Chapter 1

Anatomical and Physiopathological Aspects of Oral Cavity and Oropharynx Components Related to Oropharyngeal Dysphagia

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1. Introduction

Dysphagia (from the Greek words *dys,* difficulty, and *phagein,* to eat) is a congenital or acquired swallowing disorder that has structural and functional causes that promote a delay or difficult in the passage of food and liquids from the oral cavity to stomach. Remarkably, dysphagia is an underestimated neuromuscular disorder, although its consequences frequently are associated with high rates of morbidity and mortality. Estimates of oropharyngeal dysphagia prevalence vary broadly (ranging from 10% to 80%) according to screening methods used and especially the type of study population [1-3]. Dysphagia exhibits a multifactorial etiology, with partipation of exogenous and endogenous factors. The most common causes of dysphagia are divided into the categories of iatrogenic (such as patients with previous history of intubation, tracheostomy, or nasogastric feeding tubes, or a history of infection or metaboic disorders), medications (such as polypharmacy, depressors of the central nervous system, anticholinergics, sympathomimetics, and diuretic drugs) neurological diseases (such as stroke, dementia, amyotrophic lateral sclerosis, Parkinson's disease, Alzheimer's disease, extrapyramidal disorders), neuromuscular (such as myasthenia gravis and inflammatory myopathies), or structural obstruction (such as Zenker's diverticulum, oropharyngeal tumors, and factors that causes extrinsic compression of the upper aerodigestive tract), as well as other causes [4-7]. Clinically, dysphagia might be classified into three major types: oropharyngeal dysphagia, esophageal dysphagia, and functional dysphagia. Oropharyngeal dysphagia is the inability to initiate the act

of swallowing, whereas esophageal dysphagia is the perception of difficulty of passing solids or liquids from the throat to the stomach. Functional dysphagia refers to a condition in which some patients complain of dysphagia but do not have an organic cause for a swallowing disorder. The most common symptoms of oropharyngeal dysphagia include difficulty in manipulating food, problems with saliva production, and difficulty in chewing the food and swallowing the bolus (a soft mass of chewed food mixed with saliva at the point of swallowing), and an associated impaired quality of life. Frequently, patients with oropharyngeal dysphagia exhibit a series of complications such as nasal regurgitation, coughing, suffocating, gurgle or wet voice after swallowing, unexplained weight loss, anxiety, depression, low-tract respiratory infections and, it's most serious complication, aspiration pneumonia. Problems with social isolation and poor quality of life are a common feature of individuals with dysphagia. Notably, the occurrence of dysphagia is associated with high mortality rate [8].

The different parts of the oral cavity and oropharynx are made up of several cell types and tissues (nerves, fibrovascular, cartilaginous, lining and salivary glandular epithelia, and smooth and striated muscles) along with mineralized tissues (enamel and dentin of the teeth and bones) [8, 9]. Notably, there is an intimate relationship between dysphagia and anatomical, functional, and regulation disturbances of oral cavity and oropharynx components related to physiological salivation, chewing, and swallowing. Salivation depends of the anatomical and functional integrities of the minor and major salivary glands. The saliva lubricates the oral cavity and oropharynx, and an adequate salivary flow assists the initial digestive process by reducing the bolus size of food, begins the enzymatic digestion of some types of carbohydrates, and provides moisture and lubrication of the food particles in order to facilitate the swallowing mechanism, i.e., the movement of the bolus from oropharynx to esophagus [10]. Chewing and swallowing are likely complex and well-coordinated motor programs, combined together as a sequence. During chewing, the food particles are reduced in size and consistency. Chewing is highly dependent of an efficient participation of the teeth, a mineralized tissue whose occlusal surfaces are frequently used for cut off, rip, knead, and grind food during feeding. Moreover, the masticatory muscles have a pivotal role in establishing the muscle strength necessary for the implementation of chewing activity in order to manipulate and grind the food [11-13]. During swallowing two essential and vital functions must be executed: bolus transport and airway protection. After adequate bolus preparation, it needs to be swallowed through an involuntary transport process from the oral cavity and pharynx to the esophagus without allowing the entry of food particles or liquid in the respiratory trac [14, 15]. Together, salivation, chewing, and swallowing, therefore, plays a critical role in alimentary events, allowing food to be initially processed, formed into a bolus, and subsequently transported in the digestive system. Individuals with health problems related to these mechanisms often present with complaints of oropharyngeal dysphagia.

In this present chapter, we will highlight a series of morphological and physiological aspects related to the oral cavity and oropharynx. Moreover, we will discuss the physiopathological aspectos of the salivation, chewing, and swallowing mechanisms in order to allow to health professionals to obtain essential knowledge for management of oropharyngeal dysphagia.

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2. Anatomical and functional aspects of the oral cavity and oropharynx

2.1. The oral cavity and the oropharynx

Anatomically, the oral cavity or mouth is an organ of the digestive system that is anteriorly delimited by the lips, posteriorly by the oropharynx, superiorly by the hard and soft palates, and inferiorly by the tongue (anterior 2/3) and floor of the mouth, and surrounded by a buccal mucosa that lines the cheeks, along with the upper and lower teeth and periodontum. The upper teeth are embedded in the maxilla and the lower teeth are embedded in the mandible, which articulates with the temporal bones of the skull. The oropharynx is the part of the throat just behind the mouth. It includes the base of the tongue, the soft palate, the tonsils, and the side and back wall of the throat. The oropharynx is the middle part of the pharynx (between the nasopharynx and hypopharynx/laryngopharynx) that is located behind the oral cavity (the palatoglossal arch) extending from the uvula to the level of the hyoid bone. It opens anteriorly into the mouth through the isthmus faucium. In this site, the oropharynx is delimited by the base of the tongue (posterior 1/3) and the upper border of the epiglottic vallecula. Laterally, it is formed by the palatine tonsils, tonsillar fossa, and tonsillar pillars located between the palatoglossal and palatopharyngeal archs. Superiorly, its wall consists of the inferior surface of the soft palate and the uvula [8, 16] [Figure 1].

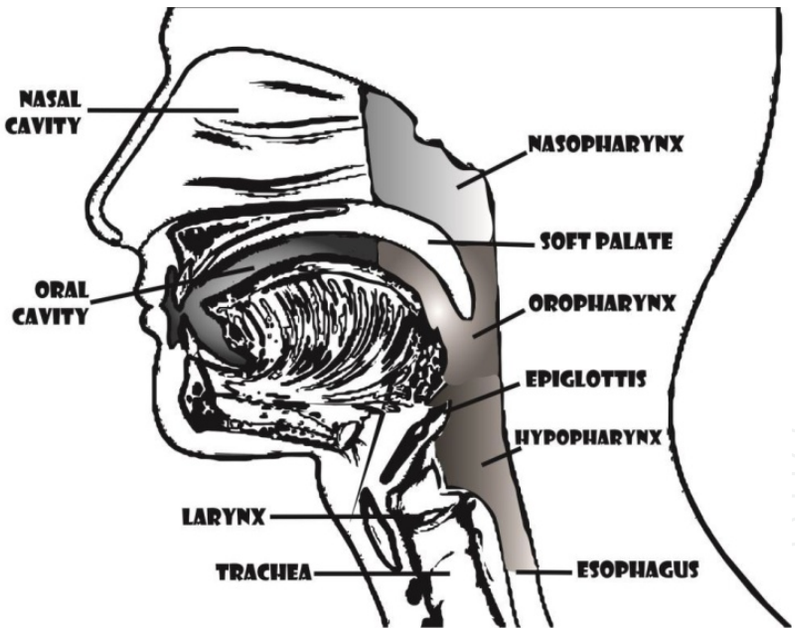

Figure 1. Anatomical aspects of the oral cavity and oropharynx.

2.2. The teeth

The tooth is an organ that consists of a mineralized, inert, and acellular superficial tissue (enamel, an exclusive hard tissue produced by epithelial cells of ectodermal origin known as ameloblasts) but supported by a less mineralized, more resilient, and vital hard tissue (dentin,

which is secreted by a cells of neural crest origin known as odontoblasts) which is formed from and supported by a rich innervated and vascular connective tissue (the dental pulp, which is rich in fibroblast-like cells, blood vessels and nerves). Mammalian tooth development is regulated by means of sequential and reciprocal interactions between the cranial neural crest-derived mesenchymal cells and the ectoderm-derived dental epithelium. The teeth are found in the entrance of the oral cavity and constitute about 20% of the structural area of mouth. Anatomically, the tooth consists of a crown and a root, and the junction of the two regions is known as cervical margin (Figure 2A). The teeth have an important role in food processing due to different actions performed by their occlusal surfaces during chewing (Figure 2B). However, the teeth also exhibit other functions, such as defense, proper phonetic articulation, and esthetics in humans. Due to a specialized supporting biological apparatus that consist of the cementum (a mineralized and avascular tissue composed of apatite and organic matrix rich in collagen whose function is to anchor the fiber bundles of the periodontal ligament to the tooth root), periodontal ligament (a highly specialized connective tissue composed for collagen fibers bundles that connect the cementum that cover the tooth root to the alveolar bone whose roles are related to teeth flexibility and sensorial receptor functions), and the alveolar bone (mineralized tissue that support the teeth), the teeth are found firmly attached to the jaws. The hardness of teeth, which is determined by a rich hydrated biologic apatite crystal amid an organic matrix, the number of teeth, the total superficial area formed by the occlusal surfaces, and the supporting tissues allow the teeth to withstand to the forces of the chewing [17, 18].

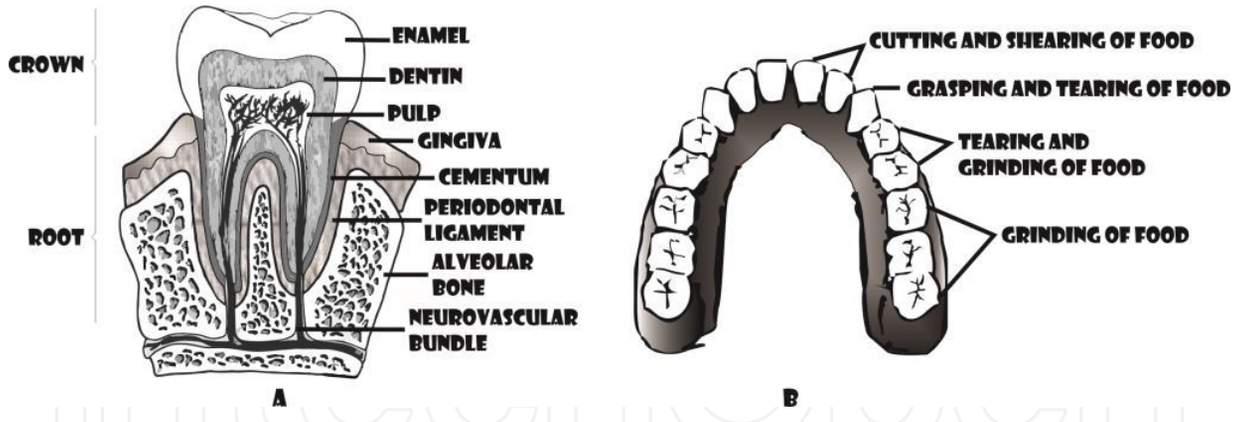

Figure 2. Structural components of the tooth and periodontal support (A). Occlusal surfaces of the human teeth (B).

2.3. The oral mucosa

The oral mucosa is lined by a mucous membrane that consists of a lining epithelial tissue and an underlying connective tissue. The oral mucosa can be classified as follows: lining, masticatory, and specialized. Among its functions, the oral mucosa has been related to protection, taste sensation, and chewing. The lining of oral mucosa must be as flexible as possible in order to be protective. Related to chewing, the oral masticatory mucosa permits a free movement of the lips, tongue, and cheek muscles. It exhibits a covering of keratinized epithelium and its

connective tissues is strongly attached to the bone to withstand the constant mastication of food. The lips exhibit cutaneous (external face, skin), semi-mucosa (transition between external and internal surfaces), and mucosa (inner surface in contact with the anterior teeth) lining. The specialized mucosa that is found in the dorsal surface of the tongue exhibits papillae and taste buds responsible for taste sensation. In the case of the oropharynx mucosa, it is lined by non-keratinized squamous stratified epithelium [9].

2.4. The salivary glands

Three paired sets of major salivary glands (parotid, submandibular, and sublingual) and about 600 to 1,000 minor salivary glands scattered throughout the oral cavity and oropharynx make saliva that keeps the mouth moist and play a pivotal role for chewing, swallowing, and digestion. Histologically, the salivary glands exhibit a secretory unit known as an acinus that is composed by numerous secretory glandular epithelial cells (acinar cells) that surround a central space where the secretion is released. From this space, occurs the formation of the ductal structure, a closed channel lined by epithelial (ductal) cells that run through the gland and end as an opening on the oral mucosa surface. The ductal system contains specialized segments with distinct functions characterized as intercalated, striated/granular (middle portion), convoluted tubule, and excretory ducts (located next to the opening on oral mucosa). Beyond its transport role, the ductal cells alter the salivary electrolytic composition. Other important cell types found between the acinar/intercalated ductal epithelial cells and basal lamina are the myoepithelial cells. These cells show contractile capacity that helps to expel the salivary secretion from the acinus through the ductal system [19, 20]. The parotid glands are located inferior and anterior to the external acoustic meatus, lying posteriorly to the mandibular ramus and anteriorly to the mastoid process of the temporal bone. It drains its secretions (rich in proteins due to presence of acini with serous glandular epithelial cells) into the superior vestibule of oral cavity through Stensen's duct or the parotid duct. The parotid gland is responsible for providing about 25% of the total salivary volume. The submandibular glands are located superiorly to the digastric muscles and it is divided into superficial and deep lobes by the mylohyoid muscle. It drains its secretions (rich in glycoproteins due to presence of predominantly mucous acini, alonmg with some serous acini) into the submandibular duct in the sublingual caruncles, a small papilla near the midline of the floor of the mouth on each side of the lingual frenum. The submandibular gland is responsible for producing about 65% of the total salivary volume. The sublingual salivary glands are anteriorly located to the submandibular gland and inferiorly to the tongue and closest to the oral mucosa lining in the floor of the mouth. It drains its secretions (predominantly mucous, but it also contains some serous epithelial glandular cells) through 8-20 minor excretory ducts (the ducts of Rivinus) and one largest ducts, the sublingual duct or duct of Bartholin. These ductal structures join the submandibular duct to drain through the sublingual caruncle. The sublingual salivary gland is responsible for the production of 3-5% of the total salivary volumen. The minor salivary glands are mainly located in the lips, tongue, buccal mucosa, and palate, although they can also be found along the tonsils, supraglottis, and paranasal sinuses. Each minor salivary gland has a single duct which secretes serous, mucous, or mixed saliva directly into the oral cavity [21, 22] (Figure 3).

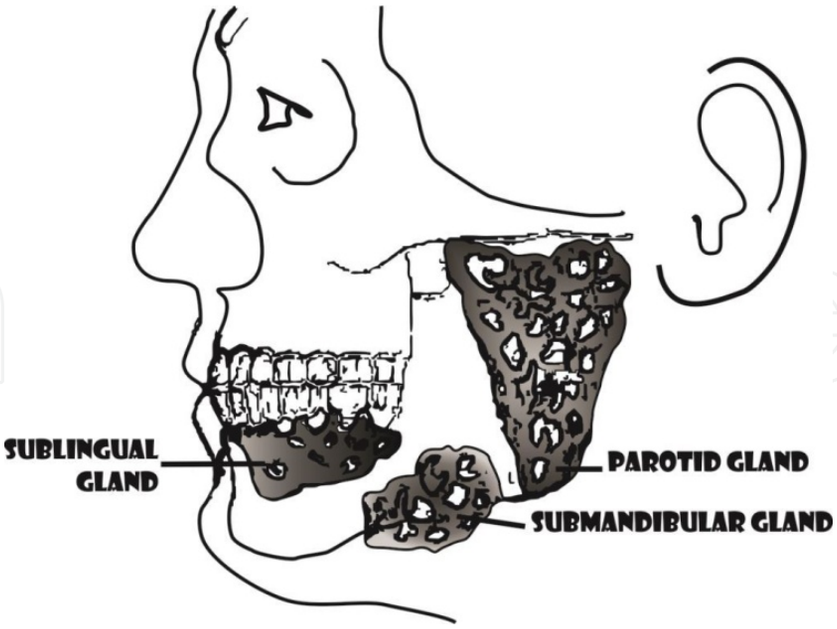

Figure 3. Anatomical localizations of major salivary glands.

2.5. The masticatory muscles

The masticatory muscles are voluntary striated muscles that are responsible for chewing actions, grinding the teeth, moving the mandible from side to side, opening the mouth, and also assisting in speech. During embryogenesis they develop from the mesoderm of the first brachial arch, also known as mandibular arch. In humans, the mandible is connected to the temporal bone of the skull via the temporomandibular joint, an extremely complex joint which permits movement in all planes. The masticatory muscles originate on the skull and insert into the mandible, thereby allowing for jaw movements during contraction. That group of muscles is represented by the masseter, temporalis, medial pterygoid, and lateral pterygoid muscles. The masseter muscle represents the most important masticatory muscle. The masseter is made up of outer and inner parts. The origin point of the outer masseter muscle is along the zygomatic arch, the bony arch of the cheek formed by the connection of the zygomatic and temporal bones. The insertion point of the outer masseter is on the surface of the ramus of the mandible. The origin point of the inner masseter muscle is from the rear of the zygomatic arch and it's insertion point is on the upper surface of the ramus of the mandible. The temporalis muscle is the muscle which assists in closing the mouth, grinding the teeth and moving the mouth from side to side when chewing. Its origin point is along the entire rim of the temporal fossa of the skull and its insertion point is the coronoid process of the mandible and temporal crest. The pterygoid muscle, made up of lateral and medial parts, is located on the inside of the ramus of the mandible. The lateral pterygoid muscle is located higher to the medial pterygoid muscle. These muscles work in tandem with the masseter muscles to assist in chewing, jaw rotation, side to side movement of the mouth, and the projection of the lower jaw. The lower head of lateral pterygoid muscle also assists in opening the mouth. Both lateral and medial parts of the pterygoid muscle have two heads and exhibit two origin points. The

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upper head of the lateral pterygoid muscle's origin point is from the lateral plate of the sphenoid bone and the origin point of the lower head is in the lateral pterygoid plate. Both heads merge to share the same insertion point which is the pterygoid fovea, but the upper head's insertion reaches the capsula and articular disc. The deep head of the medial pterygoid muscle has an origin point from the lateral pterygoid plate and the superficial head has an origin point from the palatine bone and maxillary tuberosity. Both heads merge to form a broad insertion point on the inner surface of the ramus of the mandible. Other muscles associated with the hyoid bone (such as sternohyoid, middle pharyngeal constrictor, hyoglossus, digastric, stylohyoid, geniohyoid, and mylohyoid muscles), also cooperate for opening the jaw in addition to the lateral pterygoid. The hyoid bone provides attachment for the muscles of the floor of the mouth and the tongue above, the larynx below, and the epiglottis and pharynx behind [13, 23] (Figure 4).

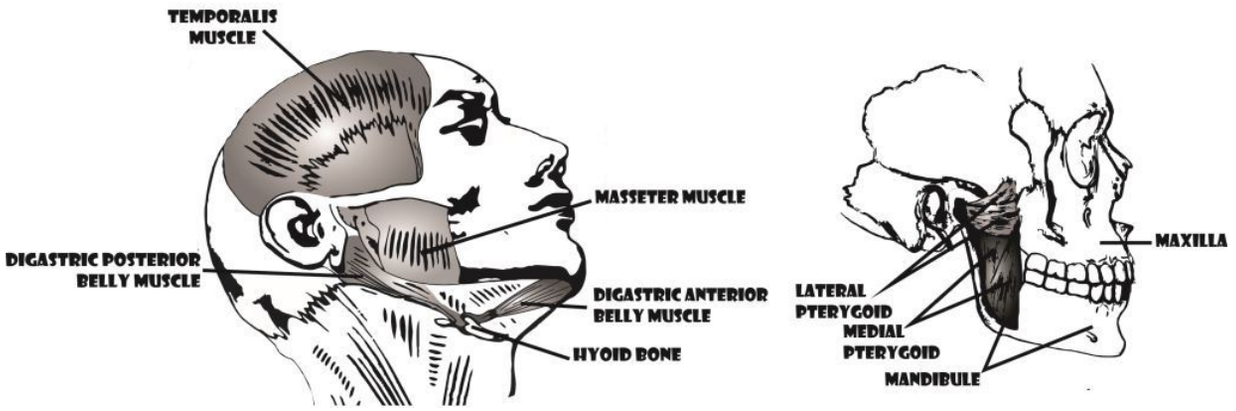

Figure 4. The muscles of mastication

2.6. The tongue

The tongue is an organ lined by an oral epithelium that contains numerous specialized structures related to taste sensation (receptor cells that can sense particular classes of tastes located in the taste buds, including filiform papillae, fungiform papillae, vallate papillae, and foliate papillae) (Figure 5A). Internally, however, the tongue exhibits its most remarkable characteristic in that it is predominantly composed of striated muscle. According to its embryological origin, the tongue might be classified by anterior and posterior regions. The anterior region, that represents about 2/3 of the length tongue, is visible, highly mobile, and directed forward against the lingual surfaces of the lower incisor teeth. The posterior region, which represents 1/3 of the length of the tongue, has its base on the floor of the mouth, connected with the hyoid bone, epiglottis, and soft palate, styloid process, and approximates the oropharynx. Both regions of the tongue exhibit a distinct nerve supply and are delimited by the terminal sulcus anteroposteriorly and by the lingual septum mediolaterally. The striated muscle tissues of the human tongue are classified as intrinsic (i.e. they originate and insert within the tongue, running along its length, and are responsible for changing the shape of the

tongue, lengthening and shortening it, curling and uncurling its apex and edges, and flattening and rounding its surface in order to execute eating, swallowing, and speech) (Figure 5B) and extrinsic (i.e. they originate from bone and extend to the tongue, and are responsible for change the tongue position, allowing for protrusion, retraction, elevation and side-to-side movement) (Figure 5C). The intrinsic muscles of the tongue are: 1) the superior longitudinal muscle, that runs along the superior surface of the tongue under the mucous membrane, and elevates, assists in retraction of, or deviates the tip of the tongue. It originates near the epiglottis, the hyoid bone, and from the median fibrous septum, 2) the inferior longitudinal muscle, that lines the sides of the tongue and is joined to the styloglossus muscle, 3) the verticalis muscle, which is located in the middle of the tongue and joins the superior and inferior longitudinal muscles, and 4) the transversus muscle, which divides the tongue at the middle and is attached to the mucous membranes that run along the sides. On the other hand, the extrinsic tongue muscles are represented by: 1) the genioglossus, which arises from the mandible and depresses and protrudes the tongue, 2) the hyoglossus, which arises from the hyoid bone and retracts and depresses the tongue, 3) the styloglossus, which arises from the styloid process and elevates and retracts the tongue, and 4) the palatoglossus, which arises from the palatine aponeurosis and depresses the soft palate, moves the palatoglossal fold towards the midline, and elevates the back of the tongue [24-26] (Figure 5).

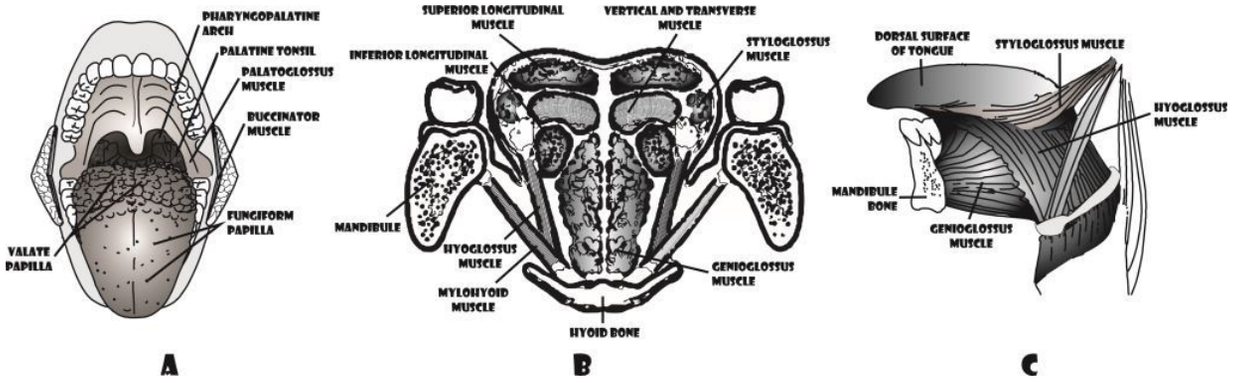

Figure 5. Anatomical aspects of dorsal (A), transversal (B), and lateral (C) views of the tongue.

2.7. The vascular and nervous network of the components of the oral cavity and oropharynx related to salivation, chewing, and swallowing

Almost all of the soft and hard components of the oral cavity and oropharynx exhibit rich vascular and motor-sensitive innervation networks [8, 27-32].

The maxillary teeth are supplied by the maxillary artery (a branch of the external carotid artery) and its branches: the middle and posterior superior alveolar arteries. The mandibular teeth are supplied by the inferior alveolar artery (a branch of the maxillary artery) and its branches: the mental artery and the incisive artery. The venous drainage of the maxillary and mandibular teeth occurs via the anterior, middle, posterior, and inferior alveolar veins. Like enamel, dentin is avascular. The odontoblasts located within the dentin receive nutrition through dentinal

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tubules from tissue fluid that originates from the blood vessels located in the dental pulp, the vital tissue of the tooth, characterized as a connective tissue with many cells (odontoblasts, fibroblasts, immune cells, and undifferentiated mesenchymal cells) and an extensive nerve and vascular supply. In the pulp chamber, there is the plexus of Raschkow that monitors painful sensations and participates of inflammatory events. In this plexus, there are two types of nerve fibers (A and C fibers) that mediate the sensation of pain. A-fibers conduct rapid and sharp pain sensations and belong to the myelinated group, whereas C-fibers are involved in dull aching pain and are thinner and unmyelinated. Within each dentinal tubule may contain an odontoblastic process and possibly an afferent axon characterized as an A-delta type sensitive fiber.

In the oral cavity, the palate is supplied by the maxillary and, sphenopalatine arteries (a branch of the maxillary artery) and its branches: the lesser and greater palatine, facial (a branch of external carotid artery) and its branches: the ascending palatine, tonsilar, submentual, upper and lower labial and angular arteries. The floor of the oral cavity is supplied by arteries: facial, ascending palatine, submental (a branch of the facial artery), and lingual (a branch of the external carotid artery). The masticatory muscles receive a blood supply from branches (the pterygoid portion) of the maxillary artery (masseteric, superficial temporal, anterior and posterior deep temporal, pterygoid branches, and buccal arteries). The tongue receives its blood supply primarily from the lingual artery but a secondary blood supply is supported by the tonsillar branch of the facial artery and the ascending pharyngeal artery. The tissues of the cheeks receive a blood supply from the facial artery. The tissues of the superior and inferior lips receive blood from the superior and inferior labial arteries, respectively, that are branches of the facial artery. The venous drainage of the palate and the floor of the mouth occur via thelesser and greater palatine veins, the sphenopalatine vein, the lingual vein, the submental vein and the pterygoid plexus. Veins of the tongue drain into the sublingual vein and the internal jugular vein. There is also a secondary blood supply to the tongue from the tonsillar branch of the facial artery and the ascending pharyngeal artery.

The salivary glands also receive a rich vascular network. When the oral mucosal surface is stimulated, afferent nerve signals travel to the salivatory nuclei in the medulla. The medullary signal may also be affected by cortical inputs resulting from stimuli such as taste and smell. Efferent nerve signals, mediated by acetylcholine, also stimulate salivary gland epithelial cells and increase salivary secretions. The parotid gland is mainly irrigated by the external carotid artery via its branches, the posterior auricular and transverse facial arteries. However it also receives a blood supply from the superficial temporal artery (a branch of the external carotid artery when it bifurcates into the superficial temporal artery and maxillary artery). The venous drainage of parotid glands is supported by the retromandibular vein (formed by the union of the superficial temporal and maxillary veins) while its lymphatic drainage is supported by the preauricular or parotid lymph nodes which ultimately drain to the deep cervical chain. The submandibular gland receives its blood supply from the facial and lingual arteries. Its venous drainage occurs through the anterior facial vein. The sublingual gland receives its blood supply from the sublingual and submental arteries.

The oropharynx receives vascular irrigation from the ascending pharyngeal artery, a branch of the external carotid. Iti s a long, slender vessel, deeply seated in the neck, beneath the other branches of the external carotid. Palatine tonsils are vascularized by the tonsillar branches of the facial, descending palatine and ascending pharyngeal arteries. The oropharynx venous drainage occurs through the parapharyngeal spaces to the region of the midportion of the peritonsillar plexus, which drain into the lingual and pharyngeal veins, which in turn drain into the internal jugular vein, particularly the jugulodigastric nodes.

Regarding innervation, the motor innervation of the oral cavity is supported by some branches of the mandibular division of the trigeminal cranial nerve (CN V) and the sensitive innervation is supported by branches of maxillary and some branches of the mandibular nerve. The maxillary teeth and their associated periodontal ligament are innervated by the branches of the maxillary division of the trigeminal nerve, the posterior, middle, and anterior superior alveolar nerves. The mandibular teeth and their associated periodontal ligament are innervated by the inferior alveolar nerve, a branch of the mandibular division. This nerve runs inside the mandible, within the inferior alveolar canal below the mandibular teeth, giving off branches to all the lower teeth.

The oral mucosa of the anterior region of maxilla (maxillary incisors, canines and premolar teeth) is innervated by the superior labial branches of the infraorbital nerve. The pterygopalatine nerve (nasopalatine nerve) is responsible for innervation of the anterior mucosa of maxilla (emerging from beneath the incisive papillae). The lingual nerve, a branch of the mandibular division of the trigeminal nerve, is responsible for innervation of the gingiva of the lingual aspect of the mandibular teeth. The mental nerve, a branch of the inferior alveolar nerve, is responsible for innervation of the facial aspect of the mandibular incisors and canines. The buccal nerve is responsible for innervation of the gingiva of the buccal region of the mandibular molars. The palate is innervated via the maxillary nerve, the nasopalatine nerve, the greater palatine nerve, the lesser palatine nerve and the glossopharyngeal nerve. The floor of the oral cavity is innervated through the lingual, mylohioid, hypoglossal, glossopharyngeal, internal laryngeal, and chorda tympani nerves. Unlike most of the other facial muscles, which are innervated by the facial cranial nerve (CN VII), the muscles of mastication are all innervated by the trigeminal nerve (CN V), more specifically they are innervated by its mandibular branch. The motor function of the trigeminal nerve activates the muscles of mastication and other accessory muscles (the tensor tympani, tensor veli palatini, mylohyoid, and anterior belly of the digastric). All intrinsic and extrinsic muscles of the tongue are supplied by the hypoglossal nerve (CN XII), with the exception of the palatoglossus muscle that is innervated by the vagus nerve (CN X). Regarding sensory nerves, the sensation of taste in the anterior region of the tongue is passed along the chorda tympani, a branch of the facial nerve. Sensation is passed along the lingual nerve, a branch of the trigeminal nerve. Posteriorly, both taste and sensation are passed along the glossopharyngeal nerve (CN IX).

Innervation of salivary glands is entirely autonomic. They are innervated by parasympathetic fibers (via cranial nerves V, VII, and IX) and sympathetic fibers (via preganglionic nerves in the thoracic segments T1-T3 and via postganglionic sympathetic fibers C2-C3) of the autonomic nervous system (Figure 6). The parotid salivary gland is innervated by the facial nerve

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and anterior branch of the great auricular nerve (composed of branches of spinal nerves C2 and C3 from the cervical plexus). Postganglionic sympathetic fibers from the superior cervical sympathetic ganglion reach the parotid gland as the periarterial nerve plexuses around the external carotid artery and their function is mainly vasoconstriction. Preganglionic parasympathetic fibers leave the brain stem from the inferior salivatory nucleus in the glossopharyngeal nerve (CN IX) and then through its tympanic and then the lesser petrosal branch pass into the otic ganglion where they synapse with postganglionic fibers that reach the parotid gland via the auriculotemporal nerve. The sympathetic nervous system also affects salivary gland secretions indirectly by innervating the blood vessels that supply the glands. Both sympathetic and parasympathetic stimuli result in an increase in salivary gland secretions. The submandibular and sublingual glands receive their parasympathetic input from the facial nerve (CN VII) via the submandibular ganglion. Their secretions are also regulated directly by the parasympathetic nervous system and indirectly by the sympathetic nervous system. The sympathetic nervous system regulates submandibular secretions through vasoconstriction of the arteries that supply it. Parasympathetic innervation of both submandibular and sublingual glands is provided by the superior salivatory nucleus via the chorda tympani nerve. Parasympathetic activity increases salivary flow, makingsaliva watery. On the other hand, increased sympathetic activity reduces glandular blood flow, making the saliva thicker, rich in glycoproteins and glycosaminoglycans. The lingual nerve is responsible by the postganglionic parasympathetic innervation of minor salivary glands. However, the minor salivary glands located on the superior jaw are innervated by fibers of the palatine nerve.

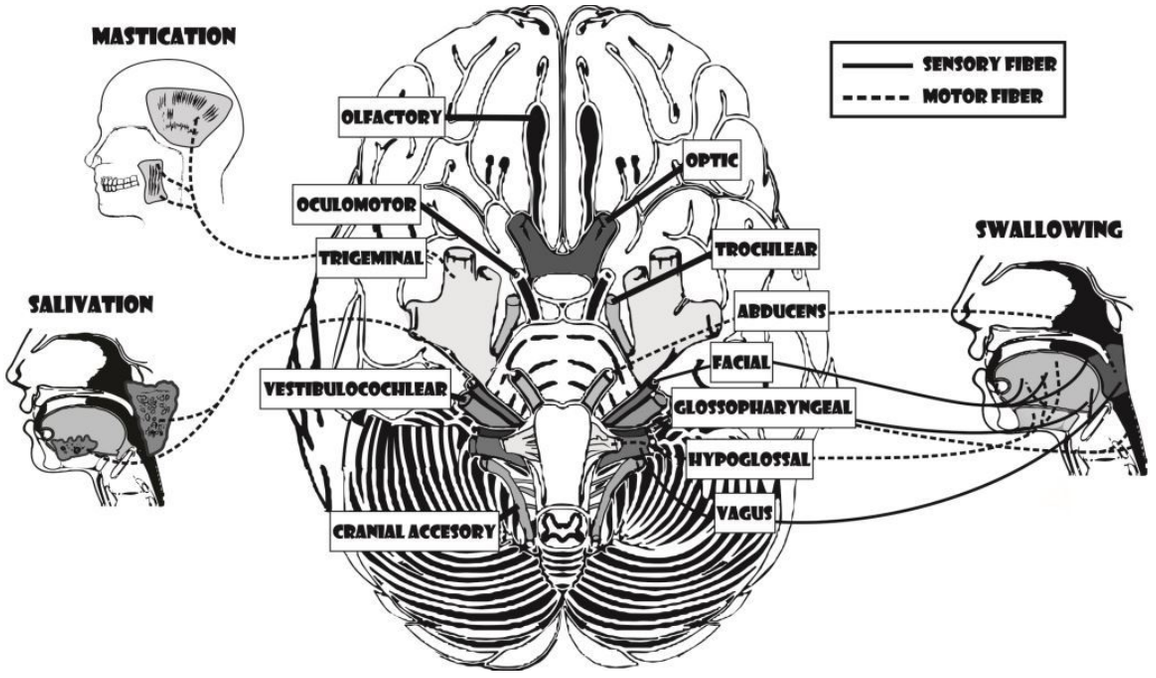

Figure 6. Cranial nerves and their relationship with salivation, chewing, and swallowing mechanisms.

The muscles of the oropharynx receive innervation from the pharyngeal plexus of the vagus nerve (CN X). This network of nerve fibers provides sensory and motor innervation from the pharyngeal branches of the glossopharyngeal nerve (CN IX), the pharyngeal branch of the vagus nerve (CN X), and superior cervical ganglion sympathetic fibers (a component of the autonomic sympathetic nervous system responsible for maintaining homeostasis of the body). However, the motor innervation of the oropharynx also receives nerve fibers from the cranial part of accessory cranial nerve (CN XI). The palatine tonsils receive afferent innervation via the tonsillar plexus, which has contributions from the general somatic afferent fibers of the maxillary division of the trigeminal nerve (CN V) via the lesser palatine nerves, and general visceral afferent fibers from the tonsillar branches of the glossopharyngeal nerve (CN IX) (Figure 6).

3. Physiological aspects of salivation, chewing, and swalloing

3.1. Salivation

Minor and major salivary glands are responsible for saliva production. These exocrine glands consist of a few fundamental cell types: i) acinar cells, which are responsible for secretion of salivary components; ii) myoepithelial cells, which usually form a thin layer above the basement membrane that support acinar and ductal cells and have contractile functions that help to expel the salivary secretions from the lumen to the duct; iii) ductal cells, which are distributed in specialized segments for transportation and modulation of salivary composition. Moreover, there are putative stem cells that reside in the ductal compartment and are related to repair and regeneration of salivary glandular tissue [19, 20].

Saliva is an exocrine secretion made by minor and major (parotid, submandibular, and sublingual) salivary glands that is expressed into the oral cavity. Saliva consists of approximately 99% water, containing a variety of electrolytes (sodium, potassium, calcium, chloride, magnesium, bicarbonate, phosphate), carbohydrates (glucose, glycosaminoglycans), proteins (represented by enzymes, immunoglobulins and other antimicrobial factors, mucosal glycoproteins, traces of albumin, and some polypeptides and oligopeptides), and traces of other biological products (urea, ammonia). Saliva exhibits pH is from 6 to 7 and varies in accordance with the salivary flow. Saliva is critical for preserving and maintaining the health of the oral tissues, allowing them to adequately perform a number of pivotal functions for taste sensation, mastication, digestion, deglutition, and speech. Also aided by the effects of saliva are tissue protection and repair, dilution of toxic substances and cleaning of microorganisms and residues. Saliva can be a useful auxiliary means of diagnosis, an indicator of risk, anda way to monitor local and systemic diseases [10, 33-37] (Table 1).

Saliva can be categorized as *whole saliva* and *specific glandular-derived saliva*. Whole saliva is representative of the typical status of the oral cavity, as it is a complex mixture of fluids from the salivary glands, the gingival crevicular fluid, mucous secretions from the oral cavity, nasal cavity, and pharynx, non-adherent oral bacterial, food remainders, desquamated epithelial

Function	Main chracteristics
Taste sensation	· The low levels of glucose, sodium, chloride, and urea hypotonicity in the saliva provides the ionic environment capable to dissolve substances and maintain taste cells; · Secretions from lingual salivary glands of von Ebner may modulate taste; · A salivary protein (gustin) seems to play a role in the growth and maturation of gustatory buds.
Hydration and lubrication of soft oral tissues	· Presence of mucin glycoprotein that forms seromucosal coverage on the soft oral tissues, hydrating and lubricating the soft oral tissues.
Chewing, swallowing, and digestion of carbohydrates and fats	· Saliva is fundamental for adequate crushing and lubrication of food particles and bolus formation ; · The presence of the digestive enzyme α-amylase (ptyalin) and lingual lipase (produced by minor salivary glands of tongue; the enzyme is only activate in the stomach) in saliva are responsible for the initial digestion of carbohydrates and triglycerides, respectively.
Protection of soft and hard oral tissues	· The coverage of mucin protects the oral tissues against the adhesion and colonization of pathogenic agents. · Prevent the colonization by potentially pathogenic microorganisms once saliva selectively modulates the adhesion of microorganisms to the oral tissue surfaces. · Saliva contains a spectrum of immunologic (immunoglobulins = IgA, more frequent, and IgM and IgG – less frequent, complement system) and non-immunologic (enzymes = lysozyme, lactoferrin, and peroxidase and non-enzymatic proteins = mucin glycoproteins, agglutinins, histatins, proline-rich proteins, statherins, and cystatins) which present antibacterial and antiviral properties. · Protection for tooth enamel through the formation of a biofilm, presence of antibodies, buffer capacity (sialin, urea, and carbonic acid-bicarbonate and phosphate buffer systems), and concentration of calcium, phosphate, and fluoride, which are decisive for the stability of teeth and enamel minerals. Moreover, its buffer capacity neutralizes gastric juices to protect the oral cavity and esophagus · Maintain the structural integrity of tooth enamel by modulating the remineralization and demineralization processes.
Tissue repair	· Saliva can stimulates blood coagulation, shortening bleeding time. · Components of saliva (such as EGF = epidermal growth factor) can increase wound contraction and stimulate rapid tissue repair.
Dilution and cleaning of toxic substances and residues	· Elimination of desquamated oral mucosa epithelial cells, non-adherent microorganisms, toxins, and food waste. · The salivary flux tends to eliminate excess nutrients (such as carbohydrates) that could favor the growth and colonization of microorganisms.
Auxiliary means of diagnosis, indicator of risk, and monitoring of diseases	· Saliva offers a highly potential non-invasive means for monitoring health status, disease onset and progression, treatment outcome, and decision making for patient care for both local and systemic diseases.

Table 1. Functions of saliva and main characteristics.

and blood cells, as well as traces of medications or chemical products. On the other hand, specific glandular-derived saliva is characterized as salivary production collected directly and exclusively from the output of the salivary ducts in the oral cavity. Another way to categorize the saliva flow rate is as *unstimulate*d (resting) or *stimulated*. Unstimulated salivary flow rate has a continuous and low salivary flow (basal unstimulated secretion). With regard to the stimulated salivary flow, it is responsible for most of the daily production of saliva and it is usually stimulated by several mechanical, pharmacological, gustatory, and olfactory factors. In healthy subjects, the mean whole saliva production ranges from 1 to 1.5L daily. The clinical evaluation regularly monitored of the stimulated and unstimulated *salivary flow index* (SFI) can be used to categorize the individuals as exhibiting normal, high, low, or very low salivary flow. Adults with normal stimulated salivation exhibit SFI variations between 1 and 3 mL/min, low SFI ranges from 0.7 to 1.0 mL/min, and very low SFI (hyposalivation) is characterized as less than 0.7 mL/min. On the other hand, adults with normal unstimulated salivation exhibit SFI variations between 0.25 to 0.35 mL/min, low SFI ranges from 0.1 to 0.25 mL/min, and very low SFI is less of 0.1 mL/min. The sensation of oral dryness in healthy individuals usually occurs when the whole (both unstimulated and stimulated) SFI is reduced by more than 50% of the daily production [34, 38-40].

Salivary flow and composition varies between individuals and also in the same individual once it is affected by several exogenous and endogenous factors such as age, gender, anatomic and functional aspects of salivary glands, sensory stimuli to food, posture, weight, circadian and circannual cycles, type of nutritional characteristics of the diet, frequency of chewing and bite force, physical exercises, tobacco and alcohol drinking, use of medication, fasting and nausea, and presence of systemic diseases. Decreased salivary flow and alterations in salivary composition cause a clinically significant oral imbalance manifested by increased susceptibility to oral infections (dental caries, periodontal disease, oral candidosis); burning mouth; sore tongue (glossodynia); difficulties with speech, mastication, and swallowing; altered taste sensation (dysgeusia); and halitosis [35, 36, 41-44] (Table 2).

Factors	Main characteristics
Age	· Although with increasing age the salivary gland tissue undergoes atrophy and gradually is replaced by a fibro-adipose tissue, increasing age *per se* seems to not interfere significantly in production and salivary composition; · Unstimulated salivary flow seems to be more affected by advanced age (it is lower in elderly) compared to stimulated salivary flow.
Gender	· Although the findings are conflicting, it appears that women exhibit low unstimulated and stimulated salivary flow compared to males as a result of a smaller size of the salivary glands and the influence of female sex hormones.
Anatomic and functional aspects of salivary glands	· Salivary flow and its composition depend on the individual contribution of each major salivary gland (parotid, submandibular and sublingual) and minor salivary glands. In unstimulated salivary flow, the parotid, submandibular, sublingual, and minor glands contribute 20%, 65%-70%, 7%-8%, and <10%, respectively. When the salivary flow is stimulated, the parotid gland contributes with over 50% of the total salivary secretion;

Factors	Main characteristics
	· Salivary secretions may be serous, mucous, or mixed. The parotid gland contributes a rich serous secretion while the submandibular, sublingual and minor salivary glands contribute a mucous or mixed secretions rich in mucin.
Sensory stimuli for food	· A series of mechanical, chemical, memory, visual, and olfative stimuli may promote alteration in salivary production; Mechanical stimulus (such as chewing) is related to increased salivary secretion; · Food containing acid substances promote intense salivary flow; Greater stimulus to saliva production can occur when humans see or imagine favorite foods.
Posture, lightning, circadian and circannual cycles	· Higher saliva production is verified in individuals kept standing up; · Lower saliva production is observed in individuals in the dark or blindfolded;
Type of nutrition, consumption of water, and characteristics of diet	· Nutritional deficiencies promote lower salivary function and composition; · Higher the body hydration results in greater salivary flow; · The consumption of food with greater consistency promotes an increased salivary flow rate.
Frequency of chewing and bite forcé	· Decreases in the frequency of chewing or a reduced bite force are related to lower salivary flow.
Physical exercises	· Physical activities can affect both production as well as composition of saliva. During exercise, there is less production of saliva and changes in the concentration of salivary proteins and electrolytes levels.
Tobacco and alcohol drinking use	· Tobacco components affect the production of saliva from the salivary glands. Smokers have increased production of saliva; · In contrast, the ingestion of alcoholic beverages promotes a decrease of salivary components (enzymes and electrolytes) as well as salivary flow.
Use of medication	· A comprehensive class of drugs may direct or indirectly influence salivary production and composition, especially those with anticholinergic effects (antidepressants, anxiolytics, antipsychotics, antihistaminics, and antihypertensives).
Presence of systemic diseases	· A series of physical and emotional systemic diseases may directly or indirectly affect the production of saliva and its composition, such as Sjögren syndrome, celiac disease, diabetes mellitus, chronic renal insufficiency, anorexia, bulimia, mood disorders (anxiety and depression), obesity, paraneoplastic syndrome, nausea, vomiting, and others

Table 2. Factors associated with production and composition of saliva.

3.2. Chewing

Chewing is a complex integrative physiological mechanism involved in the early stages of digestion and absorption of nutrients. During chewing, the food particles are reduced in size and consistency due to manipulation of food by oral soft tissues and crushing of food by the occlusal surfaces of teeth. The saliva facilitates the chewing mechanism by moistening and coalescing the food particles. The chewed food is kept trapped laterally by the tongue and buccal mucosa and becomes soft and slippery to ensure an adequate swallowing and further processing in the gastrointestinal tract. Chewing involves the participation of diverse ana-

tomical structures such as teeth, bone (mandible and maxilla), accessory and masticatory muscles, and sensory-motor activities. Biomechanically, the chewing in humans occurs in a stereotypical way: after ingestion of food it is transported from the front of the mouth to the occlusal surfaces of the post-canine teeth. Following this, the food is subjected to a series of masticatory cycles needed to process the food, making it softer and disintegrated until the food becomes more difficult to chew. At this stage, when food is ready to be swallowed, it is propelled posteriorly into the oropharynx where the food accumulates until it is finally swallowed [11, 13].

The muscles of mastication exhibit an adductor action (i.e., a motion that pulls a structure or part towards the midline of the midsaggital plane of the body) of mandible, while the lateral pterygoid muscle abducts (i.e., a motion that pulls a structure or part away from the midline of the midsaggital plane of the body). All four move the mandible laterally. Another accessory muscle, the sternohyomastoid is responsible for opening the mandible in addition to the lateral pterygoid. Anteriorly, the muscles of the lips (orbicularis oris) ensure the closing of the mouth in order to avoid escaping of the food. Posteriorly, at the moment of swallowing, the simultaneous contraction of perioral and jaw muscles, together with opening of the pharyngeal ring, causes a squeeze of the food toward the fauces. The coordinated activity of the trigeminal (CN V) and facial (CN VII) nerves during chewing may be controlled by the direct projections from the parvocellular reticular nucleus to the trigeminal and facial nuclei. Muscle activity during chewing requires complex neuromuscular control with a central pattern generator and peripheral feedback regulatory mechanisms. A distinctive population of brainstem neurons located on the corticobulbar projections play a role at the onset of rhythmicity of the chewing movements and swallowing mechanisms while peripheral afferent nerves modulate the activity of the brainstem central pattern generators. The rhythmicity of chewing movements may also be affected by basal ganglia dysfunctions. Only a small part of the muscle activity observed during chewing is needed for the basic rhythmic movements of the jaw. The peripheral feedback mechanisms that control masticatory muscle activity are supported by periodontal mechanoreceptors (rapidly conducting trigeminal sensory afferent neurons that innervate specialized receptors in the periodontal ligament of the teeth, located closest to collagen fibers) and muscle spindles (afferent sensory proprioceptors located within the belly of a muscle that primarily detect changes in the length of this muscle). Notably, muscle activity seems to start just when the intake food promotes resistance during chewing. The information provided by these mechanoreceptors is important for the specification of the forces used when food is manipulated and positioned between the teeth and prepared for chewing. Moreover, information about mechanical properties of food as well as the spatial contact patterns with the dentition is also provided by these mechanoreceptors. In this way, during cortical stimulation, the central pattern generator produces stereotyped open-close cycles that are mainly dependent on peripheral information about the position and velocity of the mandible, the forces acting on the mandible and on the teeth, and the length and contraction velocity of the muscles involved [45-47].

There are several factors that determine the performance of the masticatory system and, so, of chewing. Among them: the quantity and the total occlusal area of teeth, bite force, neuromus-

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cular control of jaw movement, performance of food manipulation by the tongue, cheeks, and teeth, breakage behavior, taste and texture of food, number of chewing cycles until swallowing of food, and salivary flow [11, 48-50] (Table 3). Experimentally, the masticatory function of individuals might be evaluated and measured by using subjective and objective strategies. The subjective masticatory function (chewing ability) might be evaluated by interviewing subjects as to their own assessment of that function. In turn, the objective masticatory function (chewing performance) has often been measured by determining an individual's capacity to fragment a test food. The chewing performance can be determined by quantifying the degree of fragmentation of an artificial test food (Optosil, a silicon rubber commonly used as a dental impression material, 8 cubes of edge size of 5-8 mm) after a fixed number of chewing cycles (range from 10 to 160 cycles). The degree of fragmentation considers the size of food fragments forced through a stack of sieves. The distribution of particle sizes of the comminuted food can be adequately described by a Rosin-Rammler distribution function which is characterized by two variables only. In each sieve, the amount of test food is weighed and the median particle size of the particles is calculated from the weight distribution of particle sizes [51, 52].

Factors	Main characteristics
Teeth	· Form the occlusal area where the food particles are fragmented; · The fragmentation of food is directly dependent on the number of teeth and thus of its total occlusal area.
Bite forcé	· Depends on jaw muscle volume, activity, and the coordination between the chewing muscles; · Important for reduction of food particle size.
Neuromuscular control	· The brain stem has been shown to be an essential part of the central nervous system that is necessary for basic rhythmic activity of the jaw-opening and jaw-closing muscles. This type of control (central pattern generator) may be switched on by activity of higher centers or by intra-oral stimuli. Moreover, peripheral feedback mechanisms provide information (position and velocity of the mandible, forces acting on the mandible and on the teeth, length and contraction velocity of the masticatory muscles involved) to the central nervous system to improve the performance of mastication; · Responsible for the neurologic control of masticatory muscles and movement of the mandible, playing an important role in the fragmentation of the food; · Act to exert muscle forces that close, and abduct-adduct the mandible allowing the teeth cut and crush food; · A small part of the muscle activity observed during chewing is needed just for the basic rhythmic movements of the jaw, and additional muscle activity is required to overcome the resistance of the food; · The texture of food might simulate different breakage behavior. Crispy food decelerates (induces a decrease of the chewing muscle electrical activity after food breaks) and accelerates (induces an increase of the chewing muscle electrical activity with intact food) the mandible as result of resistance and breakage of food particles.

Factors	Main characteristics
Performance of food manipulation and trituration	· Depends on how well the tongue and cheeks manipulate the food while the teeth crushe the food and turn it in small particles; · Individuals with a high masticatory performance will, on average, swallow finer food particles (median particle size of about 1mm) than subjects with a less high performance (median particle size of about 3mm).
Characteristics of the food	· Depends of factors such as percent water content, fat percentage, taste, texture, volume, and consistency; · Consistency of food can make the jaw decelerate and accelerate as a result of resistance and breakage of food particles; · These characteristics also affect masticatory force, jaw muscle activity, and mandibular jaw movements.
Number of chewing cycles until swallowing of food	· During chewing cycles, the food particles are reduced in size and consistency. Together, the saliva, due to the presence of water and mucin glycoprotein, facilitates chewing by moistening the food particles for swallowing; · The number of chewing cycles needed to prepare the food for swallowing is importantly influenced by food characteristics; · The number of chewing cycles needed to prepare food before swallowing is rather constant within an individual for certain foods but varies among different individuals.
Salivary flow	· Responsible for mixing of food particles into a bolus that can be swallowed; · Salivary flow rate is weakly correlated to variation in the swallowing threshold, i.e., individuals with a relatively high salivary flow rate do not necessarily swallow the food after fewer chewing cycles compared to a subject with less saliva; · Saliva might modify food properties, which may lead to changes in chewing force, mandibular jaw movements, number of chewing cycles to prepare the food for swallowing, and, perhaps, the visual and sound perception of the food.

Table 3. Factors related to chewing and mains characteristics.

3.3. Swallowing

The process of swallowing includes the voluntary effort to ingest food and an involuntary effort of bolus preparation and transport. During early stages of swallowing, the bolus, which represents food particles bound together under viscous forces determined by components of saliva, is transported from the oral cavity and pharynx to the esophagus [14, 15, 53-55].

Briefly, swallowing mechanisms might be summarized as follows: in the oral cavity, where the initial preparation of swallowing occurs, the food is chewed, moistened, and coalesced. During chewing, the food particles are continuously moved towards the occlusal surface of the teeth through the actions of the tongue and masticatory muscles. A premature spillage of the bolus into the pharynx is avoided by the approach of the soft palate to the tongue that creates a glossopalatal seal, while various movements of the mandible are important for the adequate grinding of the bolus. When the bolus is ready for swallowing, the tongue forms a

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slit containing the bolus with the lateral edges curved upwards. The soft palate closes the nasopharynx, the larynx is elevated and closed, the pharyngeal constrictor muscles (superior, middle, and inferior) contract and the cricopharynx muscles relaxes. The tongue then contracts from anterior to posterior pushing the bolus back into the pharynx, the whole process taking about one second. The true cords, the false cords, the epiglottis and the aryepiglottic folds constrict to form a barrier of several layers to protect the airway and prevent aspiration. Elevation of the larynx occurs by the contraction of the suprahyoid musculature and relaxation of the cricopharyngeus musculatures. With this relaxation, negative pressure is created in the upper oesophagus, which helps in the movement of the bolus towards to stomach. The oropharyngeal stage has a short duration (less than a second), although it may vary depending on size of the bolus. Moments before the bolus enters the oropharynx, the soft palate elevates to close the nasopharynx, avoiding the regurgitation of the bolus through the nose, while the hyoid bone elevates and draws the larynx upwards and epiglottic folds down to protect its entrance. The tongue base moves in contact with the posterior pharyngeal wall, accompanied by an anterior movement of the hyoid. The cricopharyngeus muscle begins its relaxation resulting in the opening of the upper esophageal sphincter. The rhythm and pattern of the swallowing mechanism is involuntary controlled by a central pattern generator located in the medulla. Moreover, during the oropharyngeal stage of swallowing, it the participation of the voluntary cranial nerves trigeminal (CN V), abducens (CN VI), and hypoglossal (CN XII) is fundamental for muscle voluntary control. The involuntary control (sensitive) of the oropharyngeal stage of swallowing involves the pivotal participation of the glossopharyngeal (CN IX) and vagus (CN X) cranial nerves. The esophageal phase starts after bolus passage through the upper esophageal sphincter and is characterized by a single primary peristaltic wave movement travelling at 3-4 cm/sec. After opening of the lower esophageal sphincter, the bolus is directed towards the stomach. Several secondary peristaltic waves occur spontaneously in an hour helping to clear residue and any gastric reflux. The oesophageal transit time varies with age [15, 53, 54, 56, 57].

4. Local and systemic physiopathological conditions associated to oropharyngeal dysphagia

A plethora of endogenous and exogenous etiologic factors might disrupt the physiology and anatomical integrity of oral cavity and oropharynx components and, therefore, have an negative affect on salivation, chewing, and swallowing mechanisms. The occurrence of disturbances in these mechanisms play pivotal roles in the occurrence of oropharyngeal dysphagia [1, 5] (Figure 7).

4.1. Age

The aging process is currently viewed as the result of an accumulation of insults to orofacial structures and function. Dental caries on root surfaces uncovered by gingival tissue, periodontal disease, tooth loss, oral cancer, infectious diseases (such as oral candidosis), traumatic

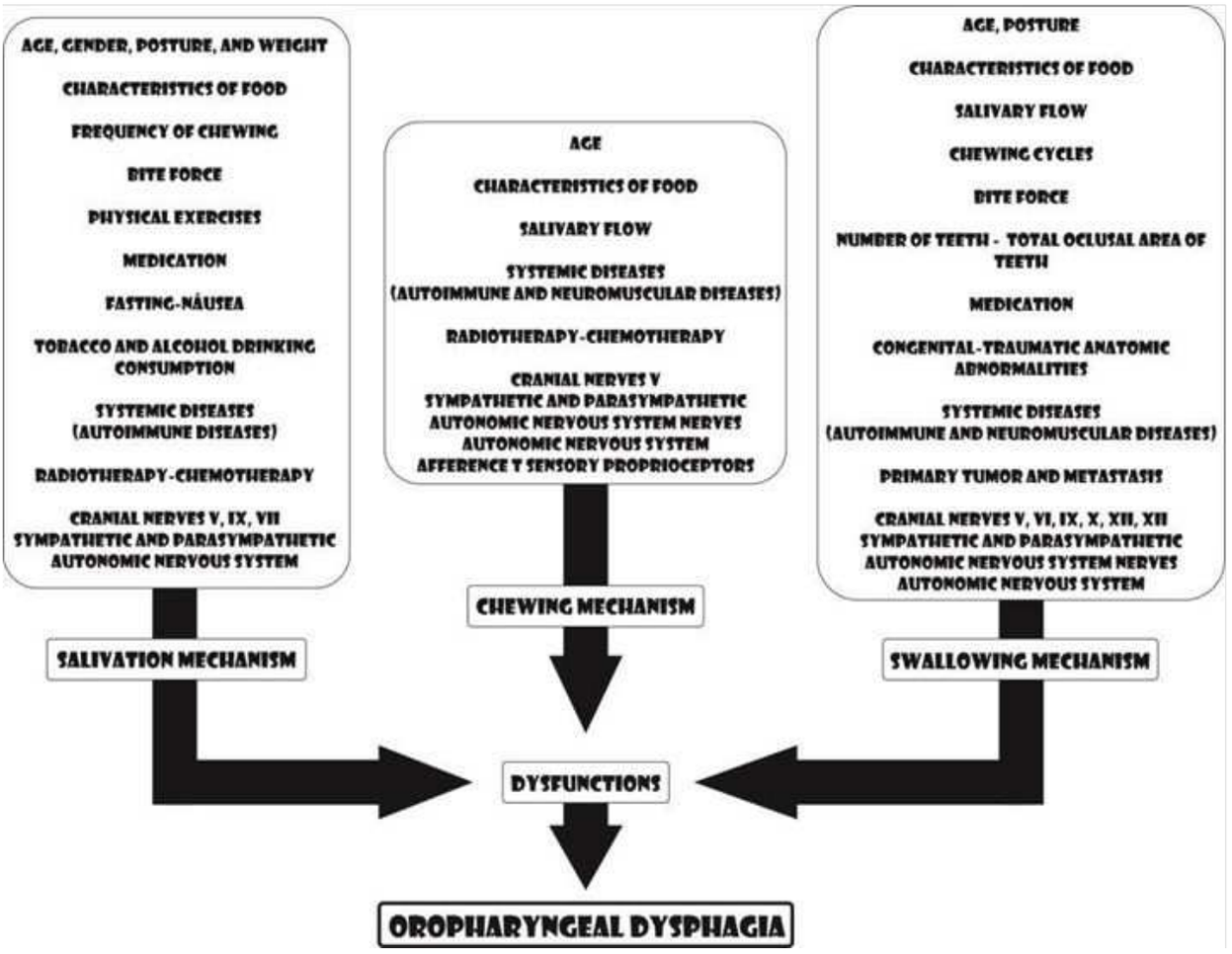

Figure 7. Schematic representation of the main etiological factors associated with dysfunction in salivation, chewing, and swallowing mechanisms, with promotion of oropharyngeal dysphagia.

lesions (hyperplastic and ulcerations caused by removable prosthodontic appliances), vesiculobullous autoimmune diseases (such as pemphigus vulgaris and cicatricial pemphigoid), and others remain as a constant threat to the maintenance of oral health in older individuals. Although many of these diseases might be promptly prevented, diagnosed and treated, a majority of older persons have encountered significant oral and pharyngeal problems that can ultimately have a profound impact on the quality of their lives. Many of these changes are directly connected to problems with genetic and epigenetic susceptibilities, socioeconomic inequalities, health behavior, access to oral health services, and systemic diseases and their treatment rather than the simple passage of time [5, 7, 58-62].

Dysfunctions related to salivary supply may negatively influence the masticatory and swallowing process by making them impossible for individuals to prepare food into a bolus before swallowing adequately. It has been frequently reported that salivation mechanisms seem to be affected with the advancing age. Indeed, mouth dryness (xerostomia) promoted by hyposalivation in older individuals is the most common complaint among the elderly. However, it has been demonstrated that in healthy older individuals there is not a significant

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alteration in volume and composition of saliva compared to a younger adult. Therefore, hyposalivation in older persons seems to be more associated with age-related diseases, masticatory disturbances, and use of certain therapeutic drugs [36, 63, 64].

Masticatory motor performance function is frequently affected in older individuals, especially those with poor subjective (self-perception of oral health) and normative (decay, periodontal disease, tooth loss) conditions of oral health. Older individuals exhibit a reduced bite which, in turn, determines a higher number of cycles needed to chew a standard piece of food, with increased particle size reduction and longer chewing sequence duration. Due to this, elderly frequently avoid highly textured foods, which are one of the important masticatory muscle activators, and might reduce the salivary flow. Other factors reported to affect masticatory performance in elderly persons include loss and restoration of posterior teeth, number of residual teeth, occlusal force, stimulated salivary flow rate, and oral motor function, which seem to accelerate masticatory dysfunction with ageing. However, it has become increasingly evident that masticatory performance need not decline with age if natural dentition is maintained. Therefore, if tooth loss and hyposalivation are not considered as characteristics of physiological ageing, ageing by itself may not be a risk factor for masticatory dysfunction. With increasing age, maintaining an adequate number of healthy natural teeth is the best guarantee to maintain adequate masticatory ability. Although the loss of teeth may be compensated for by dentures, and the dentures contribute to breaking down of food, it has been noted that prosthetic treatments seem to be unsuccessful for approaching the efficiency of a complete natural dentition, and so, of the masticatory mechanism [49, 65, 66].

Regarding swallowing, older healthy persons do not seem to experience major changes in this mechanism. However, the participation of certain risk factors, such as use of depressor central nervous system drugs or neurological diseases, might increase risk for dysphagia in aged persons. During mastication and swallowing, tongue activity creates a pressure that facilitates the manipulation of food particles and transport of the bolus. Older individuals exhibit a lower isometric swallowing (palate-tongue) pressure compared to younger persons. However, the influence of swallowing changes attributable to age appear to be important only for deglutition of certain types of food with different physical characteristics (consistency, texture). Generally, age does not seem to influence swallowing pressure. Moreover, healthy elderly individual exhibit a higher oral transit time with a prolonged oropharyngeal phase, upper esophageal sphincter relaxation, reduced pharyngolaryngeal sensory discrimination, and a higher threshold to trigger the pharyngeal phase of swallowing [55, 67].

A high occurrence of dysphagia in elderly persons has been documented although it is frequently neglected by health professionals and patients themselves. Clinical signs of dysphagia are not specific in geriatric patients and the clinical manifestation of swallowing disorder in these individuals may fluctuate over time, and therefore needs repeated clinical evaluation. In that population, the occurrence of dysphagia has been associated with malnutrition and/or dehydration while compromised safety increases the risk of aspiration pneumonia. With regards to oral health, elderly persons with oropharyngeal dysphagia frequently present with high prevalence of dental caries, periodontal diseases, and edentulism. Moreover, the occurrence of dysphagia in older persons has significant social and psychological conse-

quences. Other health problems such as modifications of respiration or coughing at mealtime, reduction or refusal of food intake, changes in types of meal texture, recurrent pulmonary infections and unexplained bouts of fever or unintentional weight loss correlate with dysphagia in older individuals [7, 61, 68].

In this way, many of the pathological conditions that affect salivation, chewing, and swallowing therefore can cause dysphagia in an older population, but are not an inevitable consequence of advancing age of individuals. Early recognition and appropriate management of dysphagia in elderly patients is pivotal.

4.2. Congenital and traumatic anatomic abnormalities

A series of primary congenital anatomic abnormalities (such as laryngeal, palate and lip clefts, tracheoesophageal fistula, and jaw micrognathia) and traumatic injuries (such as fibrosis of the upper aerodigestive tract mucosa for physical and chemical etiologic factors, and post-operatory insults after surgical procedures) can affect the oral cavity and oropharynx and have been associated with immobility of organs/structures or respiratory difficulty during feeding. These congenital disturbances manifest early in childhood, occurring independently or in combination with other anatomic abnormalities, associated or not with syndromes. Traumatic anatomic abnormalities are typically caused by direct injury. All these types of abnormalities can lead to significant oropharyngeal dysphagia [69, 70].

4.3. Medication

Drugs that directly or indirectly affect the mechanisms of protection of normal oropharyngeal mucosa (antibiotics, cytotoxics/immunossupressors), production of saliva (anxiolytics, anticholinergics/antireflux, anticonvulsants, antidepressants, antihypertensives, antipsychotics, antihistamines/decongestants, diuretics, and opiates), and chewing (botulinum toxin type A) and swallowing (antipsychotics, antihypertensives, local anesthetic agents) mechanisms may potentiate the development of dysphagia. The dimensions and impact of these side effects vary depending on the response of the individual patient and the duration of medication use [71-73].

4.4. Neurological disorders

Chewing and swallowing are complex motor tasks characterized by a coordinated and synchronized activation of an afferent system (cortical and subcortical areas and oropharyngeal afferents), the brain stem swallowing center (interneuronal network organizer) and the efferent system (motoneurons). Some cerebrovascular diseases that affect the motor control of the cranio-cervical region may interfere with the appropriate performance of chewing and swallowing mechanisms, which can explain the occurrence of dysphagia. To date, dysphagia is found in about 50% of individuals with dementia and cerebrovascular accident (stroke), 30% with deconditioning, and 90% with Parkinson disease [74].

Dementia is a condition in which there is progressive deterioration in cognition that affects day to day function of patients. All types of neurodegenerative or vascular dementia (vascular,

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multi-infarct, Lewy body dementia, Alzheimer's disease, and Parkinson's disease), may affect cortical regions involved in chewing and swallowing. Development of deglutition disorders differs with the type of dementia, but most frequently they occur during late stages. The most drastic complication in patients with dementia is the aspiration pneumonia [75]. Alzheimer's disease (AD) represents the most frequent form of dementia. In AD, brain areas underlying swallowing function show early compromise, probably before clinical dysphagia diagnosis. In these patients there occurs a delayed oral transit time due to deficits in the sensory aspects of swallowing. The common reported symptoms in these patients would be pocketing of food in the mouth, difficulties with mastication, coughing or choking with food or fluid, and the need for reminders to swallow food. In Parkinson disease (PD), drooling, persistent food residues, slow transit and repeated tongue movements can be observed during the oral phase. Delayed triggering of the pharyngeal swallow, prolonged opening of the upper esophageal sphincter and vallecular stasis are also reported. In less frequent extrapyramidal disorders (progressive supranuclear palsy, corticobasal degeneration, dementia with Lewy bodies, and multiple system atrophy), deglutition may be severely impaired even at early stages of the disease [76, 77].

Stroke is a devastating group of neurological diseases responsible for high rates of disabilities and death worldwide. Patients with stroke frequently exhibit a need for artificial feeding and higher length of hospital stay. Swallowing aspiration is reported in up to 50% of patients with stroke, with the major complication being pneumonia, a likely consequence of bacteria-infected secretions or ingested food repeatedly transgressing into the airway. Stroke patients have a worse outcome in terms of mortality and length of hospital stay when dysphagia is present [74, 78].

4.5. Neurodegenerative disorders of the motor system

Muscles need a patent motor innervation to maintain their functionality and trophism. In diseases characterized with progressive degeneration of motor neurons (both higher, cortical, and the brain stem and spinal cord), denervation atrophy occurs with consequent muscle wasting, progressive difficulty in performing movements, and loss of muscle strength. Many neurodegenerative disorders that affect the motor system exhibit a genetic etiology. However, it has been evidenced that immune system disturbances also contribute for development of that group of diseases [79]. Amyotrophic lateral sclerosis (ALS) is a neurodegenerative disorder that causes muscle spasticity and rapidly progressive muscle weakness and atrophy throughout the body due to the degeneration of the upper and lower motor neurons, which is often asymmetric at least in the early stages. Individuals affected by the disorder may ultimately lose the ability to initiate and control all voluntary movement. With respect to neuromuscular function occurring in the upper aerodigestive tract, ALS patients usually present with difficulty in speaking, swallowing, and breathing. Up to 30% of patients with ALS present with muscle bulbar (jaw, face, tongue, soft palate, pharynx, and larynx) symptoms, such as dysphagia and dysarthria (poor articulation of phonemes due to neurological injury of the motor component of the motor-speech system) at the onset of the disease, while almost all patients develop such symptoms at later stages of the disease. These patients often

lose weight in part because of dysphagia, but also because there is denervation of limb muscles and significant reduction of appendicular muscle mass. Aspiration pneumonia and respiratory difficulties are severe complications of ALS. Bulbospinal muscular atrophy or Kennedy's disease is an X-linked chronic motor neuron disease that also promotes progressive degeneration of bulbar motor neurons. Other chronic motor neuron disorders such as spinal muscular atrophy sometimes affect the muscles of swallowing [80, 81].

4.6. Other muscle disorders

Dystonia represents an involuntary sustained (tonic) or spasmodic (rapid or clonic) muscle contraction that can occur in various regions of the body producing twisting, repetitive, and patterned movements, or abnormal postures. Its etiology is complex and includes genetic predisposition, peripheral or central nervous system injuries, drug induced or metabolic disturbances, paraneoplastic syndromes, and neurodegenerative or cerebrovascular diseases. Oromandibular dystonia (OD) manifests as focal disturbances on perioral movements performed by masticatory, lower facial, and tongue muscles which may result in trismus, bruxism, involuntary jaw opening or closure, involuntary tongue movement, dysphonia, difficulty with chewing, and dysphagia. OD can manifest alone or in association with other motor control disturbances, such as Meige's syndrome and Brueghel's síndrome [80, 82].

Muscular dystrophies are a group of muscle diseases that have in common a progressive weakening in the musculoskeletal system, defects in muscle proteins, and the death of muscle cells and tissue. Although it has been suggested that this could be caused by environmental factors, the muscular dystrophies are caused by a mutation of the dystrophin gene. Among the muscular dystrophies, oculopharyngeal muscular dystrophy (OMD), myotonic dystrophy (Steinert's disease), and advanced stages of Duchenne muscular dystrophia (DMD) are most commonly associated with dysphagia. Frequently, all phases of swallowing (oral, pharyngeal, and esophageal) are impaired in these disorders. As a consequence, these patients have a delayed onset of swallowing and slowed bolus transit times. Moreover, in these dystrophic diseases, dysphagia occurs as a consequence of a progressive weakness of the tongue, palatal, and pharyngeal muscles. In the onset of OMD, dysphagia is mainly pharyngeal, but the lingual and oral phases are also affected. In DMD, the occurrence of macroglossia complicates the oral phase of swallowing [75, 83, 84].

The primary inflammatory myopathies are polymyositis (generalized and chronic inflammatory myopathy), dermatomyositis (microangiopathy that affects skin and muscle), and inclusion body myositis (progressive acquired myopathy of late stage and slow progression). They all have in common infiltration of chronic inflammatory cells within muscle tissue and tissue destruction. It has been hypothesized that the presence of specific autoantigen(s) in the muscle tissue initiates the disease, though this has not yet been identified in each disease. These diseases can promote the progressive weakening of the oropharyngeal musculature. Consequently, dysphagia occurs in about in 60% of patients with primary inflammatory myopathies. Swallowing disorders may be severe in these patients, and complicate significantly any respiratory dysfunction they may have (aspiration, interstitial lung disease, respiratory muscle

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deficiency). In inclusion body myositis the inflammatory process can promote a prominent cricopharyngeus and inferior constrictor muscles [85, 86].

Myositis ossificans (MO) is a disease that is characterized by non-neoplastic, heterotopic bone formation within a muscle. MO is divided broadly into progressive and traumatic forms. The progressive form of MO is an autosomal dominant disease in which multiple heterotopic ossifications develop in the systemic muscle, fascia, tendons, and ligaments, sometimes within families. Traumatic MO is a disease in which muscles are ossified presumably following acute trauma, burns, surgical manipulation, or repeated injury. When affecting the masticatory muscles, MO exhibits higher frequency of involvement in the masseter, followed by the medial pterygoid, lateral pterygoid, and temporal muscle. In these cases, MO might cause swelling, trismus, pain, and dysphagia [87, 88].

In certain mitochondrial myopathies (Kearns-Sayre disease, chronic progressive external ophthalmoplegia, and mitochondrial myopathy, peripheral neuropathy, gastrointestinal disease, and encephalopathy syndrome), patients present with dysphagia owing to the weakness of the pharyngeal constrictor muscles that, in turn, impairs swallowing mechanisms. Peripheral neuropathy seldom involves the pharyngeal muscles, given the short length of the pharyngeal nerve fibers. However, disorders that are independent of fiber nerve length (Guillain-Barré syndrome and chronic inflammatory demyelinating polyradiculoneuropathy) tend to affect the bulbar muscles. In these cases, dysphagia usually involves the pharyngeal phase, but oral manipulation mainly for solid food is also compromised [89, 90].

4.7. Autoimmune diseases

The autoimmune diseases represent a broad spectrum of diseases that occur when the immune system turns against components of the body itself, attacking as if it were a foreign molecule. The autoimmune human diseases comprise more than 50 distinct diseases in which oral manifestations are encountered with high frequency, sometimes as the first clinical signs or symptoms of the autoimmune disease [91].

Sjögren's syndrome (SS) is a human chronic autoimmune disorder of the exocrine glands, with a population prevalence of about 0.5% and most commonly found in postmenopausal females. SS may clinically manifest as primary (primary SS, exocrinopathy form) or in the context of underlying connective tissue disease (secondary SS). Secondary SS exhibits connective tissue disorders that can affect the skin, ears, nose and throat, joints, lungs, heart, kidneys, liver, and the neurologic (peripheral and central), haematological, and lymphoproliferative systems [92]. The etiology of SS remains unknown but probably it is multifactorial. Exogenous and endogenous factors (virus infections, stress, and hormonal factors) are thought to trigger chronic inflammation in individuals with a genetic predisposition to the disorder. The initial steps in pathogenesis of SS probably involve disturbances in endothelial, acinar, stromal, and dendritic cells, with consequent upregulation of adhesive proteins that appear to drive the migration and retention of lymphocytes into the gland. The progressive accumulation of lymphocytes is associated with subsequent activation of cytotoxic cells and release of metalloproteinases that are responsible for the tissue destruction [93]. Lesions that affect lacrimal and salivary glands characterize both primary and secondary clinical forms of SS. In the oral cavity, the hallmark

of SS is the lymphocytic infiltration of the salivary glands, particularly in the periductal areas, promoting a progressive destruction of the glandular tissues. The development of SS in the salivary gland results in dysfunction in glandular secretion with consequent hyposalivation (xerostomia). SS patients with hyposalivation exhibit loss of the lubricating, buffering, and antimicrobial capacities of saliva with an increased incidence of oral/dental infection, mucosal friability, objective and subjective findings of dryness, irritation, burning sensation, higher difficulty for dry and water bolus swallows, and significantly prolonged pharyngeal transit times as compared to controls. Another important factor that might increase the perception of dysphagia in SS patients is the occurrence of gastrotracheal reflux. Since saliva has high pH that normally neutralizes acid refluxed from the stomach, SS patients can be predisposed not only to gastro-oesophageal reflux but also to reflux into the trachea, which can mimic upper respiratory-tract infection [94, 95].

Major bullous conditions that involve the oral cavity (such as pemphigus vulgaris, cicatricial pemphigoid, bullous pemphigoid, and oral lichen planus) and idiopathic chronic inflammatory conditions (recurrent aphtous ulcers, Behçet's disease, Crohn's disease, ulcerative colitis, chronic graft-versus-host-disease) clinically manifest as painful and bleeding chronic ulcers in the oral mucosa. These ulcerative alterations may promote adverse affects on taste and smell sensation and impair mastication and swallowing by disrupting the integrity of oral mucosa without specifically affecting the anatomical structures directly related to salivary function and swallowing mechanisms [91].

Myasthenia gravis is an autoimmune disease in which self-antibodies are produced against nicotinic acetylcholine receptors on the neuromuscular junctions that connect the nervous system to the muscular system. This results in modification of the synaptic cleft and destruction of the postsynaptic neuromuscular membrane. In this way, myasthenia gravis is also considered a disorder of the neuromuscular junction [96]. Patients with this disease frequently exhibit fatigable muscle weakness that is clinically the hallmark of this disease. The clinical severity ranges from mild, purely ocular forms to severe generalized weakness and respiratory failure. Progressive destruction of neuromuscular junctions in muscles involved in masticatory swallowing mechanisms leads to disordered oral, masticatory, and pharyngeal phases, with consequent dysphagia. Clinically, due to progressive muscle weakness promoted by the disease, patients present with problems with manipulation and transport of food, whereas others have difficulties restricted to the pharyngeal phase. Some patients have greater problems with chewing food or moving it in their mouth, whereas others have difficulties restricted to the pharyngeal phase. About one third of myasthenia gravis patients with dysphagia aspirate food particles [97, 98].

4.8. Cysts and primary neoplasms in the head and neck

The initial dysphagia associated with hyperplastic and neoplastic lesions located on the upper aerodigestive tract mucosa is attributed to the combination of disrupted normal anatomy secondary to exophytic or infiltrative nature of the tumor growth, along with any muscle, vascular, and nerve involvements, soft tissue tethering, or tumor induced pain. In this way, the presence and development of hyperplastic lesions, large cysts and benign or malignant

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tumors might potentially promote disturbances in salivation (adenomas, adenocarcinomas, and carcinomas of the major salivary glands), chewing (large cysts, or benign or malignant tumors that occur in the jaws or in soft tissues of the oral cavity and oropharynx, such as dentigerous cysts, ameloblastomas, and infiltrative carcinomas), as well as swallowing mechanisms. Dysphagia also can occur in these patients as a consequence of therapeutics [70].

4.9. Treatment of the upper aerodigestive tract cancer

Upper aerodigestive tract cancer (UADTC), also known as head and neck cancer, represents a broad term, which encompasses a group of human malignancies that arise in the epithelial lining of the upper aerodigestive tract mucosa. Approximately 90% of UADC are diagnosed as squamous cell carcinoma and it represents the sixth most common type of human cancer, and is responsible for high morbidity and mortality rates worldwide every year. A plethora of socio-demographical, economic, and cultural factors associated with a background of genetic and epigenetic molecular disturbances are pivotal to progression of UADTC [99-102].

All UADTC patients will undergo surgery, chemotherapy, radiotherapy, or a combination of any of these three therapeutic modalities. The choice of modality is dependent on patient and tumor variables (presence of physical disabilities of patients, clinical stage, primary site, type, and resectability of tumor). Patients presenting with early-stage malignancy can be managed by curative surgery or radiotherapy. Frequently, patients diagnosed with a late stage tumor might be treated with complete surgical excision followed by post-operative radiotherapy or with concomitant chemoradiotherapy. The uses of organ-sparing treatments have been recommended in recent years; however, they have not necessarily translated into functional preservation of head and neck tissues. Dysphagia is recognized as a potentially devastating UADC post-treatment complication, occurring in up to 50% of UADTC survivors. The most significant consequences of dysphagia in UADTC patients are alteration of the sense of taste (dysgeusia), xerostomia, malnutrition, dehydration, weight loss, reduced functional abilities, fear of eating and drinking socially, anxiety, depression, reduced quality of life, and food aspiration [103].

During reconstruction or surgical procedures for treatment of tumors located in the head and neck (enucleation of primary tumor, tracheostomy, endoscopic laser surgery on the larynx, partial/supraglottic/supracricoid/total laryngectomy, hypopharyngeal surgery and skull base surgeries), notably for malignant tumors in late stages, the structure and function of specific cranial nerves (CN V, VII, IX, X, XII) or other specific anatomic structures related to salivation, chewing, and swallowing mechanisms are often affected and, therefore, patients might present with site-specific patterns of dysphagia. Surgical resection can have devastating effects on swallowing. When surgical resection extends beyond the tongue to laryngeal or pharyngeal structures the patients may never be functional oral eaters and always have dysphagia [104, 105].

Radiation therapy disrupts cell division in healthy tissue as well as in tumors and also affects the normal structure and function of upper aerodigestive tract tissues, including the oral and pharyngeal mucosa, salivary glands and bones. Frequent and distressing acute and chronic side-effects might occur in UADTC patients during and after radiotherapy. Acute side-effects

of radiotherapy are mucositis, xerostomia, dysphagia, hoarseness, erythema and desquamation of the skin (dermatitis). Other late complications that frequently are observed in radiotherapy post-treatment are dental decay, trismus, hypogeusia, subcutaneous fibrosis, thyroid dysfunction, esophageal stenosis, hoarseness, damage to the middle or inner ear, and osteoradionecrosis (infection in a hypovascularized tissue with consequent tissue destruction). These post-radiotherapy sequelae are dependent on radiation field, radiation dose, use of anti-xerostomic medication, and post-radiotherapy time. In xerostomic patients, irreversible damage can occur to the salivary glands, resulting in dramatic hyposalivation and increases in oral and systemic infections. Moreover, oral mucositis induced by radiation therapy frequently occurs, leading to painful oral ulcerations and local and systemic infection. In patients treated with high dose radiation, swallowing can be affected several years after treatment due to a series of complications such as fixation of the hyolaryngeal complex, reduced range of tongue motion, reduced glottic closure, and cricopharyngeal relaxation, resulting in the potential for aspiration. Irradiated patients have longer oral transit times, increased pharyngeal residue, and reduced cricopharyngeal opening times [106].

Concurrent chemoradiation was introduced to improve prognosis of UADTC patients by increase the tumor cell killing with chemotherapy, which also acts as a radiosensitizer. However, although inoperable tumors showed a better prognosis, the toxicity of the two modalities combined resulted in more significant side-effects. Various side-effects like nausea, vomiting, mucositis induced by chemotherapy, dysphagia, neutropenia, and generalized weakness might occur. The anti-metabolites such as methotrexate and 5-fluorouracil are the cytotoxic agents most commonly associated with oropharyngeal and esophageal dysphagia. Chemotherapeutic agents can impact the ability of UADTC patients to swallow. Severe dysfunction of the base of the tongue, larynx and pharyngeal muscles are observed after chemoradiation, leading to stasis of the bolus, vallecular residue, dysmotility of the epiglottis, and food aspiration [106-108].

5. The role of the dentist in an interdisciplinary effort to manage oropharyngeal dysphagia

Oropharyngeal dysphagia arises as a result of so many types of endogenous or exogenous injuries that affect, isolated or combined, the salivation, chewing, and swallowing mechanisms. Injuries in these mechanisms usually impair the physical and mental health and quality of life of individuals. Undoubtedly, successful management requires an interdisciplinary collaboration among health professionals, which need to promote an accurate diagnostic workup, promote effective therapeutic strategies, and formulate an adequate management strategy of dysphagic patients in cases where cure of that condition is not currently possible.

The dentist is a health professional that clinically evaluates the oral health of patients at frequent intervals. However, in most curriculums of dental schools, very little is presented and discussed about chewing and swallowing disturbances regarding the diagnosis, management, and treatment of these conditions. Moreover, nor is it strongly discussed about salivary gland

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dysfunction (hyposalivation) and its interrelation as an important risk factor for swallowing disorders. However, as dysphagia is affected by salivation, chewing, and swallowing disturbances, it is clear to realize that the dentist can perform proper educational and clinical activities that might provide preventive approaches, fast and accurate diagnosis, and participate in treatment decisions in a multidisciplinary health care team. Moreover, the dentist may be the health professional with whom patients feel comfortable in reporting their salivation, chewing, and swallowing disorders.

With regards to the role of the dentist, the maintenance of oral health conditions in healthy individuals or the restoration of an adequate oral health in those individuals where these conditions are unfavorable (such as presence of dental caries, periodontal disease, oral candidiasis) must be prioritized before or during the treatment of oropharyngeal dysphagia. In addition, due to the high prevalence of edentulism (especially in older individuals), the replacement of missing teeth should be provided through the use of dental implants or dentures. The control of oral infections is mandatory. The aspiration of the pathogenic bacteria populations (mostly gram-negative) by dysphagic patients is responsible for development of pneumonia, the worse consequence of dysphagia. In healthy oral conditions, oral biofilm is colonized by commensal microflora, which acts as a barrier against the colonization of respiratory pathogens. However, poor oral health conditions reduce that commensal microflora, allowing the colonization and growth of pathogenic bacteria populations. Concerning the management of dysphagia due to hyposalivation, the dentist must identify the underlying cause, minimize or eliminate the effect of the underlying cause and, therefore, its effect on dental health and quality of life. In the same way, the dentist must prevent, identify, and treat many dental occlusion, articular (temporomandibular) and neuromuscular diseases that promote parafunction of masticatory muscles. Maintenance of oral health is fundamental for hospitalized patients and its impact appears to be more significant in medically compromised or long-stay hospitalized patients. Hospital-based dentistry might play an important role in the delivery of oral health care to long-term hospital dysphagic patients with disabilities (such as traumatic and congenital anatomical abnormalities and neurological and neuromotor disorders) who are unable to receive their required dental care in their community practice settings. In patients with deglutive disorders promoted by the side-effects of the usual oncologic treatments (surgical, chemotherapy, and radiotherapy) the oral care programs aim to remove mucosal irritating factors, cleanse the oral mucosa, maintain the moisture of the lips and the oral cavity, relieve mucosal inflammation and prevent and treat the inflammation.

In this way, a policy of systematic evaluation of dysphagic patients by oral health professionals is highly recommended, with routine assessment of oral health, improvement of oral hygiene, and appropriate treatment of diseases related to salivation, chewing, and swallowing mechanisms.

6. Conclusions

The different parts of the oral cavity and oropharynx are made up of several cell types of tissues (nerves, fibrovascular tissues, cartilaginous tissues, lining and salivary gland epithelium, and

smooth and striated muscles) along withenamel and dentin tissues of the teeth and the supporting bones. The morphophysiology of the oral cavity and oropharynx components is responsible for preservation and maintenance of oral health which contributes to systemic health and a better quality of life to individuals. These components are parts of the body that are highly accessible, sensitive to the action of environmental factors and, at the same time, are able to reflect changes occurring internally in the body. Oropharyngeal dysphagia represents a neuromuscular disorder which characterizes individuals with a difficulty in swallowing. That disorder may result from an accumulation of many factors caused by both endogenous and exogenous etiologic agents which compromise, directly or indirectly, mechanisms of salivation, chewing, and swallowing. The dentist is the health professional that clinically evaluates the oral health conditions of individuals regularly. That health professional must be considered as an integral component of the multidisciplinary health team in order to perform proper educational and preventive approaches, management, and therapeutical actions that might restore oral health to their patients.

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