Review Article

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DRY MOUTH: AN OVERVIEW

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ABSTRACT:

Salivary secretory dysfunction and/or salivary gland pathology are conditions that can be found in all segments of the population. The aged are uniquely susceptible to some of these entities which may reflect manifestations of local, systemic or even imagined disorders. xerostomia will be defined as a subjectively perceived complaint of dryness by a patient whose salivary volume, when measured, is within normal limits. Salivary hypofunction is the term that represents a decreased production of saliva substantiated by an objective measurement. in this overview the dry mouth will be defined and the causes and most important causes, that can lead to occurrence of dry mouth, will be explained. Lastly, there will be a paper description of the diagnosis and management of dry mouth.

Keywords: Saliva, mouth dryness, xerostomia, salivary gland.

INTRODUCTION:

Xerostomia is the subjective complaint of oral dryness. It is a frequent annoying condition. It is estimated that 12-47% of the elderly and 10-19.3% of people in their early 30's have been suffering from dry mouth.[1,2,3] The symptoms of xerostomia are as follows: Cracked peeled atrophic lips, glossitis, progressive cervical, or cusp tip caries even with optimum oral hygiene, candidiasis, and pale corrugated dry buccal mucosa. The size, texture and tenderness of salivary glands should be assessed. Xerostomia can lead to dysphagia, dysgeusia, oral pain, dental caries, oral infection, periodontal disease, and finally can affect the health-related quality-of-life.[4,5,6,7] Malnutrition and psychosocial problems could be associated with dry mouth as well.[7] The basis of xerostomia is the alteration in both quantitative and qualitative function of salivary glands.[6] There are multiple causes with various mechanisms of xerostomia such as systemic diseases, anticholinergic effects of many drugs, psychological conditions, alcohol, head and neck radiation therapy, and physiological changes, but xerostomia-related systemic diseases have not been addressed as much as they worth it.[8] Multiple methods have been described to manage xerostomia. Saliva substitutes, topical stimulants, and parasympathetic agonists such as pilocarpine and cevimeline are approved medications to treat xerostomia.[9] Early detection of these diseases may aid to timely treatment of xerostomia.

Saliva and the Salivary Glands in the Elderly

It is generally accepted that approximately 1500 ml of saliva are produced in a 24 hour period by all of the salivary glands.[10] When nothing is in the mouth and the glands are at rest, normal levels of whole saliva are calculated as 0.4 ml/min,[11] mostly

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originating from the submandibular/sublingual gland complex. However, with stimulation, salivary volumes from the parotid glands equal that produced by the submandibular/sublingual glands. The normal level of stimulated whole saliva has been reported as 2.0 ml/min.[12] Measured values that are above these normal resting and stimulated volumes represent salivary hyperfunction, while values belowreflect salivary hypofunction. However, volumes can vary from individual to individual, are lower in women and dependent upon collection technique and time of day of the collection. xerostomia will be defined as a subjectively perceived complaint of dryness by a patient whose salivary volume, when measured, is within normal limits. Salivary hypofunction is the term that represents a decreased production of saliva substantiated by an objective measurement. The patient's awareness of a dry mouth develops when resting whole saliva falls below the range of 0.1–0.2 ml/min. [11,13] Sialorrhea will be used to describe a patient's notion of the presence of excessive saliva when quantitative evaluation does not indicate salivary hyperfunction. If objective measurements testify to increased salivary volume, salivary hyperfunction is the appropriate description. Drooling represents the leakage of saliva on to the chin which may originate from hyperfunction.Where hyperfunction is not a factor, drooling may have its roots in disturbed neuromuscular swallowing mechanisms, mental retardation, habit or loss of oro-facial muscle tone. With aging, a significant change occurs in the histologic features of the salivary glands. Parenchymal depletion develops with a corresponding increase in fibroadipose tissue and the number of dilated ducts.[14] A 30%–40% decrease in parenchymal volume of individual salivary glands has been reported.[15] Besides the microscopic evidence, acinar loss has also been observed in CT scans of the parotid gland. A significant decrease in parotid gland density, reflecting loss of parenchyma and increased presence of fibro-adipose tissue, is seen.[16]

Functions of saliva

Saliva possesses the following functions in the edentulous patient:

- It is responsible for the physical retention of complete dentures

- It aids in mastication by softening foods for swallowing.

- It facilitates the sense of taste, Taste is expedited because saliva dissolves the chemical tastants within food and delivers these to taste bud receptors.

- It lubricates and protects the oral mucosa: Saliva's lubricating presence also acts to prevent glossodynia. Futhermore, mucosal lubrication by saliva is important for a patient using a removable dental prosthesis.

• It helps in food digestion, which begins in the oral cavity with the ability of salivary amylase to metabolize carbohydrates.

• It promotes clear speech, Speech impairment will become apparent without saliva moistening the oral mucosa.

• It buffers endogenous and exogenous acids and thus inhibit dental demineralization. A significant complementary function, remineralization, is facilitated by the availability of salivary calcium and phosphate ions. Antibacterial, antifungal and antiviral activities of saliva are mediated through salivary IgA, lactoferrin, lysozyme, peroxidase systems and histatins. [17,18]

Problems of reduced salivary flow

A reduction, or absence of saliva (xerostomia) is a particular problem for edentulous patients, because it causes a reduced retention of denture. There may also be an increased susceptibility to denture trauma resulting in complaints of pain and in some case the burning mouth syndrome. A complaint of dry mouth can occur in the absence of the clinical signs of dryness (symptomatic xerostomia). Under such circumstances the physical retention of the dentures would not be expected to be diminished. In clinical xerostomia there are intra-oral signs of dryness such as a dry, atrophic mucosa and lack of saliva pooling in the floor of the mouth. The dentist can check the dryness of the buccal mucosa simply and quickly during the examination of the patient by carrying out the 'mirror test'. For this the dentist lightly presses the face of the mirror against the buccal mucosa and then tries to remove it. If the mirror comes away easily the mucosa is still covered by a substantial film of saliva; if the mucosa adheres to the mirror then it is dry.

Aetiology of reduced salivary flow

Old age per se does not result in reduced salivary flow rates.[19] However, the condition is relatively common in middle-aged and older people, the main candidates for complete dentures, with between 12% and 16% complaining of a dry mouth. [20,21] The commonest causes of dry mouth.[21,22] are: Medications, Dehydration and Acute Parotitis, Mouth Breathing, Diabetes mellitus, Radiation, Gastroesophageal Reflux Disease (GERD), Sjogren's Syndrome, Human immunodeficiency virus, Sarcoidosis, Hepatitis C virus, Alzheimer's Disease and Parkinson's disease (PD).

Medications

With aging, the elderly inevitably require an increasing number of medications as a means of controlling illness. Among those that are 65 years of age or older, 75% are reported to be taking at least one medication. Many of these drugs, over 400 of them, decrease salivation usually through an anticholinergic activity.[23] The psychotherapeutic agents, sedatives, many cardiovascular drugs, the antihistamines, diuretics and belladonnas are some of the medicinals

that cause glandular hypofunction.[24]These drugs make their presence known by decreasing the volume, of resting saliva.[17, 25] Saliva is a very significant factor in maintaining dental integrity and avoiding root caries to which the elderly are prone. The aged take many anticholinergic medications which cause a hypofunction of resting saliva. Kitamura, Kiyak and Mulligan have reported that these medications are the cause of increased coronal and root caries in the elderly.[26] Conversely, most reports indicate that caries incidence in the aged is in direct proportion to oral hygiene, sugar intake and periodontal breakdown, while the use of medications are poor predictors of increases in caries.[27,28,29]. A study by Saunders and Handelsman (1992) demonstrated no statistical increase in either coronal or root caries in patients 65 years or older, who are in a long-term care facility and are taking medications that induce hyposalivation when compared to a non-medicated control group. [30]

Dehydration and Acute Parotitis

Dehydration is a common and underdiagnosed condition in the elderly. The hospital rate of dehydration per 10,000 discharges has been reported as 12.1 for the 55 to **-64**year age category, but this rate increases to 287 per 10,000 for those in the 65 to 75-year age range[23,31] In the ambulatory patient, dehydration is usually caused by a decreased fluid intake or excessive sweating**.** Hospitalized patients have an increased incidence for several reasons. Sweating from a febrile condition, loss of blood from surgery, vomiting, diarrhea, prescribed medications and inadequate intravenous fluid replacement are all contributing factors**.**

Frequently the problem arises when the patient is forbidden oral nourishment following a gastrointestinal surgical procedure**.** Salivary hypofunction becomes an inevitable consequence of dehydration**.** Decreased unstimulated and stimulated parotid salivary flow rates have been reported and cause lip dryness and complaints of thirst.[31] Additionally, the hypofunction leads to a loss of the protective aspects of saliva and encourages oral infections. Furthermore oral pain and problems with mastication**,** swallowing and digestion are encountered as saliva's abilities to lubricate, soften and digest food are compromised**.** Acute parotitis (AP) represents a more severe outcome of dehydration. The associated decreased salivary productionwith its failure to flush the duct system can lead to an ascending duct infection and an acute parotid abscess. Many factors play a role in the development of the AP. Elderly patients use a variety of medications, many of which decrease salivary production. The diagnosis of a true AP is based largely upon the clinical recognition of the pre-existing conditions for its development. The AP is identified by the sudden onset of a painful parotid swelling. The overlying skin is erythematous and a fever is usually present. An intraoral examination will reveal pus exiting from the parotid duct

orifice. Eating greatly accentuates pain as retained stimulated saliva adds to the already existing inflammatory pressure within the gland. Parotid swelling associated with the abscess is inhibited to some degree by the presence of the gland's dense fibrous capsule. The increased intracapsular pressure, exerting its force upon the sensory nerves within the gland, is the source of severe patient discomfort**.** Therapeutic surgical intervention is usually not necessary because the duct system acts as a natural drainage mechanism. The key to successful therapy is rehydration and supportive therapy. Increased fluid intake will bring about increased salivary production and duct lavage. Supportive therapy includes oral nourishment, oral hygiene, bed rest and antibiotic therapy as indicated.

Mouth Breathing

Laxity of the peri-oral musculature not only causes angular drooling, but also plays a significant role in the patient's complaint of xerostomia. The loss of muscle tone that occurs with aging often leads to increased mouth breathing. The flow of air across the oral mucosa, as the patient inspires and expires, causes rapid evaporation of the aqueous serous element of saliva. Subjective dryness results from the loss of saliva's moisturizing effect. The problem is further emphasized during sleep when gravity encourages an open mouth and snoring occurs. Snoring is commonly seen in the aged. These patients will state that the dryness is most severe upon awakening in the morning or if they awaken in the middle of the night. The dryness that develops during sleep is accentuated by the fact that only minimal amounts of saliva are produced during sleep because no oral stimuli are present. The sparse nocturnal secretions rapidly evaporate when mouth breathing is present.

Diabetes mellitus

Diabetes mellitus is an endocrine disease characterized by the deficit in production of insulin with consequent alteration of metabolism and balance of glucose concentration. According to its etiology, it is classified as Type 1 and 2. [32] Type 1 diabetes mellitus is a metabolic dysfunction characterized by hyperglycemia resulting from definite shortage of insulin secretion caused by autoimmune illness and genetic factors.[33] Type 2 diabetes mellitus (formerly known as non-insulin-dependent diabetes mellitus) is the most common form of disease featured by hyperglycemia, insulin resistance, and relative insulin deficiency. Type 2 diabetes results from interaction between genetic, environmental and behavioral risk factors.[34] It is estimated that there will be 380 million persons with diabetes mellitus in 2025**.** Patients with uncontrolled diabetes often report dry mouth, which is believed to be due to polyuria, dehydration, and autonomic dehydration.[32] The prevalence of xerostomia was reported in 14-62% of diabetes mellitus 2 cases,[35,36,37] and it was found in 38.5% and 53% of children

and adolescents subjects with diabetes mellitus 1, respectively.[37,38]

Radiation

Surgery with or without radiation and/or chemotherapy is the treatment of choice of cancers. Cancericidal radiation doses involve treatments with 60 Gy or more. Such intensive dosages lead to a radiation sialadenitis and permanent salivary hypofunction if the beam inadvertently targets the salivary glands. Collateral salivary gland damage from the therapeutic use of dosages even as low as 25Gy will cause an almost immediate decrease of 60%–90% in salivary volume. Some recovery can be expected with time if less than 25Gy has been used.[23] The serous cells of the parotid gland are very susceptible to radiotherapy (RT) while the mucus cells of the submandibular salivary gland are less affected by the destructive effects of the RT. Clinical evidence for the relative resistance of the mucous cell to RT can be derived from the fact that the diminished saliva in patients who have received RT is very viscous. This reflects the relative proportional increase in viscous mucus over the decreased aqueous serous production of a RT-damaged parotid gland. However, with the higher RT cancericidal doses, total loss of all salivary gland secretions can be expected**.** It is believed that even low doses of radiation have an effect on the secretory granules present in the serous cells.[39] The RT interacts with the metallic ions present in the granules. Free oxygen radicals are produced and are thought to be the cause of the cell's destruction and the resultant decreased salivation.[40]

Gastroesophageal Reflux Disease (GERD)

Gastroesophageal reflux disease (GERD) is a common condition affecting at least 30% of the elderly with an increased prevalence occurring with age. [41] Reflux of gastric contents into the esophagus, because of an incompetent lower esophageal sphincter, causes heartburn. The pyrosis is most pronounced after meals, particular after ingesting spicy foods. Gravity favors its occurrence with the problem becoming accentuated when the patient is prone while sleeping. Classically heartburn is recognized by a midline retrosternal burning sensation that may extend from the xiphoid process to the throat. Repeated incursions of gastric acids into the esophagus over prolonged periods inevitably lead to esophageal erosions and ulcerations and dysphagia. Clearance of most of the gastric acid from the esophagus is accomplished by physiologic peristalsis initiated by swallowing and/or local esophageal stimulation.

However, peristalsis alone is not sufficient to clear the residual gastric acid affecting the lower esophageal wall, and further acid clearance is necessary.[42] Final and effective clearance of the acid results from an esophagosalivary reflex (ESR) mechanism mediated through a vagal afferent

innervation. Stimulation of the ESR causes a salivary hypersecretion known as “water brash”. The ESR is intimately involved in the inhibition of the development of GERD symptomatology. With increased salivary production and the inevitable swallow, saliva with its buffering capacity neutralizes the residual gastric acids and simultaneously lavages and protects the esophageal wall from the irritative effect of the gastric acids.[43,44]. Elderly patients frequently are seen in the SGC with a history of GERD and complaints of excessive salivation. When questioned, they characteristically relate the hypersecretion to episodes of heartburn. Often they state that their pillow is wet upon awakening in the morning. Their horizontal position during sleep favors regurgitation and a resulting salivary hypersecretion. Inevitably, the increased saliva tends to drool out of the corner of the mouth on the side that the head is turned during sleep. The chronic escape of saliva mixed with the regurgitated gastric acids displaced into the mouth results in excoriated and macerated soft tissue at the angle area of the mouth and serves as a telltale marker of uncontrolled GERD.[45] There are many treatment options for the management of GERD. A variety of medications are available to inhibit gastric secretions. In addition, preparations are prescribed to neutralize the gastric acids or to form a protective coating on the esophageal wall. Elevating the head during sleep, avoiding large meals and not consuming substances such as alcohol, caffeine and spices will lessen GERD symptomatology. Clinical control of GERD will eliminate the drool that represents salivary hypersecretion and regurgitated contents.

Sjogren’s Syndrome

Sjogren’s syndrome (SS) is a debilitating systemic autoimmune exocrinopathy, with the elderly representing 20% of the patient cohort.[46] It is characterized by the presence of decreased salivation and lacrimation, and in this form it is referred to as the sicca complex, or primary SS. Primary SS predominates in the older age groups.[47] Systemic autoimmune diseases, usually rheumatoid arthritis, lupus erythematosus or scleroderma, are associated with the salivary and lacrimal hypofunction. This triad of symptoms represents secondary SS and its occurrence in the elderly is in direct relation to the age range of the accompanying systemic autoimmune disease. After rheumatoid arthritis, SS represents the second most common autoimmune disease, affecting approximately 1%–4% of the adult population.[23] with more than 90% of the patients being post-menopausal women.[15,23,47] Because there is no cure, SS can only be treated symptomatically. Hydroxychloroquine is prescribed because of its proven efficacious effect in other autoimmune diseases. Corticosteroids or other immunosuppressive agents are indicated in the presence of serious extraglandular manifestations. The cholinergic agonists pilocarpine and cevimeline have been of

some value in increasing salivation. The use of sugarless chewing gum or sour candy is also helpful as a sialogogue. Artificial salivas and oral lubricants are available. Aggressive fluoride therapy, to abort the increased incidence of dental caries, should be instituted.

Human immunodeficiency virus

Human immunodeficiency virus (HIV) infection is one of the most devastating infections in modern times. Oral manifestations of HIV infection occur in approximately 30-80% of patients.[48] Oral lesions are among the early signs of HIV infection and can predict progression to acute immunodeficiency syndrome. The more common HIV-related lesions include oral candidiasis, herpes simplex infection, oral Kaposi's sarcoma, oral hairy leukoplakia, parotid gland enlargement, periodontal diseases (linear gingival erythema and necrotizing ulcerative periodontitis), human papilloma virus-associated warts, and ulcerative conditions including herpes simplex virus lesions, recurrent aphthous ulcers, neutropenic ulcers, and xerostomia.[49,50] Xerostomia is due to the side effects of HIV medications (e.g., didanosine) or the proliferation of CD8 + cells in the major salivary glands (50). Parotid hypertrophy as a compensative reaction has been also found more commonly in HIV-positive children.[51,52]. Xerostomia has been estimated as 1.2-40% in HIV-positive patients.[50,53,54,55,56,57] There is conflict about xerostomia-related HIV medications in the literature, for example Lin claimed that xerostomia was not compounded by medications.[58,59] In contrast, some researchers believe that taking anti-HIV drugs is effective on the prevalence of xerostomia.[56]

Granulomatous diseases (Sarcoidosis)

Sarcoidosis is a multisystem non-caseating granulomatous disorder of unknown etiology**.** Typically bilateral hilar lymphadenopathy, pulmonary infiltrations and skin or eye lesions are present. Respiratory symptoms such as coughing or dyspnea often develop, and a linkage between sarcoidosis and lymphoma has been reported.[60,61,62] Sarcoidosis usually affects young adults in the 30 to 40-year-old age range. the aged population is also affected. [63,64]

Salivary glands are prone to the disease process but the pattern of glandular involvement varies. The salivary glands could be affected by sarcoidosis as well, which was reported in 6% of the cases. [65] The submandibular salivary glands are infrequently involved, but minor salivary glands may have granulomatous infiltrates. Because of the granulomatous infiltration into major and minor salivary glands, some decrease in salivary volume can be expected**.** Diagnosis is mostly dependent on the radiologic identification of distinctive lung infiltrates and a hilar lymphadenopathy. Blood studies may show elevated calcium and alkaline phosphatase levels along with increases in angiotensin converting enzyme. Confirmation of a clinical diagnosis of sarcoidosis is based

on the accumulated pathologic signs and symptoms and often substantiated by a labial gland biopsy. Because 58% of the labial gland biopsies have proven to be positive.[66] such an intervention is a reasonable part of the diagnostic approach**.** Treatment of the disease is generally symptomatic because spontaneous resolution does occur. Corticosteroids and other immunosuppressives are prescribed for the more serious cases, especially in patients with ocular involvement.[64,65] Blindness is a known complication, and its prevention may require aggressive pharmacologic management.

Hepatitis C virus

The hepatitis C virus (HCV) is a linear, single-stranded RNA virus of the *Flaviviridae* family that was first identified in 1989 [60]. HCV infection is a major health problem among the general population, and its extrahepatic manifestations have also been reported like Sicca syndrome.[61] Several autoimmune and immune complex-mediated disorders have been proposed to be related to HCV infection such as essential mixed cryoglobulinemia, which is frequently associated with Sjögren's syndrome. The association between HCV and Sjögren's syndrome may be related to the following reasons: (1) close association between HCV infection and mixed cryoglobulinemia, (2) the salivary gland tropism of HCV. [67] However, some studies did not find any relationship between xerostomia and the presence of HCV infection.[68,69] Xerostomia has been found among 5-55% of HCV-infected patients.[68,69,70,71] Xerostomia is also an adverse event during ribavirin-interferon therapy. [72,73]

Alzheimer's Disease

Dementia is considered an important, usually progressive neurologic disorder of old age and is the fifth leading cause of death.[74] It is characterized by a loss of cognitive abilities that impedes the performance of normal daily activities with loss of memory being the most prevalent sign. Alzheimer's disease (AD) is the most common variety of dementia. patients with Alzheimer's disease are known to have submandibular salivary gland hypofunction. Often the complaint of dryness is not objectively present and represents somatoform disease, a perceptual condition associated with a psychiatric abnormality[75] The first features of AD are characterized not only by memory loss but by a gradual deterioration in personality and intellectual ability. Anxiety, problems with sleeping, depression and an aggressive or even an apathetic behavior are often present. Abstract reasoning, ability to learn, judgment and language skills are impaired. Patients cannot care for themselves and eventually lose motor function with accompanying weakness and wasting from lack of food. Death usually results from a fatal pneumonia, probably caused by aspiration of food and oral microorganisms, facilitated by a developing salivary hypofunction and difficulty with swallowing. [76]

The etiology of AD is unknown, but a familial relationship has been shown to exist in about 5% of the patients [76]. A variety of neurochemical abnormalities develop. Most consistently, deficiency in the cholinergic system results and is distinguished by decreased activity of choline acetyl-transferase, an enzyme needed for the synthesis of acetylcholine. It can be anticipated that with the decrease in cholinergic activity, salivary hypofunction will result. However, when unstimulated and stimulated salivary volumes are measured, only the submandibular salivary gland demonstrated a decreased secretion. The parotid gland showed no changes in salivary volume.[74] The cause for this selective effect on the submandibular gland is unknown. Patients with AD receive numerous psychotherapeutic and mood-stabilizing medications to control symptoms of dementia. Most of the drugs are anticholinergic in their activity and will decrease the production of resting saliva. In addition, levodopa, a common neurotherapeutic agent for AD, has been reported to decrease salivary volume. Treatment may also include cholinesterase inhibitors whose aims are to boost cholinergic transmission at the remaining functional synapses and bring about improved cognitive performance and ameliorate behavioral symptoms. Simultaneously the increased cholinergic activity causes an increased salivary volume. Consequently salivary production in patients with AD is varied and is the result of the interplay of two diametrically opposite therapeutic approaches. The psychotherapeutics and levodopa decrease the salivary production that is supplemented by the pre-existing hypofunction of the submandibular gland, while the cholinesterase inhibitors tend to increase salivary volume.

Parkinson's disease (PD)

Parkinson's disease (PD) is a relatively common, progressive, debilitating, and neurological disorder. It can occur in individuals in their thirties, the peak age of onset is generally in the seventh decade of life. This chronic slowly progressing degenerative neurologic disease is characterized by tremors, muscle rigidity, loss of postural reflexes and slowing of voluntary movement.[77] A shuffling slow gait and a tendency to fall develop. Difficulty in initiating movement and a blank or staring facial appearance are features of PD. The salivary production in patients with PD is considered to be normal.[78] Any dryness that may develop is a side effect of prescribed medications**.** The neuromuscular dysfunction associated with PD causes a reduced rate and increased difficulty with swallowing. Salivary pooling in the mouth floor develops as a result of this dysphagia, which is not a result of salivary hyperfunction.Because there is an increased accumulation of saliva volume and because PD patients usually have their head in a bent forward position, saliva tends to drool out.[78] This visible

drooling of saliva has been interpreted in the past as excessive salivary production.

The therapeutic approach to PD includes the use of levodopa as well as several other medications that have a side effect of reducing salivary production.[78] Therapeutically, the anticholinergic agents can reduce the normal but unmanageable salivary volume. Recently Botox injections into the salivary glands to reduce salivary production have met with success.

Management of dry mouth

Close collaboration with the patient's general medical practitioner or with a specialist in oral medicine is often necessary. It might be possible, for example, to change an existing xerostomic drug to one less liable to reduce salivary flow. As there is a definite relationship between fluid intake and secretory performance it is essential that the patient is kept well hydrated. Chewing and energetic exercise improve salivary flow, possibly because of improved blood circulation to the glands.[22] Where some functional salivary tissue remains the problem may be alleviated by sialogogues such as sugar-free chewing gum or ascorbic acid. In cases where flow rate cannot be improved limited relief may sometimes be obtained by the use of artificial saliva. It is very important for a denture patient with a dry mouth to maintain an excellent level of denture hygiene. The likelihood of proliferation of Candida albicans is increased in xerostomia and therefore unless denture hygiene is maintained at a high level the denture is likely to be rapidly colonised by the micro-organism, resulting in denture stomatitis. Motivation and instruction of the patient, followed by monitoring the quality of denture hygiene are essential. In cases where an intractable dry mouth gives rise to a persistent problem of loose dentures a denture adhesive will usually provide some improvement in denture function.

CONCLUSION:

With the aging of the population, an increased frequency of visits by the elderly to the dental practitioner's office can be expected. These patients will often bring a variety of complaints associated with salivary secretory dysfunction and/or salivary gland disease.

Salivary glands are involved due to many systemic diseases with the resultant complication of xerostomia as autoimmune diseases, diabetes mellitus, HIV, mouth breathing, alzheimer's disease, hepatitis C virus, parkinson's disease (PD), sjogren's syndrome, sarcoidosis, gastroesophageal reflux disease, dehydration and acute parotitis. Some problems may be unique to the aged while other conditions reflect pathologic entities that affect all age ranges. It is necessary for the profession to be cognizant of the manifestations of the diverse abnormalities which may develop in this patient group. The purpose of this article is to alert the

profession to the symptomatology of these patients with salivary problems. Knowledge will result in prompt and accurate diagnoses. Identification of the main reason of xerostomia helps attain timely diagnosis and more appropriate treatment plan.

REFERENCES:

1. Thomson WM. Issues in the epidemiological investigation of dry mouth. Gerodontology 2005; 22:65–76.
2. Murray Thomson W et al. Xerostomia and medications among 32-year-olds. Acta Odontol Scand 2006; 64:249–54.
3. Guggenheimer J, Moore PA. Xerostomia etiology, recognition and treatment. JADA 2003; 134:61–9.
4. Fox PC. Xerostomia: Recognition and management. Dent Assist 2008; 77:44–8.
5. Grötz K et al. Long-term oral Candida colonization, mucositis and salivary function after head and neck radiotherapy. Support Care Cancer 2003; 11:717–21.
6. Momm F et al. Different saliva substitutes for treatment of xerostomia following radiotherapy. Strahlenther Onkol 2005; 181:231–6.
7. Turner M et al. Hyposalivation, xerostomia and the complete denture. A systematic review.JADA 2008; 139:146–50.
8. Daniels TE. Evaluation, differential diagnosis, and treatment of xerostomia. J Rheumatol Suppl 2000;61:6.
9. Rayman S et al. Xerostomia: Diagnosis and management in dental practice. N Y State Dent J 2010; 76:24–7.
10. Atkinson, JG, Wu AJ. Salivary gland dysfunction: Causes, symptoms, treatment. JADA 1994; 125:409–416.
11. Sreebny LM. Recognition and treatment of salivary induced conditions. International Dental Journal 1989; 39:197–204.
12. Astor FC et al. Xerostomia: A prevalent condition in the elderly. Ear Nose & Throat Journal 1999; 78:476–9.
13. Longman LP et al. Salivary gland hypofunction in elderly patients attending a clinic. Gerodontology 1995; 12:67–72.
14. Drummond JR, Chisholm DM. A qualitative and quantitative study of the ageing human labial salivary glands. Archives of Oral Biology 1994; 29:151–5.
15. Baum BJ et al. Salivary gland functions and aging: A model for studying the interaction of aging and systemic disease. Critical Reviews in Oral Biology & Medicine 1992; 4:53–64.
16. Drummond JR et al. Tomographic measurements of age changes in the

human parotid gland. Gerodontology 1995; 12:26–30.
17. Vissink A et al. Aging and saliva: a review of the literature. Special Care in Dentistry 1996; 16:95–103.
18. 18.Vitali, C et al. Classification criteria for Sjogren's syndrome: A revised version of the European criteria proposed by the American–European Consensus Group. Annals of the Rheumatic Diseases 2002; 61:554–58.
19. Scott, J Prominence of the submandibular glands in the aged. Journal of the American Geriatrics Society 1960; 8:53–4.
20. Locker D. Xerostomia in older adults: a longitudinal study. Gerodontology 1995; 12:18–25.
21. 21.Field EA et al. Age and medication are signifi cant risk factors for xerostomia in an English population, attending general dental practice. Gerodontology 2001; 18:21–4.
22. Niedermeier, W et al. Significance of saliva for the denture-wearing population. Gerodontology 2000; 17:104–18.
23. Ship JA et al. Xerostomia and the geriatric patient. Journal of the American Geriatrics Society 2000; 50:535–43.
24. Sreebny LM, Schwartz SS. A reference guide to drugs and dry mouth. Gerodontology 1997; 14:33–47.
25. Kagami H et al. Assessment of the effects of aging and medication on salivary gland function in patients with xerostomia using 99m TC-scintigraphy. Nagoya Journal of Medical Science 1995; 58:149–55.
26. Kitamura M et al. Predictors of root caries in the elderly. Community Dentistry and Oral Epidemiology 1986; 14:34–8.
27. Persson RE et al. Differences in salivary flow rates in elderly subjects using xerostomic medications. Oral Surgery Oral Medicine Oral Pathology 1991; 72:42–6.
28. MacEntee M I et al.. Predictors of caries in old age. Gerodontology 1993; 10: 90–7.
29. Younger H et al. Relationship among stimulated whole, glandular flow rates, and root caries prevalence in an elderly population; a preliminary study. Special Care in Dentistry 1993; 18:156–63.
30. Saunders RH, Handelsman SL. Effects of hyposalivary medications on salivary flow rates and dental caries in adults aged 65 and older. Special Care in Dentistry 1992; 12:116–21.
31. Ship JA, Fischer DJ. The relationship between dehydration and parotid salivary gland function in young and older healthy adults. Journals of Gerontology Series A-Biological Sciences and Medical Sciences 1997; 52:310–9.
32. Vasconcelos ACU, Soares MSM, Almeida PC, Soares TC. Comparative study of the concentration of salivary and blood glucose in type 2 diabetic patients. J Oral Sci. 2010;52:293–8.

33. Bakianian Vaziri P, Vahedi M, Mortazavi H, Abdollahzadeh Sh, Hajilooi M. Evaluation of salivary glucose, IgA and flow rate in diabetic patients: A case-control study. J Dent (Tehran) 2010;7:13–8.
34. Olokoba AB et al. Type 2 diabetes mellitus: a review of current trends. Oman Med J 2012; 27:269-270.
35. Bajaj S et al. Oral manifestations in type-2 diabetes and related complications.Indian J Endocrinol Metab 2012; 16:777–9.
36. Khovidhunkit S-oP et al. Xerostomia, hyposalivation, and oral microbiota in type 2 diabetic patients: A preliminary study. J Med Assoc Thai 2009; 92:1220–8.
37. Busato IMS et al. Impact of xerostomia on the quality of life of adolescents with type 1 diabetes mellitus. Oral Radiol Endod 2009; 108:376–82.
38. Costa CC et al. Study of the oral manifestations in diabetic children and their correlation variables. Arq Bras Endocrinol Metabol. 2004; 48:374–8.
39. Nagler R et al. Novel protection strategy against Xrayinduced damage to salivary glands. Radiation Research 1998; 149:271–6.
40. Bohuslavizki KH et al. Radioprotection of salivary glands by amifostine in high-dose radioiodine therapy. Journal of Nuclear Medicine 1998; 39:1237–42.
41. Cassel CK et al. Geriatric medicine; an evidenced based approach 4th ed.; New York: Springer, 2003
42. Helm JF. Role of saliva esophageal function and disease. Dysphagia 1989; 4:76–84.
43. Helm JF et al. Salivary response to esophageal acid in normal subjects and patients with reflux esophagitis. Gastroenterology 1987; 93:1393–7.
44. Kahrilas PJ. Esophageal motor activity and acid clearance. Gastroenterology Clinics of North America 1990; 19: 537–50.
45. Mandel L, Tamari K. Sialorrhea and gastroesophageal reflux. JADA 1995; 126:1537–41.
46. Al-Hashimi I. Xerostomia secondary to Sjogren's syndrome in the elderly: Recognition and management. Drugs & Aging 2005; 22:887–99.
47. Cassel CK et al. Geriatric medicine; an evidenced based approach 4th ed, New York: Springer 2003; pp. 838.
48. Okoje V et al. Orofacial lesions in 126 newly diagnosed HIV/AIDS patients seen at the University College Hospital, Ibadan. Afr J Med Med Sci. 2006; 35:97–101.
49. Nokta M. Oral manifestations associated with HIV infection. Curr HIV/AIDS Rep. 2008; 5:5–12.
50. Reznik D. Oral manifestations of HIV disease. Top HIV Med. 2005; 13:143–8.
51. Expósito-Delgado A et al. Oral manifestations of HIV infection in infants: A review article. Med Oral Patol Oral Cir Bucal 2004; 9:415.

52. Pinto A, De Rossi SS. Salivary gland disease in pediatric HIV patients: an update. J Dent Child (Chic)2004; 71:33–7.
53. Sharma G. Oral manifestations in HIV/AIDS infected patients from India. Oral Dis. 2006; 12:537–42.
54. Taiwo O et al. Oral manifestation of HIV/AIDS in Plateau state indigenes, Nigeria.West Afr J Med 2006; 25:32–7.
55. Freed JR et al. Oral health findings for HIV-infected adult medical patients from the HIV Cost and Services Utilization Study. JADA 2005; 136:1396–405.
56. Sontakke SA et al. Comparison of oral manifestations with CD4 count in HIV-infected patients. Indian J Dent Res 2011; 22:732.
57. Pinheiro A et al. Dental and oral lesions in HIV infected patients: a study in Brazil. Int Dent J. 2004; 54:131–7.
58. Nittayananta W. Hyposalivation, xerostomia and oral health status of HIV-infected subjects in Thailand before HAART era. J Oral Pathol Med 2010; 39:28–34.
59. Lin A et al. Alteration in salivary function in early HIV infection. J Dent Res 2003; 82:719–24.
60. Ramos-Casals M et al. Therapeutic management of extrahepatic manifestations in patients with chronic hepatitis C virus infection. Rheumatology (Oxford) 2003; 42:818–28.
61. Prunoiu C et al. Sjogren's syndrome associated with chronic hepatitis C-the benefit of the antiviral treatment. Rom J Morphol Embryol 2008; 49:557–62.
62. Olewiecki, S et al. Sarcoidosis-lymphoma syndrome. Journal of the Royal Society of Medicine 1992; 85:176–7.
63. Surattanont F. Bilateral parotid swelling caused by sarcoidosis. JADA 2002; 133:738–41.
64. Poate T et al. Orofacial presentations of sarcoidosis: A case series and review of the literature. Br Dent J 2008; 205:437–42.
65. Mansour M et al. Coexistence of Sjögren's syndrome and sarcoidosis: A report of five cases. J Oral Pathol Med 2007; 36:337–41.
66. Nessan V, Jacoway J. Biopsy of minor salivary glands in the diagnosis of sarcoidosis. New England Journal of Medicine 1979; 301:922–4.
67. Grossmann SdMC et al. Xerostomia, hyposalivation and sialadenitis in patients with chronic hepatitis C are not associated with the detection of HCV RNA in saliva or salivary glands. J Clin Pathol 2010; 63:1002–7.
68. de Mattos Camargo Grossmann S. Detection of HCV RNA in saliva does not correlate with salivary flow or xerostomia in patients with chronic hepatitis C. Oral Radiol Endod. 2010; 109:851–6.
69. Carrozzo M, Gandolfo S. Oral diseases possibly associated with hepatitis C virus. Crit Rev Oral Biol Med 2003; 14:115–27.

70. Nawito Z et al. Sicca complex among Egyptian patients with chronic hepatitis C virus infection. Clin Rheumatol 2011; 30:1299–304.
71. Giordano N et al. Immune and autoimmune disorders in HCV chronic liver disease: Personal experience and commentary on literature. New Microbiol 2005; 28:311–7.
72. Nagao Y et al. Candidiasis and other oral mucosal lesions during and after interferon therapy for HCV-related chronic liver diseases. BMC Gastroenterol 2012; 12:155.
73. Aghemo A et al. Ribavirin impairs salivary gland function during combination treatment with pegylated interferon alfa-2a in hepatitis C patients. Hepat Mon.2011; 11:918–24.
74. Ship JA et al. Diminished submandibular salivary flow in dementia of the Alzheimer type. Journals of Gerontology 1990; 45:61–6.
75. Ship JA, Fischer DJ. The relationship between dehydration and parotid salivary gland function in young and older healthy adults. Journals of Gerontology Series A-Biological Sciences and Medical Sciences 1997; 52:310–9.
76. Friedlander AH. Alzheimer's disease: psychopathology, medical management and dental implications. JADA. 2006; 137:1240–51.
77. Cersósimo M et al. Hyposialorrhea as an early manifestation of Parkinson disease. Auton Neurosci. 2009; 150:150–1.
78. Proulx M et al. Salivary production in Parkinson's disease. Mov Disord 2005; 20:204–7.