Anatomy, Functions, and Diseases of the Esophagus

The esophagus, or oesophagus, is a hollow organ that is commonly known as the gullet or food pipe. With the aid of peristaltic (wavelike) contractions, food passes through the esophagus from the pharynx (throat) to the stomach. The esophagus, or gullet, is a long tube that is approximately 25 cm long in adults and 8-10 cm long in infants. The following article will provide a compact overview of the anatomy, most important diseases, and physiology of this integral organ.

Location of the Esophagus

The esophagus is located behind the trachea (windpipe) and heart and in front of the spine and connects the pharynx to the stomach. The esophagus begins at the same height as the 6th/7th cervical vertebrae on the lower edge of the cricoid cartilage. The gullet also begins with the superior esophageal sphincter at the bottom of the hypopharynx (entrance into the esophagus) adjacent to the left pyriform sinus, then runs dorsal to the trachea in the chest area and ventral to the spine, and continues in a caudal fashion through the diaphragm, ultimately ending at the junction with the cardia of the stomach, parallel to the 11th thoracic vertebra.

The esophagus has 2 gentle curves in the coronal plane. The first curve begins slightly below the commencement of the esophagus, moves to the left by the root of the neck, and returns to the midline at the level of the 5th thoracic vertebra. Access to the
esophagus can be gained from the right chest. The second curve to the left is formed as the esophagus bends to cross the descending thoracic aorta before it pierces the diaphragm. The esophagus also has anteroposterior curvatures that correspond to the curvatures of the cervical and thoracic parts of the vertebral column.

Sections of the Esophagus

The esophagus is divided into three sections: the pars cervicalis, the pars thoracica, and the pars abdominalis.

The pars cervicalis, the cervical area, is the part of the esophagus inside the neck. It begins at the cricoid, runs ventral to the spine, and after entering the chest cavity it becomes the pars thoracica.

The pars thoracica, the thoracic area, is the part of the esophagus in the chest. It is the longest section, runs dorsal to the trachea in the mediastinum, and after passing through the hiatus oesophageus of the diaphragm it becomes the pars abdominalis.

The pars abdominalis, the abdominal area, is the part of the esophagus that leads to the stomach. It enters into the cardia of the stomach after about 1-3 cm.

The Three Constrictions of the Esophagus

Adjacent anatomical structures cause three constricted areas in the esophagus. These constrictions are as follows:

Constrictio cricoidea – is caused by the cricoid cartilage and is constricted by the upper esophageal sphincter (UOS) until the sphincter relaxes in response to a bolus.

Constrictio partis thoracicae/aortal constriction – is caused by the proximity to the aortic arch (arcus aorta) and the left main bronchus.

Constrictio diaphragmatica – is caused by the hiatus esophagus of the diaphragm.

Wall Structure of the Esophagus

The esophagus generally has the typical wall structure of the gastrointestinal tract.

Histologically, the esophagus has the following 4 concentric layers (see image below):

- Mucosal layer;
- Submucosal layer;
- Muscular layer; and
- Adventitial layer.

The esophagus lacks a continuous covering layer.
Mucosal layer (mucous membrane)

Mucosa

The mucosa forms the innermost layer and is formed by a non-keratinizing stratified squamous epithelium that is consistent with that of the pharynx. Mucosal epithelium changes from squamous cell epithelium to columnar cell epithelium at the gastroesophageal junction called the “Z line” or squamocolumnar junction. It is also subjected to heavy mechanical strain.

Below the mucosa lies a smooth lamina propria that hosts capillaries, lymphatics, and muscularis mucosae.

Submucosal layer

The second layer is formed by submucosa, and it loosely connects the mucous membrane...
and the muscular coat. This layer also contains the larger blood vessels, the submucosal (Meissner) nerve plexus, and esophageal glands.

**Side note on clinical presentations:**

This venous plexus is important for portocaval anastomoses (e.g., with cirrhosis of the liver). If the portal system is under increased pressure, it finds already present anastomoses with the systemic circulation in which to dump blood to decrease that pressure. One of these connections is between gastric veins and esophageal veins. Esophageal varices may form. Esophageal varices are enlarged, sub-mucosal veins in the lower third of the esophagus. This condition occurs most often in people with serious liver diseases or represents a consequence of portal hypertension, commonly due to cirrhosis. Esophageal varices develop when normal blood flow to the liver is blocked by a blood clot or scar tissue in the liver. Patients with esophageal varices have a strong tendency to develop severe bleeding or hemorrhaging which, left untreated, can be fatal.

**Muscular layer**

The third layer is formed by circular and longitudinal muscle fibers. Between the circular and longitudinal layer is a portion of the enteric nervous system—the myenteric plexus of Auerbach (plexus myentericus). It controls the motility and peristalsis of the esophagus and the opening of the lower esophageal sphincter.

- **Inner circular muscle fibers:** These fibers are continuous superiorly with the fibers of the cricopharyngeal part of the inferior constrictor and inferiorly with oblique fibers of the stomach.

- **Outer longitudinal muscle fibers:** The longitudinal muscle fibers form a continuous coat around the whole of the esophagus, except posterosuperiorly, 3-4 cm below the cricoid cartilage; here, they diverge as 2 fascicles that ascend obliquely to the anterior aspect of the esophagus. The longitudinal layer is generally thicker than the circular layer.

The esophageal muscles are not uniform. The muscular fibers in the cranial part of the esophagus are red and consist chiefly of striated muscle. The intermediate part is mixed, and the lower part, with rare exception, contains only smooth muscle. Accessory bands of muscle connect the esophagus and the left pleura to the root of the left bronchus and the posterior of the pericardium.

The proximal 1/3 of the esophagus consists primarily of striated muscle. Smooth muscle predominates in the distal portion.

**Side note on clinical presentations:**

Laimer’s Triangle: This is a missing piece of longitudinal muscle in the dorsocranial area of the esophagus; this weak spot can lead to an increase in intraluminal pressure and an outward eversion of the mucosa and submucosa in the muscular layer which is called pseudodiverticulum. It was named in 1877 by German pathologist, Friedrich Albert von Zenker, and is commonly caller Zenker diverticulum. It may be seen more frequently in elderly men who present with complaints about regurgitation of undigested, malodorous food. True diverticula are eversions of all wall layers.

Note: True **diverticula** are eversions of all wall layers!
Adventitial layer

The tunica adventitia is the shifting outer fascial layer that allows for free mobility of the esophagus while swallowing. It surrounds the esophagus and fills the spaces between the esophagus and surrounding organs such as the trachea, bronchi, and pleural. The following are located here:

- Large supply vessels
- Lymphatic vessels
- Nerve fascicles of the vagus nerve and the esophageal sympathetic plexus

The esophagus has no serosa which makes it unique to the rest of the gastrointestinal tract.

Vascular Supply of the Esophagus

The three segments of the esophagus are supplied by various arteries.

**Pars cervicalis** - from the inferior thyroid artery (from the truncus thyrocervicalis)

**Pars thoracalis** - from the aorta and the intercostal arteries

**Pars abdominalis** - from the esophageal of the left gastric artery (from the celiac trunk)

Venous drainage occurs through the small esophageal veins—into the v. azygos and v. hemiazygos—and into the superior v. cava.

Note: Collateral circulation—there are connections to the v. portae hepatitis (portocaval anastomoses) through the v. gastrica dextra which enlarge when blood drainage through the liver is disrupted.

Lymph Discharge of the Esophagus

The lymphatic vessels generally follow the flow of the arteries. Lymph discharge also occurs through various lymph nodes depending on the segment of the esophagus.

The pars cervicalis drains its lymph through the deep lymph nodes of the throat (nll. cervicales profundi) into the jugular trunk that follows the jugular veins. Metastatic disease of the upper esophagus invades the upper jugular chain.

The upper portion of the pars thoracica runs cranially through the mediastinal lymph nodes (paratracheal nodes, superior and inferior tracheobronchial nodes) into the bronchomediastinal trunk while the lower portion of the pars thoracica, like the pars abdominalis (through the left gastric and coeliac nodes), drains into the intestinal trunk.

This joins with the cisterna chyli which can be pictured as a type of “reservoir” from which the lymph flows solely in the thoracic duct into the left venous angle between the subclavian and jugular veins.

Innervation of the Esophagus

Like other parts of the digestive system, the esophagus is mainly controlled by the autonomous/enteric nerve system with the assistance of the parasympathetic and sympathetic nervous systems.
The sympathetic innervation occurs through post-ganglionic sympathetic fibers from:

- The stellate ganglion
- The sympathetic trunk in the chest
- The thoracic ganglia II-V (the post-ganglionic fibers which extend into the esophageal plexus)

Activation of the sympathetic nervous system causes inhibition of secretion of the esophageal glands.

The parasympathetic innervation carries over in the upper esophageal segment of the recurrent laryngeal nerve, and the lower portion of the esophagus is supplied by the vagus nerve.

Beneath the bifurcation of the trachea, the left and right vagal trunks merge into the esophageal plexus from which the vagal trunks proceed in a distal manner and pass through the esophageal hiatus, together with the esophagus.

Activation of the parasympathetic nervous system induces an increase in gland secretion and heightened peristalsis.

Sensory Innervation

Afferent fibers containing the viscerosensory information of pain and stretching reach the brain from the esophagus through the recurrent laryngeal and vagus nerves.

The Closing Mechanism of the Esophagus

When resting, the esophageal muscles are exposed to greater longitudinal pressure, and the lumen is closed (resting pressure between 10-30 mmHg).

The esophagus possesses closing mechanisms at both its upper and lower ends which are called the upper esophageal sphincter (UOS) and the lower esophageal sphincter (LOS).

Upper esophageal sphincter (UOS)

The upper esophageal sphincter (UOS) consists solely of striated skeletal muscles. Caudal segments of the inferior pharyngeal constrictor muscle, as well as a portion of the upper esophageal musculature, are part of its functional entity. The sphincter is innervated by the glossopharyngeus and the vagus. The upper esophageal sphincter is a barrier against reflux, and it prevents aerophagia.

Lower esophageal sphincter (LOS)

To be precise, the lower esophageal sphincter (LOS) is not a true sphincter. Rather, various mechanisms interlock and form a functional entity that ultimately facilitates the sealing of the esophagus. The insufficient closing of the lower sphincter causes reflux of the stomach’s contents.

Clinical Presentations

Reflux is the return flow of gastric juices into the esophagus which can become inflamed as a result (esophagitis).

Another complication of reflux disease is a peptic ulcer.
Chronic reflux induces a long-lasting state of irritation and can transform the multi-layered, non-keratinizing stratified squamous epithelium into the columnar epithelium. This restructuring of the tissue is called Barrett’s esophagus and is a precancerous condition because of its risk of degeneration to cancer.

The following mechanisms form the functional sealing entity of the lower sphincter:

- **Wringing mechanism** – the smooth musculature in the tunica muscularis runs inward in a spiral shape along the caudal end of the esophagus.
- **Angle of His** – the esophagus joins with the cardia at an acute angle which prevents reflux of gastric juices.
- **Phreno-esophageal ligament** – the ligament allows independent movement of the diaphragm and esophagus during respiration and swallowing.
- **Constriction effect of the diaphragm** – the sides of the diaphragm muscle located at the hiatus adhere firmly around the esophagus.
- **Venous plexus** – extensive venous plexuses are located in the mucosa (namely: lamina propria mucosae) and the submucosa.

The lower esophageal sphincter (LOS) loosens reflexively (via the plexus myentericus) thereby allowing for the transit of food.

Functional disruptions of the sphincter resulting from an insufficient opening lead to achalasia.

**How does Swallowing Happen?**

From an anatomical perspective, the swallowing of food and fluids is facilitated by a highly tensile yet firmly established tissue in the esophagus.

The actual act of swallowing is a semi-reflexive process—i.e. controlling it is partially voluntary, partially involuntary. We willingly decide when we will swallow a bolus of nutrients. However, if it has touched the base of the tongue or the back of the throat, the swallow reflex is triggered and is involuntary.

The swallowing process is subject to sensomotoric fine-tuning where it is adjusted to the consistency of the bolus. In this manner, information on the scent, taste, texture, and size of the bolus are constantly being sent to the brain. With peristaltic movements of the esophageal muscles, food ultimately makes its way to the stomach. When ingesting fluids, for instance, there are rarely any peristaltic movements of the esophagus. The upper and lower esophageus sphincters briefly open while the base of the mouth and the tongue push the fluid down into the stomach by the ‘splash swallow’.

The process is somewhat more complex for solid items.

The swallowing process can be divided into four different phases to illustrate how it works:

**The oral preparation phase**

The oral preparation phase serves to break down and coat the bolus with saliva and is performed voluntarily.

**The oral transportation phase**
The oral transportation phase includes the closing of the lips and jaws as well as the beginning of the lifting of the velum to the nasopharynx; it is a voluntarily triggered, reflexive process.

**The pharyngeal phase**

The pharyngeal phase begins when the bolus has passed through the pharyngeal isthmus and describes the transportation of the bolus through the pharynx while being protected by the airways by lifting the glottis against the epiglottis; this phase is reflexive (swallow reflex).

**The esophageal phase**

The esophageal phase describes the transportation of the bolus through the esophagus into the stomach; it is reflexively controlled.

**Swallowing Reflex**

The broken-down, saliva-coated bolus is passed over the tongue toward the pharynx. If the bolus makes contact with the base of the tongue or the back of the throat, afferents in the n. glossopharyngeus and the n. vagus lead the mechanical stimulation to the deglutition center in the medulla oblongata—the pharyngeal muscles are subsequently activated.

During the swallowing process, the upper and lower airways are sealed, and respiration ceases.

**Closing of the upper airways**

The closing of the upper airways when swallowing is ensured by Passavant’s bar. The superior pharyngeal constrictor muscle contracts and produces a bulge-like swelling of the lateral and posterior epipharyngeal wall. Together with the backward motion of the velum, the bulge seals the nasopharynx while swallowing.

**Trip to the clinic**

If the velum is paralyzed, as may be the case with diphtheria, this closing mechanism becomes insufficient, and food and fluids may enter the nose. Fortunately, diphtheria’s vaccine has nearly eradicated diphtheria.

**Closing of the lower airways**

The vocal folds and epiglottis close, the base of the mouth tightens, and the larynx elevates—meaning that the larynx is lifted upward—causing a closure of the lower airways.

If the airways are secured, the upper esophageal sphincter opens by way of the n. vagus and primary esophageal peristalsis is initiated.

The mid and lower pharyngeal constrictor muscles contract thereby moving the bolus to the esophagus. After the food has entered the esophagus, the upper esophageal sphincter (UES) closes, and the airways reopen.

The bolus now “slides” downward toward the stomach because of gravity and the peristaltic motions of the esophageal muscles.
**Esophageal Peristalsis**

Primary esophageal peristalsis describes contractive waves of the esophageal muscles toward the lower esophageal sphincter (LES).

Secondary esophageal peristalsis is caused by the stretching of the esophageal wall induced by the bolus.

The lower esophageal sphincter (LES) must relax for the bolus to enter the stomach which occurs reflexively under the control of the myenteric plexus.

**Important USMLE Question. LES-tone is lowered by:**

Lower esophageal sphincter (LES) tone is lowered by:

- Secretin
- Cholecystokinin
- GIP (gastric inhibitory peptide)
- Progesterone (pregnancy heartburn)

Note: A tone reduction of the lower esophageal sphincter (LES) leads to insufficient closing to heartburn. Fat, alcohol, coffee, and nicotine lower the muscle tone.

Lower oesophageal sphincter (LOS) tone is increased by:

- Motilin
- Gastrin
- Substance P

Note: Achalasia is a degenerative disease that leads to expansion of the esophagus upon insufficient relaxation of the lower esophageal sphincter (LES).

After entering the cardia, the LES closes again. Passage of one bolus of solid food takes between 5 and 25 seconds.

**Diseases of the Esophagus**

Reflux disease is also known as gastroesophageal reflux disease or GERD.

This widespread disease describes a chronic condition of increased reflux of gastric juices into the esophagus which may lead to regurgitation, retrosternal pain, and hoarseness.

Since the mucous layer of the esophagus does not offer enough protection against the aggressive action of hydrochloric acid, the result may be esophagitis or even erosion (ulcers). The cause of reflux disease is related to a dysfunctional lower esophageal sphincter.

Reasons for weakness in muscle tone may include:

- Endogenous substances (e.g., progesterone during pregnancy)
- Stimulants and depressants (such as nicotine, caffeine, and alcohol)
- Poor nutrition (excessive sugar and fat; carbonated beverages = “antacids”)
- Vitamin B12 deficiency
- Stress (activating an increase in the sympathetic nervous system)
- Increased pressure on the lower esophageal sphincter (LES)/intra-abdominal (pregnancy, obesity, digestion problems, lying down)
- Hiatal hernias

Therapy is contingent on the triggering factors and primarily consists of removing these triggers. While simpler, stress-induced discomfort can often be improved by learning relaxation procedures (e.g., PMR) and the avoidance of poor nutrition habits, surgery may be necessary for severe or chronic progression.

Furthermore, highly effective medications (antacids, H2 blockers, and proton-pump inhibitors = PPIs) are available.

**Esophagitis**

In esophagitis, the esophagus is inflamed due to a chronic state of irritation. Most of the causes lie in the long-term effects of noxes on the mucosa and may come in many different forms:

- HCL (gastro-esophageal reflux disease, constant vomiting from bulimia, etc.)
- Alcohol (high percentage – from alcohol abuse)
- Mucous-damaging medication
- Infections (such as cytomegaly, herpes, and candida)

**Barrett’s Esophagus**

Chronically untreated reflux disease may result in metaplasia of the esophageal epithelium where the normal multi-layered, non-keratinizing stratified squamous epithelium is turned into columnar epithelium. This epithelium is resistant to stimuli, but it is also susceptible to dysplasia which may, in turn, become an early stage of cancer. For that reason, Barrett’s esophagus is also considered precancerous.

**Esophageal Cancer**

Esophageal cancer refers to the name of a malignant tumor in the esophagus. This type of tumor is more frequent in patients exhibiting nicotine, alcohol abuse, or an overload of nitrosamines- substances present in soy sauce, cured meats, and beer. Squamous epithelium carcinoma is more frequently encountered than adenocarcinoma and occurs in the upper 2/3 of the esophagus while the GRED-related columnar epithelium type of cancer occurs in the lower 1/3 of the esophagus.

**Hiatal Hernia**

Normally, the esophageal hiatus is constrained by the contraction of the diaphragm during inhalation. This ensures that no stomach contents enter the esophagus as a result of the intra-abdominal pressure increase during inhalation.

In the case of a hiatal hernia, the esophageal hiatus forms a type of hernial orifice through which parts of the stomach or the entire stomach are permanently or temporarily displaced into the chest cavity. One cause of a hiatal hernia is the acquired expansion of
the esophageal hiatus.

This is facilitated by age-related loss of the conjunctive tissue’s elasticity or the increase of intra-abdominal pressure, as is the case during pregnancy, obesity, or chronic coughing.

There are various types of hiatal hernias:

- Sliding hernias (axial hernias)
- Paraesophageal hernias
- Hybrids
- Upside-down stomach

Depending on the type of a hernia, the function of the lower esophageal sphincter may be disrupted which may result in heartburn with all of its after-effects. Para-esophageal hernias often cause a feeling of pressure in the heart area, or shortness of breath, and have a high rate of complications—esophageal/gastric incarceration, a twisted stomach, or similar conditions. Paraesophageal hernias must be treated surgically due to the high rate of complication.

**Esophageal Varices**

With congestion of the portal vein (e.g., resulting from cirrhosis of the liver), the blood must be re-routed through a circumventing route (portocaval anastomoses). One of these anastomoses occurs through the esophageal veins:

**Vena portae hepatis**

- Gastric veins
- Esophageal veins
- Azygos/hemiazygos veins
- Superior vena cava

This may result in the expansion of the vv. oesophagea—the esophageal varices. Rupture of the varices is associated with haematemesis and represents an absolute emergency.

**Diverticula**

**Pulsation diverticulum / pseudo-diverticulum**

Pulsion diverticulum occurs as a result of increased intraluminal pressure. There are three types of pulsion diverticulum based on the location along the oesophagus: the Zenker’s diverticulum (ZD), the mid-oesophageal diverticulum (MD) and the epiphrenic diverticulum (ED). Pulsion diverticulum is also considered a pseudo (fake) diverticulum as it does not involve all layers of the esophageal wall, and it does not have a muscle coat. This type of diverticulum forms through dilation in the mucosa and submucosa in the esophagus, in the weak area of the Laimer’s triangle.

**Traction diverticulum / true diverticulum**

The traction diverticulum has all the layers of the oesophageal wall and is a true diverticulum. These diverticula form when adjacent structures form scarring, pulling on the whole wall of the esophagus (e.g., parabronchial diverticula, epiphrenic diverticula).
Foetor ex-ore (stinky breath) and dysphagia are common in traction diverticulum. This may especially result in regurgitation and aspiration during the night. Diverticula can easily become inflamed or form fistulae with the accumulation of food particles or food remains.

**Achalasia**

A damaged myenteric plexus leads to a disruption of esophageal peristalsis and insufficient slackening of the lower sphincter, resulting in the typical “champagne glass” dilation of the esophagus.

Patients may present with complaints about difficulty swallowing, regurgitation, and a feeling of retrosternal pressure.

**Review Questions**

The solutions are found beneath the bibliography.

1. **The tone of the lower esophageal sphincter is lowered by:**
   - A. Secretin
   - B. Motilin
   - C. Estrogen
   - D. Gastrin
   - E. Substance P

2. **Peristalsis of the esophagus is controlled by:**
   - A. Plexus myentericus
   - B. Plexus submucosus
   - C. Meissner’s plexus
   - D. Glossopharyngeus

3. **The so-called Laimer’s triangle...**
   - A. ...leads to achalasia.
   - B. ...forms varices in the esophagus.
   - C. ...is the cause of Barrett’s esophagus.
   - D. ...can form traction diverticula.
   - E. ...is a missing piece of longitudinal muscle in the dorsocranial area of the esophagus.

**References**


**Correct answers:** 1A, 2A, 3E

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