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Salivary Hypofunction and Xerostomia: Diagnosis and Treatment

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Saliva plays a central role in the maintenance of oral homeostasis. The complex mixture of proteins, glycoproteins, mucins, and ions helps prevent dental caries, promotes remineralization of early carious lesions, buffers acids generated by oral bacteria, and prevents other types of oral infections [1]. Proteins such as salivary peroxidase, lysozyme, and lactoferrin are antibacterial and limit the growth of cariogenic bacteria. The film of salivary mucins on the teeth and mucosal surfaces is believed to protect these oral structures from wear. Histatins, a family of salivary proteins, have potent antifungal properties that limit the growth of oral yeast. These salivary components, in conjunction with the mucosal tissues, form part of the innate immune system that continually protects the human body from infection. The oral cavity also is protected by secretory immunoglobulins A and M, which are produced locally by B cells within the salivary glands. These antibodies include those with specificity against oral cariogenic bacteria. Other evidence suggests that saliva may be important in protecting and healing the esophagus. It neutralizes acid that protects the esophagus from damage after gastric reflux and contains growth factors that could stimulate epithelial growth to promote healing [2].

When salivary volume is reduced significantly, patients are at risk for serious oral complications. Several clinical studies have demonstrated that caries and salivary flow rates are associated. Reports that root caries rates

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are inversely proportional to flow rates and to the use of medications with xerostomic effects [3,4] and that adults with a whole stimulated salivary flow of less than 1.0 mL/min were more likely to lose teeth [5] provide evidence that saliva is crucial to the prevention of dental caries. However, an evidence-based review published in 2001 concluded that no evidence linked a "missing salivary element" to the development of caries in humans [6]. The only clear association between caries and saliva was between the amount of caries and the volume of salivary flow.

Another sequela of low salivary flow rates is an increase in oral infections such as candidiasis. Saliva contains histatins, a family of low molecular weight proteins that both inhibit and kill candidal organisms. Lysozyme also contributes to the antifungal properties of saliva, and recent evidence suggests that saliva collected from elders has a reduced ability to inhibit attachment of *Candida* to surfaces [7]. Fungal load in the mouth is inversely proportional to stimulated salivary gland flow rates [8,9], and overt candidal infections occur more often in patients with decreased salivary flow [10].

Saliva participates in the formation of the initial food bolus, begins digestion, and aids the swallowing process. Other complications associated with severe reductions in salivary flow rates include swallowing difficulties [11], decreases or alterations in taste [12], and aggravation of conditions such as gastroesophageal reflux disease [13]. Decreases in taste and difficulties in swallowing can force elders to alter food choices [14].

Prevalence of xerostomia and decreased salivary flow in elders

Many elder groups have been studied to determine their frequency of dry mouth complaints and their salivary flow rates. To appreciate these studies, it is first necessary to understand two related concepts. First, complaints of dry mouth (or xerostomia) may not reflect reduced salivary function and may instead reflect dehydration or other systemic conditions. Therefore, studies that only examine the complaints of dry mouth will not reflect the true risk for oral diseases in the population. Second, it is very difficult to determine values for normal salivary function, because normal values vary considerably, and large patient groups must be compared to make meaningful conclusions about changes in salivary flow rates.

Several healthy patient cohorts were examined in the 1980s and 1990s to determine whether salivary gland function declined with age. Some studies suggest that there are small declines in the salivary function of women with age [15], whereas earlier studies found no effect [16]. Although salivary function generally is conserved in healthy, unmedicated persons aged more than 65 years, these individuals constitute a very small percentage of this age group. Other studies suggest that salivary flow rates in elders are related to the number of medications they take on a regular basis [17,18], the number of systemic disorders they report, and the length of time for which they consume the drugs [19].

Medications and salivary hypofunction

Medication use is believed to be the most common reason for reduction of salivary flow in older individuals. This effect is primarily mediated through medications' anticholinergic actions or their effect on fluid balance. Many diuretics, antihypertensives, antihistamines, sedatives, opioid analgesics, tricyclic antidepressives, and major antipsychotics will reduce flow. It is estimated that 50% of the noninstitutionalized adult population in the United States takes at least one prescription medication, and those using the highest number of medications are women aged 65 years or older [20]. In one study of women aged more than 65 years, 12% took at least 10 medications, and 23% took at least five prescription drugs. Polypharmacy in elders increases the risk for adverse side effects [21], including oral dryness. Evidence that medication usage by the aged increased during the 1990s comes from a Finnish study [22] that assessed changes in medication use among community-dwelling persons aged 64 years or older in 1990 to 1991 (n = 1131) and 1998 to 1999 (n = 1197). Among those surveyed, 78% in 1990 to 1991 and 88% in 1998 to 1999 used prescription drugs. The number of medications per person increased from 3.1 to 3.8, and polypharmacy (concomitant use of more than five medications) increased from 19% to 25%. Other evidence suggests that the salivary glands of older persons may be more susceptible to medications with anticholinergic effects and that there is a reduced "reserve" in the glands of older individuals [23,24].

Several studies of nursing home patients have documented a high incidence (up to 63%) of dry mouth complaints and decreased salivary flow rates in a large percentage of the residents (Table 1) [14,25–27]. Xerostomia is also a frequent complaint of the community-dwelling population over 65 years of age, with an estimated prevalence of 11% to 57% (see Table 1) [11,17,28–32]. Both salivary flow rates and complaints of oral dryness are related to medication use in the elderly (see Table 1). Data from these studies also suggest that elderly women may have more dryness complaints than men, that oral dryness causes swallowing and chewing difficulties, and that tooth loss is related to salivary flow rates.

Autoimmune diseases

Sjögren's syndrome (SS) is an autoimmune exocrinopathy that primarily affects salivary and lacrimal glands. Although case reports appeared in the literature before 1930, the syndrome is named for the Swedish ophthalmologist Henrik Sjögren, who first described a group of women with xerostomia, rheumatoid arthritis, and a type of eye dryness termed keratoconjunctivitis sicca. Further studies by Bloch et al [33] defined primary and secondary forms. Patients may have primary Sjögren's syndrome (only salivary and lacrimal gland involvement) or salivary or lacrimal gland dysfunction in association with another major connective

Table 1
Representative studies of xerostomia complaints and salivary flow rates in elders

Author	Residence of subjects: hospital, resident facilities, or community dwelling	Number of elderly subjects	Findings
Thorelius et al (1988) [25]	Resident facilities	149	11% had very low stimulated whole flow; number of drugs taken inversely correlated with flow; women more likely to have dryness complaints.
Handelman et al (1989) [26]	Resident facilities	157	61% reported oral dryness; number of drugs taken inversely correlated with stimulated whole salivary flows.
Loesche (1995) [14]	Hospital, resident, and community dwelling	123 resident homes or community; 218 outpatient controls; 132 long-term VA facilities; 81 hospitalized	Stimulated flow rates lower in those complaining of oral dryness; 55% using at least one xerogenic medication; 86% xerogenic drug use by those in long-term facilities. Patients with xerostomia had more difficulty chewing and swallowing and avoided certain foods. Ipratropium, oxybutynin, triazolam, amitriptyline, and tropical triamcinolone were related to oral dryness.
Pajukoski et al (2001) [27]	Hospital and community dwelling	175 hospitalized; 252 community-dwelling	63% of hospitalized subjects had oral dryness versus 57% of community-dwelling subjects. In all patients, psychiatric drug use was the strongest predictive factor for dry mouth.

Österberg et al (1984) [28]	Community dwelling	973 for interview; subset of 58 men and 51 women for salivary flow rates	Women more likely to have dryness complaints; dryness complaints related to number of medications taken and to consumption of diuretics and anticholinergic medications. Salivary flow rates negatively correlated to tooth loss.
Ben-Aryeh et al (1985) [29]	Community dwelling	259	28% had oral dryness; anticholinergic medications, sympatholytic agents, and diuretics used more frequently by those with oral dryness.
Sreebny et al (1988) [17]	Outpatients	185 subjects aged 55 or older (subset of 529)	Oral dryness related to number of medications taken; 29% of entire group had complaints of oral dryness.
Gilbert et al (1993) [30]	Community dwelling	600	39% had oral dryness complaints; dryness complaints associated with xerogenic medication use; 10% of those with dryness complaints had difficulty swallowing.
Locker (1995) [32]	Community dwelling	Longitudinal study of 611 patients aged 50 or older	Oral dryness complaints in the group increased from 15% to 29.5% in 3 years. Those reporting poor health were more likely to have oral dryness. Onset of oral dryness and eating complaints may be associated.

tissue disease, such as rheumatoid arthritis, systemic lupus erythematosus, scleroderma, or primary biliary cirrhosis. Most academic health centers have adopted the modified European criteria for the diagnosis of Sjögren's syndrome [34], and estimates of its prevalence range between 0.5% and 1.5% worldwide [35].

The hallmarks of Sjögren's syndrome are intense, activated plasmolymphocytic infiltrates of the lacrimal and salivary glands [36], high-titered autoantibodies, hypergammaglobulinemia, and a loss of secretory function by the salivary and lacrimal glands [37]. The loss of saliva and tears can produce severe oral and ocular dryness and associated diseases. Other patient complaints include difficulty in eating, speaking, and swallowing and burning or itching of the eyes and mouth. Over 90% of patients with primary SS are female, often in their fifth decade of life when diagnosed. However, it is not uncommon to diagnose the syndrome in children, young women, and patients aged 65 to 80 years.

Primary SS is a systemic disease [37]. Infiltrating lymphocytes can compromise many organ systems, producing what are known as extraglandular manifestations of SS. Most serious is malignant lymphoma, which is estimated to occur 20 to 30 times more frequently in these patients. A longitudinal study of patients with primary SS found that those with low complement fraction 4 in their serum had a significantly increased mortality [37]. Other studies in recent years have examined quality of life issues in SS patients. Fatigue is a common complaint, as well as an increased incidence of depressed mood, reduced sense of well-being, and impaired vitality [38,39]. Treatment for Sjögren's syndrome consists of preventive and palliative treatments (see further discussion). Unfortunately, clinical trials with systemic anti-inflammatory agents and other immunosuppressives have not been successful to date in this patient group, but several trials are ongoing. Topical anti-inflammatory agents, such as cyclosporine eye preparations and an alpha-interferon oral lozenge [40], have had some positive results in clinical trials.

Radiation therapy

It is estimated that 28,000 patients per year in the United States have high-dose, external beam radiation to eradicate a tumor of the head and neck region. New cases are concentrated in patients aged 50 years or older. Approximately 90% of the tumors are squamous cell carcinomas, and treatment consists of surgery or external beam radiotherapy. Unfortunately, the salivary gland tissue that is included in the field of radiation suffers a permanent loss of function, usually within the first 2 weeks of fractionated radiation treatment [41]. The reasons for the high radio-sensitivity of salivary tissues are not well understood, but at present the best option for treatment is to avoid irradiating some salivary tissue (see later discussion of treatment). The known mechanisms of radiation damage and other

treatment issues for the head and neck radiation patient were recently reviewed [41]. The internal radiation agent I_{131} (radioactive iodine) also can damage salivary tissue, although usually not to the same extent as external radiation [42].

Other diseases

Several other diseases have been associated with reduced salivary function or xerostomia. These include hypertension (both treated and untreated), diabetes, depression, and dementia of the Alzheimer's type.

Hypertension was associated with decreased flow rates and complaints of oral dryness in very early studies. It was hypothesized that the salivary glands of individuals with hypertension received inadequate parasympathetic stimulation [43]. Recent studies suggest that medications, rather than hypertension, depress salivary flow in hypertensive individuals [44,45], but these studies were conducted with small patient groups. The antisalivation properties of clonidine have been studied in clinical trials [45], whereas beta-blocking agents alter the protein composition of saliva [46]. Antihypertensive agents such as diuretics are frequently associated with complaints of oral dryness (see Table 1).

Patients with poorly controlled diabetes were found to have decreased salivary flow rates in a study by Chavez et al [47]. Patients with dementia of the Alzheimer's type [48] and untreated depression also are reported to have decreased salivary flow [49].

Treatment

Treatments for salivary gland diseases vary according to their cause, and potential treatments for diseased or nonfunctional salivary glands are active areas of research (Box 1). However, many recommended treatments have not been tested in well-designed clinical trials [50]. Currently available treatments for salivary gland hypofunction and xerostomia can be classified into four major categories: (1) prevention, (2) symptomatic treatment, (3) local or topical salivary stimulants, and (4) systemic therapies.

Prevention

Preventive measures must be emphasized with every patient who has decreased salivary function. Frequent dental examinations are essential. For patients receiving radiation therapy, strategies are available to limit salivary gland exposure. Radiation stents can be fabricated to shield the ipsilateral side when unilateral radiation treatment is required. Another method of limiting radiation to salivary glands is conformal and intensity-modulated irradiation (IMRT). This technique, first reported by Eisbruch et al [51], targets the lesion while sparing the major salivary glands from radiation. After 1 year, patients treated using IMRT had fewer xerostomia complaints,

Box 1. Guidelines for treatment of patients with decreased salivary function

I. Establish the cause of the decreased salivary flow rate.
- a. Does the patient have Sjögren's syndrome?
- b. Is the patient taking multiple medications daily (more than five) or a medication with significant anticholinergic effects?
- c. Is there a history of internal or external radiation treatment?
- d. Does the patient have multiple systemic illnesses?

II. Take preventive measures.
- a. Remind the patient that the use of sugared products throughout the day to stimulate flow will significantly increase dental caries.
- b. Prescribe a supplemental fluoride that is appropriate for patient's caries risk.
- c. Recall the patient every 3–4 months until caries control is achieved.
- d. Discuss with the physician the feasibility of altering the type of medication to one with fewer anticholinergic effects or of changing the time of day the medication is taken.
- e. Discuss with the patient's oncologist the possibility of using radiology techniques that spare one or more salivary glands from radiation.

III. Administer symptomatic treatment.
- a. Encourage the patient to sip water throughout the day.
- b. Recommend salivary substitutes or coating agents.
- c. Recommend humidifying the environment.

IV. Promote salivary stimulation.
- a. Advise the patient to use *sugarless* candies, mints, or gum to stimulate flow.
- b. Prescribe pilocarpine or cevimeline for 2 months if not medically contraindicated. Discontinue if there is no improvement of signs or symptoms after 2 months.

V. Plan restorative treatment.
- a. Early diagnosis of caries and intervention is essential.
- b. Select a direct resin restorative material based on its mechanical and fluoride-releasing properties.
- c. Make every effort to ''do no harm.'' In some cases, it is acceptable to do less rather than more.
- d. Before providing extensive restorative treatment, determine that the patient can maintain it.

a higher quality of life, and less loss of total parotid gland function than patients treated with conventional radiotherapy [51,52].

Amifostine is an oxygen scavenger that may protect salivary glands from free-radical damage during radiation therapy. It has a broad spectrum of cyto-protective and radio-protective functions. Amifostine is reported to protect the salivary glands and reduce xerostomia during head and neck radiation therapy. However, it requires intravenous drug administration before each radiation treatment and has associated side effects [53–56].

Patients with reduced salivary flow also have an increased incidence of oral fungal infections and salivary gland infections. Sugarless antifungal agents such as nystatin powder and clotrimazole vaginal troches can be used to treat infections without increasing the caries risk. In addition, any intraoral acrylic prosthesis used by an infected patient must be soaked in an antifungal agent. Patients should be encouraged to maintain an adequate fluid intake and remain hydrated to prevent bacterial infections of the glands. Milking the salivary glands daily by gentle massage, sucking on sugarless candies, and wiping the oral cavity with glycerine swabs will help prevent mucous plug formation and salivary gland infections.

Modification of a patient's medication regimen can reduce the degree of medication-induced dryness complaints. Substituting different medications with fewer anticholinergic effects can reduce oral dryness. For example, patients report fewer side effects with the medication donepezil than with other medications used to treat memory loss associated with Alzheimer's disease [57]. Serotonin-specific reuptake inhibitors are reported to cause xerostomia less frequently than do tricyclic antidepressants [58,59]. Olanzapine is an antipsychotic medication that has markedly reduced xerostomia side effects but similar efficacy when compared with chlorpromazine [60]. Another method of reducing xerostomia is to alter the time of day when medications are taken. Salivary flow declines at night. Taking a medication that reduces flow and causes xerostomia in the morning or dividing medication doses when possible may improve oral comfort.

Symptomatic treatment

Water is the most important treatment for symptoms of dry mouth. Sipping water throughout the day keeps the oral mucosa hydrated and clears debris from the mouth. Sipping water during meals aids in chewing, swallowing, and taste perception. Caffeine-containing beverages should be avoided. The geriatric population is more susceptible to dehydration and should be reminded to drink water on a regular basis. Using a room humidifier increases environmental humidity and may improve patients' oral comfort, resulting in more restful sleep.

Many over-the-counter products are available for the symptomatic relief of oral dryness. Patients should be advised not to use products containing alcohol or strong flavors, which may irritate the mucosa. Patients should

avoid sugar-containing products because of their increased susceptibility to dental caries. Artificial salivas can provide some relief for patients with low salivary gland function. Most available in the United States contain carboxymethylcellulose, whereas mucin-based products are available in Europe. Saliva substitutes and the oral lubricant Oralbalance (Biotene, Rancho Dominguez, California) [61] may increase patient comfort, but these substitutes have not been shown to reduce the risk for caries or other oral infections associated with reduced salivary output.

Salivary stimulation with local or topical regimens

Sugar-free candies, gums, and mints can stimulate salivary flow. The combination of chewing and taste can provide significant relief for patients who have some remaining salivary gland function. Xylitol is a low-calorie sugar product in gums and mints that suppresses growth of cariogenic streptococci and reduces caries [62]. Electrical stimulation has been used as a treatment for xerostomia. Low-voltage electrical stimulation was shown many years ago to increase salivary output, but only limited evidence of its efficacy exists. Recently, Domingo [63] investigated the effects of electrostimulation with a hand-held transcutaneous electrical nerve stimulator and found that the unit improved parotid salivary flow in 6 of 18 patients.

Systemic stimulation

Although several agents have been proposed as systemic sialogogues to treat salivary gland dysfunction and xerostomia, most have not been tested in randomized clinical trials with objective measures of salivary function. Only two secretagogues, pilocarpine and cevimeline, have been approved by the US Food and Drug Administration.

Pilocarpine hydrochloride is derived from the *Pilocarpus jaborandi* plant. Field workers in Brazil would chew this plant while working to increase salivary flow, prompting Coutinho, a Brazilian physician, to suggest *P jaborandi* as a treatment for dry mouth. Pilocarpine is a parasympathetic agent that functions as a nonspecific muscarinic agonist with mild beta-adrenergic activity. This alkaloid causes pharmacologic stimulation of exocrine glands. Pilocarpine acts by stimulating functioning salivary gland tissue; consequently, patients with little functioning salivary gland parenchyma may have no improvement of symptoms with its use. However, patients with severe salivary gland destruction may still report improvement of symptoms with the medication. Therefore, a 2-month trial of pilocarpine is recommended. If the patient does not feel any improvement in xerostomia or the physician sees no improvement in the clinical signs of salivary hypofunction, it should be discontinued.

Pilocarpine is the most widely studied systemic sialogogue. In 1991, Fox et al [64] published a double-blind, placebo-controlled study of 39 patients with salivary hypofunction (mostly from SS and postradiotherapy to the

head and neck). A dose of 5 mg three times a day reduced complaints of oral dryness and increased unstimulated flow rates. Multicenter trials found similar results for patients after head and neck radiation and those with SS [65]. Gotrick et al [66] found pilocarpine effective in the treatment of opioid-induced xerostomia, suggesting that it may be beneficial for the treatment of some medication-induced oral dryness.

The usual oral dosage for pilocarpine is 5 to 10 mg three times per day. The initial recommended dose is 5 mg three times per day, which can be increased up to 30 mg/d depending on response and tolerance. The onset of action is 30 minutes, and the duration of action is approximately 2 to 3 hours. Common side effects include gastrointestinal upset, sweating, tachycardia, bradycardia, increased pulmonary secretions, increased smooth muscle tone, and blurred vision. Contraindications include gall bladder disease, angle closure glaucoma, and renal colic. Risk to the patient must be considered when administering to patients with heart disease, asthma, angina pectoris, chronic bronchitis, chronic obstructive pulmonary disease, or a history of myocardial infarction. Pilocarpine may interact with various medications, including beta adrenergic antagonists and other parasympathomimetic drugs, and could antagonize the therapeutic anticholinergic effects of medications such as oxybutynin.

Cevimeline is a cholinergic agonist with selectivity for two of the five known muscarinic receptors. Studies suggest that these receptors are the primary mediators of salivation. In 2000, the US Food and Drug Administration approved the drug for the treatment of autoimmune-associated xerostomia. The effectiveness of this medication is limited in that it only stimulates remaining functioning salivary gland tissue. Unlike pilocarpine, which is a nonselective muscarinic agonist, cevimeline selectively binds to the M1 and M3 receptors [67]. Although fewer cardiac and respiratory side effects should be experienced with cevimeline, those experienced in clinical trials are similar to those of pilocarpine. Cevimeline is only taken three times daily, because its duration of action is longer than that of pilocarpine [68]. The same precautions apply to use of cevimeline and pilocarpine. Patients with uncontrolled asthma, cardiac disease, or angle closure glaucoma should not take the drug.

Restorative considerations in the dry mouth patient

Two crucial principles should be observed when dealing with the restorative needs of the patient with decreased salivary flow or xerostomia. The first of these is the necessity for early diagnosis and intervention in caries development [69]. Any patient at high risk for the development of dental caries requires frequent recall. Patients should be maintained at a 3-month recall frequency until the level of caries risk and activity falls to moderate and then placed on 6-month recall until the risk falls to a low level. At recall appointments, dental hard tissues should be examined closely for

primary and secondary caries in a dry field, using magnification and caries detection dyes where appropriate.

The rate of caries development in even the highest-risk patient will usually allow for some monitoring of early smooth-surface nonproximal lesions. Where patient compliance is good and the lesion is visible, accessible, in enamel, and noncavitated, an arresting treatment with a concentrated fluoride varnish (such as 5% sodium fluoride containing fluoride at 25,000 ppm) and monitoring at 3-month recall is a viable option [70]. If cavitation, even superficial, is detected or suspected or the lesion is proximal or in a high-risk area of the tooth, plaque entrapment and progression should be assumed in the dry mouth patient [71]. Restoration with a fluoride-releasing restorative material is the preferred treatment [72].

The second principle to apply to the restorative needs of the patient with complaints of oral dryness is "do no harm." Where restorative treatment is necessary to restore carious tooth structure, careful selection of the technique and restorative material is extremely important. Conservative cavity preparation techniques and adhesive material systems should be used wherever possible to avoid unnecessary removal of sound tooth structure and achieve retention. Several aspects of each restorative material should be considered before selection: retentive mechanism, tendency to protect remaining tooth structure, prevention of secondary caries, tendency to assist remineralization of tooth structure, longevity under functional load, and aesthetics.

Materials currently available can be divided broadly into direct and indirect materials, based on their method of clinical placement. Direct restoratives are so named because they are deformable when mixed and placed. After placement, they can be molded into an appropriate form before they set. These materials vary in their physical properties and ability to support remaining tooth structure [73]. The direct restoratives include composite resins, polyacid-modified composite resins, resin-modified glass ionomers, conventional glass ionomers, and dental amalgam. Indirect restorative materials are formed in the laboratory, and the finished restoration is luted into the preparation. Consequently, preparation of a cavity form without undercuts is required. This group of materials includes indirect composite inlays, onlays, and veneers, ceramic inlays, onlays, and veneers, and gold and porcelain-fused-to-gold full and partial veneers. Because a common path of withdrawal is needed for these materials, the preparations are far less conservative of tooth structure than those required for direct restoratives.

Keeping these factors in mind, one observes that a direct plastic restorative material is the obvious choice for the small to moderately sized carious lesion in a patient with dry mouth. Removal of tooth structure can also be kept to a minimum through the use of materials with adhesive capabilities. The fact that these restorations can be readily and inexpensively added to, modified, repaired, or replaced makes a direct restorative the

obvious choice for the dry mouth patient in whom caries activity is unpredictable or difficult to monitor. It is also wise to use direct materials until the caries risk and activity levels have stabilized.

Where extensive damage to tooth structure exists, adhesive techniques can be used in conjunction with direct and indirect materials to optimize marginal seal and minimize unnecessary removal of tooth structure for retention of the restoration. Restoration margins should be placed in areas that facilitate monitoring and cleaning whenever possible. Fluoride-releasing luting cements such as a glass ionomer, which also possess good film-thickness properties, should be used when possible with indirect restoratives [74].

When extensive treatment is provided to a dry mouth patient, it must be predetermined that the patient can maintain the restored dentition. In theory, all restorative options should be available, but, in reality, the oral and systemic health of the patient often dictates modification of treatment.

Direct restorative materials for patients with reduced salivary flow

Matching the properties of restorative materials to patients' needs for oral and systemic health is the key to prognosis. For patients with some salivary flow, the selection of fluoride-releasing restorative materials is advantageous because of their ability both to prevent secondary caries and to assist in remineralization [75–77].

Fluoride-releasing restorative materials can be classified into four major categories on the basis of their physical, chemical, and clinical properties [71]. At one end of the continuum lie the conventional (chemical set) glass ionomers; at the other, the fluoride-releasing composite resins. The polyacid-modified composite resins (compomers) are more closely aligned with resin composites, whereas resin-modified glass ionomers behave similarly to conventional glass ionomers [78].

Resin composites have superior mechanical and aesthetic properties and greater wear resistance than the other materials listed. Some resin composites have the ability to release fluoride, albeit in far smaller amounts than glass ionomer restoratives. Glass ionomers have inherent adhesion to dentin and release comparatively high amounts of fluoride (sufficient to remineralize enamel but not dentin), but their mechanical properties and wear resistance are inferior. Resin-modified glass ionomers have improved aesthetics but in other respects behave like glass ionomers and hence should not be used on load-bearing areas [78].

Compomers behave more like fluoride-releasing composite resins because they contain more resin than glass ionomers. In vitro, it has been noted that the adhesive systems used by composite resins and compomers prevent the uptake of fluoride released from the restorative material by tooth structure [79]. The clinical significance of this finding is still unknown. Fluoride released from glass ionomer restorative materials can be measured in whole

saliva in vivo, and its incorporation into tooth structure can be measured by microbiopsy techniques [80,81]. However, the fluoride release in tooth structure has been found to be localized to within 1 mm of the restoration margin [82]. Fluoride released from conventional glass ionomer restoratives has been measured over time and found to be at its highest level 1 to 2 days after placement [83]. It then drops substantially but is still detectable up to 1 year after placement. [84]. The lower salivary pH values often found in severe dry mouth patients appear to accelerate fluoride release [85]. This effect is hypothesized to result from erosion of the alumino-fluorosilicate glass particle surface.

Probably the most important feature of fluoride-releasing restorative materials in the high-caries-risk dry mouth patient is not their initial fluoride release but their capacity to be ''recharged'' with fluoride from external sources [86]. Conventional glass ionomers show the greatest fluoride ''recharge'' capacity, followed by compomers and resin-modified glass ionomers. Fluoride-releasing composite resins do not exhibit this property [87]. Fluoride may be replenished with toothpastes, rinses, or solutions. Frequent application is necessary, because fluoride release on ''recharge'' has been shown to be of short duration (approximately 1 day) [87]. Daily topical application is therefore recommended. Acidulated phosphate fluoride products should not be used, because they cause etching and degradation of the restoration surface of glass ionomers, resin-modified glass ionomers, and compomers [88]. Neutral sodium fluoride gels, pastes, and rinses are the easiest means of ''recharge'' in dry mouth patients.

Theoretically, it should be possible to control all new caries through the ''recharge'' of fluoride-releasing restorative materials. However, when the salivary pH stays below 4.5 for prolonged periods (as it can in a dry mouth patient), the potential for remineralization is inhibited. The most common fluoride products that can be applied topically for ''recharge'' are sodium fluoride, stannous fluoride, and sodium monofluorophosphate. The vehicle most commonly used is a dentifrice containing fluoride at around 1000 ppm. Neutral sodium fluoride gels containing fluoride at around 10,000 ppm or neutral sodium fluoride rinses containing fluoride from 100 to 1000 ppm can also be applied daily [89].

Summary

Salivary gland hypofunction and complaints of xerostomia are common in elderly patients, irrespective of their living situation. Medication use is frequently related to dry mouth symptoms and reductions in salivary flow rates. Patients with reduced salivary flow are at increased risk for caries, oral fungal infections, swallowing problems, and diminished or altered taste. Oral health care providers should institute aggressive preventive measures and recommend palliative care for patients with significant reduction in salivary gland function. The systemic agents pilocarpine and cevimeline may

help selected patients. Selective use of fluoride-releasing restorative materials and conservative treatment plans are recommended for this patient group.

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