

Oropharyngeal Dysphagia: Understanding the Etiology

Sujatha S. Reddy, Priyadharshini R, Sujatha S. Reddy*

¹Department of Oral Medicine and Radiology, Ramaiah University of Applied Sciences, India

ARTICLE INFO

Article history:

Received: 29 September 2017

Accepted: 13 October 2017

Published: 19 October 2017

Keywords:

Oropharyngeal;
Dysphagia;
Etiology;
Causes;
Stomatognathic

Copyright: © 2017 Reddy SS et al.,
J Otolaryngol Res

This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Citation this article: Reddy SS, Priyadharshini R. Oropharyngeal Dysphagia: Understanding the Etiology. J Otolaryngol Res. 2017; 1(1):115.

ABSTRACT

The stomatognathic system performs various functions of speech, mastication, perception of taste, deglutition and digestion. Swallowing is a complex function involving cordial functioning of the orofacial structures. The dentition and salivary gland secretions are involved in mastication and formation of bolus. The muscles involved in swallowing are regulated by cranial and spinal nerves. Myriad of conditions affecting this harmony of the stomatognathic system like structural variations, neuronal abnormalities, systemic conditions, auto immune disorders, neoplastic and mucosal lesions can deter mastication and normal deglutition. A detailed and systematic case history helps in early and prompt diagnosis of cause of dysphagia. This review focuses on recognizing stomatognathic conditions affecting the oral cavity and associated structures that could contribute to oropharyngeal dysphagia. Early recognition and prompt treatment of these local or systemic conditions decreases the morbidity associated with oropharyngeal dysphagia.

Introduction

On an average an individual swallows 500 times per day. Mastication and deglutition involves coordinated action of the stomatognathic system which includes skeletal and dental components, orofacial soft tissues, the Temporomandibular Joint (TMJ), and the masticatory muscles. Muscles of face, tongue, mastication, pharynx, soft palate, larynx, supra hyoid and infra hyoid muscles as well as the muscles of oesophagus are involved in the complex process of deglutition. The "swallowing center" in medulla oblongata closely regulates the functions of these muscles through the cranial nerves (trigeminal, facial, glossopharyngeal, vagus and spinal accessory) and peripheral nerves (C1-C3) [1,2].

Deglutition occurs in four distinct yet chronological phases: the oral preparatory phase, the oral transport phase, the pharyngeal phase, and the oesophageal phase [2,3]. While the first 2 phases are under voluntary control, the latter two phases are monitored involuntary. The consistency of food further decides if this is a two or four step process [4].

Dysphagia is a symptom that refers to difficulty or discomfort during the propagation of bolus from the mouth to the stomach. The term "dysphagia" is derived from the Greek words 'dys', with difficulty, disordered or ill and 'phago', to eat or swallow, that describes difficulty in swallowing [3,5,6].

Correspondence:

Dr. Sujatha S. Reddy,
Department of Oral Medicine and
Radiology, Ramaiah University of
Applied Sciences, India,
Tel: 9448974887;
Email:
s_sujathajanardhan@yahoo.com

Anatomically dysphagia can be oropharyngeal or oesophageal, pathophysiologically dysphagia can be caused by organic factors, structural diseases or impaired physiology [3,5,6]. A lesion above or proximal to the oesophagus precipitates oropharyngeal dysphagia. A patient with oropharyngeal dysphagia reports of uneasiness in initiating a swallow and propelling the food into the pharynx and esophagus. Swallowing is not usually painful. Associated phenomena include nasal regurgitation, coughing during swallowing, choking, residual food in the oral cavity, spilling of food from the mouth, drooling of saliva, heartburn, dysarthria, or nasal speech due to weakness of muscles of soft palate. Other clinical features that may present important diagnostic clues include the presence of a speech disorder, presence of cranial nerve deficits, limb weakness, changes in sleep pattern, sleep apnea and snoring [3,6,7].

While variants to normal swallowing pattern do exist, neurological and structural abnormalities of the head and neck and loss of skeletal muscle mass and decrease in muscle strength compromises swallowing resulting in dysphagia.

Causes of Oropharyngeal Dysphagia

Various endogenous and exogenous factors disrupting the physiology and anatomical harmony of oral and pharyngeal structures have detrimental effect on salivation, chewing, and swallowing mechanisms (Table 1).

1. Neuromuscular conditions affecting the stomatognathic system

Conditions affecting the stomatognathic system undermine mastication and deglutition thereby causing dysphagia. Pathology affecting the TMJ and muscles of mastication affects the ability to masticate food and initiate swallow [4,8].

The oral phase of deglutition is markedly affected by facial nerve palsy as it innervates orbicularis oris, zygomaticus, buccinator, digastricus posterior and stylohyoid muscles as well as provides parasympathetic innervation to salivary glands and taste to the anterior two-thirds of the tongue [2,9]. Patients with Bell's palsy

often complaint of pooling of saliva and food in the vestibule of oral cavity on the affected side [10,11].

Table 1: Causes of Oropharyngeal Dysphagia.

Congenital
Elongated styloid process
Ectopic thyroid gland
Inflammatory
Retropharyngeal pharyngeal abscess
Tonsillitis
Pharyngitis
Syphilis
Neoplastic
Oral submucous fibrosis
Trauma to head and neck
Autoimmune disorders
Systemic lupus erythematosus
Myasthenia gravis
Sjogren's syndrome
Rheumatoid arthritis
Systemic conditions
Diabetes mellitus
Scleroderma
Amyloidosis
Plummer Vinson disease
GERD
Stomatognathic
Temperomandibular disorders
Salivary gland hypofunction
Neuromuscular
Cerebrovascular lesions
Peripheral facial nerve palsy
Parkinson's disease
Multiple sclerosis
Oromandibular Dystonia
Muscular dystrophy
Radiotherapy for head and neck cancer
Drug induced
Ageing

Fibromyalgia characterized by diffuse and chronic musculoskeletal pain is associated with TMJ joint noise, limited mouth opening, pain during palpation and joint movement and dysphagia [12]. Sarcopenia affecting the muscles of mastication, tongue and suprahyoid muscles can contribute to oropharyngeal dysphagia. The decrease in muscle volume and strength secondary to ageing and nutritional deficiencies compromises effective swallowing [10,13].

Impaired mastication and dysphagia has been associated with Oromandibular Dystonia(OMD). OMD is a rare focal neurological disorder affecting the facial muscles characterized by repetitive, involuntary, sustained muscle contractions with spastic movements of tongue, facial and masticatory muscles. The patients present with compromised mastication, dysphagia, altered speech, unconscious opening and closing of the mandible, pulling and twisting of the mandible forward or laterally, nasal contractions, facial grimacing, lip

pursing or sucking, bruxism, tongue dyskinesia, retraction of commissure of lip, and spasms involving platysma [14].

The oral phase of deglutition has been noted to be progressively affected in children presenting with Duchenne Muscular Dystrophy (DMD). DMD is a neuromuscular disorder resulting from mutations of the dystrophin gene coding for protein dystrophin. The progressive muscle weakness involving the tongue musculature and submental group of muscles confers feeding, chewing and swallowing difficulties [15].

The swallowing disorders most frequently observed in Parkinson's disease patients are related to the oral and pharyngeal phase, resulting in abnormal bolus formation, delayed swallowing response, and prolonged pharyngeal transit time, with repetitive swallows to clear the throat [16].

Autoimmune conditions like Myasthenia Gravis affecting orofacial muscles, weaken the muscles resulting in easy fatigability, compromising mastication [4,17]. Patients suffering from multiple sclerosis can have reduced tongue control, impaired tongue base retraction and delayed or absent swallowing reflux. Dysphagia resulting from neurological disturbances is referred to as neurogenic dysphagia [18].

Cerebrovascular diseases that affect the motor areas of the cerebral cortex regulating the initiation of swallowing interfere with mastication and initiating deglutition. The disordered pattern of oropharyngeal swallow in stroke involves delayed swallowing reflex with pooling or stasis of residue, reduced pharyngeal peristalsis and poor tongue control [4,19].

2. Anatomical Variations, Trauma and Infections

Skeletal abnormalities like elongated styloid process noticed in Eagle syndrome are associated with dysphagia. Dysphagia in this condition is attributed to muscular hyperactivity as compared to healthy individuals due to interferences of the elongated styloid process [20]. Large lingual thyroid which is ectopic thyroid tissue present at the base of the tongue can also contribute to dysphagia. The ectopic lingual thyroid contributing to dysphagia and oropharyngeal obstruction is usually diagnosed at puberty owing to the

hypertrophy of the ectopic thyroid tissue with the increase in circulating levels of Thyroid Stimulating Hormone (TSH) [21].

Trauma to mandible and stylomandibular complex can present with dysphagia, odynophagia, trismus, pain in pharyngeal, tonsillar, preauricular and retromandibular region [22-24].

Deep infections or abscesses that spread along the fascial planes like sublingual space infection, Retropharyngeal space infection, Ludwig's angina secondary to periapical or periodontal infections can also present with dysphagia [25,26].

3. Systemic conditions

Dysphagia may also be a presenting symptom of amyloidosis. The dry mouth associated with amyloid infiltration of salivary glands and associated macroglossia can hinder deglutition [27].

Dysphagia, though not commonly associated, may be one of the symptoms of Gastro Esophageal Reflux Disease (GERD). According to the International Consensus of the Montreal, GERD is defined as "the condition that develops when the retrograde passage of gastric contents causes troublesome symptoms and/or complications that result in an impairment of the quality of life of these patients". Obstructive lesion and/or neuromuscular disorder contributing to severe reflux in GERD may elicit dysphagia. Patients present with dry mouth, halitosis, pharyngeal tightness, erosions of palatal surfaces of teeth, water brash and erythema of the palatal mucosa and uvula [3,28].

Scleroderma is a systemic disease characterized by excessive deposition of collagen and other matrix elements by fibroblasts in skin and, sometimes, in multiple internal organs. Limited cutaneous Systemic Sclerosis (SSc) is characterized by skin involvement limited to the hands, face, feet, and forearms and includes the CREST variant (Calcinosis, Raynaud's phenomenon, Oesophageal dysmotility, Sclerodactyly, Telangiectasias). When Gastrointestinal (GI) involvement occurs, it tends to be late in the course of disease, predominantly affecting the oesophagus. Diffuse skin involvement and early, significant visceral involvement are seen in diffuse cutaneous SSc. Perioral skin

tightening causes significant reduction in opening the mouth and hence contributes to oropharyngeal dysphagia. Muscular fibrosis superimposed over smooth muscle atrophy and arteriolar changes leading to neural dysregulation alters GI motility in these patients [29-31]. Plummer-Vinson or Paterson-Kelly syndrome presents as a typical triad of dysphagia, iron-deficiency anemia and esophageal webs with increased risk of squamous cell carcinoma of upper aerodigestive tract. The dysphagia is usually painless and intermittent or progressive over the years, limited to solid foods and sometimes associated with weight loss. Oral mucosal atrophy, pain, glossitis, angular cheilitis and koilonychia are associated presentations. Chronic severe iron deficiency anemia results in depletion of iron dependent enzymes resulting in mucosal atrophy and web formation. This condition is seen more frequently in women [32].

4. Oral submucous fibrosis

Oral Submucous Fibrosis (OSF) is a chronic, insidious disease that affects the lamina propria of the oral mucosa characterized by fibrosis of the upper digestive tract involving the oral cavity, oropharynx and frequently the upper third of the oesophagus and, as the disease progresses; it involves tissues deeper in the submucosa of the oral cavity with consequential loss of fibroelasticity. Blanching of the oral mucosa, loss of elasticity and stiffening of the mucosa, restricted mouth opening, development of fibrous bands in lips, cheeks and soft palate, rigid lips, cheeks, pharynx and upper third of the esophagus and progressive trismus contributes to dysphagia. The activation of procollagen genes, Tissue Inhibitors of Matrix Metalloproteinases (TIMPs), increased production of growth factors like Transforming Growth Factor β (TGF β), increased activity of lysyl oxidase upregulates collagen synthesis and downregulates degradation. The over activity of the masticatory and peri oral musculature from constant chewing of arecanut results in depletion of muscular glycogen leading to muscle fatigue and degeneration contributing to muscular fibrosis and scarring [33,34].

5. Neoplasm of head and neck

60-70% of the patients treated for head and neck cancer suffer from oropharyngeal dysphagia. Altered anatomy, mass effect and the consequences of treatment instituted cause significant changes contributing to dysphagia [35]. Tumors in oral cavity interfere with formation and transport of the bolus [4]. Dysphagia seen in early radiation injury secondary to radiotherapy in head and neck cancer patients is attributed to reduced retraction of base of tongue, poor epiglottic retroflexion, decreased laryngeal elevation, delay in pharyngeal transit, and/or poor coordination of swallowing muscles. The radiation induced fibrosis results from progressive collagen accumulation, permanent fiber disorganization, altered microvasculature, production of pro-fibrotic growth factors (i.e., TGF), and/or eventual loss of elasticity. Dysphagia at 6–12 months post treatment can be correlated to early inflammatory damage to mucosa (i.e., xerostomia, mucositis) [5,36,37].

6. Salivary gland hypofunction

Xerostomia acts as a contributing factor for oropharyngeal dysphagia. The oral phase of deglutition involves the mechanical disruption of food into smaller particles by chewing and addition of saliva which aids in taste, bolus formation for swallowing and initiates digestion of starch and lipids. Additionally, saliva helps in cleansing of the oral cavity, solubilization of food substances, bolus formation, facilitation of mastication and swallowing, food and bacterial clearance, dilution of detritus and lubrication of mucosa as well as facilitates speech [38]. Xerostomia is the subjective feeling of dry mouth, a symptom that may or may not be accompanied by hyposalivation, an objective decrease in salivary flow [39]. Salivary gland hypofunction (SGH) can have a detrimental effect on oral health, generalized oral discomfort, halitosis, difficulty in chewing and swallowing, altered taste perception, altered speech and compromises retention of prosthesis. It contributes to increased risk of dental caries, gingivitis, periodontitis, candidiasis, poor oral hygiene, decreased lubrication, dehydration and atrophy of the mucosal surfaces leading to loss of integrity, injury and ulceration, infection risk, difficulty in

tolerating dentures, delayed wound healing, and pain. Other problems associated with SGH include burning mouth syndrome, taste changes, aspiration, altered sensation, and difficulty in chewing, swallowing, and speech [40-42]. Decreased salivation along with oesophageal dysmotility can lead to dysphagia in patients with Sjögren's syndrome [31].

7. Drug induced

Drugs like antidepressants, antipsychotics, anticholinergics, clonidine, diuretics, psychotropic medications, cyclophosphamide, epirubicin or methotrexate, and 5-fluorouracil contribute to dysphagia by decreasing the salivary secretion either through anticholinergic effects, activation of alpha 2-adrenoceptors in the lateral hypothalamus or by inducing dilation of the excretory duct, acinar degeneration and inflammation of glandular tissue. Decreased salivation interferes with mastication, bolus formation and lubrication, leading to laborious swallowing [4,16,40].

Medications altering cognitive alertness like antiemetics, antiepileptics, anti-anxiety and centrally acting anti-hypertensives can delay neuromuscular responses and affect sufficient oral intake [7].

8. Ageing

Dysphagia in elderly can be caused by age-related changes of the musculatures, demyelination of nerves, altered sensory functions, medications, sarcopenia and age-related diseases [7]. Presbyphagia refers to dysphagia in elderly due to naturally diminishing physiologic and neural mechanisms leading to alterations in swallowing pattern. Individuals with age-related loss in swallowing function might experience a loss or decline of functional reserves of swallowing [4,5,10,16,43]. The time required for the oral transit of bolus is also increased with ageing [44]. With ageing the decrease in tongue pressure and hence the limited tongue movement is also said to interfere with the ability to swallow. Sarcopenia involving the muscles of tongue with ageing is associated with increase in non-contractile fibers and decrease in contractile muscle fibers and hence affects the strength of tongue musculature. This

makes the tongue stiffer, restraining the tongue movements during mastication and swallowing [45].

Complications of Oropharyngeal Dysphagia

Eating and drinking are basic life functions. Difficulty in eating and swallowing can significantly affect the quality of life of an individual. The severity of oropharyngeal dysphagia can vary from moderate difficulty to complete inability to swallow. The difficulty associated with swallowing has also shown to create a sense of anxiety and panic among these individuals. Progressive dysphagia can precipitate depression and can affect the psychological wellbeing of the individual [46,47]. Strenuous swallowing interferes with regular intake of food resulting in malnutrition, dehydration, choking and tracheobronchial aspiration. Aspiration can lead to pneumonia, exacerbation of chronic lung disease, asphyxia and death. Aspiration pneumonia remains one of the serious complications of oropharyngeal dysphagia [7,46,48,49].

Conclusion

With significant morbidity and mortality associated with the condition, early and prompt diagnosis and subsequent treatment of dysphagia becomes inevitable. Clinical methods and specific complementary studies involve comprehensive personal and medical history, complete physical examination of the mouth, head, and neck structures with appropriate radiological and functional evaluation including endoscopic evaluation, specific anatomic studies, CT, barium swallow, MRI, video fluoroscopy and electromyography [50]. The elderly individuals at risk of dysphagia and aspiration have to be monitored for warning signs associated with dysphagia. This includes increase in the amount of food remaining on the plate, reluctance to eat, lack of enthusiasm, increased meal time, intermittent cessation of intake, laborious chewing, repetitive swallowing, coughing and choking upon swallowing, increased need to clear throat, peculiar head and neck movements, change in voice, regurgitation of food, unexplained weight loss, etc [7].

A multidisciplinary approach involving speech-swallow therapists, gastroenterologists, ENT specialists,

neurologists, surgeons, rehabilitation physicians, dietitians, radiologists, and geriatricians is essential in diagnosis and management of oropharyngeal dysphagia. Depending on the cause and severity of dysphagia, preventive, supportive or interventional form of therapy is instituted ranging from medical, surgical management, pharmacological management, functional swallowing therapy, safety strategies, tube feeding and tracheostomy. Swallowing sexercise interventions like the Shaker (head lift exercise), Mendelsohn manoeuvre (larynx elevation exercise) and Masako manoeuvre (tongue-holding) can improve swallowing in patients with head and neck cancer [18]. Isometric exercises for neck, tongue and oropharyngeal musculature has shown to improve swallowing by strengthening these muscles [7]. Early identification, timely diagnosis and characterization of the underlying abnormality can improve the quality of life of these patients. Dietary modifications including intake of small frequent meals, reducing quantity of each intake (using a teaspoon instead of tablespoon), adequately wetting the food to assist bolus formation, chewing from the unaffected normal side in case of unilateral weakness and using eating and drinking aids can be helpful. Posture alterations like eating in upright posture, chin down swallow for less viscous liquids, turning the head towards the affected side while swallowing in case of hemiparesis can facilitate safe swallowing and eliminate the risk of aspiration. Minor life style modifications, appropriate supportive measures, physiotherapy and psychological counseling can thus improve the quality of life of these individuals [6,31,50,51].

References

- Okeson JP. (2013). Functional anatomy and Biomechanics of the masticatory system. In: Management of Temporomandibular Disorders and Occlusion. 7th ed. Elsevier; p. 2–21.
- Shaw SM, Martino R. (2013). The Normal Swallow Muscular and Neurophysiological Control. *OtolaryngolClin N Am*. 46: 937–956.
- Castell DO, Donner MW. (1987). Evaluation of Dysphagia: A Careful History Is Crucial. *Dysphagia*. 2: 65–71.
- Sasegbon A, Hamdy S. (2017). The anatomy and physiology of normal and abnormal swallowing in oropharyngeal dysphagia. *NeurogastroenterolMotil*. 1–15.
- Clave P, Shaker R. (2015). Dysphagia: current reality and scope of the problem. *Nat Rev GastroenterolHepatol*. 12: 259–270.
- Clave P, Terre R, Kraa M De, Serra M. (2004). Approaching oropharyngeal dysphagia. *Rev EspEnferm Dig*. 96: 119–131.
- Ney DM, Weiss JM, Kind AJH, Robbins J. (2009). Senescent swallowing: impact, strategies, and interventions. *NutrClinPract*. 24: 395–413.
- Gilheaney O, Zgaga L, Harpur I, Sheaf G, Kiefer L, et al. (2017). The Prevalence of Oropharyngeal Dysphagia in Adults Presenting with Temporomandibular Disorders Associated with Rheumatoid Arthritis: A Systematic Review and Meta-analysis. *Dysphagia*. 7; 1–14.
- Moverare T, Lohmander A, Hultcrantz M, Sjogreen L. (2017). Peripheral facial palsy: Speech, communication and oral motor function. *Eur Ann Otorhinolaryngol Head Neck Dis*. 134: 27–31.
- Maeda K, Akagi J. (2016). Treatment of sarcopenic dysphagia with rehabilitation and nutritional support: a comprehensive approach. *J AcadNutr Diet*. 116: 573–577.
- Tiemstra JD, Khatkhate N. (2007). Bell's palsy: diagnosis and management. *Am Fam Physician*. 76: 997–1002.
- Pimentel MJ, Gui MS, Aquino LMM de, Rizzatti-Barbosa CM. (2013). Features of Temporomandibular Disorders in Fibromyalgia Syndrome. *J Craniomandib Sleep Pract*. 31: 40–45.
- Maeda K, Akagi J. (2016). Sarcopenia is an independent risk factor of dysphagia in hospitalized older people. *GeriatrGerontol Int*. 16: 515–521.
- Raofi S, Khorshidi H, Najafi M. (2017). Etiology, Diagnosis and Management of Oromandibular

Dystonia: an Update for Stomatologists. *J Dent Shiraz Univ Med Sci.* 18: 73–81.

15. van den Engel-Hoek L, Erasmus CE, Hendriks JC, Geurts AC, Klein WM, et al. (2013). Oral muscles are progressively affected in Duchenne muscular dystrophy: implications for dysphagia treatment. *J Neurol.* 260: 1295–1303.

16. Fusco S, Cariati D, Schepisi R, Ganzetti R, Sestili M, et al. (2016). Management of oral drug therapy in elderly patients with dysphagia. *J GerontolGeriatr.* 64: 9–20.

17. Weijnen FG et al. (2002). Masticatory performance in patients with myasthenia gravis. *Arch Oral Biol.* 47: 393–398.

18. Prosiegel M, Schelling A, Wagner-Sonntag E. (2004). Dysphagia and multiple sclerosis. *Int MS JOURNAL.* 11: 22–31.

19. Hamdy S, Rothwell JC, Aziz Q, Thompson DG. (2000). Organization and reorganization of human swallowing motor cortex: implications for recovery after stroke. *Clin Sci.* 99: 151–157.

20. Das S, Suhaimi FH, Othman F, Latiff AA. (2008). Anomalous styloid process and its clinical implications. *BratisLek List.* 109: 31–33.

21. Gallo A, Leonetti F, Torri E, Mancio V, Simonelli M, et al. (2001). Ectopic Lingual Thyroid as Unusual Cause of Severe Dysphagia. *Dysphagia.* 16: 220–223.

22. Gayathri G, Elavenil P, Sasikala B, Pathumai M, Raja VBK. (2016). “Stylo-mandibular complex” fracture from a maxillofacial surgeon’s perspective – review of the literature and proposal of a management algorithm. *Int J Oral MaxillofacSurg [Internet].* 45: 297–303.

23. Minton G, Tu HK. (1984). Pneumomediastinum, pneumothorax, and cervical emphysema following mandibular fractures. *Oral surgery, oral Med oral Pathol.* 57: 490–493.

24. Schwimmer AM, Greenberg AM. (1986). Management of mandibular trauma with rigid internal fixation. *Oral surgery, oral Med oral Pathol.* 62: 630–637.

25. Ogle OE. (2017). Odontogenic Infections. *Dent Clin NA [Internet].* 61: 235–252.

26. Datta G, Gupta M, Rao N. (2017). Clinical profile of cases of dysphagia presenting in ENT department : a study from rural tertiary care center. *Int J Otorhinolaryngol Head Neck Surg.* 3: 639–645.

27. Schwartz HC, Olson DJ. (1975). Amyloidosis: A rational approach to diagnosis by intraoral biopsy. *Oral Surg Oral Med Oral Pathol.* 39: 837–843.

28. Romano C, Cardile S. (2014). Gastroesophageal reflux disease and oral manifestations. *Ital J Pediatr [Internet].* 40: A73.

29. Sjogren RW. (1994). Gastrointestinal Motility Disorders In Scleroderma. *Arthritis Rheum.* 37: 1265–1282.

30. Kirby DF, Chatterjee S. (2014). Evaluation and management of gastrointestinal manifestations in scleroderma. *Curr Opin Rheumatol.* 26: 621–629.

31. Sheehan NJ. (2008). Dysphagia and other manifestations of oesophageal involvement in the musculoskeletal diseases. *Rheumatology.* 47: 746–752.

32. Novacek G. (2006). Plummer-Vinson syndrome. *Orphanet J Rare Dis.* 36: 1–4.

33. Warnakulasuriya S, Johnson NW, Waal I van der. (2007). Nomenclature and classification of potentially malignant disorders of the oral mucosa. *J oral pathol med.* 36: 575–580.

34. Tilakaratne WM, Klinikowski MF, Saku T, Peters TJ, Warnakulasuriya S. (2006). Oral submucous fibrosis: review on aetiology and pathogenesis. *Oral Oncol.* 42: 561–568.

35. Govender R, Smith CH, Taylor SA, Barratt H, Gardner B. (2017). Swallowing interventions for the treatment of dysphagia after head and neck cancer: a systematic review of behavioural strategies used to promote patient adherence to swallowing exercises. *BMC Cancer.* 17: 43.

36. King SN, Dunlap NE, Tennant PA, Pitts T. (2016). Pathophysiology of Radiation-Induced Dysphagia in Head and Neck Cancer. *Dysphagia.* 31: 339–351.

37. Rogus-Pulia NM, Larson C, Mittal BB, Pierce M, Zecker S, et al. (2016). Effects of Change in Tongue Pressure and Salivary Flow Rate on Swallow Efficiency Following Chemoradiation Treatment for Head and Neck Cancer. *Dysphagia*. 31: 687–696.
38. Pedersen AM, Bardow A, Jensen SB, Nauntofte B. (2002). Saliva and gastrointestinal functions of taste, mastication, swallowing and digestion. *Oral Dis*. 8: 117–129.
39. Napenas JJ, Brennan MT, Fox PC. (2009). Diagnosis and treatment of xerostomia (dry mouth). *Odontology*. 97: 76–83.
40. C Scully. (2003). Drug effects on salivary glands: dry mouth. 44: 165–176.
41. Liu B, Dion MR, Jurasic MM, Gibson G, Jones JA. (2012). Xerostomia and salivary hypofunction in vulnerable elders: prevalence and etiology. *Oral Surg Oral Med Oral Pathol Oral Radiol*. 114: 52–60.
42. Niklander S, Veas L, Barrera C, Fuentes F, Chiappini G, et al. (2017) Risk factors, hyposalivation and impact of xerostomia on oral health-related quality of life. *Braz Oral Res*. 31:e14.
43. Maeda K, Akagi J. (2016). Sarcopenia is an independent risk factor of dysphagia in hospitalized older people. *GeriatrGerontol Int*. 16: 515–521.
44. Robbins J, Hamilton JW, Lof GL, Kempster GB. (1992). Oropharyngeal swallowing in normal adults of different ages. *Gastroenterology*. 103: 823–829.
45. Yoshida M, Kikutani T, Tsuga K, Utanohara Y, Hayashi R, et al. (2006). Decreased tongue pressure reflects symptom of dysphagia. *Dysphagia*. 21: 61–65.
46. Eslick GD, Talley NJ. (2008). Dysphagia: epidemiology, risk factors and impact on quality of life—a population based study. *Aliment Pharmacol Ther*. 27: 971–979.
47. Verdonschot RJCG, Baijens LWJ, Serroyen JL, Leue C, Kremer B. (2013). Symptoms of anxiety and depression assessed with the Hospital Anxiety and Depression Scale in patients with oropharyngeal dysphagia. *J Psychosom Res*. 75: 451–455.
48. Rofes L, ArreolaViridiana, AlmirallJordi, Cabre M, Campins L, et al. (2011). Diagnosis and Management of Oropharyngeal Dysphagia and Its Nutritional and Respiratory Complications in the Elderly. *Gastroenterol Res Pract*.
49. Scannapieco F, Shay K. (2014). Oral Health Disparities in Older Adults : Oral Bacteria, Inflammation, and Aspiration Pneumonia. *Dent Clin NA*. 58: 771–782.
50. Etges CL, Scheeren B, Gomes E, Barbosa LDR. (2014). Screening tools for dysphagia: a systematic review. In: CoDAS. *SciELOBrasil*. 26: 343–349.
51. Force NDDT, Association AD. National dysphagia diet: standardization for optimal care. American Dietetic Association. 2002.