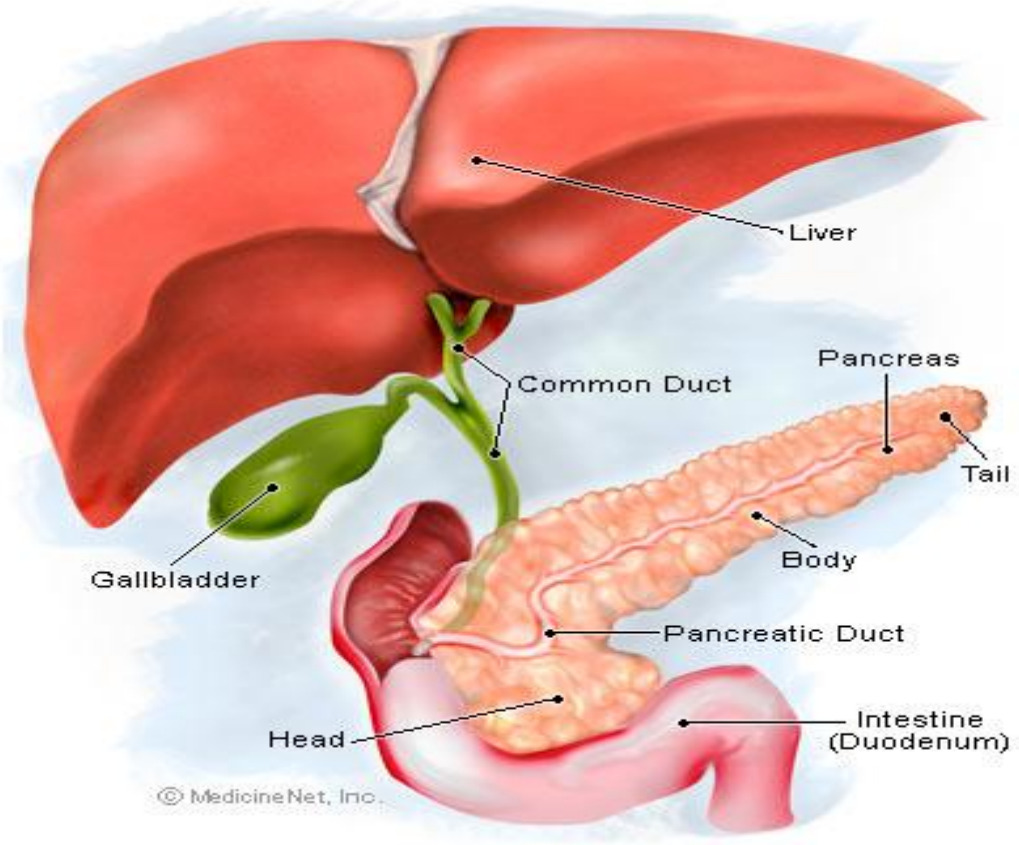
Lecture 3

Swallowing, Physiology of esophageal motility and Pathophysiology of reflux



PHYSIOLOGY TEAM – 430

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Swallowing, Physiology of esophageal motility and Pathophysiology of reflux

❖ Whatever written in green is the team's notes.

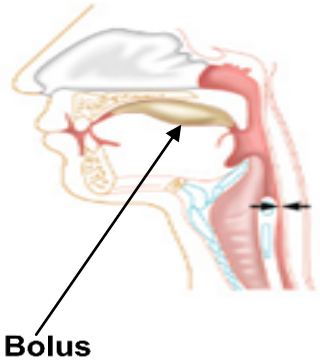
Introduction:

- Chewing process is controlled by nuclei in the brain stem. (medulla)
- Much of the chewing process is caused by a Chewing reflex & stretch reflex.
- Swallowing is initiated voluntarily in the mouth (Oral Stage), but thereafter once it reach opening of the pharynx (Pharyngeal stage) it becomes under involuntary or reflex control.
- The reflex portion is controlled by the swallowing center in the medulla.

- Stages of Swallowing:

1. Oral Stage (voluntary)
2. Pharyngeal stage (involuntary)
3. Esophageal stage (involuntary)

✓ Oral Stage (voluntary):



(Stretch reflex) the bolus turns into small particles → pushing of these particles posteriorly by pressure of the tongue against the palate → pharynx

✓ Pharyngeal stage (involuntary): lasts (2-6) sec

The bolus of food stimulates epithelial swallowing receptor areas all around the pharynx opening:

Tonsillar pillars (somatosensory receptors) → impulses pass to the medulla: nucleus tractus solitarius (swallowing center) and accordingly initiate a series of autonomic pharyngeal muscle contractions as follows:

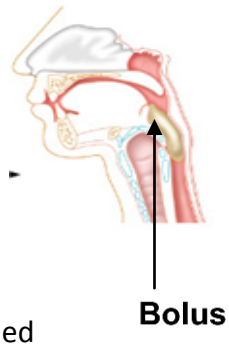
Briefly:

1. Soft palate is pulled upward
   (Close the nares, so nothing to go to the nasal cavity)
2. the epiglottis moves to cover opening of larynx
   (So nothing to go to the respiratory tract)
3. the upper esophageal sphincter relaxes
   (Allowing bolus to move from pharynx to esophagus)
4. Peristalsis wave of contraction initiated in the pharynx
   (Moving food from pharynx through the upper esophageal sphincter)

Breathing is inhibited during the pharyngeal stage of swallowing (for 4 - 6 sec)

More Details:

The soft palate is pulled upward to close the posterior nares → prevents the food from entering the nasal cavities.



The palatopharyngeal folds on each side of the pharynx are pulled medially to approximate each other → form a sagittal slit → food must pass into the posterior pharynx

The vocal cords of the larynx are strongly approximated → larynx is pulled upward and anteriorly by the neck muscles → the epiglottis swing backward over the opening of the larynx → prevent food from going into the nose and trachea.

The upward movement of the larynx pulls up and enlarges the opening to the esophagus.

The upper esophageal sphincter relaxes and allows food to move freely from the posterior larynx into the upper esophagus.

The entire muscular wall of the pharynx contracts (superior, middle, then inferior parts) propelling the food by peristalsis into the esophagus.

✓ Esophageal stage (involuntary): lasts **(8-10) sec**

- Main function of esophagus is to conduct food from the pharynx to the stomach.

✓ This process is controlled mainly by the swallowing centre. (Swallowing reflex)

- Transport is accomplished by peristalsis, with propulsive and receiving segments.

The esophageal stage is controlled by:

✓ The enteric nervous system (ENS).

- The musculature of the pharyngeal wall (striated muscle) of upper 1/3 of esophagus innervated by vagus & glossopharyngeal nerves.

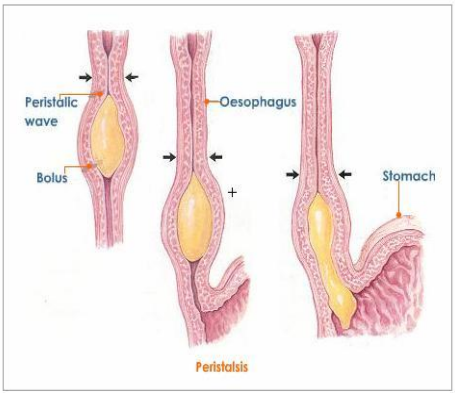
- The lower 2/3 of the esophagus (smooth muscle) is innervated by vagus nerve with Neurons of Myenteric plexus

- Both striated and smooth muscles are mainly innervated by branches of vagus nerve.

The esophageal musculatures are of 2 types:

The proximal esophagus is predominantly striated muscle, while the distal esophagus and the remainder of the GI tract contain smooth muscle. The mid esophagus contains a graded transition of striated and smooth muscle types. The muscle is oriented in 2 perpendicular opposing layers: an inner circular layer and an outer longitudinal layer, known collectively as the muscularis propria. The longitudinal muscle is responsible for shortening the esophagus, while the circular muscle forms lumen-occluding ring contractions.

Upper esophageal sphincter will contract again → prevents entry of air into esophagus → primary peristaltic movement (propulsive and receiving segments) → pushing the bolus down into the stomach → relaxation of Lower esophageal sphincter by inhibitory neurons → the bolus will enter the stomach → Lower esophageal sphincter will contract again → prevent reflux of stomach contents into the esophagus.



Esophageal peristalsis:

Initiated by vagal reflexes:

Esophagus ⇨ vagal afferent ⇨ medulla oblongata ⇨ vagal efferent ⇨ back to esophagus

- Swallowing Center (medulla) :
  - Sensory input from pharynx and esophagus
  - Coordinates activity from vagal nuclei with other centers (e.g., inhibits respiratory center).

The relaxation of lower esophageal sphincter and proximal pert of the stomach caused by:
Receptive Relaxation reflex (vagovagal reflex)

When the bolus reach to lower esophageal sphincter → activate vagovagal reflex → afferent vagus → medulla oblongata → efferent vagus → activates the inhibitory motor neurons (VIP) → relaxation of lower esophageal sphincter and proximal pert of the stomach

☒ **So swallowing in general:** chewing reflex → swallowing reflex → vagovagal reflex.

Esophageal peristalsis may occur as primary peristalsis or secondary peristalsis:

- peristaltic wave (movement):

Peristaltic wave	Primary	Secondary
Function	Normal pharyngeal movement	Start above stick bolus due to localized distension of an esophageal segment when the primary peristalsis fails to pass the bolus down.
Coordinated by	swallowing reflex	ENS

1 – Primary peristalsis (triggered by the swallowing center):

(It is initiated by **vagus**, coordinated by **Myenteric**).

It travels at 3-5 cm / sec. and traverse the entire esophagus in less than 10 sec.

Push Bolus towards Stomach:

A bolus in Esophagus ⇨ Pressure receptors ⇨ Swallowing Center ⇨ Vagus ⇨ Primary Peristalsis

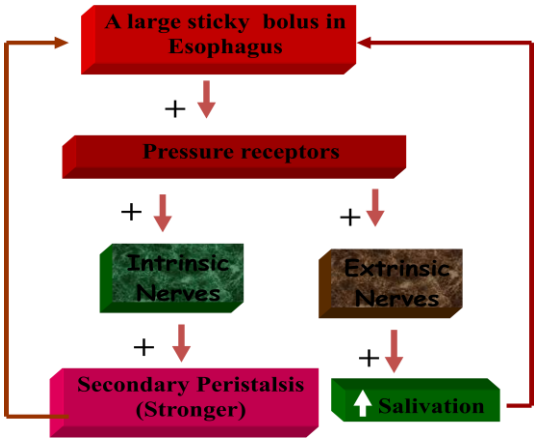
When the primary peristalsis event fails to clear the bolus from the body of esophagus or if the bolus is too big or sticky, Secondary peristalsis occurs.

- ❖ If you drink water or eat food and masticate it very well: primary is enough.
- ❖ If not: Secondary will help.

2 – Secondary Peristalsis:

- **Initiated by activation of mechanoreceptors due to localized distension of an esophageal segment by the retained food.**
- **The peristaltic wave begins above the site of distension and moves downward until all food has emptied into the stomach.**

Stretch receptors in the esophageal lining are stimulated and a local reflex response causes a secondary peristaltic wave around the bolus, forcing it further down the esophagus and these secondary waves will continue indefinitely until the bolus enters the stomach.



Intrinsic: Myenteric. Extrinsic: vagus.

The role of vagus here is to produce strong salivations.

Swallow-induced peristalsis is called primary peristalsis, and the peristalsis elicited by esophageal distention is called secondary peristalsis.

Nervous Control of Esophageal Phase:

Primary peristalsis:

- Continuation of pharyngeal peristalsis.
- Coordinated by swallowing center.
- Cannot occur after vagotomy (striated muscle).

Secondary peristalsis:

- Stretch related afferent sensory input to ENS and swallowing center are both involved.
- Can occur after vagotomy (SM).

Vagotomy is the surgical cutting of the vagus nerve to reduce acid secretion in the stomach.

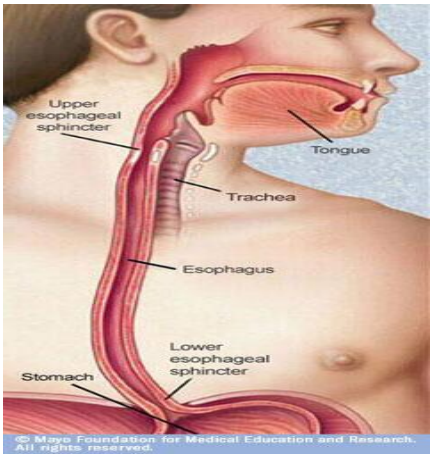
Note:

If we cut vagus nerve: paralysis If we cut Myenteric plexus: paresis (weakness) In case of vagotomy: enteric nervous system takes over.

Esophageal sphincters:

- ✓ Roles of sphincters during Swallowing:

	Upper esophageal sphincter(UES)	Lower esophageal sphincter(LES)
Function	prevents entry of air into esophagus	prevent reflux of gastric contents into esophagus
Contraction	normally remain completely relaxed	normally remain tonically constricted
Relaxation	During Pharyngeal Stage	During Esophageal Stage
Duration during relaxation	1 sec	For 7-10 sec



The LES is a high pressure zone between the esophagus and stomach. The LES is made up of muscles at the bottom of the esophagus as well as the muscles of the diaphragm (breathing muscle) that surround the bottom of the esophagus. When it is closed, the LES maintains a higher pressure than that of the stomach so that food and digestive juices cannot wash back into the esophagus. The LES normally opens or relaxes (lowers its pressure) for 7-10 seconds as food is moved down the esophagus by esophageal contractions.

It is necessary to have a barrier at the gastroesophageal junction, why? Because antireflux barriers prevent reflux of gastric contents into the esophagus, and their dysfunction leads to gastroesophageal reflux disease and or dysphagia

- Same pressure in esophagus in comparison to intrathoracic pressure i.e. mostly –ve pressure in esophagus in comparison to intra-abdominal pressure (except for a short intra-abdominal segment). So that pressure in the stomach is always higher than the esophagus.

Competence and the antireflux functions of the LES are due to: (FOR READING)

- 1- Its resting pressure (15-30 mmHg).
- 2- A valve like mechanism (one way valve) of the distal end of the esophagus that lies immediately beneath the diaphragm and is exposed to +ve intra-abdominal pressure. This flutter-valve closure of the lower esophagus by the increased intraabdominal pressure prevents the high pressure in the stomach from forcing its contents into the esophagus.
- 3- The crura of the diaphragm wrap around the esophagus. At the level LES, contraction of the diaphragm helps to increase the pressure in the LES with each inspiration.
  The crural diaphragm is tendinous structures that extend inferiorly from the diaphragm to attach to the vertebral column. It has two functions, respiratory and gastrointestinal. Respiratory function relates to ventilation, and gastrointestinal to the sphincter like action at the lower end of the esophagus.

LES closed

LES opened

Control of LES function:

- Contraction of the circular musculature of the sphincter is regulated by nerves, (extrinsic & intrinsic), hormones and neuromodulators.

- Stimulation of sympathetic nerves to the sphincter also causes the LES to contract.
  Sympathetic ⟹ fight & flight. Parasympathetic ⟹ rest & digest.

  1 –During swallowing, efferent impulses in the vagus are inhibitory causing the sphincter to relax. The transmitter probably being nitric oxide or vasoactive intestinal peptide (VIP).
  2 –The hormone gastrin → contracts LES.
  3 –Secretin and cholecystokinine (CCK) → relax the LES.

  They are digestive hormones released when food from the stomach reaches the first part of the small intestine. They affect the LES by relaxing it as well as increasing motility (movement) of the stomach and intestine.

Achalasia:

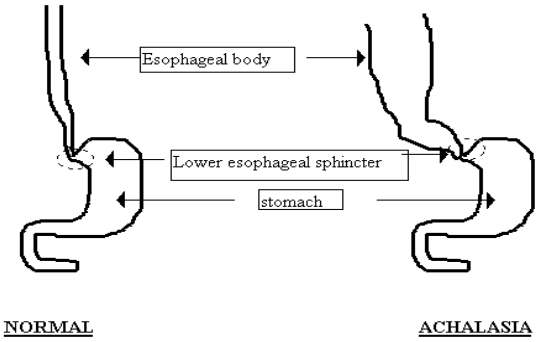
- It is a condition due to high resting pressure of the LES. So it fails to relax during swallowing.
- As a result, food transmission from the esophagus into the stomach is impeded or prevented.
- So the food accumulates in the esophagus and the organ becomes massively dilated.

  ☒ ***Causes:***

  It's caused by a pathological state of or absence of the Myenteric plexus containing VIP & NO in the lower third of esophagus.

  (Since the Achalasia is in LES "lower part of esophagus", then we know that the problem will be on Myenteric).

- *More explanation*
- The musculature of the lower esophagus instead remains contracted and the Myenteric plexus has lost the ability to transmit a signal to cause relaxation of the LES.
- When Achalasia becomes severe, the esophagus may not empty the swallowed food into the stomach for many hours.
- The esophagus becomes enlarged which may be infected and cause ulceration, severe substernal pain or even rupture and death.
- The food often reflux into the pharynx and is then aspirated into the lungs.



Therapy of Achalasia:

- Administering drugs that inhibit the tone of the LES. (Although some patients with Achalasia have improvement of symptoms with medications, most do not. oral medications are likely to provide only short-term and not long-term relief of the symptoms of Achalasia).
- Mechanically dilating LES. (Dilation of LES is done by having the patient swallow a tube with a balloon at the end).
- Surgically weakening the LES.

✓ Gastroesophageal reflux disease (GERD):

Incompetence of the LES:

- Incompetence cause esophageal reflux and result in chronic exposure of esophageal mucosa to acid. The stomach contents are highly acidic and contain many proteolytic enzymes.
- The esophageal mucosa, except in the lower eighth of esophagus, is not capable of resisting for long the digestive actions of gastric secretions.
- It can lead to reflux esophagitis, heart burn, esophageal ulcer and dysplastic changes that may become cancerous.