Xerostomia in Older Adults: Diagnosis and Management

Jonathan A. Ship, DMD, *Department of Oral Medicine and the Bluestone Center for Clinical Research; New York University College of Dentistry, New York, NY.*

Saliva is critically important for oral and pharyngeal health. Xerostomic complaints and salivary hypofunction are common in older adults, producing impaired nutritional intake, host defence and communication. Salivary function remains remarkably intact in healthy older persons. Systemic diseases, medications and head and neck radiotherapy for cancer account for the majority of salivary disorders in the elderly. Diagnosis of the underlying phenomenon is critical before implementing therapy. Management strategies include replacement therapies and gustatory, masticatory and pharmacological stimulants. Prevention of the oral and pharyngeal sequelae of salivary hypofunction requires a multidisciplinary approach to stomatological care.

Key words: *xerostomia, saliva, Sjögren's syndrome, salivary glands, radiotherapy.*

Introduction

Saliva plays a critical role in the preservation of oral-pharyngeal health. Complaints of a dry mouth (xerostomia) and diminished salivary output are common in older populations, which can result in impaired food and beverage intake, host defence and communication. Persistent xerostomia and salivary dysfunction can produce significant and permanent oral and pharyngeal disorders and impair a person's quality of life. Salivary function remains remarkably intact in healthy older persons, yet a plethora of systemic diseases (e.g., Sjögren's syndrome), medications (e.g., anticholinergics) and head and neck radiotherapy (e.g., for cancer) cause xerostomia, particularly in the older adults.

Epidemiology of Xerostomia

It is difficult to determine global estimates of xerostomia and salivary gland dysfunction due to limited epidemiological studies, yet it is probable that ~30% of the population older than 65 years experience these disorders.[1] Drug-induced xerostomia is the most common cause, since most older adults are taking at least one medication that causes salivary dysfunction. The prevalence of xerostomia is nearly 100% among patients with Sjögren's syndrome,[2] and head and neck radiation for the treatment of cancer causes permanent xerostomia.[3]

Salivary Gland Physiology

There are three bilateral pairs of major salivary glands—parotid, submandibular and sublingual—and hundreds of minor salivary glands that produce serous and mucous saliva. Salivary secretion occurs in response to autonomic stimulation. During the day and sleeping hours, salivary output is at a low level, and these secretions play a prominent role in protection of all oral and upper gastrointestinal tract tissues (Table 1). At mealtime, saliva output is stimulated to assist in deglutition and digestion. Without adequate salivary production, numerous oral and pharyngeal disorders can develop (Table 2).

Many older adults experience salivary gland dysfunction and complain of xerostomia.[4] Output from major salivary glands does not undergo clinically significant decrements in healthy individuals.[5] There are reports of age-related decrements in several salivary constituents, whereas other studies report age-stable production of salivary electrolytes and proteins in the absence of major medical problems and medications.

The cause of xerostomia and salivary hypofunction in older adults is most likely due to numerous systemic conditions (e.g., Sjögren's syndrome, diabetes, Alzheimer disease, Parkinson's disease, dehydration) and their treatments (medications, head and neck radiation, chemotherapy)[6] (Table 3). Older salivary glands are more vulnerable to the deleterious effects of disease, medication and radiotherapy,[7] which probably explains why the prevalence of xerostomia and salivary dysfunction increases with age.

Clinical Findings of Xerostomia and Salivary Dysfunction

Several of the common oral symptoms of dry mouth are associated with mealtime: altered taste and difficulty eat-

Table 1
Physiological Functions of Saliva

Function	Representative Components
Mucosal lubrication	Mucin
Mucosal repair	Epidermal growth factor
Food bolus formation/translocation	Mucin, water
Initial food processing	Amylase, DNase, RNase
Antimicrobial	sIgA, histatins, lactoperoxidase, lactoferrin, secretory leukocyte protease inhibitor (SLPI)
Remineralization	Statherin, proline-rich proteins
Buffering	Bicarbonate, histatins
Mediating gustation	Water

Table 2
Oral and Pharyngeal Effects of Salivary Hypofunction

Dental caries
Dry lips
Dry mouth
Dysgeusia
Dysphagia
Gingivitis
Halitosis
Mastication problems
Mucositis
Oral-pharyngeal candidiasis
Poorly fitting prostheses
Sleeping difficulty
Speech difficulty
Traumatic oral lesions

ing, chewing and swallowing, particularly dry foods and especially without drinking accompanying liquids (Table 2). Patients complain of impaired denture retention, halitosis, stomatodynia and intolerance to acidic and spicy foods.[6] These problems can lead to changes in food and fluid selection that may compromise nutritional status. They also can lead to choking, as well as an increased susceptibility to aspiration pneumonia, with consequent colonization of the lungs with gram-negative anaerobes from the gingival sulcus.[8] Night-time xerostomia is also common, since salivary output normally reaches its lowest circadian levels during sleep, and may be exacerbated by mouth breathing.

Extraoral findings of salivary hypofunction include dry and cracked lips that are frequently colonized with *Candida* species (angular cheilitis). Visible and palpable enlarged major salivary glands occur secondary to salivary infections and obstructions (e.g., bacterial parotitis, mumps, Sjögren's syndrome). A swollen parotid gland can displace the earlobe and extend inferiorly over the angle of the mandible, whereas an enlarged submandibular gland is palpated medial to the posterior-inferior border of the mandible.

There are numerous intraoral findings of salivary hypofunction. Oral mucosal surfaces (tongue, buccal mucosa, floor of the mouth, palate, posterior oral pharynx) become desiccated and easily friable. They are susceptible to developing microbial infections, the most common (particularly in older persons) being candidiasis. This fungal infection manifests itself as angular cheilitis of the lips, erythematous candidiasis beneath prostheses, and pseudomembranous candidiasis as a white plaque that can be removed from mucosal surfaces. There is decreased or absent saliva pooling in the anterior floor of the mouth.

A second frequent infection is dental caries, which is particularly common among today's older population due to the high prevalence of retained natural teeth and previously restored dental surfaces. Without sufficient saliva to restore oral pH and to regulate bacterial populations, the oral cavity becomes rapidly colonized with caries-associated microorganisms.

Edentulous and partially edentulous adults using removable prostheses have diminished denture retention, which will adversely impact chewing, swallowing, speech and nutritional intake. Desiccated and friable oral mucosal tissues are more likely to develop traumatic lesions, particularly in denture-wearing adults. The subsequent speech and eating difficulties that develop can impair social interactions and may cause some patients to avoid social engagements.

Pathogenesis of Salivary Dysfunction

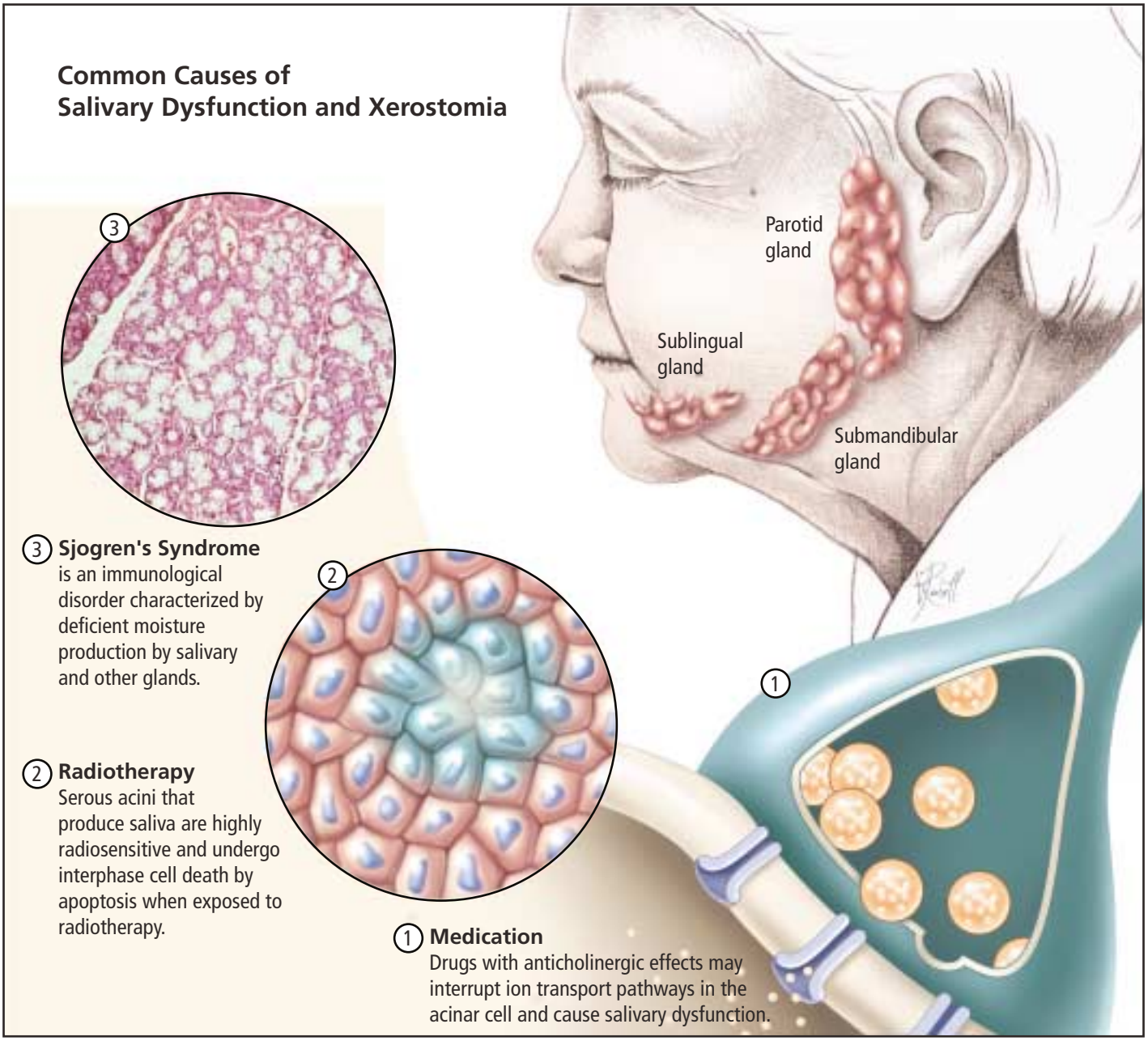
Medications

The most common causes of salivary disorders are prescription and non-prescription medications. For example, 80% of the most commonly prescribed medications have been reported to cause xerostomia, with over 400 medications associated with salivary gland dysfunction as an adverse side effect.[9] With the increased intake of prescription medications in older adults, many of which cause salivary gland dysfunction, there is a large prevalence of medication-induced xerostomia in this population.[10]

The most common types of medications that cause salivary dysfunction have anticholinergic effects. However, any drug that inhibits neurotransmitter binding to salivary gland membrane receptors, or that perturb ion transport pathways in the acinar cell, may adversely affect the quality and quantity of salivary output. These

Table 3
Etiology of Xerostomia in the Elderly

Condition	Examples
Medications	Anticholinergics, tricyclic antidepressants, sedatives, tranquilizers, antihistamines, antihypertensives, cytotoxic agents, anti-Parkinsonian drugs, anti-seizure drugs, skeletal muscle relaxants
Oral diseases	Acute and chronic parotitis, sialolith, mucocele, partial/complete salivary obstruction
Systemic diseases	Mumps, Sjögren's syndrome, diabetes, HIV/AIDS, scleroderma, sarcoidosis, lupus, Alzheimer disease, dehydration, graft versus host disease
Head and neck radiotherapy	



medications include tricyclic antidepressants, sedatives and tranquilizers, antihistamines, antihypertensives (alpha- and beta-blockers, diuretics, calcium channel blockers, angiotensin-converting enzyme inhibitors), cytotoxic agents, and anti-Parkinsonian and anti-seizure drugs.

Chemotherapy also has been associated with salivary disorders that occur during and immediately after treatment.[11] Most patients experience return of salivary function to pre-chemotherapy levels, yet long-term changes have been reported.[12] Finally, radioactive iodine (I-131) used in the treatment of thyroid tumours may cause parotid but not submandibular dysfunction in a dose-dependent fashion.[13]

Head and Neck Radiotherapy

Radiotherapy, a common treatment modality for head and neck cancers, causes severe and permanent salivary hypofunction with persistent complaints of xerostomia.[3] These patients frequently experience the spectrum of oral health problems described in Table 2.

The serous acini that produce saliva are considered to be highly radiosensitive, and undergo interphase cell death by apoptosis when exposed to external beam radiotherapy. Within one week of the start of irradiation (after 10 Gy have been delivered), salivary output declines by 60–90%, with later recovery only if the total dose to salivary tissue is < 25 Gy.[14] Most patients receive therapeutic dosages that exceed 60 Gy, and their salivary glands undergo atrophy and become fibrotic.

Sjögren's Syndrome

Sjögren's syndrome (SS), a systemic autoimmune disorder associated with inflammation of epithelial tissues, is the most common medical disorder associated with xerostomia and salivary dysfunc-

tion. SS occurs in primary and secondary forms. Primary SS involves salivary and lacrimal gland disorders with associated decreased production of saliva and tears. In secondary SS, the disorder occurs with other autoimmune diseases, such as rheumatoid arthritis, systemic lupus erythematosus, scleroderma, polymyositis and polyarteritis nodosa.[15] The American European Consensus Group recently revised the classification criteria for SS, and it can be used for diagnosing primary and secondary SS.[16]

Reported prevalence for primary SS varies from 0.05–4.8% of the population,[17] with approximately one million people in the U.S. estimated to have the disease. The onset of disease is often insidious, and accordingly, diagnosis may be delayed for many years. The female to male ratio has been estimated to be 9:1, although reported ratios vary considerably.

The pathogenesis of SS remains unknown.[2] It is possible that an environmental agent (e.g., virus) may trigger events in a genetically and otherwise susceptible host, resulting in the development of SS. Alternatively, decreased function of the hypothalamic-pituitary-adrenal axis may cause SS, as this also occurs in other autoimmune rheumatic diseases. Hormonal factors may influence the pathogenesis, since females with SS are far more common than males. Finally, SS has a genetic component: the prevalence of SS and autoantibodies (e.g., anti-Ro/SSA) may be higher in family members than in the general population.[2]

Typical oral findings in the SS patient with xerostomia are described above for other xerostomic patients (Table 2). Diminished tear output results in inflammation and damage in the lacrimal glands. Systemic manifestations are frequent, including synovitis, neuropathy, vasculitis and disorders of the skin, thyroid gland, urogenital system and respiratory and gastrointestinal tracts. There also is a reported

Table 4

Management of Dry Mouth-associated Problems[22]

Xerostomia-associated Problem	Management Strategy
Dental caries	– daily use of fluoridated dentifrice (0.05% sodium fluoride) – daily use of prescription fluoride gel (1.0% sodium fluoride, 0.4% stannous fluoride) – application of 0.5% sodium fluoride varnish to teeth – dental examinations at least every six months and intraoral radiographs for early diagnosis every 12 months
Dry mouth	– sugarless gums, mints, lozenges – artificial salivary replacements – prescription sialogogues: pilocarpine 5mg t.i.d. and q.h.s.; cevimeline 30mg t.i.d. – lubricants on lips q2h – bed-side humidifier during sleeping hours
Dysgeusia	– use of fluids during eating
Dysphagia	– careful eating with fluids – copious use of fluids during meals – avoid dry, hard, sticky and difficult-to-masticate foods
Oral candidiasis	– antifungal rinses: Nystatin oral suspension 100,000 units/mL, rinse q.i.d. – antifungal ointments: Nystatin ointment applied q.i.d. – antifungal lozenges dissolved in mouth q.i.d.: Nystatin pastilles 200,000 units; clotrimazole troches 10mg; nystatin vaginal suppositories – denture antifungal treatment: daily hygiene, soak prosthesis for 30 min in benzoic acid, 0.12% chlorhexidine, or 1% sodium hypochlorite
Bacterial infections	– systemic antibiotics x 10 days: amoxicillin with clavulanate 500mg q8h; clindamycin 300mg t.i.d.; cephalexin 500mg q6h – increase hydration – salivary stimulation with sugarless gums, mints, lozenges
Poorly fitting prostheses	– soft and hard-tissue relines by dentist – denture adhesives

44-fold increase in the frequency of B-cell lymphomas among SS patients.[18] Laboratory tests will frequently be positive for rheumatoid factor (90%), anti-Ro/SSA or anti-La/SSB (50–90%), with the presence of hypergammaglobulinemia.[19] Antinuclear antibodies are present in ~80% of cases. Autoantibodies that precipitate anti-Ro/SSA are associated with systemic manifestations, including anemia, leukopenia, thrombocytopenia, purpura, cryoglobulinemia, hypocomplementemia, lymphadenopathy and vasculitis.

Management of Xerostomia

The initial step in the management of xerostomia is the establishment of a diagnosis. This frequently involves a multidisciplinary team of healthcare practitioners within which communication is critical, since many older persons have concomitant medical problems and the frequent complications of polypharmacy. The second step is scheduling frequent dental evaluations due to the high prevalence of oral complications.[20] Low-sugar diet, daily topical fluoride use and anti-microbial mouth rinses are critical to help prevent dental caries (Table 4). Dry mucosal surfaces and dysphagia are managed with oral moisturizers and lubricants, artificial salivas and nighttime use of bedside humidifiers. Patients must be instructed on the frequent use of fluids during eating, particularly with dry and rough foods.

If there are remaining viable salivary glands, stimulation techniques are helpful. Sugar-free chewing gum, candies and mints can stimulate salivary output. Two secretagogues, pilocarpine and cevimeline, have been approved by the FDA for the treatment of xerostomia and salivary hypofunction; they are effective for increasing secretions and diminish xerostomic complaints in patients with sufficient exocrine tissue. 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.

Oral candidiasis, as previously mentioned, is a frequent complication of dry mouth and is most commonly treated with topical antifungal agents[21] (Table 4). Oral rinses, ointments, pastilles and troches are effective for most forms of oral candidiasis, and systemic antifungal therapy (e.g., ketoconazole, fluconazole) should be reserved for refractory disease and immunocompromised patients. Dentures may harbour fungal infections and thus require immersion in solutions containing benzoic acid, 0.12% chlorhexidine or 1% sodium hypochlorite. Daily denture hygiene and topical antifungal ointment also is helpful. Angular cheilitis should be treated with a combination of antifungal and anti-inflammatory agents.

Instead of prescribing xerostomia-associated drugs, substitution with similar types of medications that have fewer xerostomic side effects is preferred. For example, selective serotonin re-uptake inhibitors have been reported to cause less dry mouth than tricyclic antidepressants. If anticholinergic medications can be taken during the daytime, nocturnal xerostomia can be diminished since salivary output is lowest at night.[9] Furthermore, if drug dosages can be divided, unwanted side effects from a large single dose may be avoided. Scrutiny of drug side effects can assist in diminishing the xerostomic potential of many pharmaceuticals used by the older population. ◆

No competing financial interests declared.

References

1. Ship JA, Pillemer SR, Baum BJ. Xerostomia and the geriatric patient. J Am Geriatr Soc 2002;50:535-43.
2. Fox RI, Stern M, Michelson P. Update in Sjogren syndrome. Curr Opin Rheum 2000;12:391-8.
3. Henson BS, Inglehart MR, Eisbruch A, et al. Preserved salivary output and xerostomia-related quality of life in head and neck cancer patients receiving parotid-sparing radiotherapy. Oral Oncol 2001;37:84-93.
4. Bergdahl M. Salivary flow and oral complaints in adult dental patients. Comm Dent Oral Epidemiol 2000;28:59-66.
5. Ship JA, Nolan N, Puckett S. Longitudinal analysis of parotid and submandibular salivary flow rates in healthy, different aged adults. J Gerontol Med Sci 1995;50A:M285-M289.
6. Atkinson JC, Wu A. Salivary gland dysfunction: causes, symptoms, treatment. J Amer Dent Assoc 1994;125:409-16.
7. Ghezzi EM, Wagner-Lange LA, Schork MA, et al. Longitudinal influence of age, menopause, hormone replacement therapy, and other medications on parotid flow rates in healthy women. J Gerontol Med Sci 2000;55A:M34-M42.
8. Loesche WJ, Bromberg J, Terpenning MS, et al. Xerostomia, xerogenic medications and food avoidances in selected geriatric groups. J Amer Geriatr Soc 1995;43:401-7.
9. Sreebny LM, Schwartz SS. A reference guide to drugs and dry mouth--2nd edition. Gerodontology 1997;14:33-47.
10. Narhi TO. Prevalence of subjective feelings of dry mouth in the elderly. J Dent Res 1994;73:20-5.
11. Epstein JB, Tsang AH, Warkentin D, et al. The role of salivary function in modulating chemotherapy-induced oropharyngeal mucositis: A review of the literature. Oral Surg Oral Med Oral Pathol Oral Radiol Endo 2002;94:39-44.
12. Meurman JH, Laine P, Lindqvist C, et al. Five-year follow-up study of saliva, mutans streptococci, lactobacilli and yeast counts in lymphoma patients. Oral Oncol 1997;33:439-43.
13. Allweiss P, Braunstein GD, Katz A, et al. Sialadenitis following I-131 therapy for thyroid carcinoma: concise communication. J Nucl Med 1984;25:755-8.
14. Eisbruch A, Ten Haken RK, Kim HM, et al. Dose, volume, and function relationships in parotid salivary glands following conformal and intensity modulated irradiation of head and neck cancer. Int J Rad Oncol Biol Phys 1999;45:577-87.
15. Bloch KJ, Buchanan WW, Wohl MJ, et al. Sjogren's syndrome. A clinical, pathological, and serological study of sixty-two cases. 1965. Medicine 1992;71:386-401.
16. Vitali C, Bombardieri S, Jonsson R, et al. Classification criteria for Sjogren's syndrome: a revised version of the European criteria proposed by the American-European Consensus Group. Ann Rheum Dis 2002;61:554-8.
17. Pillemer SR, Matteson EL, Jacobsson LT, et al. Incidence of physician-diagnosed primary Sjogren syndrome in residents of Olmsted County, Minnesota. Mayo Clin Proc 2001;76:593-9.
18. Kassan SS, Thomas TL, Moutsopoulos HM, et al. Increased risk of lymphoma in sicca syndrome. Ann Int Med 1978;89:888-92.
19. Bell M, Askari A, Bookman A, et al. Sjogren's syndrome: a critical review of clinical management. J Rheum 1999;26:2051-61.
20. Fox PC. Management of dry mouth. Dent Clin N Amer 1997;41:863-76.
21. Shay K, Truhlar MR, Renner RP. Oropharyngeal candidosis in the older patient. J Amer Geriatr Soc 1997;45:863-70.
22. Ship JA. Diagnosing, managing, and preventing salivary gland disorders. Oral Dis 2002;8:77-89.