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M1 - GI Sequence

Salivary Glands and Esophagus

John Williams, M.D., Ph.D.

Winter, 2009



SALIVARY GLANDS

Saliva is produced by a collection of three major (parotid, submandibular and lingual) and a number of minor (lingual, buccal) glands. Glands are acinar-ductular in structure. Acinar cells are histologically classified as serous or mucous. Serous cells secrete a watery fluid containing soluble proteins such as salivary amylase (parotid). Mucous cells secrete viscous fluid containing mucus. Acinar cells are surrounded by contractile myoepithelial cells. Ducts from acini to mouth include intercalated ducts, striated ducts and main duct. Cells in striated ducts are specialized for ion transport and modify the acinar secretions.

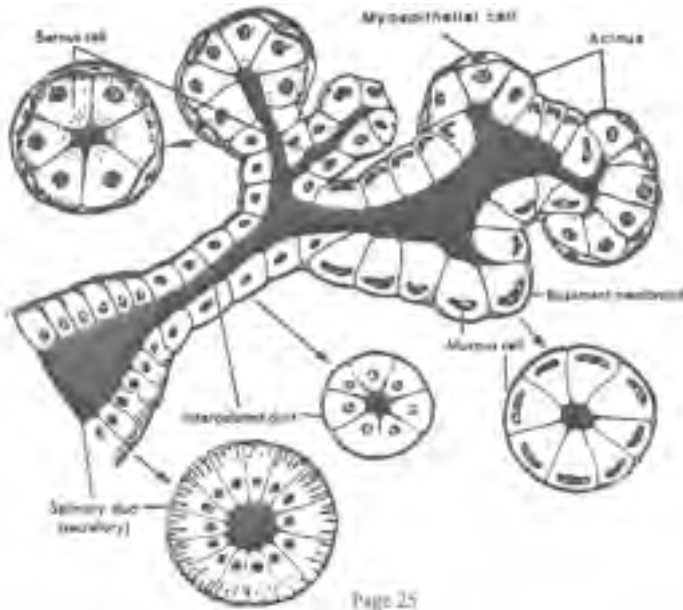


Fig. 2-3 Granger, D, et al. *Clinical Gastrointestinal Physiology*. W.B. Saunders, Philadelphia, PA; 1985:35. Modified from Berne, RM, Levy, MN. *Physiology*. C.V. Mosby St. Louis; 1983: 770.

Contents of Saliva

H₂O

Ions (HCO₃⁻)

Enzymes

Amylase

Antibacterial Compounds

Lysozyme

Lactoferrin

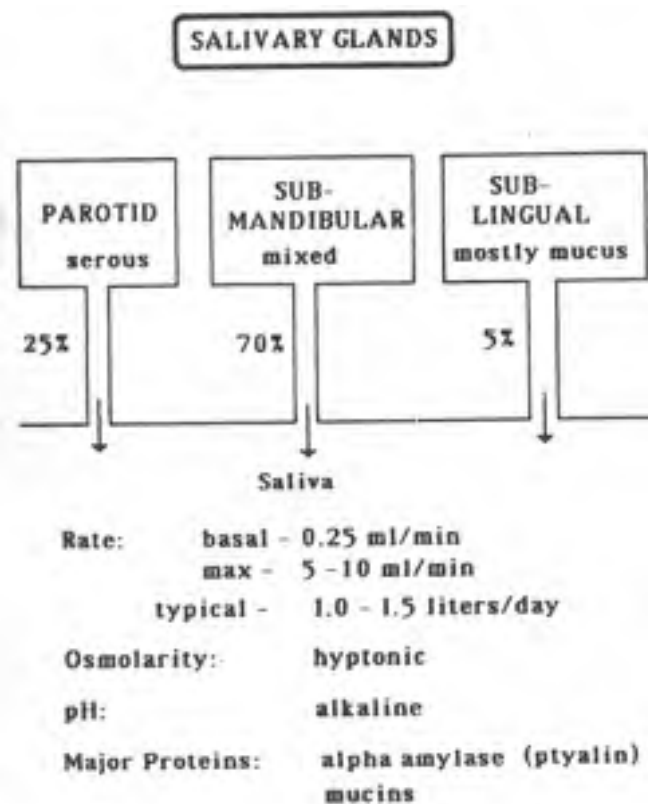
IGA

Mucus

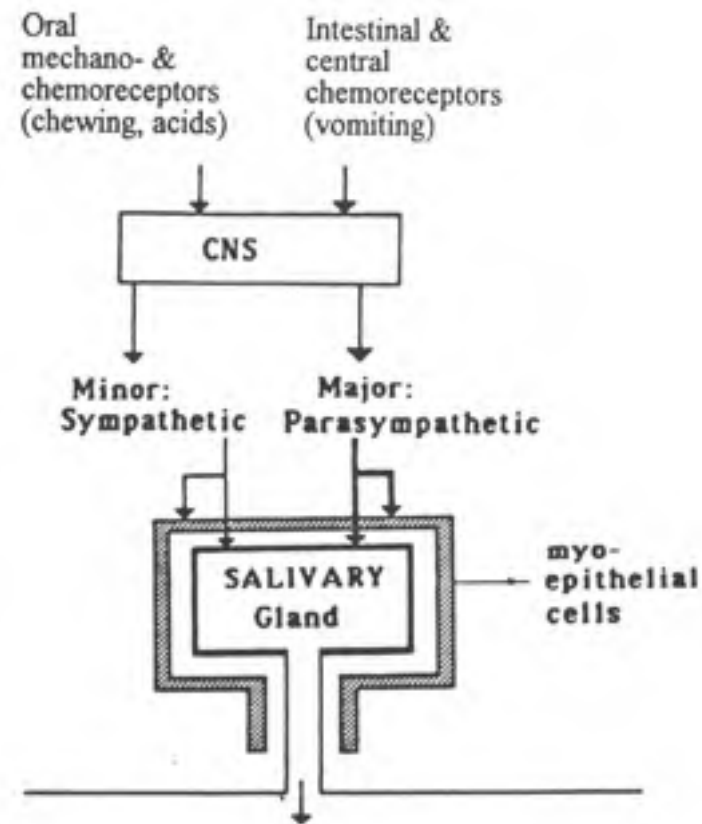
Function of Saliva

1. Lubrication of food - mucins
2. Partial digestion of polysaccharides - amylase
3. Moisten mouth and wash away dissolved food (necessary for taste)
4. Mild antibacterial - lysozyme, lactoferrin
5. Neutralize acids in food and regurgitated stomach acid
6. Maintenance of teeth - Ca²⁺, fluoride

The functions of saliva can be divided into those concerned with lubrication, digestion and protection. Salivary amylase like pancreatic amylase, cleaves internal α -1, 4 bonds in starch and can break down up to 50% of starch before being inactivated by gastric acid; however, normal digestion occurs in it's absence and in the intestine it normally makes up only 10% of amylase. The absence of saliva is termed xerostomia (dry mouth). It can be caused by drug side effects, head and neck radiation therapy or systemic disease such as Sjögrens syndrome. The absence of saliva leads to infections, tooth decay and severe discomfort.



PD-TNCL John Williams

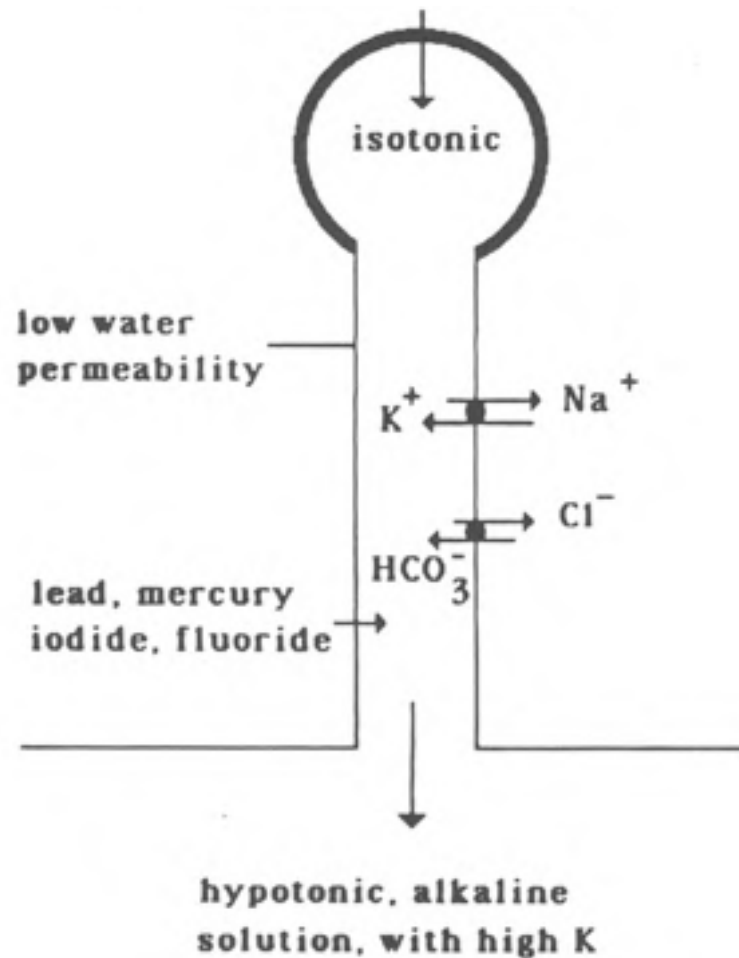


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Salivary secretion is primarily controlled by the autonomic nervous system and is the only area of the GI tract not regulated by GI hormones. Parasympathetic innervation, the predominant control begins in the Salivatory Nuclei of the medulla and is carried in the V, VII and IX cranial nerves. The salivatory nucleus is stimulated by taste, smell and chewing and is inhibited by sleep, fatigue and fear.

Because ACh is the major transmitter, anticholinergic drugs induce dry mouth. Sympathetic innervation reaches glands through the superior cervical ganglia and acts on glandular β -adrenergic receptors. Aldosterone modifies the ionic composition of saliva by decreasing Na^+ but doesn't regulate flow.

Fluid flow Model for Production of Saliva

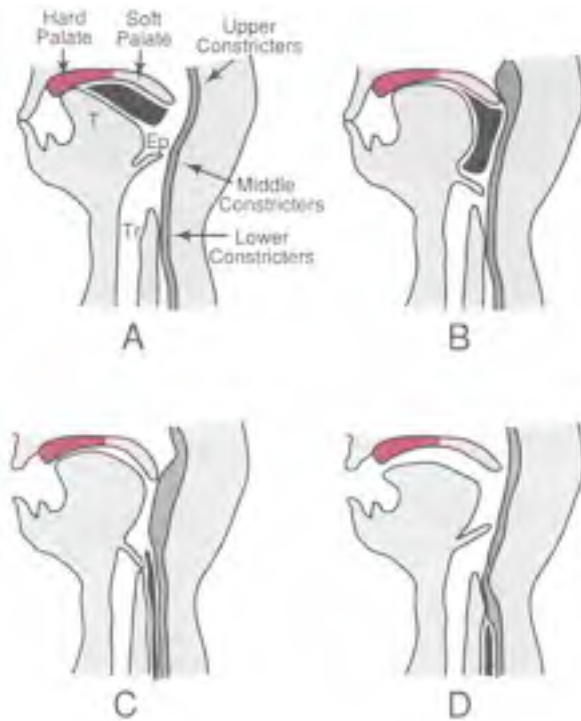


 Source Undetermined

Because of the high rate of fluid secretion, salivary glands have a high resting blood flow which is further increased upon secretion by parasympathetic innervation of blood vessels and by release of the local mediator Kallikrein which leads to production of the vasodilator, bradykinin.

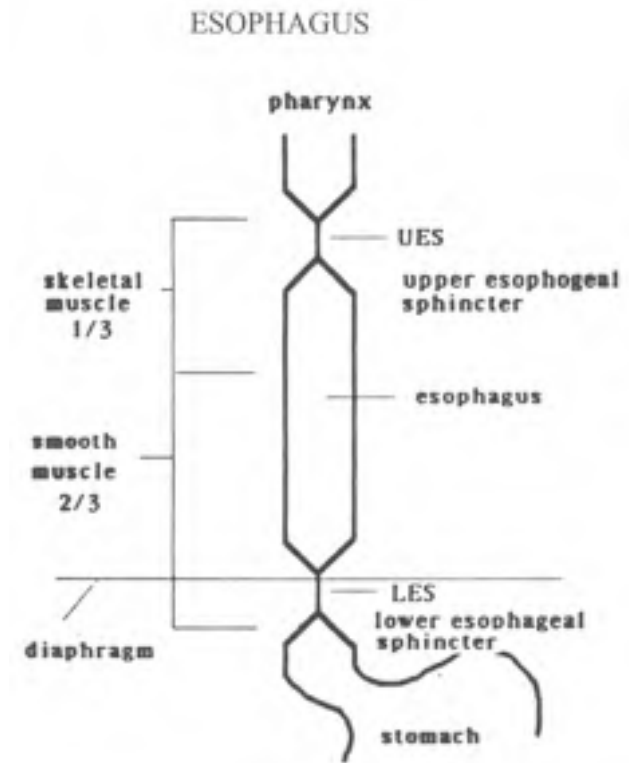
The fluid secreted by acinar cells is plasma like in its composition of Na^+ , K^+ , Cl^- and HCO_3^- . As fluid passes down the ducts Na^+ and Cl^- are reabsorbed and K^+ secreted as a result of the activity of the abundant Na^+-K^+ ATPase present in duct cells. A $Cl^-HCO_3^-$ exchanger present in the apical membrane of duct cells is responsible for the alkalization of saliva. As the duct cells are relatively impermeable to water, saliva becomes hypotonic, particularly at low flow rates. With stimulation the higher rate of flow means less time for ductular modulation and ion concentrations become more plasma-like but still with elevated K^+ and HCO_3^- . Aldosterone stimulates Na^+-K^+ exchange in the ducts.

CHEWING AND SWALLOWING



PD-INEL Fig. 3 Johnson, Leonard, *Essential Medical Physiology*. Raven Press, New York, NY, 1992: 463.

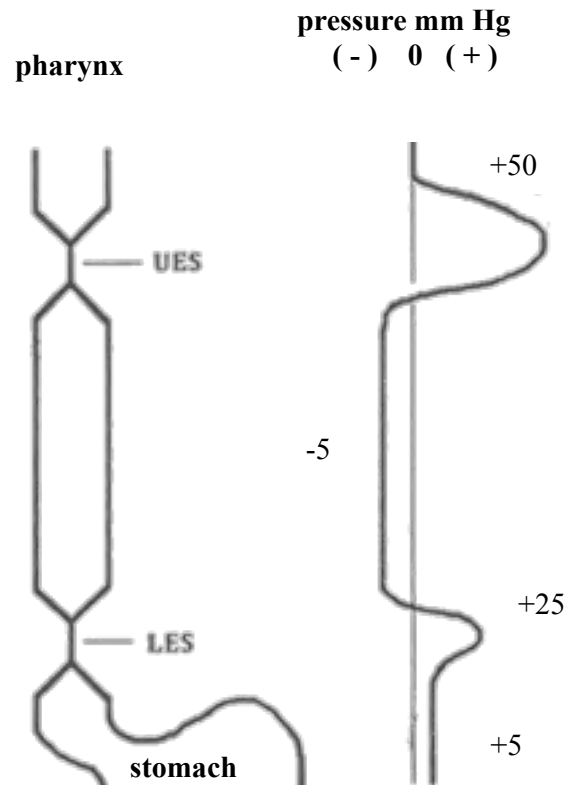
Chewing is under voluntary neuromuscular control and is controlled by the swallowing center in the brainstem. Swallowing begins as a voluntary action but proceeds reflexively through nerves from medullary centers to the pharyngeal muscles. In A) the tongue is shown pushing the soft palate up to seal off the nasal pharynx. In B) the tongue pushes food backward and in C) the superior pharyngeal muscles contract above the bolus forcing it down into the pharyngeal channel. Distal pharyngeal muscles relax and the upper esophageal sphincter opens. At the same time the glottis moves up and the epiglottis closes off the trachea to prevent food from entering the airways. With passage of food in D) the pharyngeal muscles relax and return to resting position.



PD-INEL Jim Sherman

The sole purpose of the esophagus is to convey food from the pharynx to the stomach. It is a muscular tube about 25 cm long with a sphincter at each end. The upper sphincter is anatomical while the lower is functional. Swallowing is followed by a wave of primary peristalsis which moves at 2-4 cm/sec. When the wave reaches the LES, the LES relaxes to let food enter the stomach. Secondary peristaltic waves can be initiated by distension in the absence of swallowing. Esophageal peristalsis can occur in the absence of connections to the CNS and is mediated by the enteric nervous system.

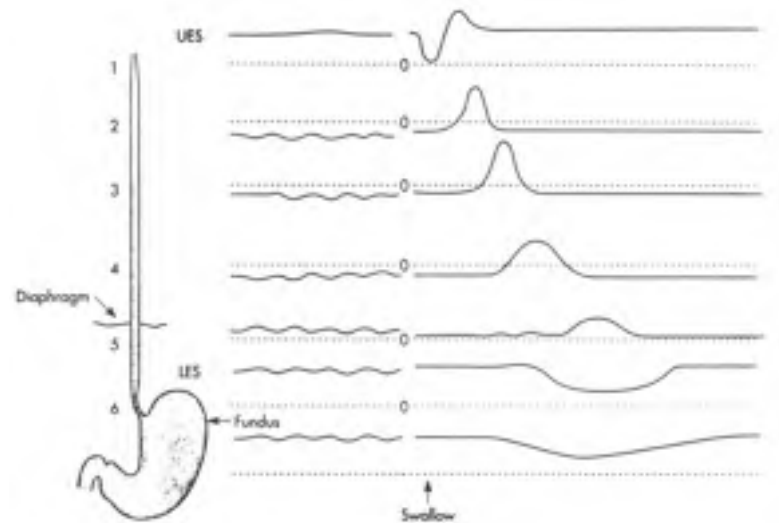
Esophageal Pressures Measured by a Catheter



PD-TNEL Jim Sherman

Reference is atmospheric pressure (0).

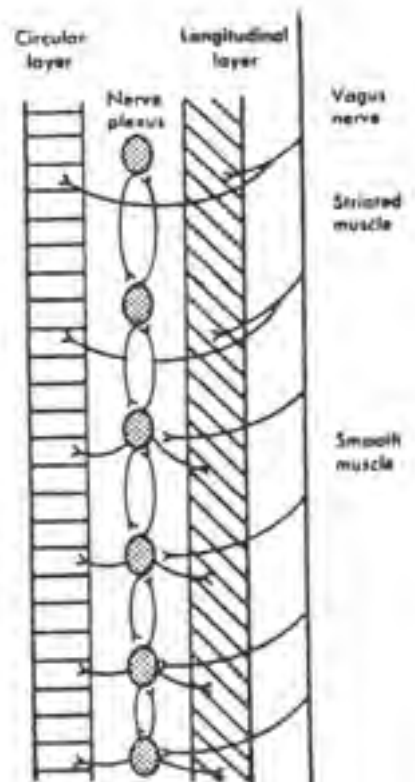
Pressure Changes During a Primary Peristaltic Wave




PD-TNEL Fig. 3-3 Johnson, L. *Gastrointestinal Physiology*, 7th ed. Mosby Elsevier, Philadelphia, PA; 2007: 26.

Between swallows the pressure at the upper and lower esophageal sphincter is positive due to intrinsic muscular tone. Pressure in the esophagus reflects intrathoracic or intra-abdominal pressure, hence negative and positive, respectively, while pressure in the fundus is due to intraabdominal pressure plus tonic contraction of fundic smooth muscle. Upon swallowing the UES relaxes and then contracts and this contraction is followed by peristaltic contraction of the body of the esophagus. The LES and fundus relax before the peristaltic contraction arrives to allow passage into the stomach. This relaxation of the gastric fundus is known as “receptive relaxation.”

Neural Innervation of the Esophagus



 Fig. 3-4 Johnson, L. *Gastrointestinal Physiology*, 6th ed. Mosby Elsevier, St. Louis, MO; 2001: 32.

The body of the esophagus receives efferent innervation through the vagus nerve. Visceral somatic fibers directly innervate the striated muscle of the upper one-third of the esophagus. In the lower portion that is made up of smooth muscle, vagal preganglionic fibers synapse on ganglion cells which then innervate smooth muscle cells. The neural circuitry mediates the peristaltic wave with contraction behind and relaxation in front of the bolus of food.

Regulation of the Lower Esophageal Sphincter

1. LES contraction regulated by intrinsic properties of smooth muscle, nerves and hormones
2. Basal tone is myogenic but increased by ACh and Gastrin
3. Transient relaxation mediated by inhibitory neurons that use VIP or NO as a neurotransmitter
4. Sphincter tone lacking in newborns and decreased during pregnancy

Abnormalities of LES

1. Failure of LES to function as a sphincter leads to reflux esophagitis, “heartburn.”
2. GERD – Gastroesophageal Reflux Disease
3. Failure of LES to relax results in achalasia. Over time leads to dilation of the esophagus.

Additional Source Information

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Slide 4 – Fig. 2-3 Granger, D, *et al. Clinical Gastrointestinal Physiology*. W.B. Saunders, Philadelphia, PA; 1985:35.

Modified from Berne, RM, Levy, MN. *Physiology*. C.V. Mosby St. Louis; 1983: 770.

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Slide 5 – (Right) John Williams

Slide 6 – John Williams

Slide 7 – (Left) Fig. 3 Johnson, Leonard, *Essential Medical Physiology*. Raven Press, New York, NY, 1992: 463.

Slide 7 – (Right) Jim Sherman

Slide 8 – (Left) Jim Sherman

Slide 8 – (Right) Fig. 3-3 Johnson, L. *Gastrointestinal Physiology*, 7th ed. Mosby Elsevier, Philadelphia, PA; 2007: 26.

Slide 9 – Fig. 3-4 Johnson, L. *Gastrointestinal Physiology*, 6th ed. Mosby Elsevier, St. Louis, MO; 2001: 32.