Human physiology

Digestive system

Digestive system (DS) is a long hollow muscular tube, can be referred to as; digestive tract, gastrointestinal tract, and alimentary control. DS is specialised along its length to break down complex foods into smaller foods that can be absorbed by the intestinal epithelia in the small intestine. DS is a complex, elaborate, multi-organ, physiological system.

Energy
The energy requirements of the human body at rest is 30/kcal/Kg of body weights per day. Energy is obtained through the ingestion of food which is a blend of complex nutrient macromolecules; proteins, lipids, carbohydrates. Macromolecules need to be broken up for our cells to use them as energy, this is the function of the digestive system. The subunits of the macromolecules can then be used by our cells for energy generation and cell growth and repair.

Digestive process
- Ingestion, introducing food into the digestive system via the oral canal
- Propulsion, moves food through the DS at a rate that permits optimal digestion and absorption, includes deglutition and peristalsis. Peristalsis involves alternate waves of contraction and relaxation of muscles in the organ walls in order to squeeze food along the DS. peristalsis is induced by pressure or distension in the gut wall created by food. Involuntary process.peristalsis causes contraction of circular muscles behind food mass then contraction of longitudinal muscles ahead of food mass, these contractions propel food forward.
- Mechanical digestion, increases surface area of ingested food preparing it for digestion by enzymes. In the oral cavity solid food is mechanically digested by chewing, the food is mixed with saliva by the tongue to form food bonuses. In the oral cavity mechanical digestion is important for speeding up the initial enzymatic breakdown of carbohydrates and lipids by salivary amylase and lingual lipase, respectively. In the stomach mechanical digestion occurs by churning. Churning is powerful muscular contractions of the gut wall in the stomach to break food bonuses into smaller particles. The stomach is adapted for this function by having three muscle layers. In the small intestine mechanical digestion occurs by segmentation, which involves rhythmic local contractions to mix food with digestive enzymes.
- Chemical digestion, involves a series of catabolic steps in which enzymes secreted into the lumen of the alimentary canal break down macromolecules into their subunits. Initiated in the oral cavity, occurs in small intestine.
- Secretion, the release of water, acids, enzymes, buffer by the gut epithelium and accessory glands to aid chemical digestion of food
- Absorption, the movement of breakdown products of digestion across the gut epithelium into the interstitial fluid of the gut epithelium then into the lymph vessels or the circulatory system for distribution to cells. The small intestine is adapted to maximise the surface area for absorption
muscle fibres are confined in the tongue and are not attached to the bone. These fibres allow the tongue to change shape for speaking and swallowing. Extrinsic muscle fibres extend to the tongue from the bones on the skull or the soft palate. These fibres alter the tongue's position. The tongue has a median septum of connective tissue. A fold of mucosa called the lingual frenulum secures the tongue to the floor of the mouth and limits its posterior movement. The tongue forms the inferior boundary of the oral cavity.

Most of the oral cavity is lined by a stratified squamous epithelium. However, regions exposed to severe friction, eg. gingive (gums), are covered by a protective layer of keratinised epithelial cells.

Cleft palate and the development of the palate
The external human face develops between the fourth and sixth weeks of embryonic development. Facial swellings arise on the frontonasal process and the first pharyngeal arches (two mandibular and two maxillary processes). These processes come together and form the continuous surfaces of the external face. The primary palate is formed in this period by fusion of the medial nasal and maxillary processes. Between the sixth and the twelfth embryonic weeks, the secondary palate is formed as the result of fusion between palatal processes growing from the oral surfaces of the maxillary processes. Each fusion site is also the site of a potential facial or palatal cleft.

Tissues of the craniofacial complex are derived from neural crest cells, a population of transiently migratory cells that originate from the dorsal aspect of the neural tube during embryogenesis, then migrate to populate the frontonasal processes and pharyngeal arches.

In the sixth week of embryonic development the palatine shelves form on each side of the tongue as downward oblique outgrowths from the maxillary processes. The nasal cavity is open to the oral cavity at this point.

In the seventh week of embryonic development the tongue moves downward within the oral cavity. The palatine shelves elevate to occupy horizontal positions relative to the tongue. Fusion of the palatine shelves with the primary palate separates the oral cavity from the nasal cavity. This isolation helps keep ingested food in the oral cavity.

Cleft palates are common congenital disorders that result in the malformation of the hard palate. Molecular mechanisms control migration of neural crest cells, defects can cause cleft in various parts of the oral cavity. Defects on the mechanisms that coordinate the fusion of the mandibular and maxillary processes cause congenital clefting of the palate.

Clefting may be caused by mutations in genes that operate during embryonic development. Clefting can also be caused by exposure to teratogens during pregnancy. Folic acid deficiency during pregnancy can cause clefting, as folic acid is required for DNA synthesis and neural crest cell proliferation.

Cleft plates cause feeding problems in children.
**Tongue**

The congenital condition, ankyloglossia, is when children are born with an extremely short lingual frenulum, this restricts the tongue's movement causing distorted speech. This condition is corrected surgically by snipping the frenulum.

The tongue's superior surface and lateral edges are covered by papillae, peg-like projections of the underlying lamina propria covered with stratified epithelium. There are three types of papillae; filiform papillae that align in parallel rows on the tongue dorsum these papillae roughen the tongue's surface helping us lick semi solid food and contain keratin which stiffens these papillae, the fungiform papillae are mushroom shaped and are scattered widely over the tongue's surface these papillae have a highly vascular care, vallate papillae are located in a v-shaped row at the back of the tongue, and the foliate papillae which are located on the lateral aspect of the posterior tongue. The fungiform, valliate, and foliate papillae contain gustatory receptors (taste receptors). These receptors detect the main taste modalities; sweet, sour, salty, bitter, and umami. Foliate papillae function in taste primarily in childhood. Serous cells beneath the foliate and vallate papillae secrete lingual lipase. Posterior to the vallate papillae is the terminal sulcus, a groove that distinguishes the portion of the tongue that lies in the oral cavity from its posterior portion in the oropharynx.

**Salivary glands**

Minor salivary glands are exocrine glands producing small quantities of saliva, and are located in the mucous membrane of the cheeks, lips, palate, and tongue.

Major salivary glands -

- The parotid gland lies in the anterior to the ear between the masseter muscle and the skin. Its duct parallels the zygomatic arch, pierces the buccinator muscles and opens into the vestibule next to the second upper molar. This gland is responsible for 50% of saliva production during digestion.
- The submandibular gland lies along the medial aspect of the mandibular body. Its duct runs beneath the mucosa of the oral cavity floor and opens at the base of the lingual frenulum. This gland is responsible for the saliva produced between meals.
- The sublingual gland lies anterior to the submandibular gland under the tongue and its ducts open into the floor of the mouth. This gland is responsible for 10% of saliva production.

The major salivary glands use a branching system of ducts to deliver their secretory products into the oral cavity. Each gland is divided into small globules that contain the structural and functional secretory units- acinus and intercalated ducts. Each acinus contains 15 to 100 acinar cells that synthesise and secrete their protein products into intercalated ducts for delivery to the oral cavity.

The salivary glands are composed of two types of secretory cells; serous and mucous. Serous cells produce a watery secretion containing enzymes and ions. Mucous cells produce mucus. The parotid gland contains only serous cells, the submandibular gland contains serous and mucous cells, and the sublingual gland contains only mucous cells.
sensory neurons to the swallowing centre in the nucleus tractus solitarius located in the medulla and lower pons. The sensory input is processed in the nucleus tractus solitarius (NTS), the NTS then activates the adjacent motor nucleus ambiguus (NA). Motor commands are then sent from the NA, via cranial nerves ix and x, to the skeletal muscles involved in deglutition. The NTS coordinates the contractions of all the muscle groups involved in deglutition and the NA carries out the motor commands of the NTS. Once food enters the pharynx respiration is momentarily inhibited and all routes except the desired one into the digestive tract are blocked off: the tongue blocks off the mouth, the soft palate rises to close off the nasopharynx, the larynx rises do that the epiglottis covers the opening into the respiratory passageways, and the upper esophageal sphincter relaxes. Wavelike peristaltic contractions propel food through the pharynx and into the esophagus. Just before the peristaltic wave, and food, reaches the esophagus, the gastroesophageal sphincter relaxes reflexively to allow food to enter the stomach. After food entry that sphincter closes, preventing regurgitation and acid reflux.

**Dysphagia**

Dysphagia is a common complication of neurological disorders such as amyotrophic lateral sclerosis (ALS). ALS is characterized by selective death of motor neurons in the brain cortex, brain stem and somatic nervous system. Motor neuron loss in ALS results in skeletal muscle atrophy and progressive paralysis. ALS symptoms include, impaired speech, laboured breathing as intercostal muscles become de- elevated, and dysphagia due to death of neurons in NA causing elevation of muscle groups required for deglutition leading to eventual death by asphyxia. ALS is a progressive disease, and death usually occurs within 2-5 years of disease onset.

**Esophagus**

A muscular tube which transports food from the laryngopharynx to the stomach. The resting muscle tone of the circular muscle layer of the proximal esophagus is high to prevent air entering the esophagus. This region is the upper esophageal sphincter. The comparable region in the distal esophagus is the lower esophageal sphincter, which is almost constantly contracted to prevent food reflux from the stomach. The esophagus pierces the diaphragm at the esophageal hiatus to enter the abdomen. It joins the stomach at the cardiac orifice within the abdominal cavity. The cardiac orifice is surrounded by the gastroesophageal or cardiac sphincter, which is a physiological sphincter. That is it acts as a sphincter but the only structural evidence of this sphincter is a slight thickening of the circular smooth muscle layer at that point. The muscular diaphragm surrounding the sphincter helps it keep closed. Mucous cells protect the sphincters and esophagus from stomach acid.

**Esophageal wall**-

The esophagus has all four basic alimentary canal layers-

- The Esophageal mucosa contains a non keratinised stratified epithelium. At the esophagus - stomach junction, the barsaian - resistant epithelium changes abruptly to the simple columnar epithelium of the stomach, which is specialised for secretion.
- When the esophagus is empty, its mucosa and submucosa are thrown into longitudinal folds, when food is in transit, these folds flatten out.
- The diaphragm relaxes and domes upwards, decreasing the volume of the thoracic cavity
- The external intercostal muscles relax, further decreasing the volume of the thoracic cavity
- The decreased volume of the thoracic cavity increases the pressure in the lungs
- Pulmonary pressure rises above atmospheric pressure, and air leaves the lungs down a pressure gradient

Factors affecting ventilation

Airway resistance-
Airflow is inversely proportional to resistance, so airflow decreases as resistance increase. Airflow is directly proportional to the difference in pressure between the external atmospheric and the alveoli. Airways resistance increases as airway diameter decreases.

Lung compliance-
Lung compliance is the ease with which lungs expand. The higher the lung compliance, the easier it is to expand the lungs at any given transpulmonary pressure. Lung compliance is determined by two factors; distensibility of elastic fibres within the lungs, and alveolar surface tension. Lung distensibility is generally high and surfactant keeps alveoli surface tension low, healthy lungs tend to have high compliance.

Gas law and gas exchange

Dalton's law
The total pressure of a gas mixture is the sum of the partial pressures of the components gas.

Partial pressure
Partial pressure is the pressure contributed by a single gas in a given mixture of gases.

Henry's law
The amount of gas which dissolves in a liquid is proportional to the partial pressure of the gas, and its solubility. At a given temperature, the amount of a particular gas in solution is directly proportional to the partial pressure of that gas. At a given pressure, the number of dissolved gas molecules will rise until an equilibrium is reached.

External respiration
Carbon dioxide diffuses from pulmonary capillaries into alveoli, and oxygen diffuses from alveoli into pulmonary capillaries. Factors affecting external respiration include; partial pressure gradients and gas solubilities, thickness and surface area of respiratory membrane, and ventilation - perfusion coupling i.e. matching alveolar ventilation with pulmonary blood perfusion. A steep oxygen pressure gradient exists across the respiratory membrane, as the partial pressure of oxygen in deoxygenated blood in the pulmonary arteries is lower than the
blood cell membrane protein. In the lungs this process is reversed. As blood moves through the pulmonary capillaries, its partial pressure of carbon dioxide declines. For this to occur carbon dioxide must first be freed from its ‘bicarbonate housing’, bicarbonate ion re-enter red blood cells and combines with hydrogen ions to form carbonic acid, which is split into carbon dioxide. Carbon dioxide then diffuses along its partial pressure gradient from the blood into the alveoli.

The haldane effect- the lower the partial pressure of oxygen and the lower the haemoglobin saturation with oxygen, the more carbon dioxide that the blood can carry. As carbon dioxide enters the systemic bloodstream, it causes more oxygen to dissociate from haemoglobin. The dissociation of oxygen allows more carbon dioxide to combine with haemoglobin. The Haldane effect encourages carbon dioxide exchange in both the tissues and the lungs. As haemoglobin becomes saturated with oxygen the hydrogen ions released combines with bicarbonate ions helping to unload carbon dioxide from the pulmonary blood. The hydrogen ion released during carbonic acid dissociation is buffered by the haemoglobin. The bicarbonate ions generated in red blood cells diffuses into the plasma where it acts as the alkaline reserve part of the bloods carbonic acid-bicarbonate buffer system/ this system resists shifts in blood pH. If the hydrogen ion concentration rises, excess hydrogen ions is removed by combining with bicarbonate ions. If the hydrogen ion concentration drops, carbonic acid dissociates.

Matching oxygen unloading with metabolic need
Tissues of high metabolic need has; a higher oxygen consumption, a lower partial pressure of oxygen, a higher carbon dioxide production, a higher partial pressure of carbon dioxide, increased muscle temperature, and a lower pH than resting tissues. All these factors promote oxygen release.

Ventilation and regulation of breathing
Voluntary breathing has its pathways in the motor cortex, and involuntary breathing is controlled by sensory feedback receptors in the brain stem and is sensitive to arterial partial pressure of oxygen, the partial pressure of carbon dioxide, and pH.

Respiratory muscle innervation
Dorsal respiratory group-
The neurons of the dorsal respiratory group are active just prior to and during inspiration and are mainly inspiratory neurons. These neurons receive input from higher brain centres, including the cerebral cortex and pons. They also receive inputs from carotid bodies and the vagus nerve. Inspiration is initiated by the dorsal neurons. The dorsal respiratory groups are responsible for innervation of the diaphragm, they contract the diaphragm increasing the volume of the thoracic cavity during inspiration.

Ventral respiratory group-
The neurons of the ventral respiratory group consist if both inspiratory and and expiratory neurons. Expiratory neurons inhibit phrenic nerves, thus inhibiting the contraction of the diaphragm. Ventral neurons stimulate internal and external intercostal muscles.
Pontine respiratory group-
Neurons in the pons and medulla oblongata establish the respiratory rhythm. Some pontine neurons are only active during expiration and some pontine neurons are active during both inspiration and expiration. The Pontine neurons may have a possible role in switching between inspiration and expiration.

Respiratory rhythm
Breathing is a rhythmic process, and the origin of this rhythm may be pacemaker neurons. Normal respiratory rhythm results from reciprocal inhibition of interconnected neuronal networks in the medulla. Inspiration begins with the firing of inspiratory neurons, which causes progressively more inspiratory neurons to be activated, thus increasing the strength of contraction of respiratory muscles. Then inhibitory neurons are activated causing inhibition of inspiratory neurons, thus causing the relaxation of respiratory muscles.

The respiratory centre inspiratory neurons send nerve signals causing the contraction of the diaphragm and internal intercostal muscles leading to inspiration. Inhibitory neurons send signals that inhibit inspiratory neurons, thus preventing contraction of muscles and allowing the diaphragm and internal intercostal muscles to relax, leading to expiration.

The basic rhythm of breathing is modified by input from specialized receptors.
Central chemoreceptors-
Central chemoreceptors are located in the ventrolateral surface of the medulla oblongata. These neurons are anatomically distinct from, but synaptically connected to, the respiratory centre. This connection allows rapid communication between chemoreceptors and the respiratory centre. Central chemoreceptors are sensitive to pH. However, hydrogen ions cannot cross the blood brain barrier. So as the partial pressure of carbon dioxide in the blood rises, arterial carbon dioxide crosses the blood brain barrier and diffuses into the cerebrospinal fluid, here carbon dioxide is hydrated to carbonic acid. Carbonic acid then dissociates, thus liberating hydrogen ions, lowering the pH of cerebrospinal fluid. Hydrogen ions directly stimulate chemoreceptors. As a result the rate and depth of breathing increases to remove carbon dioxide. This response is slow as it takes time for carbon dioxide to diffuse across the blood brain barrier.

Peripheral chemoreceptors-
Peripheral chemoreceptors are located in the aortic arch, as receptors located here can detect oxygen pressure in blood fresh from the lungs. These receptors are also located in carotid arteries, as blood here is going to the rain and so oxygen content must be sufficient. Carotid and aortic bodies send sensory information to the medulla via the glossopharyngeal nerve and the vagus nerves, respectively. Response by peripheral chemoreceptors is much greater than that by central chemoreceptors, as carbon dioxide does not need to diffuse across the blood brain barrier. Peripheral chemoreceptors are stimulated by hydrogen ions in the same way as central chemoreceptors. When the partial pressure of oxygen falls, peripheral chemoreceptors become excited and stimulate respiratory centres to increase ventilation. Changes in arterial pH resulting from carbon dioxide retention or metabolic factors act indirectly through peripheral chemoreceptors to alter ventilation.