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Salivary Gland Dysfunction: Etiology, Epidemiology, Clinical Manifestations, Diagnosis, and Treatment

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This course is no longer offered for Continuing Education credit.

This continuing education course presents the etiology, epidemiology, clinical manifestations, diagnosis, and treatment of salivary gland dysfunction.

Conflict of Interest Disclosure Statement

- Dr. Sankar reports no conflicts of interest associated with this work.
- Dr. Terézhalmy has done consulting work for P&G.

Overview

Salivary gland dysfunction may be characterized by either hyposalivation or hypersalivation. To provide competent care to patients with salivary gland dysfunction, clinicians must understand its many causes and associated complications, and develop preventive and therapeutic strategies accordingly.

Learning Objectives

Upon completion of this course, the dental professional should be able to:

- Discuss the etiology and epidemiology of salivary gland dysfunction.
- Recognize the clinical manifestations of salivary gland dysfunction.
- Diagnose salivary gland dysfunction.
- Develop strategies for the prevention and treatment of hyposalivation and hypersalivation.

Course Contents

Introduction

Salivary gland function is under the control of the autonomic nervous system.[1,2,3] In response to smell, taste, and mastication, afferent fibers of the salivary reflex arch activate the salivary nuclei located in the medulla oblongata.[4] Sympathetic and parasympathetic fibers innervating the salivary glands, with acetylcholine as the neurotransmitter, comprise the efferent part of the secretory reflex arch.[3] Following a circadian rhythm, the major salivary glands (parotid, sublingual, and submandibular) are responsible for the secretion of about 90% of all saliva; minor salivary glands, located in labial, buccal, and palatal oral mucosa, account for the balance.[4]

A healthy person secretes up to 1.5L of saliva per day.[5] Whole saliva is composed mostly of water and contains many organic and inorganic substances.[5,6,7,8,9] Two defining constituents are amylase[10,11] and mucin[12]. The parotid glands secrete predominantly amylase-rich serous saliva; the sublingual and minor salivary glands secrete predominantly mucin-rich mucous saliva; and the submandibular glands secrete mixed seromucous saliva. The parotid glands contribute most of the stimulated saliva, while the submandibular glands contribute most of the unstimulated saliva. Minor salivary glands produce up to 70% of the mucin.

Saliva provides a physical barrier against local irritants and promotes mucosal repair (epidermal growth factor); lubricates and cleans oral tissues (mucin); maintains a stable intraoral pH with its bicarbonate buffering system; and maintains electrolyte balance.[13] Saliva prevents demineralization and contributes to the remineralization (proline-rich proteins) of dental hard tissues.[14] Saliva contains antibacterial (lysozymes), antiviral (secretory leukocyte protease inhibitors), and antifungal (histatins) components, which help to maintain a favorable ecosystem for the normal oral flora.[15] Finally, saliva facilitates mastication, the formation of a bolus (mucin and water), swallowing, initiates food processing (amylase), and has a salutary effect on the sense of taste.[4]

Salivary gland dysfunction may be characterized by either hyposalivation or hypersalivation. To provide competent care to patients with salivary dysfunction, clinicians must understand its many causes and associated complications, and develop preventive and therapeutic strategies accordingly.

Hyposalivation

A patient is considered to have reduced salivary flow if the unstimulated salivary flow rate measured for 5 to 15 minutes is ≤0.1 mL/min; or if the stimulated salivary flow rate measured for 5 minutes is ≤0.5 mL/min.[5]

Etiology and Epidemiology

Hyposalivation may be related to rare congenital absence or aplasia of one or more major salivary glands or ducts; non-infectious and infectious sialoadenitis; benign and malignant salivary gland tumors; and systemic conditions that directly affect salivary gland function, or more commonly secondarily, as side effects associated with pharmacotherapeutic agents prescribed for the treatment of a primary disease.[16,17] Hyposalivation is also a major complication of cancer chemotherapy and head and neck radiotherapy.[18,19]

There is a paucity of data on the true prevalence of hyposalivation in the general population; reports vary from 0.9% to 64.8%.[20] In a 15-year longitudinal study of xerostomia, it was found that hyposalivation increased from 6% at age 50 to 15% at age 65.[21] Other investigators estimated that among the elderly ≥65 years old, the incidence of xerostomia may be as high as 30%.[22] However, there is no strong evidence that saliva production by the major glands is significantly decreased with age.[22,23] It is more likely that the increased incidence of systemic disease in the elderly and associated polypharmacy is the primary contributor to clinically significant salivary gland hypofunction.[24,25,26] A 5-year longitudinal study in a cohort of older persons found that the severity of hyposalivation was higher among women and related to the use of xerogenic medications.[27] Clearly, the incidence of hyposalivation increases with exposure to polypharmacy and over 400 drugs have been identified as xerogenic.[28] The prevalence of hyposalivation for patients with Sjögren's syndrome and those who have received head and neck radiotherapy is nearly 100%.[22]

Non-infectious Sialoadenitis

Non-infectious sialoadenitis is most commonly due to obstruction of a salivary gland duct. **Mucoceles** are common reactive minor salivary gland lesions of the lower lip. As a duct is severed by trauma, saliva leaks into the surrounding connective tissue, which results in a non-epithelium-lined fluid-filled cavity (Figure 1). **Ranulas** present as either circumscribed lesions (subsequent to ductal obstruction and cystic dilatation) or plunging lesions (following extravasation of saliva into tissues of the floor of

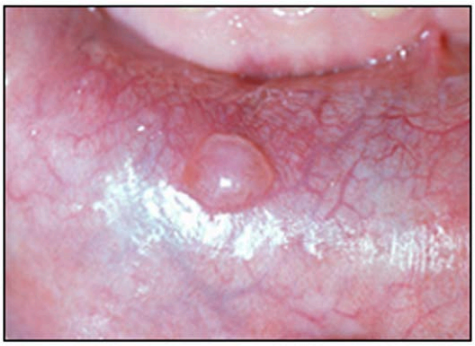


Figure 1. Mucocele

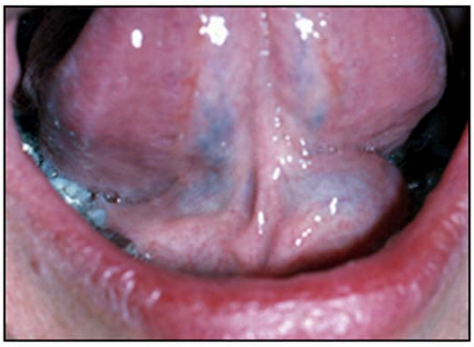


Figure 2. Ranula

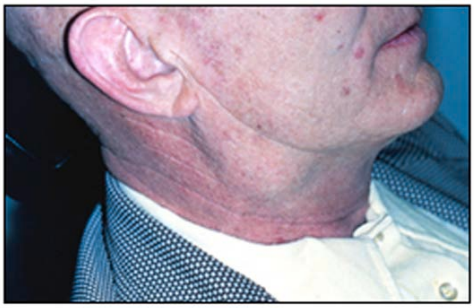


Figure 3. Sialolith

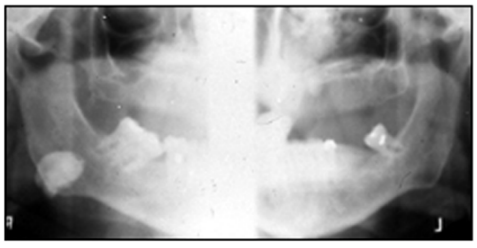


Figure 4. Sialolith Radiograph

the mouth) in association with a submandibular or sublingual gland (Figure 2). **Sialoliths** develop primarily in the submandibular ductal system and are caused by calcification of mucous plugs and cellular debris (Figures 3 and 4). Sialoliths tend to occur much less frequently in association with the parotids and are considered to be rare in sublingual and minor salivary glands. **Mucous cysts** of minor salivary glands are caused by ductal obstruction by a mucous plug, other cellular debris, or calcification and may be characterized as mucous extravasation surrounded by granulation tissue or as true cysts lined with ductal epithelium.[29]

Infectious Sialoadenitis

Bacterial
Bacterial infections, typically affecting the parotid glands, are more common in the elderly who are dehydrated or experience salivary hypofunction secondary to medications, head and neck radiation, and systemic diseases. Organisms implicated include Staphylococcus aureus, *Streptococcus viridians*, *group B streptococci*, *Haemophilus influenza*, *Escherichia coli*, and *Pseudomonas aeruginosa*. Cases of tuberculous parotitis have also been documented.[30,31,32]

Viral
Viral infections can occur in persons of all ages and preferentially affect the parotid glands. Organisms commonly implicated in children and adolescents include the mumps virus; in adults the *Cytomegalovirus*, *Coxsackievirus*, *Epstein-Barr virus*, *hepatitis C virus*, and the *human immunodeficiency virus (HIV)*.[29]

Mumps, an acute viral infection caused by an RNA virus of the *Paramyxoviridae* family, is characterized by unilateral or bilateral tender swelling of the parotid and/or other salivary glands.[33,34] Characteristic findings include an outward lifting of the ear lobe, obscuration of the angle of the mandible, and erythema affecting Stensen's ducts. The parotitis progresses over 2-3 days and persists for about a week. While the hallmark of mumps is parotitis (92%), the sublingual and submandibular glands are also affected in about 7% of cases.[35]

Approximately 5-10% of HIV infected individuals and those with AIDS experience salivary gland disease.[36] Parotid gland enlargement usually occurs bilaterally and is associated with cervical lymphadenopathy. Diffuse infiltrative lymphocytosis syndrome (DILS) results from CD8 lymphocytic infiltration and is often followed by lymphoepithelial cyst formation. This will affect either the parotid or submandibular glands with a predilection for the parotids. Salivary gland disease is considered a Group 2 oral condition, i.e., a condition less commonly associated with HIV infection.[37]

Benign and Malignant Salivary Gland Tumors
Benign and malignant salivary gland tumors primarily affect the palate followed by the upper lip. Signs and symptoms of a malignant salivary gland tumor include a swelling with facial nerve paralysis, pain, or facial paresis. The majority of tumors that arise within the salivary glands originate from epithelial tissues; however some are derived from adjacent tissues or structures (i.e., adipose, nerves, blood vessels, lymph nodes, lymphatics). Most salivary gland neoplasms are benign. Pleomorphic adenoma and papillary cystadenoma lymphomatosum represent 75% and 5% of all salivary gland tumors, respectively.[38] Adenoid cystic carcinoma (Figure 5) and mucoepidermoid carcinoma (Figure 6) are the most common malignant salivary

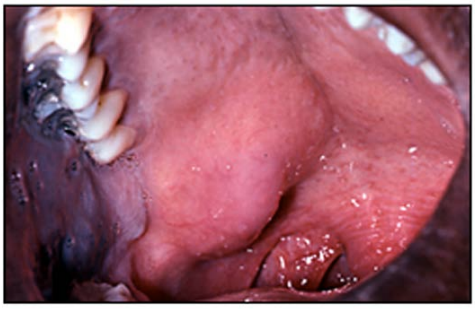


Figure 5. Adenoid cystic carcinoma

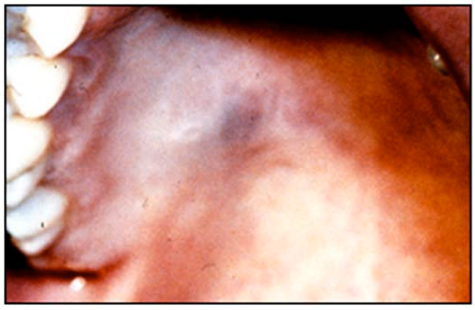


Figure 6. Mucoepidermoid carcinoma

gland tumors, representing 10% (5% and 5%) of all salivary gland tumors. Less common malignant tumors include acinic cell carcinoma, adenocarcinoma, squamous cell carcinoma, and carcinoma arising in a pleomorphic adenoma.[39]

Sialoadenosis

Sialoadenosis is seen in association with a number of systemic conditions (e.g., chronic inflammatory/autoimmune diseases [Sjögren's syndrome, systemic lupus erythematosus, scleroderma, graft-versus-host-disease (GVHD)], endocrine/metabolic disorders [diabetes mellitus, thyroid dysfunction, adrenal dysfunction], nutritional abnormalities and eating disorders [anorexia nervosa, bulimia, alcohol abuse, and neuropathic diseases [depression, Alzheimer disease, Bell's palsy], and others [fibromyalgia, chronic fatigue syndrome]).[40,41] In addition, hyposalivation is a predictable sequel of polypharmacy, cancer chemotherapy and head and neck radiotherapy.[15,16,40]

Sjögren's Syndrome

Sjögren's syndrome is a relatively common condition primarily affecting women (it may be as prevalent as 1 out of every 2,500 females) with a typical onset during the fourth or fifth decade of life.[42,43,44] Primary Sjögren's syndrome is characterized by dry mouth and dry eyes, which are the result of a progressive loss of salivary and lacrimal gland function.[42] As the disease progresses, lymphocytic infiltrates of salivary glands ultimately produce acinar gland

Table 1. Criteria to define primary Sjögren's syndrome[46]

I.	**Ocular symptoms:** A positive response to at least one of the following questions:	1. Have you had daily, persistent, troublesome dry eyes for more than 3 months? 2. Do you have a recurrent sensation of sand or gravel in the eyes? 3. Do you use tear substitutes more than 3 times a day?
II.	**Oral symptoms:** A positive response to at least one of the following questions:	1. Have you had a daily feeling of dry mouth for more than 3 months? 2. Have you had recurrently or persistently swollen salivary glands as an adult? 3. Do you frequently drink liquids to aid in swallowing dry food?
III.	**Ocular signs**: That is, objective evidence of ocular involvement defined as a positive result for at least one of the following two tests:	1. Schirmer's I test, performed without anesthesia (<5 mm in 5 minutes) 2. Rose bengal score or other ocular dye score (>4 according to van Bijsterveld's scoring system)
IV.	**Histopathology:** In minor salivary glands focal lymphocytic sialoadenitis, evaluated by an expert histopathologist:	Focus score >1, defined as a number of lymphocytic foci (which are adjacent to normal-appearing mucous acini and contain more than 50 lymphocytes) per 4 mm2 of glandular tissue.
V.	**Salivary gland involvement:** Objective evidence of salivary gland involvement defined by a positive result for at least one of the following diagnostic tests:	1. Unstimulated whole salivary flow (<1.5 ml in 15 minutes). 2. Parotid sialography showing the presence of diffuse sialectasias (punctate, cavitary or destructive pattern), without evidence of obstruction in the major ducts. 3. Salivary scintigraphy showing delayed uptake, reduced concentration and/or delayed excretion of tracer.
VI.	**Autoantibodies:** Presence in the serum of the following autoantibodies:	Antibodies to Ro(SSA) or La(SSB) antigens, or both.

degeneration, necrosis, atrophy, and complete salivary gland destruction.[45] Patients frequently present with intermittent enlargement of the parotid and/or submandibular glands. The diagnosis of primary Sjögren's syndrome is predicated on meeting at least 4 of the 6 criteria in (Table 1).[46] In its secondary form, Sjögren's syndrome is associated with co-morbidities such as rheumatoid arthritis, systemic sclerosis, systemic lupus erythematosus, or scleroderma.[47,48]

Diabetes Mellitus (DM)

In well-controlled diabetic patients, salivary gland function does not seem to be significantly affected.[49] However, several studies have reported subjective complaint of dry mouth among diabetic patients in general.[50,51,52] In one study, investigators reported an association between lower resting and stimulated salivary flow and elevated HbA1c levels.[50] In another study, investigators have demonstrated reduced resting and stimulated salivary flow rates in type 1 diabetic patients with elevated fasting blood glucose (FBG) concentrations and diabetic neuropathy.[52] Abnormalities in parotid gland basement membranes have also been demonstrated in subjects with DM.[53] Sialoadenosis, associated with excessive accumulation of secretory granules and enlargement of the acinar cells characterized by a diffuse, noninflammatory, bilateral enlargement of the parotid glands was reported with both forms of DM.[54] In addition, a significant reduction in resting salivary flow rates was reported in patients with type 1 DM taking parasympatholytic drugs in association with elevated fasting blood sugar levels.[52] A similar association was reported between parasympatholytic drugs and autonomic neuropathy in patients with type 2 DM.[54]

Polypharmacy

Xerostomia is a common complication of both prescription and non-prescription drug therapy and over 400 active medications have been implicated (Figures 7 and 8).[28] Reduced salivary flow is generally related to a drug's parasympatholytic or antimuscarinic effect. The site of action of these drugs may be in the central nervous system, at parasympathetic and some sympathetic ganglia, and at parasympathetic and some sympathetic effector junctions.

Other drug-induced salivary gland problems include fluid and electrolyte imbalance, glandular vasoconstriction, and altered fluid movement from plasma through salivary acinar cells to the ductal system, and ultimately into the oral cavity. The major classes of drugs causing xerostomia are included in (Table 2).[28,40] Salivary gland dysfunction associated with cytotoxic drugs appears to be temporal, during and immediately after treatment, and salivary function returns to pre-chemotherapy levels in most patients after completion of therapy.[19,55]

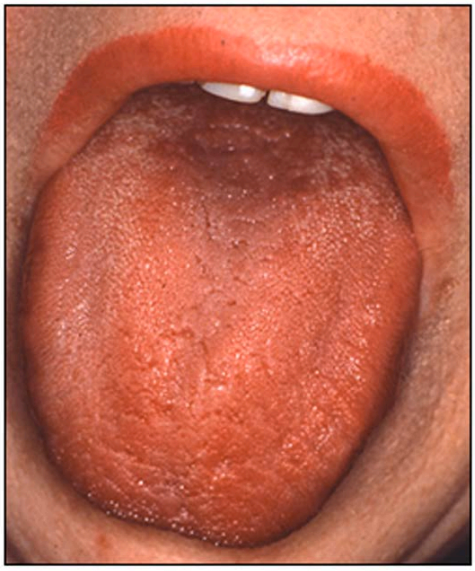


Figure 7. Antihistamine-induced dry mouth

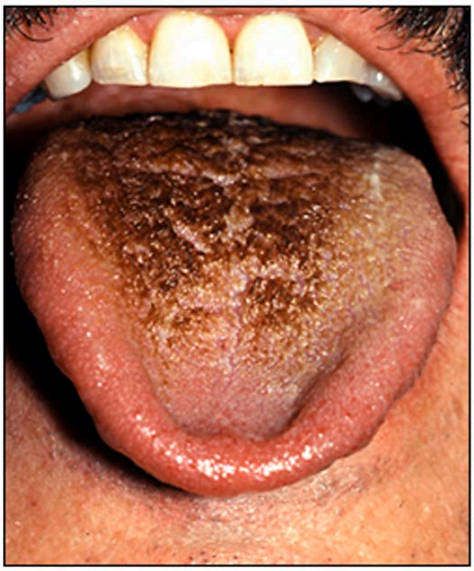
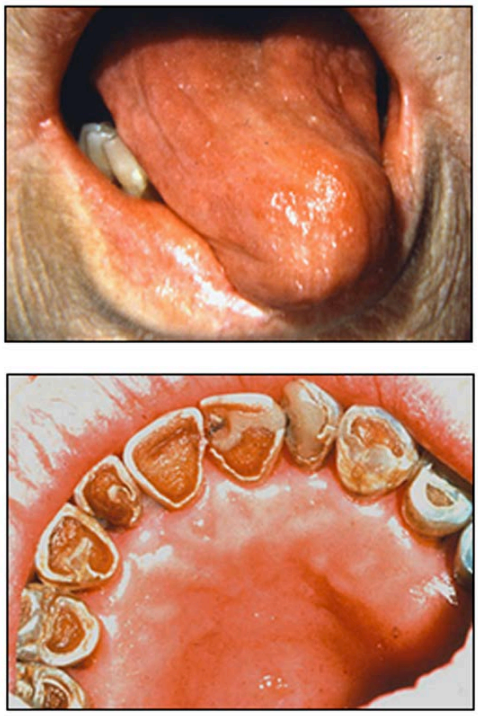


Figure 8. Diuretic-induced dry mouth

Table 2. Major drug categories causing hyposalivation
• Anticholinergic agents • Anticonvulsants • Antidepressants • Antiemetics • Antihistamines • Antihypertensive agents and diuretics • Antiinflammatory agents • Antiparkinsonian drugs • Antipsychotic agents • Anxiolytic, sedative-hypnotic agents • Cancer chemotherapeutic agents • Muscle relaxants • Opioids

Head and Neck Radiotherapy

Xerostomia is the most frequently reported late side effect of radiotherapy to the head and neck (Figures 9 and 10).[56-62] For reasons not clearly understood, the well-differentiated and slow replicating salivary gland cells are highly radiosensitive.[61,63,64] The effects are profound and depend on the volume of the glands irradiated, the total dose delivered, and the functional state of the glands prior to radiotherapy.[61,63,65]



Figures 9 & 10. Radiation-induced dry mouth and caries

Radiotherapy causes acute degeneration and necrosis of acinar cells, followed by attempted regeneration. If the radiation dose to the salivary glands is sufficient, regeneration fails leading to fibrosis and atrophy of glandular tissue.[64,66,67] While a mean dose of 60 Gy is the accepted threshold for producing irreversible damage, in some cases mean doses of as little as 10 Gy have been implicated.[60,63,65] Radioactive iodine (I-131) used in the treatment for cancers of the thyroid gland may cause parotid but not submandibular dysfunction in a dose-dependent fashion.[68]

It is generally accepted that the serous acini are more radiosensitive and less likely to regenerate compared to the mucous acini, although a recent study suggests that serous and mucous acini are equally radiosensitive. Acinar recovery, if any, is dose dependent and may occur approximately 12 to 18 months following completion of therapy. As a general rule, however, patients will experience significant reduction in salivary flow for the remainder of their life. When the major glands are affected, salivary output may be reduced

by as much as 80% within the first 2 weeks of conventional radiotherapy.[69]

Clinical Manifestations

Qualitative and quantitative changes in saliva lead to reduced lubrication. The dryness affects taste and speech. The oral mucosal tissues appear dry and atrophic, the tongue is often fissured. Loss of mucosal integrity may lead to mucositis. Reduced lavage and cleansing of oral tissues promotes plaque accumulation, gingivitis, periodontitis, and dental caries.[16]

Mucositis (i.e., loss of mucosal integrity) may result in reduced lysozyme, secretory leukocyte protease inhibitor, and histatin activity, which predisposes to bacterial, viral, and fungal infections.[70] Secondary infections and mucositis may compromise the wearing of tissue-borne prostheses and lead to impaired chewing and swallowing. Impaired chewing and swallowing and / or the presence of dysgeusia, hypogeusia, or ageusia may cause the patient to alter their diet and fluid intake, increasing the risk of malnutrition and weight loss. Hyposalivation alone may decrease the retention of even well fitting prostheses, which in turn may lead to the development of traumatic ulcers. In irradiated patients, such ulcers take on added significance because they provide a portal to oral bacteria, with a potential for the development of markedly morbid osteoradionecrosis.[18,71]

Decrease in salivary immunoglobulin levels leads to a highly cariogenic oral microflora, (*Streptococcus mutans*, *lactobacillus* and *Actinomyces*). In addition, the lack of saliva promotes the availability of sugars and other substrates essential for microbial survival and the loss of salivary buffering capacity and loss of the insoluble pellicle formed by saliva on teeth interfere with normal remineralization of teeth.[72]

As a consequence, a distinctive form of caries begin on plaque forming surfaces and areas of exposed dentin resulting in circumferential lesions at the cementoenamel junction, and smooth surface caries on cusp tips and incisal edges where attrition had previously occurred. Tooth loss, a predictable sequel of advanced carious lesions, presents further difficulties for both the patient and clinician. Alteration of the normal flora may also increase the potential for aspiration pneumonia and colonization of the lungs with gram-negative anaerobes from the gingival sulcus.[73,74]

Reduced antibacterial and antifungal activity associated with hyposalivation also contributes to an increased risk of both acute and chronic infection by the *Candida* spp, which under normal conditions is inhibited by a normal bacterial flora and salivary gland function.[75] The lesions often appear as white, raised, or cottage cheese-like growths that can be scraped off, leaving a red, hemorrhagic base. Candidiasis may also appear as an erythematous lesion under prostheses or as angular cheilosis. A comprehensive review of oropharyngeal candidiasis is presented elsewhere.[76]

Diagnosis

The diagnosis of salivary gland dysfunction is predicated on a comprehensive physical evaluation. It typically begins by assessing the patient's chief complaint in an attempt to identify conditions or predisposing factors.[77] Subjective assessment of salivation may include simple questionnaire requiring a "yes" or "no" response to four questions (Box 1).[78] A visual analogue or an ordinal scale based on ranked categories, i.e., "I have no – slight – severe – annoying feeling of dry mouth," may also provide subjective data.[79] Objective evaluation requires a measurement of the patient's salivary flow rates.[80]

Box 1. Subjective assessment of salivation[76]

1. Does the amount of saliva in your mouth seem too little?
2. Does your mouth feel dry when eating?
3. Do you have difficulty swallowing any food?
4. Do you sip liquids to aid in swallowing dry food?

Serologic testing for antinuclear antibodies (e.g., rheumatoid factor, anti-Ro/anti-SS-A, anti-La/anti-SS-B), minor salivary gland biopsy (lymphocytic infiltrates), and a variety of imaging techniques (e.g., sialograms performed with radio-opaque iodine and extraoral radiographs and radioactive isotype scintiscans (T99 pertechnetate) can provide additional diagnostic data. MRI and CT scans can help rule out salivary gland tumors and other pathoses associated with the craniofacial region that may adversely affect salivation. The diagnosis of salivary gland neoplasia requires incisional or excisional biopsies and histopathological evaluation.

Therapeutic strategies

Once a diagnosis of salivary gland dysfunction is established, medical management is predicated on its etiology and to a great extent falls in the purview of the medical profession. Irrespective of the therapeutic strategy, however, the role of oral healthcare providers is critical to minimize the many potential oral complications of salivary gland hypofunction.

Palliative and Supportive Care

Good oral hygiene and adequate hydration are essential elements of palliative and supportive care. Desiccated oral tissues are friable and prone to irritation and ulceration. Patients should be instructed to avoid products irritating to oral soft tissues (such as alcohol and tobacco; hot, spicy, and coarse foods; fruits and beverages with a high acid content); to refrain from wearing removable prostheses; to eat a dental soft diet; and to frequently rinse with a sodium bicarbonate solution. Xerostomia-related mucosal lesions are susceptible to secondary infections by microorganisms of the normal flora that have become pathogenic and by transient organisms. Prompt treatment of such infections is mandatory.

Retention of removable prostheses is frequently impaired and painful in the presence of desiccated oral mucosa tissues and the lack of adequate salivary output. Daily oral hygiene of dentures and prosthesis-bearing mucosal tissues is important, as is regular observation for candidal infections. Careful chewing and swallowing is advised with the addition of frequent sips of liquids to avoid choking and aspiration. Denture problems can be diminished with frequent dental examinations to identify sore spots and to enhance adhesion with soft- and hard-tissue relines.

Patients with residual salivary gland function may benefit from using simple dietary measures such as eating carrots or celery, or by chewing sugarless or xylitol-containing gums. A variety over-the-counter saliva substitutes and moisturizers are also available. The viscosity and electrolyte concentrations of these agents are adjusted to approximate whole saliva and pleasant-tasting flavoring is added to most preparations. Patient acceptance is variable and depends on effect, duration, lubrication, taste, delivery system, and cost.[71]

Sialagogues

Pilocarpine hydrochloride (Salagen®, Pharmacia, Saint Paul, MN) a cholinergic agonist, stimulates the production of saliva from functioning salivary glands.[81] It enhances oral comfort, improves the ability to speak, and offers prolonged symptomatic relief. The recommended initial dosing regimen is 5 mg three times daily. If necessary, up to 30 mg per day may be given in divided doses not exceed 2 tablets per dose. The lowest effective dose should be used to maintain optimal salivary flow. The continuing effect of pilocarpine hydrochloride depends on regular use. Maximum benefit is typically noted after 90 days of use. Pilocarpine hydrochloride is contraindicated in patients with uncontrolled asthma or narrow angle glaucoma, and should be used with caution in patients with significant cardiovascular disease. Common dose-dependent side effects include sweating, nausea, rhinitis, flushing, and increased urinary frequency.[82]

Another sialagogue, cevimeline hydrochloride (Evoxac®, Daiichi Sankyo Inc., Tokyo, Japan) has been approved by the Food and Drug Administration (FDA) for the treatment of dry mouth in Sjögren's syndrome in a dosage of 30 mg three times daily.[71,81,83] Like pilocarpine, it is a muscarinic agonist that increases production of saliva. Pilocarpine is a non-selective muscarinic agonist, whereas cevimeline reportedly has a higher affinity for M1 and M3 muscarinic receptor subtypes. Since M2 and M4 receptors are located on cardiac and lung tissues, cevimeline can enhance salivary secretions while minimizing adverse effects on pulmonary and cardiac

function. However, patients with uncontrolled asthma, significant cardiac disease, and narrow angle glaucoma should not take cevimeline.

Caries Prevention

It must be emphasized that saliva substitutes and sialagogues do not constitute a total chemical solution to the problem of rampant caries. Daily topical fluoride use and anti-microbial mouth rinses help prevent caries in patients with reduced salivary flow. To reduce increased caries risk, the daily use of a fluoridated dentifrice (0.05% sodium fluoride) and the daily use of a prescription fluoride gel (1% sodium fluoride or 0.4% stannous fluoride) is recommended. In addition, the application of a 0.5% sodium fluoride varnish and regular (every 2 to 6 months based on risk factors) preventive care should be implemented.[71]

Candidiasis

Hyposalivation increases the risk of superinfection by *Candida* spp., which under normal conditions is inhibited by normal flora and salivary gland function. Topical antifungal agents such as the various nystatin formulations or clotrimazole troches may be prescribed. However, these agents contain sugar and may add to the caries risk. Systemic antifungal agents such as fluconazole may be better alternatives. A comprehensive discussion of the treatment of oropharyngeal candidiasis is presented elsewhere.[76]

Hypersalivation

A patient is considered to have increased salivary flow if the unstimulated salivary flow rate measured for 5 to 15 minutes is ≥1 mL/min; or if the stimulated salivary flow rate measured for 5 minutes is ≥3.5 mL/min.[5]

Etiology and Epidemiology

A less common disorder than hyposalivation, hypersalivation usually is not reflective of a true increase in salivary flow, but of an inability to coordinate control mechanisms of orofacial musculature leading to drooling (Table 3).[84] While the condition is considered normal in infants up to the age of 18 months, it is a rather uncommon condition in adults, and may lead to social, psychological, and clinical consequences. True hypersalivation may be due to inflammation or dental infections, exposure to toxins (mercury vapor), or medication side effects. In association with neuromuscular dysfunction (i.e., Parkinson's disease, cerebral palsy, etc), it may be an apparent rather than true hypersalivation. Muscle incoordination interferes with swallowing and the transition of saliva from the mouth to the oropharynx. Anatomical abnormalities such as macroglossia, malocclusion and lip incompetence can lead to an inability to manage saliva leading to the perception of hypersalivation.[84,85]

Clinical Manifestations

Drooling or excessive salivation manifests as saliva beyond the margin of the lip. Physical

Table 3. Causes of drooling[84]

- **Neuro-muscular diseases**
  - Cerebral palsy
  - Amyotrophic lateral sclerosis
  - Parkinson's disease
- **Heavy metal neurotoxins**
  - mercury
  - thallium
  - copper
  - arsenic
  - antimony

- **Intellectual disability**
  - Wilson disease
  - Angelman syndrome
  - Moebius syndrome
  - Pseudobulbar palsy
  - Bulbar palsy
  - Stroke
  - Facial palsy
- **Anatomic abnormalities**
  - Macroglossia
- **Orthodontic problems**
  - anterior open bite
  - lip seal incompetence
- **Defects after major head/neck surgery**
  - Ankylosis of temporomandibular Joint
  - Excessive saliva production (sialorrhea)
- **Irritating oral disease**
  - ulceration
  - infection
  - trauma
- **Medication side-effects**
  - iclozapine
  - risperidone
  - nitrazepam
  - bethanecol
- **Gastroesophageal reflux**
- **Other less frequent causes**
  - laryngitis
  - pharyngitis
  - tonsillitis
  - epiglottitis
  - secretory phase of menstrual cycle
  - pregnancy

signs include perioral chapping, maceration, secondary fungal or bacterial infection, malodor, aspiration related respiratory and pulmonary complications. Drooling can lead to functional, social, psychological and clinical consequences for the patient, their family and caregivers.

Therapeutic strategies

The management of these patients often requires a multidisciplinary approach, including personnel from specialties such as ENT, neurology, dentistry, speech, occupational and physical therapy. Improving posture, stabilization of body positioning, lip closure exercises and other oral motor training may be of use.

Pharmacological intervention may include medications that reduce cholinergic activity such as atropine or ipratropium, or medications that increase adrenergic activity such as clonidine. Botulinum toxin is an emerging therapy in the treatment of drooling; however side effects include weakness of the adjacent muscles resulting in difficulty swallowing and increased episodes of aspiration. Irradiation of the salivary glands has produced variable success.

Surgical therapy includes gland excision, duct re-routing, duct ligation and transtympanic neurectomy. The results of these surgical as well as the pharmaceutical treatments have been variable. Drooling continues to be a condition which is difficult to manage.

Conclusion

Once a diagnosis of salivary gland dysfunction is established, medical management is predicated on its etiology and to a great extent falls in the purview of the medical profession. Irrespective of the therapeutic strategy, however, the role of oral healthcare providers is critical to minimize the many potential oral complications of salivary gland hypofunction. Symptomatic and supportive care of the xerostomic patient should include good oral hygiene procedures, proper dietary control, the use of saliva substitutes, and/or a sialogogue. The substitutes should preferably have a pleasant taste, contain electrolytes in concentrations normally found in saliva, and have an appropriate viscosity. The use of supplemental fluoride agents to promote remineralization of the enamel is recommended. Fluoride delivery systems that provide optimal protection are now available.

Course Test Preview

To receive Continuing Education credit for this course, you must complete the online test. Please go to www.dentalcare.com and find this course in the Continuing Education section.

1. The major salivary glands are responsible for the secretion of about _____ of all saliva.

a. 10%
b. 90%
c. 50%
d. 30%

2. Most of the stimulated saliva is contributed by the _____ glands.

a. minor salivary
b. submandibular
c. parotid
d. sublingual

3. Saliva provides a physical barrier against local irritants; promotes mucosal repair; lubricates and cleans oral tissues; maintains a stable oral pH and electrolyte balance; and _____.

a. prevents demineralization and contributes to remineralization
b. contains antibacterial, antiviral, and antifungal components to help maintain a favorable ecosystem for the normal flora
c. facilitates mastication, the formation of a bolus, initiates food processing, and has a salutary effect on the sense of taste
d. All of the above.

4. Hyposalivation may be related to all of the following conditions except which one?

a. Rare congenital absence or aplasia of one or more minor salivary glands or ducts.
b. Non-infectious and infectious sialadenitis.
c. Benign and malignant salivary glands.
d. Systemic conditions that directly affect salivary gland function, pharmacotherapeutic agents, and head and neck radiotherapy.

5. All of the following statements are true relative to hyposalivation and the elderly except which one?

a. It has been found that hyposalivation increased from 6% at age 50 to 15% at age 65.
b. Among elderly ≥65 years old, the incidence of xerostomia may be as high as 30%.
c. There is strong evidence that saliva production by the major salivary glands is significantly decrease with age.
d. The increased incidence of systemic disease in the elderly and associated polypharmacy is the primary contributor to clinically significant salivary gland hypofunction.

6. Non-infectious sialoadenitis that presents as either a circumscribed lesion or plunging lesions of the floor of the mouth gland is most likely a _____.

a. mucocele
b. ranula
c. sialolith
d. mucous cyst

7. Infectious sialoadenitis (bacterial or viral) is most likely to affect the _____ glands.

a. minor salivary
b. sublingual
c. parotid
d. submandibular

8. All of the following statements are correct with respect to benign and malignant salivary gland tumors except which one?

a. Benign and malignant salivary gland tumors primarily affect the palate followed by the upper lip.
b. Most salivary gland tumors are malignant.
c. Pleomorphic adenoma and papillary cystadenoma lymphomatosum representing 75% and 5% of all salivary gland tumors, respectively.
d. Mucoepidermoid carcinoma and adenoid cystic carcinoma (cylindroma) are the most common malignant salivary gland tumors.

9. Primary Sjögren's syndrome _____.

a. primarily affect women during the fourth and fifth decade of life
b. is characterized by dry mouth and dry eyes
c. manifests as intermittent enlargement of the parotid and/or submandibular glands
d. All of the above.

10. Which of the following statements is correct relative to diabetes mellitus?

a. Investigators reported an association between lower resting and stimulated salivary flow and elevated HbA1c levels.
b. Investigators reported an association between lower resting and stimulated salivary flow in type 1 diabetic patients with elevated fasting blood glucose concentrations and diabetic neuropathy.
c. In well controlled diabetic patients, salivary gland function does not seem to be significantly affected.
d. All of the above.

11. All of the following statements a correct with respect to polypharmacy and xerostomia except which one?

a. Xerostomia is a common complication of both prescription and non-prescription drug therapy and over 400 ingredients in various formulations have been implicated.
b. Reduced salivary flow is generally related to a drug's muscarinic or parasympathomimetic effect.
c. Drug-induced salivary gland problems include fluid and electrolyte imbalance, glandular vasoconstriction, and altered fluid movement from plasma through salivary acinar cells to the ductal system, and ultimately into the oral cavity.
d. Salivary gland dysfunction associated with cytotoxic drugs appears to be temporal, during and immediately after treatment, and salivary function returns to pre-chemotherapy levels in most patients after completion of therapy.

12. Which of the following determine the effects of head and neck radiotherapy on salivary glands?

a. The volume of the gland irradiated.
b. The total dose delivered to the gland.
c. The functional state of the gland prior to radiotherapy.
d. All of the above.

13. Patients with residual salivary gland function experience increased salivary flow following the initiation of all of the therapeutic strategies except which one?

a. Simple dietary measures such as eating carrots and celery, or by chewing sugarless or xylitol-containing gums.
b. Saliva substitutes
c. Pilocarpine (Salagen®)
d. Cevimeline (Evoxac®)

14. Which of the therapeutic strategies should be implemented to reduce caries activity in association with hyposalivation?

a. The daily use of a dentifrice containing 0.05% sodium fluoride.
b. The daily use of a prescription fluoride gel (1% NaF or 0.4% SnF).
c. Regular preventive care and the application of a 0.5% NaF varnish.
d. All of the above.

15. Hypersalivation usually is not reflective of a true increase in salivary flow, but of an inability to coordinate control mechanisms of orofacial musculature leading to drooling.

a. True
b. False

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