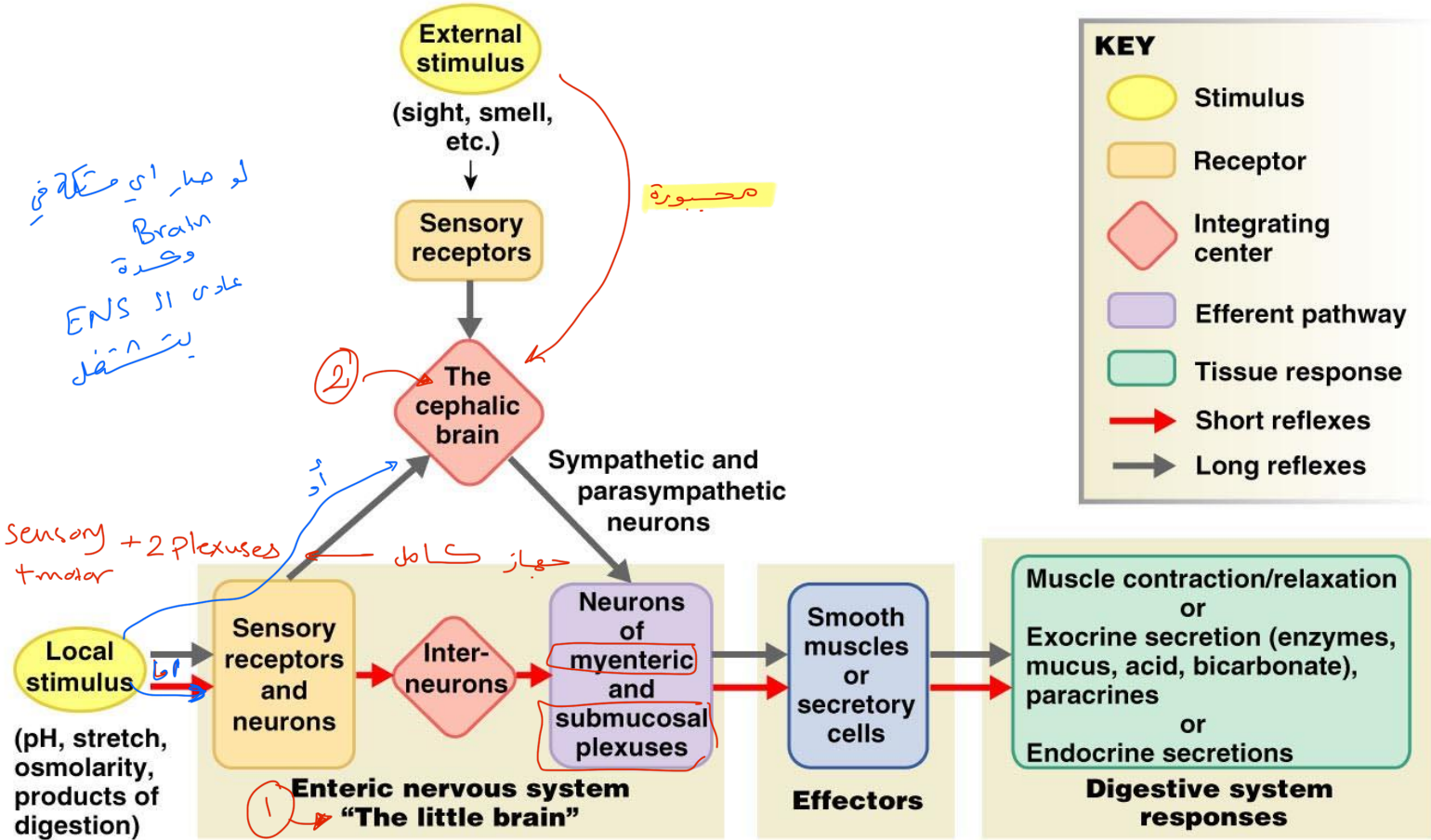
- Short reflexes:

Enteric nervous system ("little brain") ⇒  
Effect through the submucosal nervous plexus

- GI Peptides:

peptides secreted by GIT act as hormones or  
paracrines: inhibit or excite motility & secretions





# Regulating Digestion: CNS and Enteric Nervous System (ENS)

- Some reflexes originate outside the GIT: Feed Forward (stimuli: sight, smell, thought of food)

**Emotional Reflexes** (outside GIT)

- Some reflexes originate in the GIT:

**Enter-gastric reflex** (signal from colon or small intestine to inhibit stomach motility and secretion)

**Gastro-colic reflex** (signal from stomach to cause evacuation of colon)

Small intestine  
inhibit stomach  
↓  
emptying  
↓  
defecation  
↓  
gastric reflex  
↓  
defecation  
↓  
paristaltic movement  
↓  
enteric nervous system

# The gastrocolic reflex

Controlled by the **enteric nervous system**

It senses the stretch of the stomach when food arrives into it

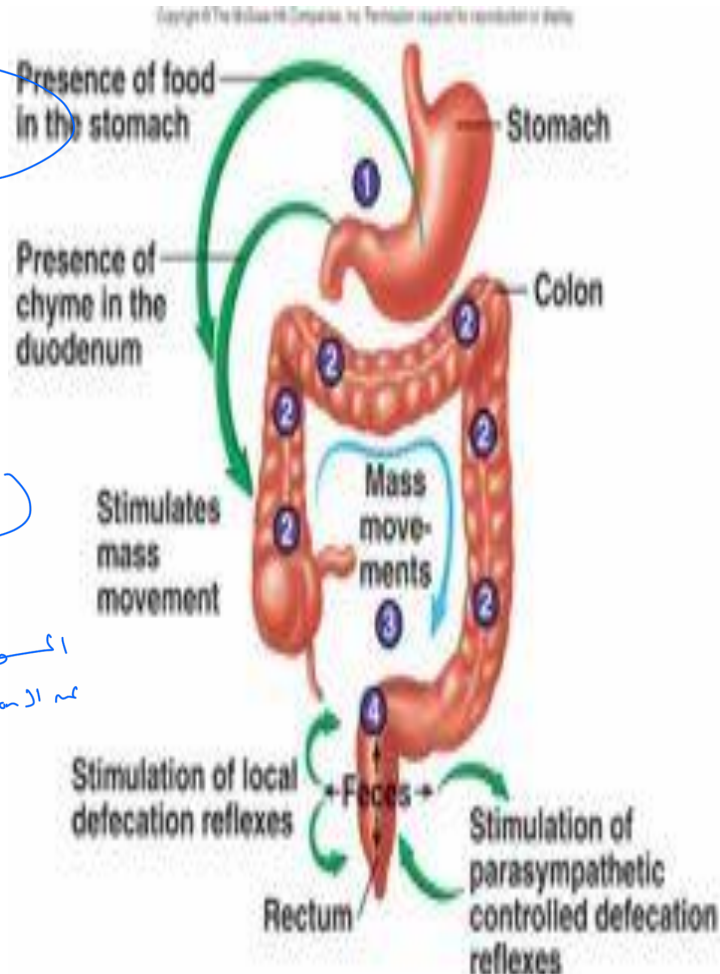
This is sensed via afferent nerves in the stomach when send impulses to the enteric nervous system

The enteric nervous system then sends efferent signals to the **colon** causing it to **increase its peristalsis**

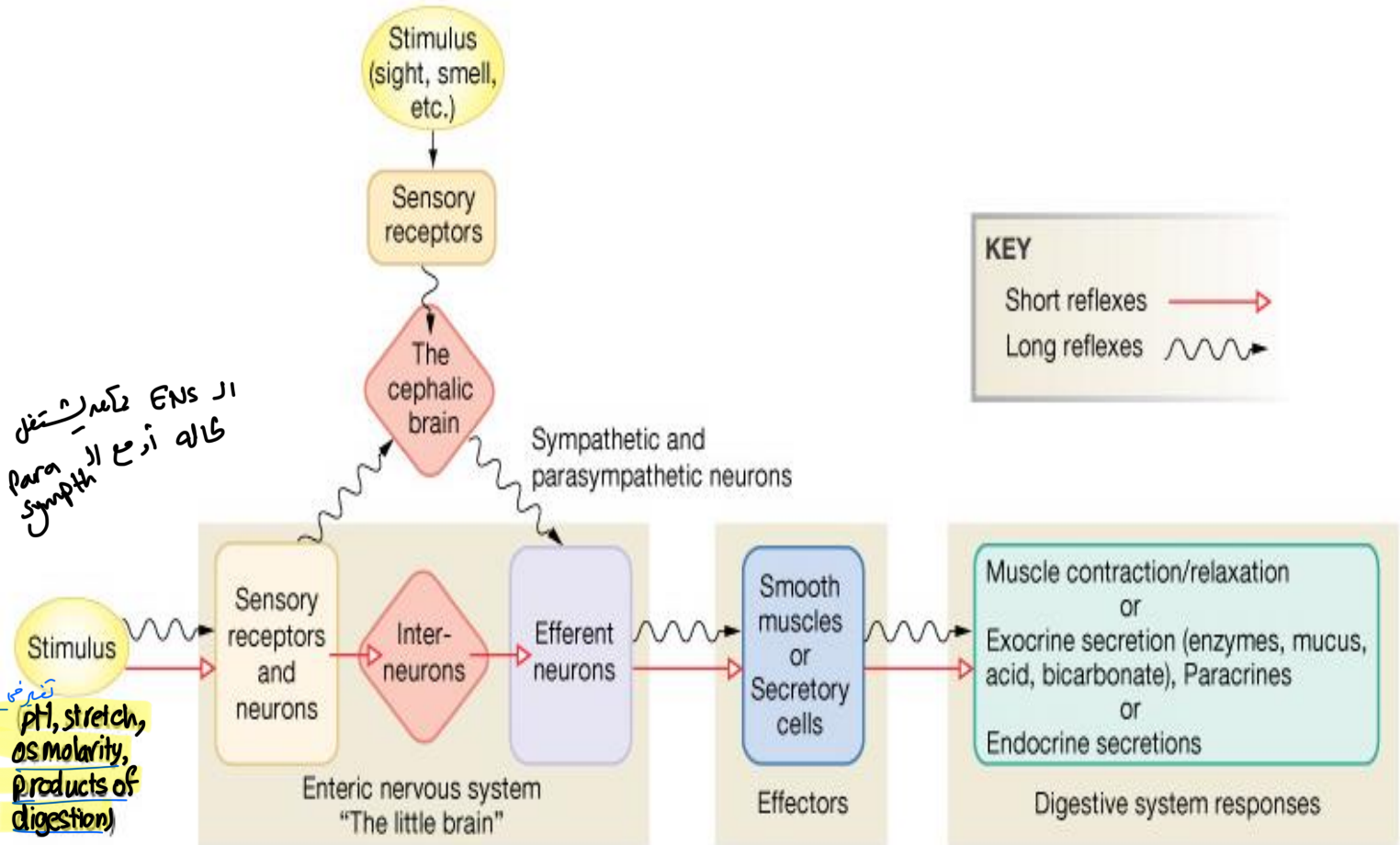
This moves food further down the colon to **make space for the new food coming in** → mass movements

This is why after we eat a meal there can be the stimulation for

**defecation**



# Regulating Digestion: CNS and Enteric Nervous System (ENS)



# Neural control of the GI system

The GI tract has its own nervous system called the **enteric nervous system**

It lies within the walls of the GI tract (beginning at the oesophagus and ending at the anus)

Contains the **same number of nerve cells as the spinal cord!!**

Made up of **two nerve plexus**

الذي يحفزه صمو ال. Parasympath.

**The myenteric plexus** - this lies between the longitudinal and circular smooth muscle layers

This controls **GI movements (peristalsis)**

When stimulated it causes:

- ① Increased **force of contraction** of the smooth muscle
- ② Increased **rate of contractions** - more rapid movements of the gut wall (PERISTALSIS)

①

Can be inhibited via substance **VASOACTIVE INTESTINAL POLYPEPTIDE (VIP)**

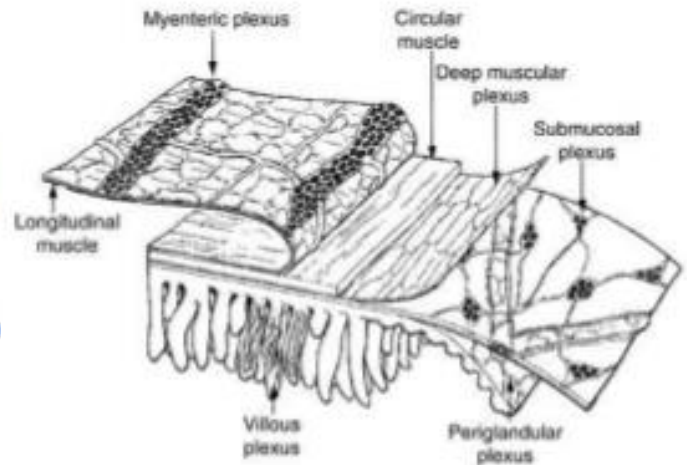
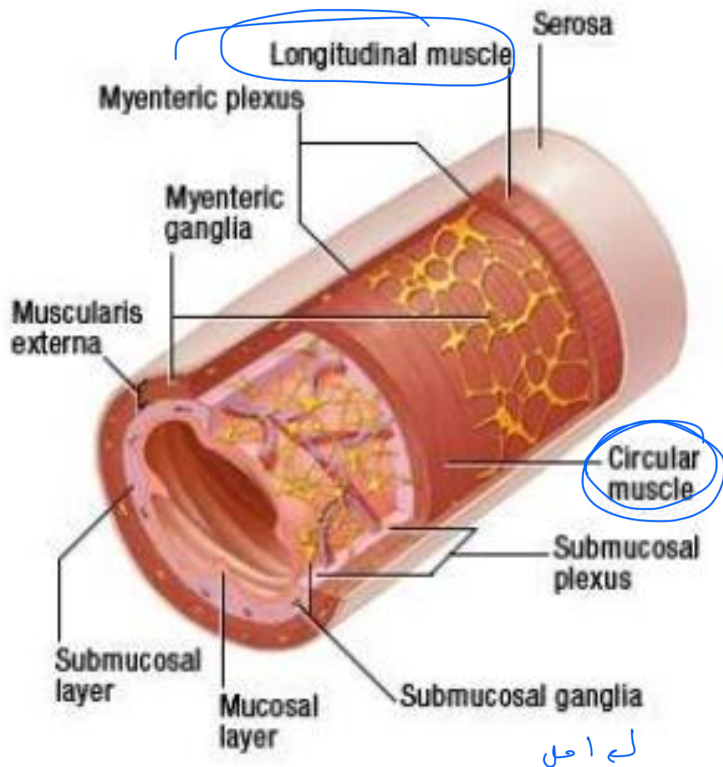
↳ ② sympathetic nerve

Used to stop muscle sphincters which inhibit the flow of blood from **contracting** - keeps them open!

The pyloric sphincter - opening from stomach to duodenum

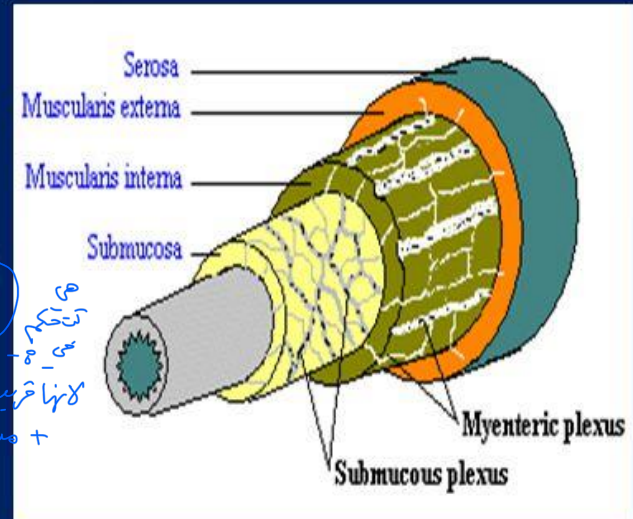
The ileocaecal valve - opening from small intestine to large intestine

# Enteric Nervous System: myenteric plexus & submucosal plexus



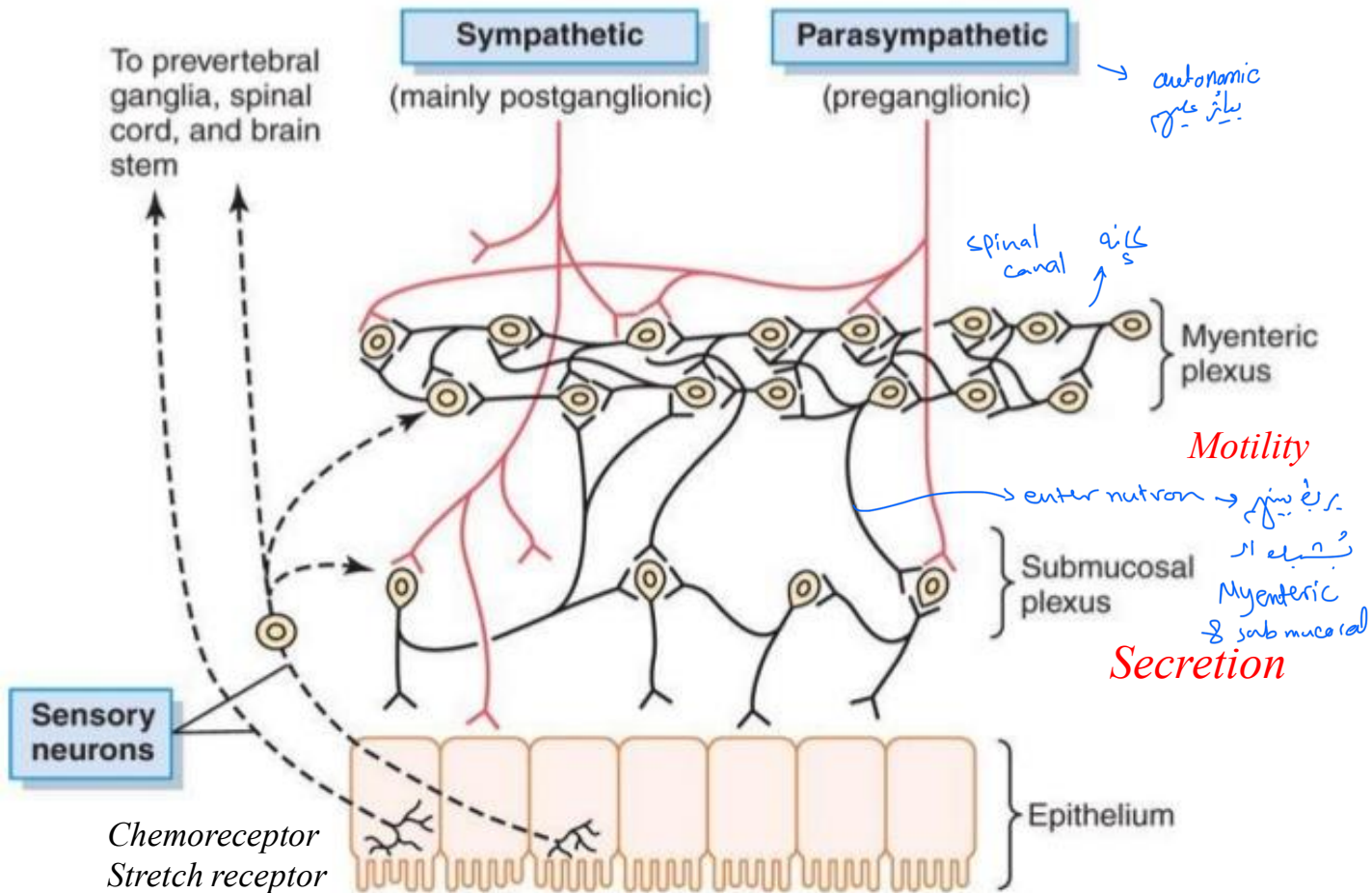
# Enteric Nervous System

- Intrinsic nervous system that directly controls the gastrointestinal system
- Composed of **two plexuses** of nerve cells and fibers located in the wall of gastrointestinal tract from the esophagus to the anal canal
- **Submucous or Meisner's plexus** lies in the **submucosa**, is mainly concerned with the control of the glands in the mucous membrane  
*Handwritten notes: "تتحكم في الغدد" (controls glands), "أجزاء قريبة من" (parts close to), "Blood vessels" (قنوات الدم), "Vasodilatory" (توسيع الأوعية), "Blood flow" (تدفق الدم)*
- **Myenteric or Aurbach's plexus** lies between the circular and the longitudinal muscle layer, controls the muscle and movements of the gut wall



Communicates with the CNS through the parasympathetic (eg, via the vagus nerve) and sympathetic (eg, via the prevertebral ganglia) nervous systems

حسب نوع ال Stimulate + كس



# Neural control of the GI system

**The submucosal plexus** - lies in the submucosa  
This controls **GI secretions** and local blood flow

Both plexus have <sup>①</sup>**sympathetic** and <sup>②</sup>**parasympathetic** inputs but it can function **ON THEIR OWN** without receiving stimulation from these nerves

← هذه يستقروا مع وجع  
او sympathetic او para  
فهمه يستقلوا بدمه اي تانيه من ال autonomic

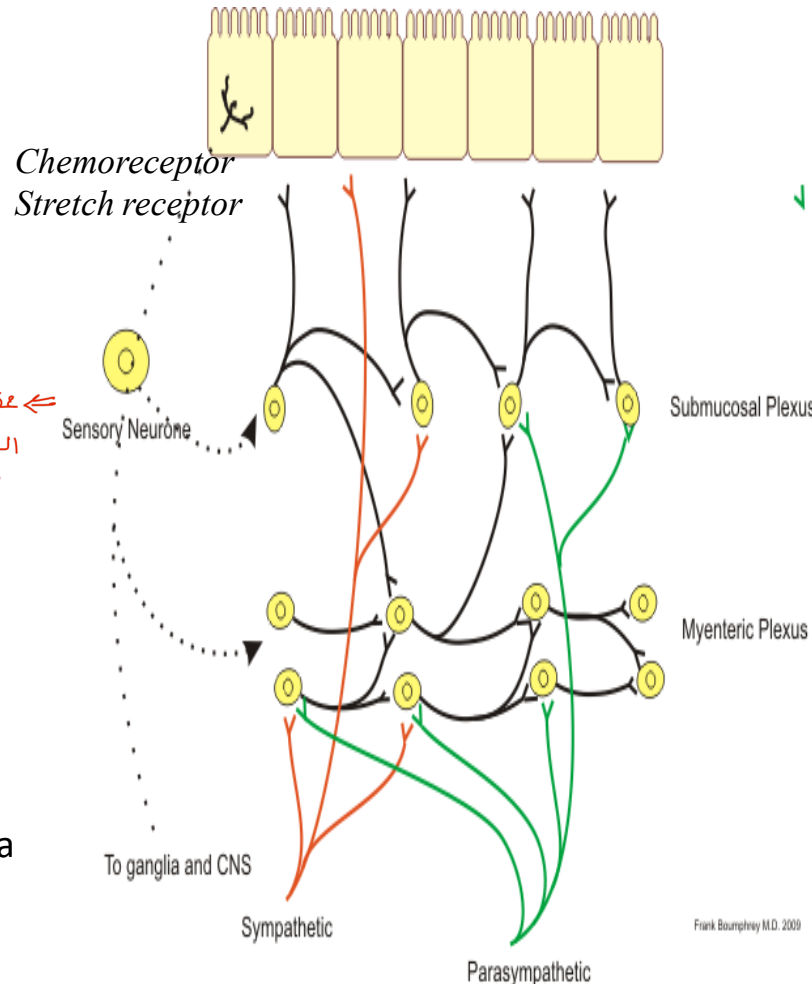
Sensory afferent nerves lie in the gut wall and are stimulated when:

**Irritation of gut wall**

**Distension of the gut wall**

**Presence of certain chemicals in the gut**

These nerves send signals which then cause inhibition or stimulation of the gut wall - via the brain in the medulla or locally via the enteric nervous system



# Electrical activity of gastrointestinal smooth muscle

## Electrical activity of the Gastrointestinal smooth muscle

Via 2 types of electrical waves

1. **Slow waves**
2. **Spikes**

### Slow waves

Most GI contractions occur rhythmically and the rhythm is determined by SLOW WAVES

These are small wave like changes in the resting membrane potential of the smooth muscle cells by 5-15 mV

### **THEY ARE NOT ACTION POTENTIALS**

Occurs at a range of 3-12 waves per minute - each wave causes a contraction

3 waves per minute in the stomach

12 waves per minute in the duodenum

9 waves per minute in the ileum

Exact cause of these waves is unknown - by likely to be due to pacemaker type cells called **CAJAL CELLS** that have ion channels which open - particularly Na channels which allow ions into the cell and K<sup>+</sup> out of the cell

# Membrane potential

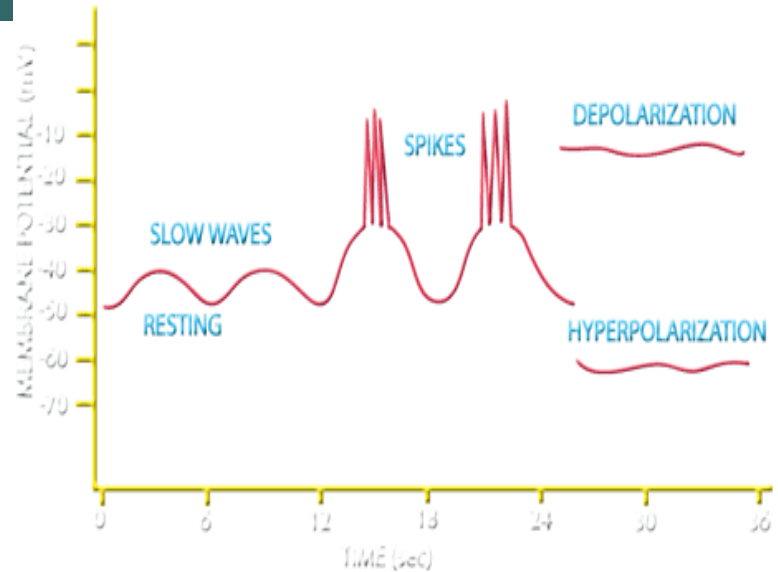
**Depolarisation**: When the action potential becomes **less negative** and the cells become **excitable**

**Hyper polarisation**: When the membrane potential becomes **more negative** and the cells become **less excitable**

Factors that make cells **excitable** (cause depolarisation):

1. **Stretching of the muscle**
2. **Parasympathetic stimulation (acetylcholine)**
3. **Stimulation by acetylcholine directly**
4. **Stimulation by certain GI hormones**

## MEMBRANE POTENTIAL



Things that make the cells **less excitable** (hyper polarisation)

1. **Adrenaline or noradrenaline**
2. **Sympathetic stimulation**

# Electrical activity of gastrointestinal smooth muscle

## Spike waves or spike potentials

**THESE ARE ACTION POTENTIALS** - because there is depolarisation of the cell membrane

Occur when the membrane potential of the smooth muscle cell becomes more positive

When the charge of the **slow wave rises above -40mV** - this **stimulates an action potential** and therefore a spike wave

The ion channels which cause the action potential in GI smooth muscle are **calcium-sodium channels** (this means Ca and Na ions move into the cell to change the charge from -ve to +ve. This is different from “normal” action potentials when only Na ions move into the cell)

These cause **long action potentials** (like in the heart) because of the slower Ca channels and therefore prolonged contraction of GI muscle

Spike waves occur when there is stimulation via efferent nerves directly from the enteric plexus to increase the membrane potential of the cells - called a **reflex mechanism**

Can be under control of the higher brain centers and autonomic nervous system

# Autonomic control of the GI tract

## Parasympathetic innervation:

2 divisions - the **cranial** and **sacral** divisions

**Cranial:** Via vagus nerve → innervates oesophagus, stomach, pancreas and first part of the large intestine

**Sacral:** Via pelvic nerves which originate from the **spinal cord** → innervates the distal part of the large intestine and anus

There are more parasympathetic nerves in the anal, rectal and sigmoid regions of the colon - to cause increase **contractions** of the smooth muscle here and promote defecation

Also causes **vasodilation** of GI blood vessels → which increases blood flow to the GI tract (remember opposite of fight or flight which wants to divert blood away)

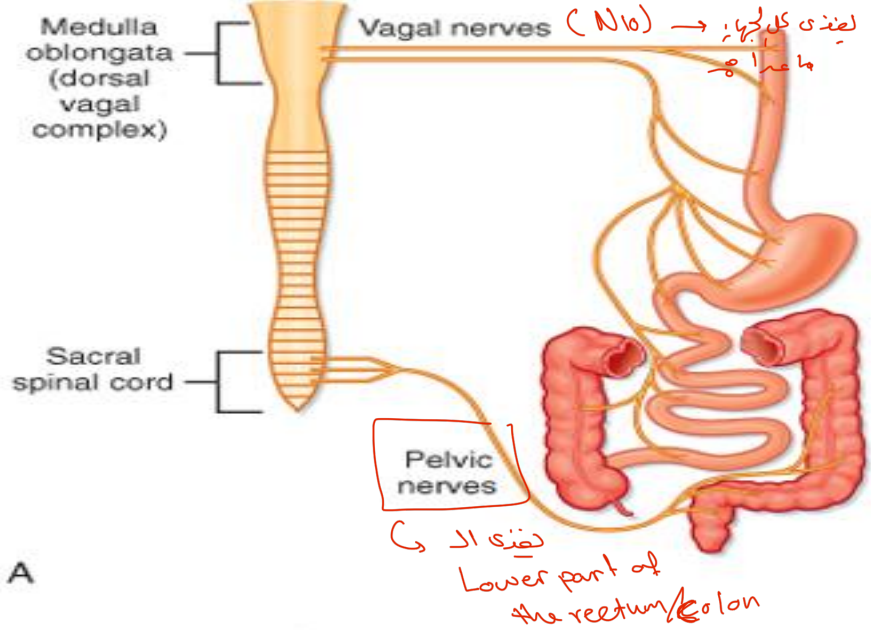
**Control glands** - increases secretion from glands (e.g. salivary that is why in a fight or flight response you have a DRY mouth as all sympathetic stimulation, not parasympathetic stimulation)

This occurs more for the glands in the upper part of the tract that are innervated by the **glossopharyngeal** and **vagus nerves**

Salivary, oesophageal, gastric, pancreatic and Brunner's glands (in the duodenum)

\*) ad sympathetic ana msh محتاج ازود فیه کما یریدة عنه التامی ، ف یک ال کبی نتی ال innervation.

## Parasympathetic



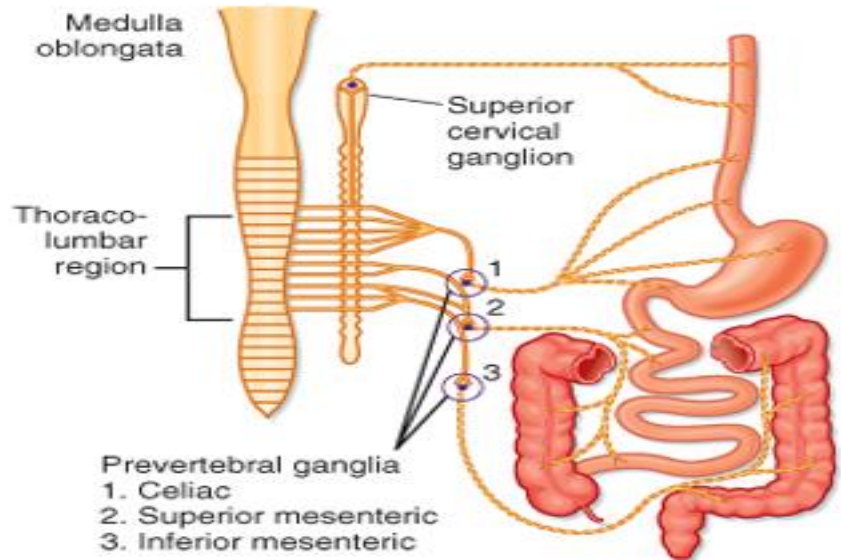
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## Sympathetic

له بيضيه ال vessel

انما يدي مع ليمفه  
للا كس نموت  
الموت، شى وقت الزور  
الsecretion و الامعاء

به الدم يربح ع الامعاء  
اكثر الدم منه ال  
heart و Brain



# Nervous control of GI tract

**Sympathetic innervation** → ال innervation لائے ني  
کی اینٹھیں حسی تھیں احنا اسے متحرک نہیں کرتے۔

These nerves originate in the **spinal cord**

Innervate all of the GI tract equally

میں لو اور سہولت زیادہ ہوگا کتر کبر  
تاکہ یوقت حرکتہ الاکل

Stimulation **inhibits** the GI tract - think fight or flight response - you need more blood to the heart and lungs and less for digestion so blood moves away!!

Strong stimulation can **inhibit the movement of the gut** walls so much that **NO**

**MOVEMENT OF FOOD CAN OCCUR!**

Causes **vasoconstriction** of the GI system **arterioles** → decreases blood flow

This can mean decreased blood flow to glands so **decreased secretion** from glands

Causes **vasoconstriction** of intestinal veins → pushing more blood back to the heart

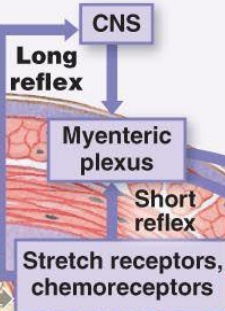
Sympathetic stimulation of the gastric system occurs **rarely** - mainly when high levels of sympathetic stimulation only → **fight or flight**

# The three major mechanisms that regulate and control digestive activities

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.



**Peristalsis and segmentation movements**

تغذية الـ ENS  
أو systemic  
نervous system.

### 1 Local Factors

Local factors are the primary stimulus for digestive activities, and include 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.

Buffers, acids, enzymes released

Secretory cells

Enteroendocrine cells

Hormones released

G-cells → Gastrin  
I-cells → CCK  
D-cells → Somatostatin  
S-cells → secretin

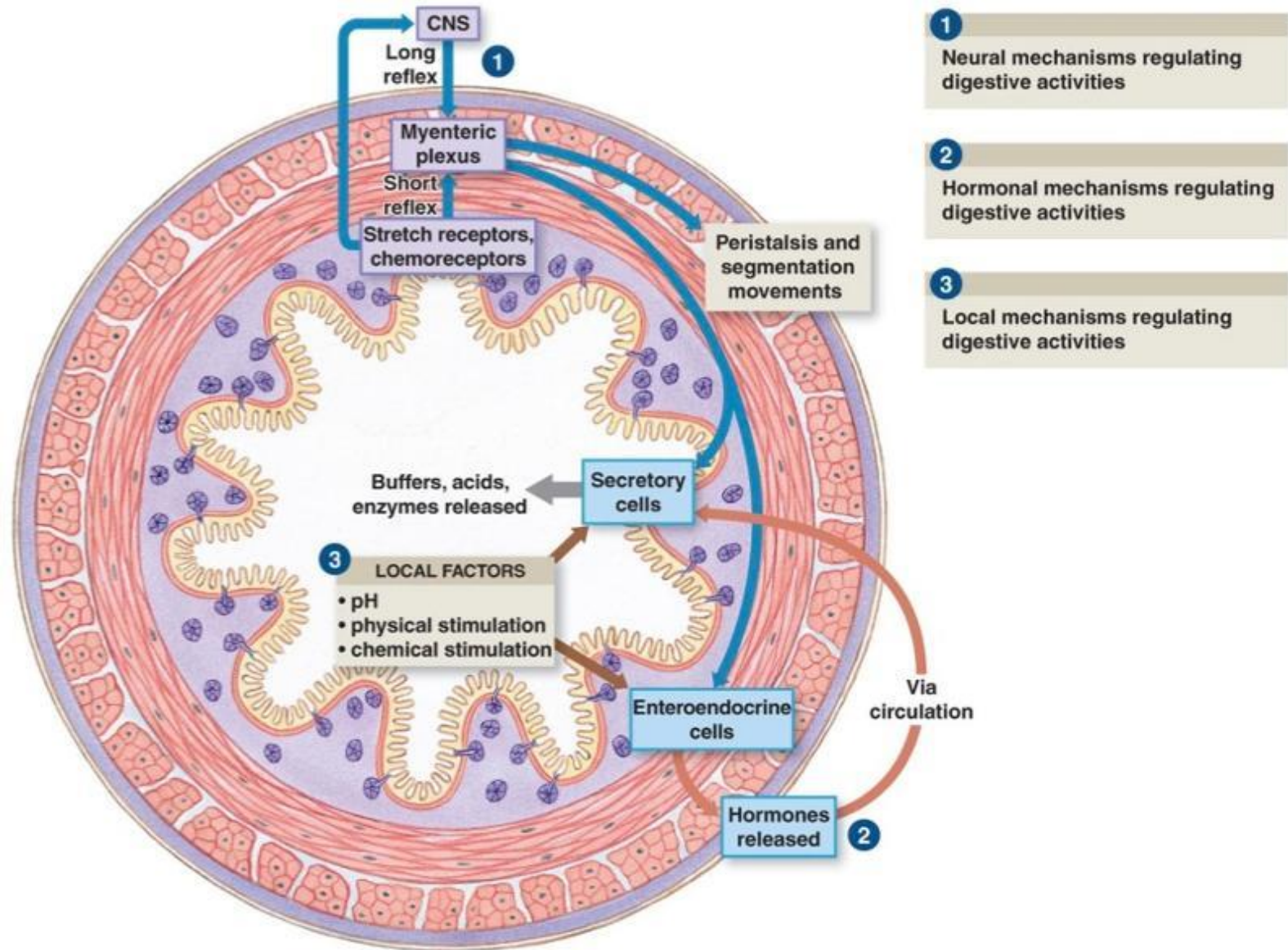
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## Hormonal Control Mechanisms

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

لـ هـرمونات بـترفع بـ الـدم و ترتبط بـ رسيـة الـهـ receptor و البـستند



# Hormonal control of GI system

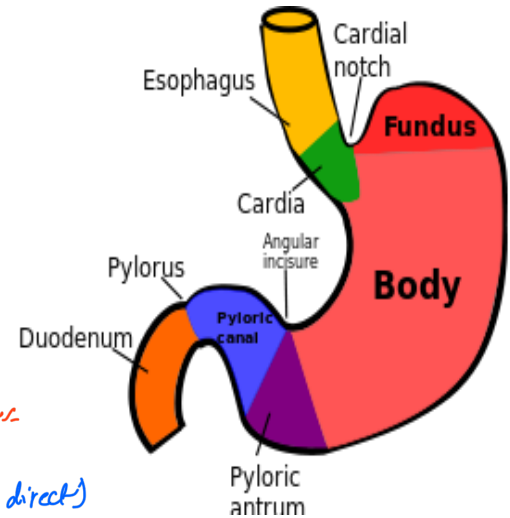
ENS

autonomic nervous syst.

Previously we mentioned glands and the fact they are under **Neural local control** via the enteric nervous system and also **higher control** from the autonomic nervous system

Glands can be controlled by **hormones** also

Hormones are released in response to **food in the GI tract** and are carried in the **blood** to the **glands** to **increase or decrease secretion**



The hormones affect the **volume** of secretions

## Gastrin:

Released from the **antrum** of the stomach. Gastrin stimulates secretions, leading to an acidic environment (direct). Handwritten notes include: "Pepsinogen secretion", "histamine release", "entero chromaffin cells", "acidic environment", "Parietal cells", "HCL", "stimulate", "secretions", "acidic environment", "direct".

Released from the **antrum** of the stomach

Released when there is **stretch** of the stomach - after a **meal**, presence of peptides and amino acids in lumen, Ach or vagal stimulation. Handwritten notes include: "stimulate", "stretch", "meal", "presence of peptides and amino acids in lumen", "Ach or vagal stimulation", "contact كثيرة".

This stimulates:

- Gastric acid secretion from parietal cells in the stomach
- Histamine release from ECL cells.
- Pepsinogen from chief cells in the stomach (inactive form of pepsin)

**Low pH** decreases gastrin secretion (a pH less than ~3). Handwritten notes include: "inhibition", "gastrin", "Low pH", "decreases gastrin secretion", "(a pH less than ~3)".

# Cholecystokinin CCK

Action of CCK → release of I-cells

- ❖ Gall bladder contraction, sphincter of Oddi relaxation
- ❖ ↑ pancreatic enzyme secretion
- ❖ Augment effect of secretin in producing alkaline pancreatic juice
- ❖ ↓ gastric emptying
- ❖ Induced satiety by acting through hypothalamus

# Hormonal control of GI motility

## Cholecystikinin CCK - secreted from I cells

Present of chyme  
rich in fat  
الذي يحفز إفرازه

Secreted from the **duodenum** and **jejunum**

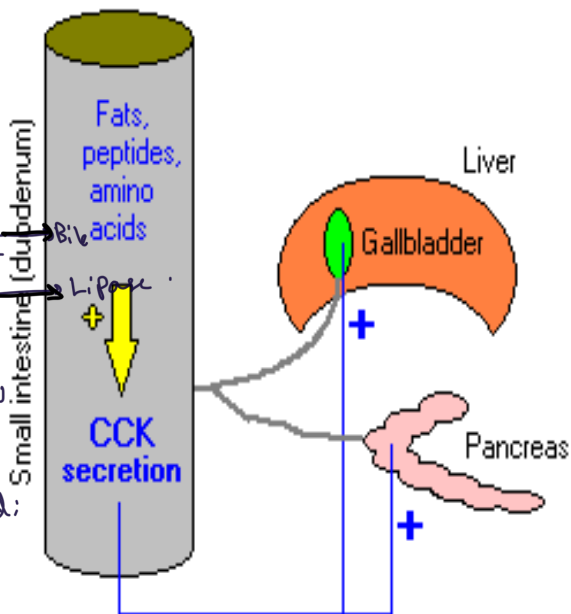
Released in response to digestive products of digestion  
specifically **fats and peptides**

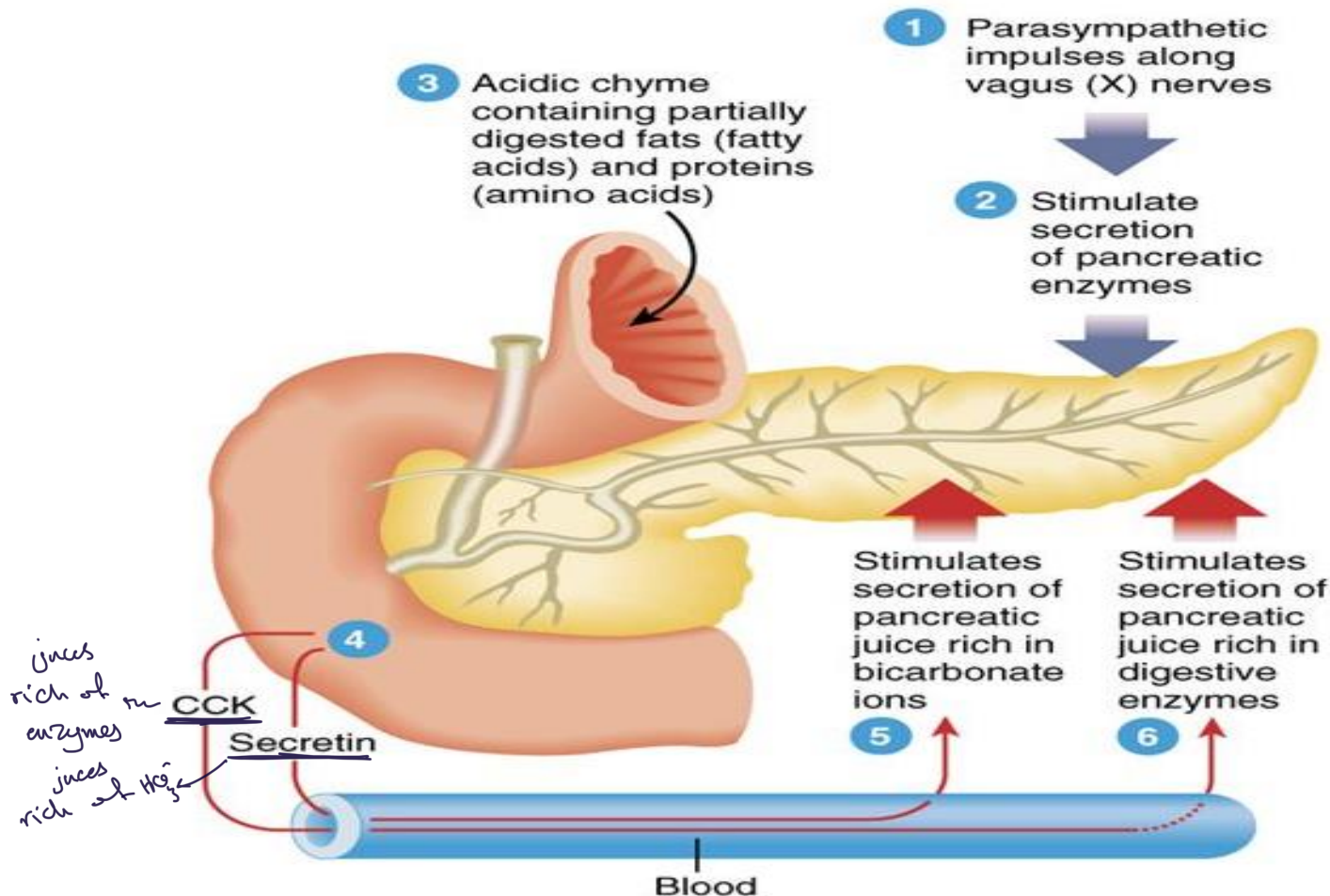
Causes **contraction of the gallbladder** which releases **bile** (Bile breaks down fat which allows them to be absorbed)

Moves to the pancreas through the blood to cause

- **increase on pancreatic digestive enzymes** e.g. **lipase** used to break down lipids
- **Stimulates growth of exocrine pancreas**
- **inhibits stomach contraction** - so can allow adequate **time** for digestion in the upper part of the GI tract
- **Inhibits gastric emptying**
- **Enhance** intestinal peristalsis
- Relaxation of oddi sphincter

contract of Gall bladder  
Pancreas  
بموجع على المر  
S +  
relax  
of sphincter  
at oddi





# Hormonal control of GI tract

## Secretin - secreted from **S cells**

Secreted from the **duodenum** سبب التحفيز :-

Released in response to **gastric acid** entering the duodenum from the stomach (pH less than 4.5)

- Stimulates the release of **bicarbonate from the pancreas** - to neutralise the pH in the intestine  
نفرز مع سكر  $\text{Na}^+\text{HCO}_3^-$  ← لا يصح الاستمرار في الحموضة  
حيث أن  $\text{H}^+$  سبب التحفيز

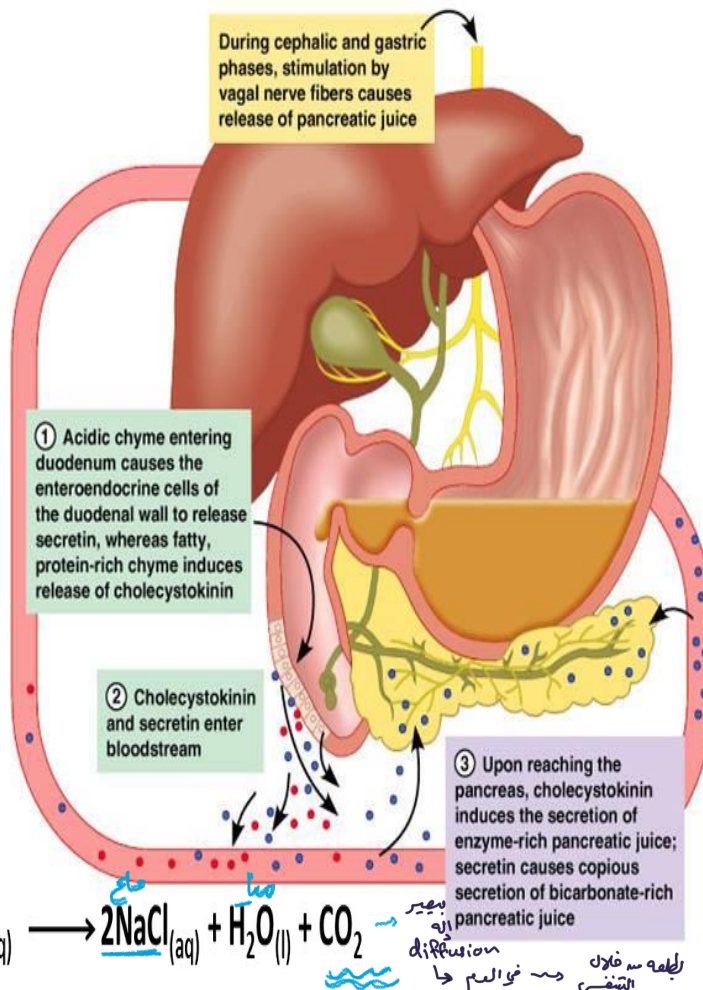
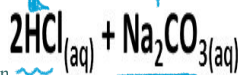
Causes **hydrochloric acid** to combine with **sodium bicarbonate** to form NaCl (salt) and  $\text{H}_2\text{CO}_3$  (carbonic acid)

The carbonic acid  $\rightarrow \text{CO}_2$  and  $\text{H}_2\text{O}$

The carbon dioxide is absorbed into the blood and expired

- Secretin also **inhibits** stomach emptying, motility and **acid release** ← عتامة مايفوت كميات كبيرة من  $\text{H}^+$  شوية شوية
- Stimulate growth of exocrine pancreas

➤ **Inhibited by Somatostatin**



# Hormonal control of GI motility

**Gastric inhibitory peptide (GIP)** <sup>من إبطاء المعدة</sup>  
gastric inhibition

Secreted by the **mucosa of the upper part of the intestine**

Released in response to **fatty acids and amino acids and monosaccharide (glucose)**

**Reduces the motility of the stomach** → slows to emptying of the stomach into the duodenum and allows more time for digestion

Stimulates insulin release from pancreatic beta cells

**Motilin** <sup>يبدأ الإلهام ٣-٤ ساعات من مأكول</sup>

Released during **FASTING!**

Released from Endocrine cells in the **duodenum**

➤ **INCREASES gastric motility** - causes waves of contracting even 90 minutes in a fasting

<sup>person</sup>  
<sup>أكل ما تأكل ما سطر يتوقف هذه الموتيلين</sup>  
Inhibited after **ingestion of food**

- **Gut hormones work by signalling to the brain which causes nerve stimulation of the parasympathetic or sympathetic nerve to cause a response**

**Ghrelin (hunger hormone)** في حالة تسمى جوع

- A hormone released by the **parietal cells** of the stomach and a little from the intestine
- Act on receptors in the arcuate nucleus of the **hypothalamus** منه هناك مراكز الشهية
- Levels of ghrelin rise **during fasting** and fall after a meal
- Increased ghrelin increases food intake in animals ?unsure in humans

**Leptin (satiety hormone)** التي يفرزها صفتها إفرازه على قصة أو (تفرز بكميات جاذبة) ↑ energy → ↑ leptin → انسان الذي يفرز خلاياهم بجلت sensitive Leptin له

- Hormone made by the adipose (fat) tissues
- Acts on receptors in the **arcuate nucleus** of the **hypothalamus** to regulate appetite to achieve **energy homeostasis**
- Inhibits hunger

In obesity, a decreased sensitivity to leptin occurs, resulting in an inability to detect satiety despite high energy stores

**Glucagon like peptide GLP (Incretins Family)** لها يكون في سكر داكل في الجسم → stimulate secretion of insulin + ↓ appetite ∴ ↓ weight.

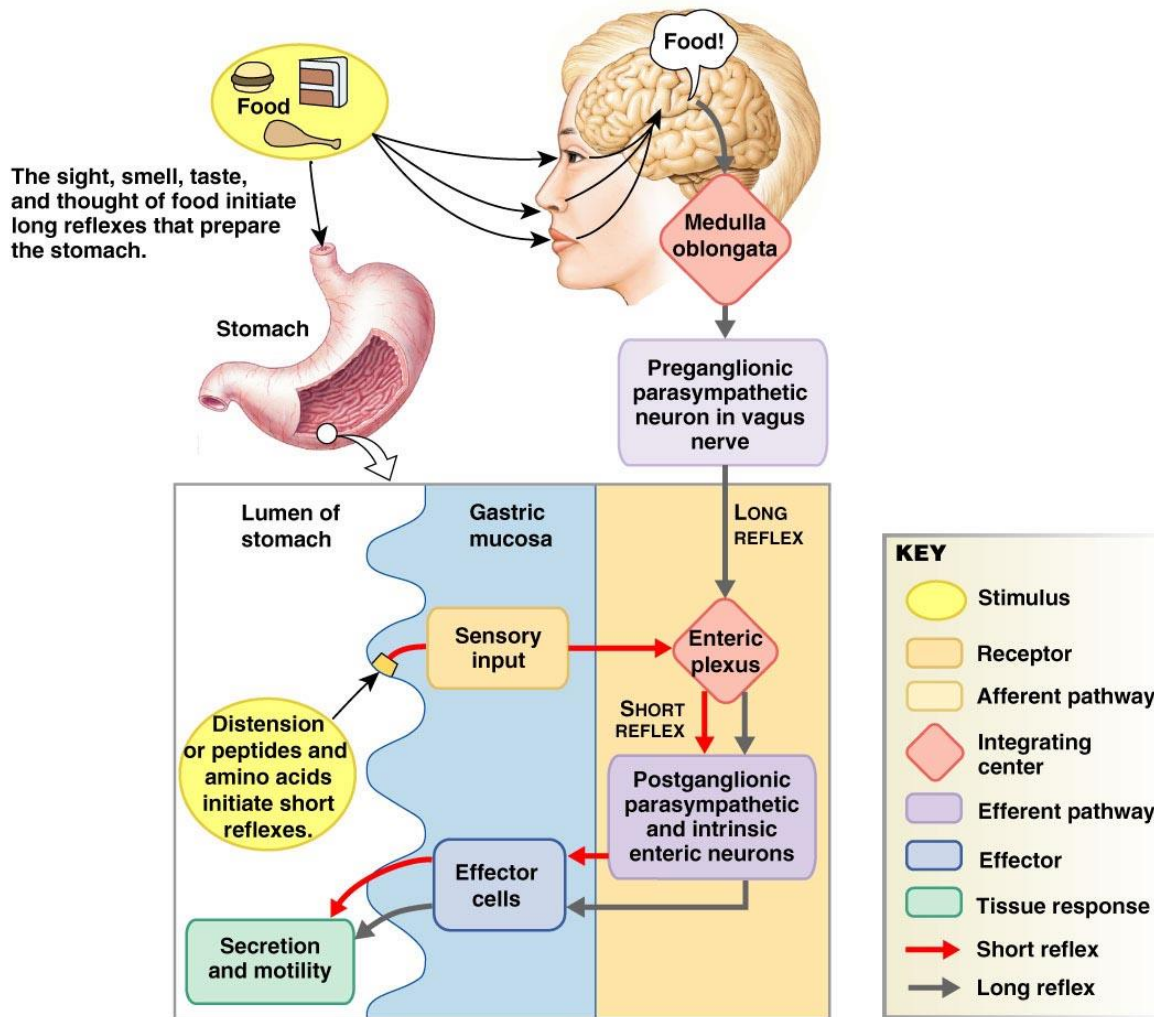
- Released from the intestines when they have food in them
- This hormone **increases insulin production** from the pancreas due to the presence of glucose
- **The glucagon like peptide and insulin both act to reduce appetite, induce satiety (reduce weight)**

**TABLE 21-1** The Digestive Hormones

|                                         | EFFECTS ON MOTILITY                                                                                          | OTHER EFFECTS                   | STIMULUS FOR RELEASE                                                                     | RELEASE INHIBITED BY   | OTHER INFORMATION                                                                   |
|-----------------------------------------|--------------------------------------------------------------------------------------------------------------|---------------------------------|------------------------------------------------------------------------------------------|------------------------|-------------------------------------------------------------------------------------|
| <b>Gastrin</b>                          | None                                                                                                         | Enhanced mucosal cell growth    | Peptides and amino acids in lumen; gastrin releasing peptide and ACh in nervous reflexes | pH < 1.5; somatostatin | —                                                                                   |
| <b>Cholecystokinin (CCK)</b>            | Stimulates gallbladder contraction for bile release; inhibits gastric emptying; promotes intestinal motility | Stimulates satiety              | Fatty acids and some amino acids                                                         | Somatostatin           | Some effects may be due to CCK as a neuropeptide rather than as a hormone           |
| <b>Secretin</b>                         | Inhibits gastric emptying                                                                                    | None                            | Acid in small intestine                                                                  | Somatostatin           | —                                                                                   |
| <b>Gastric inhibitory peptide (GIP)</b> | None                                                                                                         | Satiety and lipid metabolism(?) | Glucose, fatty acids, and amino acids in small intestine                                 | NA                     | Acid inhibition questionable at physiological concentrations                        |
| <b>Motilin</b>                          | Stimulates migrating motor complex                                                                           | Action in brain(?)              | Fasting: periodic release every 1.5–2 hours by neural stimulus                           | NA                     | Changes associated with both constipation and diarrhea, but relationship is unclear |
| <b>Glucagon-like peptide 1</b>          | Slows gastric emptying                                                                                       | Satiety                         | Mixed meal that includes carbohydrates or fats in the lumen                              | NA                     | Related to but not identical to pancreatic glucagon; acts in concert with GIP       |

# Digestive hormones in the GI tract

| <u>HORMONE</u>                       | <u>LOCALIZATION</u>                              | <u>MAIN PHYSIOLOGIC ACTIONS</u>                                                                                                                                                                                                                             |
|--------------------------------------|--------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Gastrin                              | Gastric antrum, duodenum (G cells)               | <ul style="list-style-type: none"><li>-stimulate secretion of gastric acid and intrinsic factor from parietal cells</li><li>-stimulate secretion of pepsinogen from chief cells</li><li>-promotes gastric and intestinal motility, mucosal growth</li></ul> |
| Cholecystokinin (CCK)                | Duodenum, jejunum (I cells)                      | <ul style="list-style-type: none"><li>-stimulate gallbladder contraction</li><li>-stimulates release of pancreatic enzymes</li><li>-relaxes sphincter of Oddi for release of bile and enzymes</li><li>-role in inducing satiety</li></ul>                   |
| Secretin                             | Duodenum, jejunum (S cells)                      | <ul style="list-style-type: none"><li>-stimulate secretion of <math>\text{HCO}_3</math> from pancreas</li><li>-inhibits gastrin and gastric acid secretion</li></ul>                                                                                        |
| Vasoactive intestinal peptide (VIP)  | Enteric nerves                                   | <ul style="list-style-type: none"><li>-increases water and electrolyte secretion from pancreas and gut</li><li>-relaxes smooth muscles (via nitric oxide) of the gut</li></ul>                                                                              |
| Gastric inhibitory polypeptide (GIP) | Duodenum, jejunum (K cells)                      | <ul style="list-style-type: none"><li>-reduces gastric acid secretion and intestinal motility</li><li>-stimulates insulin release</li></ul>                                                                                                                 |
| Motilin                              | Throughout the gut (Mo cells and ECL cells)      | <ul style="list-style-type: none"><li>-increases small bowel motility (MMC during fasting) and gastric emptying</li></ul>                                                                                                                                   |
| Somatostatin                         | Stomach, small intestine, and pancreas (D cells) | <ul style="list-style-type: none"><li>-inhibits secretion and action of many hormones, including all of the above</li></ul>                                                                                                                                 |



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# Vomiting

لما عدة اسباب :-  
 17 دغرة حامل  
 2 واحد لثف غير الكرسى  
 3 واحد خاينى  
 3 واحد اكل اكله غير toxin  
 14 دغرة بزدانه  
 15 واحد خاينى

(normal state) intestin ← ثم يرجع عن oral cavity  
 \* في اكله اكله طبيعيه انه ما يرجع عن لا intestin  
 \* في اكله اكله طبيعيه انه ما يرجع عن لا intestin

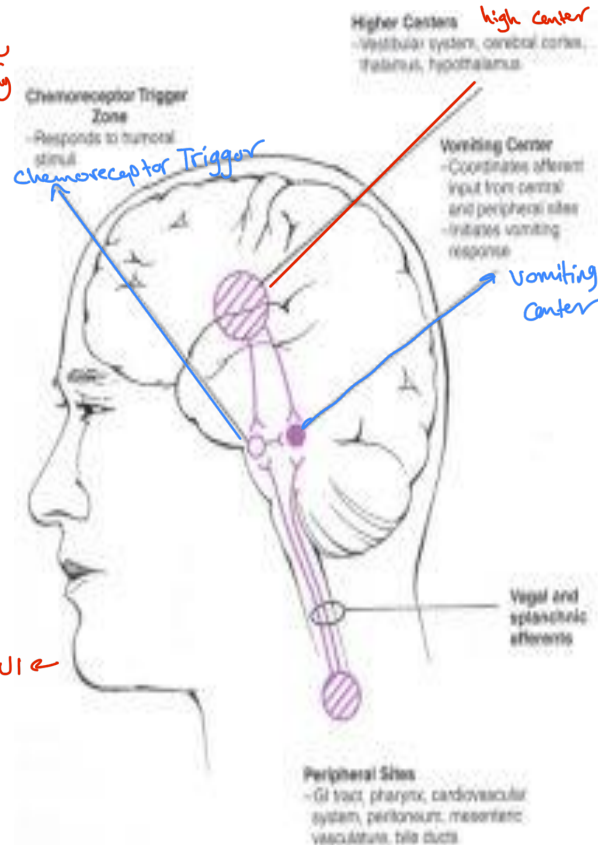
- This occurs when any part of the upper GI tract becomes excessively irritated, excited or overdistended
  - Excessive distension of the duodenum causes a very powerful vomiting reflex
  - Sensory nerves** in the pharynx, oesophagus, stomach and upper part of the intestine send afferent impulses to the “**vomiting center**” of the brainstem if they are stimulated
  - The brain then **sends efferent impulses via** the trigeminal, facial, glossopharyngeal, vagus and hypoglossal nerve to **stimulate the upper GI tract, diaphragm and abdominal muscles**.
  - This causes → deep breath, raising of the larynx, closing of the glottis and elevation of the soft palate to close posterior nares to protect the lungs and the nasal cavity
  - wave of peristalsis in the opposite direction occurs (up the GI tract). This is **called anti-peristalsis**
  - Strong contractions of the stomach and duodenum occur which are increased through contraction of the abdominal muscles
  - The upper oesophageal relaxes and vomitus moves from the stomach to the oesophagus
    - Stimulated ← Contract في المعدة وحركة في المريء (anti-peristaltic)
    - Abd. muscle & diaphragm contraction في البطن والعضلات التنفسية
    - deep breath (نفس عميق)
    - vomiting center
- Vomiting can also be stimulated separately from the “**chemoreceptor trigger zone for vomiting**” in the brain.
  - Stimulated ← Contract في المعدة وحركة في المريء (anti-peristaltic)
  - Abd. muscle & diaphragm contraction في البطن والعضلات التنفسية
  - deep breath (نفس عميق)
  - vomiting center

# Vomiting → حي نوع مع protection

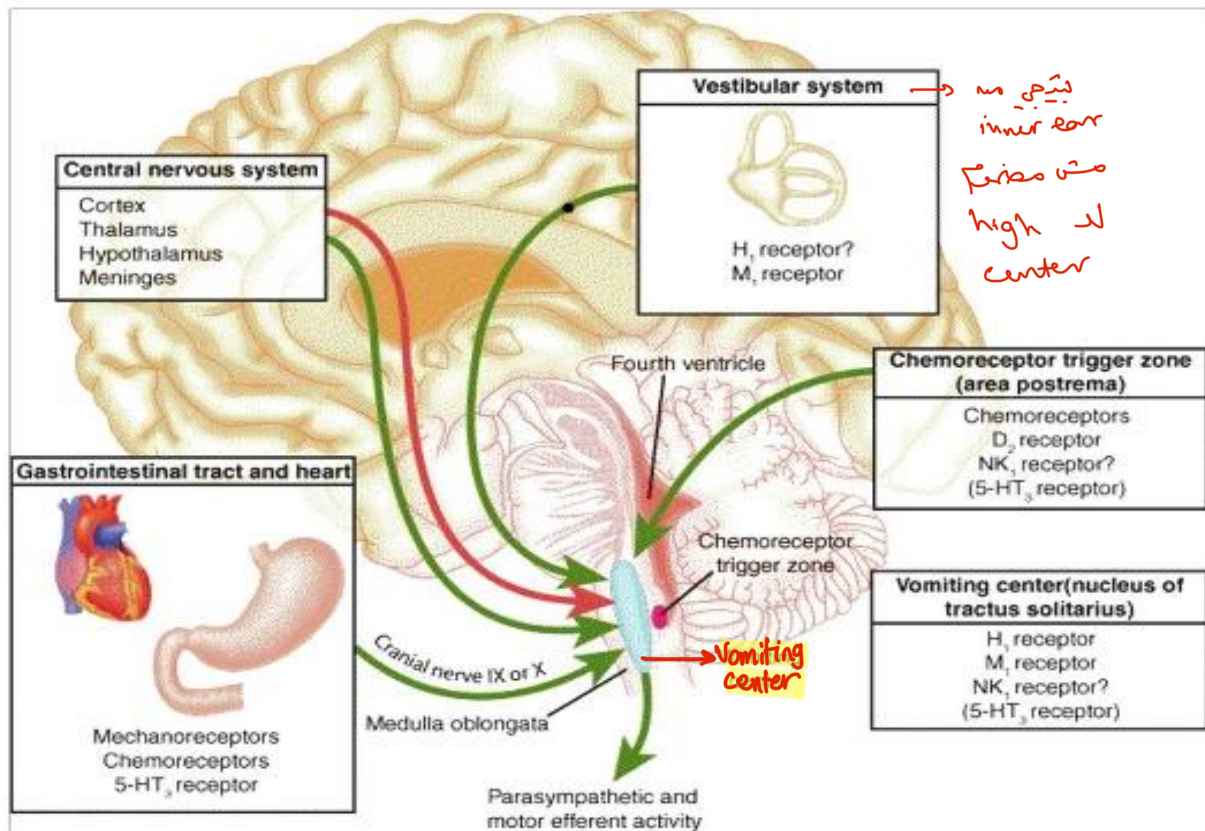
- The **vomiting center** is located in the medulla
- The **chemoreceptor trigger zone (CTZ)** for vomiting is located in the fourth ventricle
  - The CTZ is stimulated by *toxins/drugs e.g. morphine*
  - Destruction of the CTZ stops this type of vomiting but does not stop vomiting due to stimulation of the vomiting center

3. A 3rd type of vomiting comes from the stimulation of **higher centers (vestibular apparatus)**

- This occurs when a person has rapid changes in direction - spinning around, seasickness . *التقيؤ في الحركة السريعة* . Vomit
- The **vestibular apparatus of the inner ear** are stimulated and sends signals to the vestibular nuclei in the cerebellum then to the CTZ → which stimulates the vomiting center



- Nausea is stimulated by the same things as vomiting



Source: Katzung BG, Masters SB, Trevor AJ: *Basic & Clinical Pharmacology*, 11th Edition; <http://www.accessmedicine.com>

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# Neural regulation of feeding

0.8x60

- The sensation of hunger is associated with a craving for food and comes from:
  - **The Hypothalamus Contains Hunger and Satiety Centers that are influenced by neurotransmitters and hormones.**
- **Appetite** is a desire for food
  - Usually a particular **type** of food
- **Satiety** occurs when the quest for food has been successful (the opposite of hunger)
- Each of these factors are regulated by:
  - **Environmental factors**
  - **Cultural factors**
  - **Neural regulation in the hypothalamus of the brain**

\*) appetite و satiety هما مراكز في hypothalamus

يحدد قسماً يريد أكل (الكمالي) في نوع الدقيق (appetite)  
كثير شهوة تؤثر عليه هرمونات أو عوامل نفسية (مثل التوتر).

الصحة مثلاً ياكلوا شهوة زينة  
مثل الخناش، انا عيشة  
الكلية  
عند السهر  
عند السهر