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Caries Risk Assessment and Management for the Prosthodontic Patient

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Abstract

Prosthodontic patients are often at a high risk for caries, and assessing that risk prior to treatment is important. Historically, the nature of dental education and clinical practice has oriented clinicians toward recognizing and correcting the damaging effects of caries, rather than actively assessing and managing caries risk potential. New developments have led to better diagnostics and protocols for caries management, although one adapted to the specific needs of the prosthodontic patient has not been proposed. Our purpose is to outline caries risk assessment and management for the prosthodontic patient.

Treatment planning in the prosthodontic patient involves consideration of the many variables that affect the final outcome. A careful assessment of the patient's chief complaint, functional and psychological needs, relevant medical and dental history, and ability of the patient to maintain provided treatment are all important considerations in treatment planning and in establishing a prognosis. Several studies have reported that caries remains a significant risk factor that can have an impact on prognosis by limiting the longevity of prosthodontic treatment.[1-5]

Treatment planning based on risk assessment, rather than anatomic presentation, requires a shift in emphasis. Unfortunately, the nature of dental education and general clinical practice has often oriented clinicians toward simply recognizing and correcting the damaging effects of caries. Removing caries is an important therapeutic goal but does not decrease the risk of future caries.[6,7] Therefore, assessing both existing and future caries risk is important in treatment planning. Our purpose is to review how caries risk assessment of the prosthodontic patient can result in meaningful caries management and a more favorable prosthodontic outcome.

There is tremendous diversity in patients likely to seek prosthodontic care; however, the profile of the elderly patient is changing. Patients are retaining more of their dentition, living longer, and often coping with chronic diseases.[5,8] Risk factors often seen in an older population include accumulated restorative trauma to the dentition, recession and exposure of root surfaces, medication-induced xerostomia, a diet that may include frequent ingestion of carbohydrates, diminished oral hygiene because of decreased dexterity and/or motivation, and age-related cognitive impairment.[7-9]

Prosthodontic procedures often leave patients at risk for caries.[1-3] Recurrent caries on abutment teeth is a risk factor for patients with a fixed dental prosthesis (FDP) and/or a removable dental prosthesis (RDP).[6,7,9] Caries following prosthodontic procedures is a continuing risk factor because of difficulty in access for cleaning an FDP,[2] or with increased plaque accumulation and risk of caries seen with the use of an RDP.[10]

Fixed dental prostheses

A review of FPD complications by Pjetursson et al showed caries to be the second most common complication over a 5-year period, after abutment tooth devitalization.[2] A review by Goodacre et al includes 15 studies that evaluated FPD complications over an average of 8 years.[1] They reported a 27% incidence of complications, with caries the most common event, with a mean incidence of 18% on abutment teeth, 2% in post-and-core restorations, and 0.4% in single-crown restorations.[1] Reasons for increased caries activity around FDPs may include marginal gaps that create a space for plaque accumulation, resulting in increased potential for decay,[11,12] as well as difficult access for cleaning.

Removable dental prostheses

The direct effect on caries activity of wearing an RDP is controversial, and no systematic reviews have been published. Studies investigating caries activity in RDP patients are often difficult to interpret because of poor response rates to recall requests.[13,14] The high level of caries activity reported on patients wearing an RDP may be related more to continuing poor oral hygiene than the direct effects of the prosthesis. Additionally, caries risk is associated with low socioeconomic status;[5] a patient wearing a less-expensive RDP rather than a more costly FDP or implants may, therefore, be at a higher risk for caries.[5] Plaque levels, risk of caries, and risk of abutment failure have been shown to increase significantly in patients wearing an RDP.[3,4,13,15-18] Wearing an RDP significantly increases caries risk on abutment teeth,[14,17] and the risk is also higher in dentition in direct contact with an RDP[14] and in abutment teeth with recession.[3]

In a study of 1320 teeth in 137 patients over a 5-year period, Kratochvil et al showed abutment teeth were more than twice as susceptible to decay as were nonabutment teeth, which is consistent with their finding of over 60% of abutment tooth surfaces with measurable plaque.[16] Schwalm et al reported that caries lesions were found in 8.5% of abutments and that surfaces covered by any component of the RDP were at an increased risk of caries, although excellent homecare and regular dental visits decreased the risk.[14] The susceptibility of RDP abutment teeth to caries decreases when the abutment tooth is crowned,[14] but increases with gingival recession on the abutment teeth.[3] Yeung et al conducted a retrospective clinical survey in which they determined that teeth in contact with an RDP had a significantly higher plaque index and a higher gingival bleeding index.[3] They also found 8.5% of the teeth in contact with an RDP developed new carious lesions within a 5- to 6-year period and that the incidence of root caries was 3.4% for teeth in contact with an RDP.[3] In a study following 3071 teeth in 148 patients over an average 19.2 years, Miyamoto et al determined that abutments for RDPs experienced an increased risk of failure resulting in extraction (relative risk = 5.5), more than twice as high as the risk for an abutment for an FDP.[4]

Caries risk in the prosthodontic patient is high because of the general characteristics of the average prosthodontic patient: tooth loss, existing restorations, root exposure, age-associated comorbidities, and diminished skill in completing oral hygiene procedures.[4,19] Caries risk in the prosthodontic patient is also high because of the inherent risk of caries with procedures such as FDPs and/or RDPs.[2,3]

Developing a caries risk management plan for the unique characteristics of the prosthodontic patient is important, because it has been shown that risk indicators for caries vary considerably between different population groups.[20] Our purpose was to outline considerations in caries risk assessment for improved caries management in the prosthodontic patient.

Caries as a dynamic, chronic process

Dental caries results from the release of acids formed in the biofilm that covers susceptible tooth surfaces.[21,22] The so-called acidogenic biofilm bacteria ferment available carbohydrates and generate acids that can diffuse into the tooth enamel or dentin and dissolve or partially dissolve (i.e., demineralize) the carbonated hydoxyapatite tooth mineral.[21,22] If this process is not halted or reversed, a cavity forms. Dental caries is a dynamically changing but chronic process managed by mechanical removal and chemical disruption, or by neutralizing by salivary volume the effects of acids released from bacteria in the biofilm.[22,23]

There are over 500 bacterial and *Archaea* taxa in the biofilm, 50% of which are unculturable.[24] Approximately 23 bacterial strains have been identified that work synergistically to potentiate a decrease in pH and increase in risk of caries. The principal bacteria are the *Lactobacillus* species, *Streptococcus mutans*, and *Streptococcus sobrinus*, which are termed acidogenic because they produce acids from carbohydrates and aciduric because they thrive in an acid environment. *S. mutans*, and *S. sobrinus* belong to the bacterial grouping known as mutans streptococci. Additional bacteria and yeasts that produce acids, including lactic, acetic, propionic, and formic, may also contribute. All these acids can readily diffuse from the biofilm into the tooth and dissolve susceptible minerals. When these bacteria release acids they dissolve or partially dissolve mineral from crystals inside the tooth. The tooth enamel and dentin are tissues made up of crystals called a carbonated hydroxyapitite.[25,26] This is a calcium phosphate with numerous impurity inclusions, the most important of which is the carbonate ion, which makes the mineral more acid-soluble than pure hydroxyapatite. If dissolving of the mineral is not halted or reversed, the early subsurface lesion becomes a cavity. Remineralization of the tooth is possible if acid from the bacteria in the biofilm is buffered by saliva, allowing calcium and phosphate to flow back into the tooth to form new mineral on the partially dissolved subsurface crystals. The remineralized "veneer" on the surface of the individual tooth mineral crystals is much more resistant to subsequent acid attack, especially if it is formed in the presence of sufficient fluoride.[27-30]

Role of fluoride in managing caries

Fluoride's primary mechanisms of action are topical by inhibiting demineralization, enhancing remineralization, and, at high concentrations, inhibiting the metabolism of bacteria.[31-34] Caries is a dynamic process with bacterial composition, salivary content and flow, the presence of fermentable carbohydrate, and the availability of calcium, phosphate, and fluoride determining the progression or reversal of caries.[22,25,35] If the risk factors outweigh the protective factors, then caries progresses. In the reverse situation, caries is arrested or even reversed. The factors potentiating caries in the prosthodontic patient include the acidogenic bacteria, reduced salivary function by volume or content, existing restorations harboring plaque, frequent ingestion of fermentable carbohydrates reducing pH, decreased skills/compliance with oral hygiene, root exposure, and wearing an FDP or an RDP.[1-3,22] The protective factors include saliva and its numerous caries-protective components; the salivary flow rate; antibacterials, both intrinsic from saliva and extrinsic from other sources; and other factors that help raise pH and improve the potential for remineralization.[6,23,36]

In dentate individuals, there are numerous acidic daily challenges as fermentable carbohydrates are ingested, and a

struggle between the risk factors and protective factors takes place. As acid is produced by the biofilm bacteria, pH decreases, mineral dissolves, and subsequently, as the saliva neutralizes the acid, mineral is replaced. Fluoride enhances the remineralization process whether it comes from topical sources, such as drinking water, food and beverages, toothpastes/dentifrices, mouth rinses, or from higher-concentration products including fluoride varnish.[22,23]

Although fluoride is effective in reducing tooth decay, over 70% of dental services provided are replacement of existing restorations, with caries the most frequent reason.[5,37,38] There appear to be two basic reasons for this phenomenon: behavioral issues relating to lack of patient compliance with proper oral hygiene procedures and preventive measures, and the relationship between risk and protective factors. If the bacterial challenge is too high and the salivary flow too low, the beneficial effect of fluoride can be overcome by acid attack. Although fluoride helps to reduce the amount, progression, and severity of decay, fluoride alone cannot overcome a high bacterial challenge, especially if there is reduced salivary flow.[23]

Caries removal and placement of definitive restorations do not decrease the potential for future caries.[7,21,39] In a study by Featherstone et al, the levels of *mutans streptococci* and *Lactobacilli* prior to treatment and after completion of all restorations showed no statistically significant decrease in bacterial counts unless chlorhexidine was used.[21] This means that for the high-risk prosthodontic patient with high levels of cariogenic bacteria, steps to reduce bacterial loading are necessary prior to and during prosthodontic therapies. Subsequently, supplemental measures to decrease future caries activity are indicated. In summary, (1) fluoride, while effective, has limitations; (2) a high bacterial challenge cannot be completely overcome by high-concentration fluoride therapy; and (3) removing active caries and completing prosthodontic work does not reduce the patient's risk for future caries.[7,21,39]

Managing caries using diagnostics and strategies specific for the prosthodontic patient

With the emphasis on evidence-based disease management, protocols for the systematic assessment and management of caries risk have been developed.[40-42] The results of an ongoing clinical study have shown the efficacy of specific interventions,[21] and that risk indicators for caries vary among population groups.[20] Featherstone et al have developed a protocol for Caries Management by Risk Assessment (CAMBRA) as an approach for the general dental population.[9,23] CAMBRA has been validated through a 3-year randomized clinical trial at the University of California, San Francisco, dental clinics providing general restorative care.[21] In this article, we propose modifications to the original CAMBRA protocol addressing the unique characteristics of the prosthodontic patient.

Although risk assessment can be completed by the clinician, successful caries management requires patient cooperation, which is often problematic, with one study reporting a 50% noncompliance rate.[42] Despite the possibility of poor patient compliance, the need for a careful determination of caries risk and a strategy for caries management is imperative when treatment planning for many prosthodontic patients.

Table 1 Risk factors for caries based on history

(1) Existing or recent history of caries[43]
(2) No dental visit in the past 6 months[44]
(3) Limited lifetime exposure to water fluoridation[45]
(4) Brushes once a day or less[22]
(5) Saliva reducing factors (medications/radiation/systemic factors)[46]
(6) Frequent (>3 times daily) snacking between meals[4]
(7) Health behavior risk indicators (smoking, eating disorders)[47]
(8) Low socioeconomic status and/or impaired cognitive ability[5,43]
(9) Low educational level[48]

Important questions can be addressed in a health questionnaire or during the initial consultation with the patient to help determine the current and future risk for caries. Table 1 lists risk factors for caries and can be used as part of the oral history to help establish a patient's risk profile.[4-6,22,43-48]

The clinical examination of a prosthodontic patient should be comprehensive and include risk factors that may limit the success of restorative care. The clinical observations in Table 2 can be identified during a clinical exam and/or diagnostic testing procedures. These observations are among those known to be risk factors for future caries activity.[1-4,11,14,20,23,44,49,50] The most relevant protective factors to be considered based on history, clinical examination, and/or diagnostic testing are listed in Table 3 and reviewed below.

Preventive treatment plan

Involve the patient in disease control

Prosthodontic patients are often referred for treatment because of significant previous dental treatment; complex restorative needs; and high expectations for esthetic, functional, comfortable, and enduring restorative results. A satisfactory outcome is often anticipated by the prosthodontist and expected by the patient; however, the outcome of some prosthodontic services is limited in longevity, and caries is often the etiologic factor limiting success.[1-3] Because patient compliance in caries management programs is often disappointing,[42] behavioral attitudes need to be reviewed prior to treatment. Often a simple query asking how long the patient expects the proposed prosthodontic

Table 2 Risk factors for caries based on clinical exam and/or diagnostic testing

(1) Medium or high *Streptococcus mutans* and *Lactobacillus* counts[4,43]
(2) Inadequate saliva flow by observation or measurement (<0.07 ml/min stimulated)[23]
(3) Large number of filled teeth[44]
(4) Visible heavy plaque
(5) Recession with exposed roots[3]
(6) Defective restorations with open margins[11]
(7) Fixed dental prostheses[1,2]
(8) Removable dental prostheses[3,14]
(9) Deep pit and fissures[49]
(10) Noncavitated lesions[50]

Table 3 Protective factors to be considered based on history, clinical examination, and/or diagnostic testing

(1) Involving the patient in discussion of disease control[42]
(2) Brushing twice daily with fluoride toothpaste[52]
(3) Fluoride varnish use 3 times a year[51,54,76]
(4) Daily fluoride in drinking water[44]
(5) Chlorhexidine rinse daily 1 week each month[23]
(6) Xylitol gum/lozenges twice daily for 15 minutes[64,65]
(7) Amorphous calcium phosphate twice a day following use of fluoridated toothpaste[67]
(8) Stimulation of salivary flow (chewing xylitol gum, medications)[58,59]
(9) Buffering products and salivary substitutes

care to last will prompt a discussion of how caries can compromise success. This can often lead to improved patient compliance and adoption of a program to decrease future caries activity.

Recommendation: The importance of a caries reduction program during and following prosthodontic treatment should be thoroughly discussed with the patient prior to initiating treatment.

Fluoride in various forms

Fluoride in its various forms has been shown to significantly decrease caries risk.[22,23,51,52] The effect of fluoride varies depending on the patient's age, risk factors, and method and frequency of delivery as well as what fluoride products the patient uses;[53] however, the benefits of combining several forms of fluoride to reduce caries risk are generally not cumulative and can be overcome in high-risk patients.[22,54]

Toothpaste

Toothpaste containing fluoride has been shown to reduce incidence of caries by 20% to 35% depending on both dose and frequency of application.[22,54] A dose below 600 ppm will have limited benefit, but standard levels in most toothpastes (1000 to 1100 ppm) will be effective.[22] Brushing twice daily with a fluoridated toothpaste has been shown to be significantly better than brushing once a day.[52]

Recommendation: Patients should be encouraged to brush with fluoridated toothpaste a minimum of twice daily.

Varnish

In a systematic review on children and adolescents, Marinho et al evaluated fluoridated toothpaste alone or in combination with other topical fluorides, such as gels, mouthwashes, or varnishes.[54] They found in patients brushing daily with fluoridated toothpaste that the other formulations were equal in decreasing caries risk. They also determined that in patients using fluoridated toothpaste the additional caries reduction benefit of combining this procedure with a gel, mouthwash, or varnish was about 10%.[54]

Fluoride varnishes have been advocated because of the prolonged fluoride release, easy application, and lack of need for patient compliance.[51,54] In an individual not using a fluoridated toothpaste but with varnish application, the mean decrease in caries or prevented fraction[22] was 30% compared to controls without varnish applications.[51] The advantage seems to be that the varnish adheres to the tooth surface, maximizing the delivery of the fluoride for long periods of time.

Recommendation: In prosthodontic patients with gingival recession with other risk factors for caries, a fluoride varnish on root surfaces three times annually is warranted.

Chlorhexidine

Chlorhexidine digluconate (CHX) was first introduced as a disinfectant and topical antiseptic in the UK in 1954, but the beneficial effect of reducing bacterial plaque was not recognized until the 1970s.[55] The CHX mechanism of action is a strong base with cationic properties. The cationic molecules bind to the negatively charged bacteria cell walls (disrupting the osmotic balance) and weaken the bacteria over time.[56] Many clinical trials and systematic reviews have been conducted to determine the caries reduction potential of chlorhexidine.[22,57-61] In one review article, Anderson concluded there was strong support for chlorhexidine as an antimicrobial in suppressing *S. mutans* but found it less effective in suppressing *Lactobacilli*.[62] Anderson further concluded that the literature is inconclusive on the efficacy of chlorhexidine to reduce dental caries unless adjunctive fluoride is used;[57] however, CHX in combination with fluoride has been shown to be effective in reducing caries in some, but not all, adult groups who are at high risk for decay.[9,31,62] The most commonly used product is a rinse, but gels and varnishes are also available, with varnishes showing the most consistent reduction in *mutans streptococci*.[60] Because the fluoride ion is negative and chlorhexidine ion is positive, there is some interaction between the two, and they should be used at least 1 hour apart to allow each to interact with the plaque and not compete with each other.[23,63] The most common side effects of chlorhexidine rinses are staining of teeth and/or prosthesis and an adverse impact on taste. A benefit of chewing CHX-containing gum is that very little staining of teeth and prosthesis has been observed.[59]

Recommendation: Because of staining and impact on taste, chlorhexidine use should be limited to seven consecutive days a month. Recommended dosage is 10 ml of a 0.12% rinse for 1 minute at night at least 1 hour after brushing with fluoridated toothpaste and after removing any prosthesis.

Xylitol

Many artificial sweeteners, including, sorbitol, saccharin, aspartame, and xylitol, are available in a wide variety of products that replace fermentable sweeteners such as sucrose or fructose. Regular use of xylitol-containing chewing gum has been shown to help prevent decay by increasing salivary flow, raising pH after an acidic challenge from acidogenic bacteria, decreasing colony counts of *S. mutans*, decreasing the adherence of *S. mutans* to tooth structure, and enhancing remineralization of subsurface enamel lesions.[58,59,64,65] Xylitol is a so-called sugar alcohol that also has direct anticariogenic effects by decreasing viability and decreasing acid release from *S. mutans*.[65,66] A recent investigation reported that long-term chewing of xylitol gum affects *S. mutans* by altering expression of the gtfB gene, which is responsible for producing insoluble glucan on

the surface of bacterial cells that facilitates bacterial adhesion to teeth.[65]

In a systematic review, Deshpande and Jadad evaluated six studies and determined that xylitol-containing gum resulted in a caries reduction of 58% compared to not chewing gum.[64] There is a dose/response curve, and 7 to 10 g a day has been shown to be more efficacious in reducing caries activity than lower doses.[66] Potential adverse effects of xylitol include minor gastrointestinal irritation.[58]

Recommendation: Although the independent variables of unit dose, frequency of chewing, and time chewing have not been thoroughly evaluated, patients at high risk for caries should chew xylitol-containing gum twice daily for 15 minutes with a total xylitol dose of 7 to 10 g per day.[64]

Combination of xylitol and chlorhexidine

In a double-masked randomized clinical trial of 111 elderly patients, Simons et al evaluated the effect of chewing gum with xylitol (X), gum containing both chlorhexidine and xylitol (CX), and patients not chewing gum (N). Both the X and CX groups had a significant decrease in their plaque and gingival indices.[58] In a related study, the authors report that although xylitol alone was equally effective as CX in reducing plaque levels, CX was more effective than X for improving gingival indices.[59] CX use resulted in greater reductions than X in denture stomatitis (91% vs. 62%) and angular cheilitis (75% vs. 43%). All results were significantly better than in patients not chewing gum.[59]

Recommendation: Elderly partially and completely edentulous patients with caries and denture stomatitis and/or angular cheilitis would benefit from chewing gum containing both xylitol and chlorhexidine. Since the product studied by Simons et al is not currently available in the United States, the use of xylitol gum daily and 10 ml of a 0.12% rinse of chlorhexidine 1 day a week is a viable alternative.

Amorphous calcium phosphate

A product containing casein phosphopeptide-amorphous calcium phosphate (CPP-ACP) (MI paste, GC America, Alsip) is on the market, and has shown beneficial effects on reducing dental caries by remineralizing tooth structure.[67] CPP-ACP binds to the tooth surface and to bacteria in plaque, with high concentrations of calcium and phosphate ions available for remineralization of subsurface enamel lesions by diffusion of the ions back into the tooth structure. It is recommended that the paste be dispensed through the dental office and not be used by patients with milk protein allergies.

Azarpazhooh and Limeback conducted a systematic review of clinical trials using CPP-ACP complex products.[67] They identified ten caries prevention studies, of which eight were randomized clinical trials with crossover designs. Seven of the eight studies showed that CPP-ACP had caries preventive potential and the ability to remineralize the enamel subsurface; however, due to inconsistencies and potential bias in these studies, they concluded there is insufficient scientific evidence to make recommendations regarding long-term effectiveness of casein derivatives and CPP-ACP in preventing caries in vivo.[67] They also reported on the results of two studies that indicate the products may provide relief to patients suffering from xerostomia and hyposalivary function, but noted that more evidence is needed to support these statements.

Recommendation: Although studies of CCP-ACP are somewhat controversial, there appears sufficient evidence of caries reduction potential to recommend its use, especially in patients with root exposure. Application follows the use of fluoridated toothpaste twice a day using a cotton swab, a finger, or a custom tray.

Stimulation of salivary flow

Although patients may not complain about dry mouth, a thorough history and clinical examination and/or salivary testing are important because of the influence diminished salivary flow can have on diagnosis and prognosis, including high caries risk.[68,69] Normal salivary volume is approximately 1.5 l a day, but measurement varies with the time of day, body position, systemic conditions, and medications.[70] Salivary functions benefit oral health by providing buffering capacity to maintain a neutral oral pH; carrying calcium, phosphate, and fluoride ions for potential remineralization of tooth structure; containing histidine-rich peptides (the histatins) that are fungicidal and antibacterial; and providing enzymes that aid in lubricating and digesting food and mucins that lubricate the mouth.[27] Protective salivary constituents can be altered and/or diminished by a number of factors including medication-induced xerostomia, uncontrolled diabetes, head and neck radiation therapy, and autoimmune disorders such as Sjogren's syndrome. Other clinical findings that may result in diminished salivary flow are recurrent dental caries, oral yeast infections, inflamed soft tissues, chapped or cracked lips and tongue, swollen salivary glands, and difficulty swallowing or chewing.[70]

Chewing gum can stimulate increases in whole salivary flow, which can result in numerous benefits for the partially or completely edentulous patient, both by reducing the potential of caries as well as generally improving oral health.[58,59,71] Simons et al showed chewing gum stimulated whole saliva measures more than 60% compared to a control group not chewing gum.[59] Simons et al also showed nonmedicated chewing gum was helpful in decreasing food debris on dentures of frail patients and that chewing gum containing xylitol and chlorhexidine significantly decreased denture stomatitis and angular cheilitis.[59] This is consistent with another study that showed increasing salivary volume reduces the adhesion of *Candida* to acrylic resins[72] and that xylitol decreases adhesion of *S. mutans* to tooth structure.[65] Oral environments lacking adequate salivary flow support acidogenic and aciduric flora, including high levels of *Lactobacilli*.

The management of xerostomia is often palliative. A low sugar diet, daily use of topical fluoride, antimicrobial mouth rinses, xylitol gum, and avoiding mouthwashes with alcohol can help control the condition, as can sugar-free gum or candy. The use of oral moisturizers, lubricants, artificial saliva, and humidifiers at night have been reported to provide symptomatic relief of dry mouth.[73] The Food and Drug Administration has approved prescription sialogogues containing pilocarpine and cevimeline, which are parasympathomimetic-stimulating agents; however, these are contraindicated for patients with uncontrolled asthma, narrow-angle glaucoma, or acute iritis.[73]

Buffering products such as sodium bicarbonate can be used to aid in neutralizing the pH of the oral cavity, which will decrease enamel mineral loss. These can be used for patients at high risk for caries or xerostomia and those with gastric reflux or bulimia.

Saliva contains 99% water, buffering agents, and enzymes. Artificial saliva is a mixture of ions, cellulose derivatives, and flavoring agents,[36] with none of the digestive or antibacterial enzymes or other proteins present in natural saliva, but they do provide palliative relief. Products on the market include Salivart (Gebaurer, Cleveland, OH) and Optimoist (Colgate-Palmolive, New York, NY).

Recommendation: Patients with diminished saliva flow should be counseled managing the condition, including the advantages of a low-sugar diet and on possible relief provided by the products discussed above. Sipping frequently on water that contains two teaspoons of baking soda in 8 oz of water will help hydrate the mouth and neutralize bacterial acids generated in the plaque.

Other products for caries prevention

Biotene

Biotene (Los Angeles, CA) has a line of products designed for patients suffering from dry mouth. These products contain the enzymes lactoferrin, glucose oxidase, and lactoperoxides. When combined with potassium thiocyanate in saliva, hypothiocyanate, which mildly inhibits the growth of acid-producing bacteria, is formed. The different products in their line contain different ingredients. Studies performed to test the effectiveness have produced mixed results. The products are intended to mimic natural saliva, but they do not have any buffering capacity or anticaries effects. The Biotene products are effective for palliative or on an as-needed basis.

1% Iodine (10% povidone-iodine)

As a mucosal antiseptic, povidone-iodine has been used in medicine and dentistry for many years. Iodine has been shown to be microbiocidal on Gram-negative bacteria, fungi, mycobacteria, viruses, and protozoan by working directly on the microbial cell wall.[63] Iodine has been shown to be effective in controlling early childhood caries by reducing both *S. mutans* and *Lactobacillus* in children;[24] however, there is little evidence that iodine is effective in older children or adults with well-established biofilms, which may be a result of different study methodologies. The studies evaluating children were often completed in an operating room where the patients were under general anesthesia. The iodine was usually swabbed around the oral cavity and remained in contact with tooth structure for an extended period. In contrast, studies evaluating the efficacy of iodine in adults have not shown a benefit, and this may be a result of less contact time when used as a rinse, together with the existence of well-established and resistant biofilms. In addition, there are contraindications to the use of povidone-iodine such as iodine hypersensitivity, thyroid disease, and pregnancy.[24]

Recommendation: The use of iodine as an antimicrobial is not recommended since there is currently little evidence of its effectiveness in adults.

Diagnostic testing to assess caries risk in the prosthodontic patient

Saliva flow rate

Several commercial products are available to determine salivary flow rate. We are familiar with the Caries Risk Test (CRT, Ivoclar Vivadent, Amherst, NY) described below. The patient chews a paraffin pellet for 3 to 5 minutes and expectorates all saliva into a measuring cup. The amount of saliva (in milliliters) is measured and divided by the amount of time the pellet was chewed to determine the milliliter per minute of stimulated salivary flow. A flow rate of $\geq$1.0 ml/min is considered normal. A level of 0.7 ml/min is low, and anything $\leq$0.5 ml/min is dry, indicating severe salivary gland hypofunction.[23]

Bacterial testing

A number of chairside bacterial tests that provide an indication of caries risk are available.[20,23,74] The Viadent CRT bacteria product (Ivoclar) establishes a low, medium, or high cariogenic bacterial challenge.[23] It can also be used as a motivational tool for patient adherence with an antibacterial regimen. The kit includes a two-sided selective media stick that assesses S. *mutans* on the blue side and *Lactobacilli* on the green side. It has been reported that the *Lactobacilli* microbiological tests are less sensitive in predicting caries.[75]

Overall recommendation

All procedures and recommendations described above can be combined to provide CAMBRA to the prosthodontic patient. Further details of caries risk assessment and caries management in practice can be found in Featherstone et al,[23] which can be accessed online www.cdafoundation.org/journal.

Conclusions

The patient referred for prosthodontic care is often at high risk for caries. Treatment planning needs to include appropriate levels of caries risk assessment based on patient history, clinical examination, and chairside tests. Management of caries risk may include patient behavioral modification, chemical control of the biofilm, stimulation of salivary flow, and/or the supplement of constituents to favor remineralization over demineralization. Successful management of the patient's caries risk will ultimately result in more enduring prosthodontic outcomes.

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References

1. Goodacre CJ, Bernal G, Rungcharassaeng K, et al: Clinical complications in fixed prosthodontics. J Prosthet Dent 2003;90:31-41
2. Pjetursson BE, Bragger U, Lang NP, et al: Comparison of survival and complication rates of tooth-supported fixed dental

prostheses (FDPs) and implant-supported FDPs and single crowns (SCs). Clin Oral Implants Res 2007;18(Suppl 3):97-113
3. Yeung AL, Lo EC, Chow TW, et al: Oral health status of patients 5-6 years after placement of cobalt-chromium removable partial dentures. J Oral Rehabil 2000;27:183-189
4. Miyamoto T, Morgano SM, Kumagai T, et al: Treatment history of teeth in relation to the longevity of the teeth and their restorations: outcomes of teeth treated and maintained for 15 years. J Prosthet Dent 2007;97:150-156
5. Chalmers JM: Minimal intervention dentistry: part 1. Strategies for addressing the new caries challenge in older patients. J Can Dent Assoc 2006;72:427-433
6. Featherstone JDB: The caries balance: contributing factors and early detection. J Calif Dent Assoc 2003;31:129-133
7. National Institutes of Health: Diagnosis and Management of Dental Caries Throughout Life. Bethesda, MD, National Institutes of Health, 2001
8. Fontana M, Zero DT: Assessing patients' caries risk. J Am Dent Assoc 2006;137:1231-1239
9. Featherstone JDB, Adair SM, Anderson MH, et al: Caries Management by risk assessment: consensus statement, April 2002. J Calif Dent Assoc 2003;31:257-269
10. Lechner SK. A longitudinal survey of removable partial dentures. III. Tissue reactions to various denture components. Aust Dent J 1985;30:291-295
11. Totiam P, Gonzalez-Cabezas C, Fontana MR, et al: A new in vitro model to study the relationship of gap size and secondary caries. Caries Res 2007;41:467-473
12. O'Reilly MM, Featherstone JDB: De- and remineralization around orthodontic appliances: an *in vivo* study. Am J Orthod 1987;92:33-40
13. Yusof Z, Isa Z: Periodontal status of teeth in contact with denture in removable partial denture wearers. J Oral Rehabil 1994;21:77-86
14. Schwalm CA, Smith DE, Erickson JD: A clinical study of patients 1 to 2 years after placement of removable partial dentures. J Prosthet Dent 1977;38:380-391
15. Aquilino SA, Shugars DA, Bader JD, et al: Ten-year survival rates of teeth adjacent to treated and untreated posterior bounded edentulous spaces. J Prosthet Dent 2001;85:455-460
16. Kratochvil FJ, Davidson PN, Guijt J: Five-year survey of treatment with removable partial dentures. Part I. J Prosthet Dent 1982;48:237-244
17. Bergman B, Hugoson A, Olsson CO: A 25 year longitudinal study of patients treated with removable partial dentures. J Oral Rehabil 1995;22:595-599
18. Addy M, Bates JF: Plaque accumulation following the wearing of different types of removable partial dentures. J Oral Rehabil 1979;6:111-117
19. Chalmers JM: Minimal intervention dentistry: part 2. Strategies for addressing restorative challenges in older patients. J Can Dent Assoc 2006;72:435-440
20. Zero D, Fontana M, Lennon AM: Clinical applications and outcomes of using indicators of risk in caries management. J Dent Educ 2001;65:1126-1132
21. Featherstone JDB, Gansky SA, Hoover CI, et al: Cariogenic bacteria trends in a randomized caries management clinical trial. J Dent Res 2002;81(Spec Iss A):3813.
22. Llena Puy C, Forner Navarro L: Evidence concerning the medical management of caries. Med Oral Patol Oral Cir Bucal 2008;13:E325-E330
23. Featherstone JD, Domejean-Orliaguet S, Jenson L, et al: Caries risk assessment in practice for age 6 through adult. J Calif Dent Assoc 2007;35:703-707, 710-713
24. Garcia-Godoy F, Hicks MJ: Maintaining the integrity of the enamel surface: the role of dental biofilm, saliva and preventive agents in enamel demineralization and remineralization. J Am Dent Assoc 2008;139(Suppl):25S-34S
25. Featherstone JDB: The science and practice of caries prevention. J Am Dent Assoc 2000;131:887-899
26. LeGeros RZ: Crystallographic studies of the carbonate substitution in the apatite structure. Thesis, New York University, 1967
27. Lamkin MS, Oppenheim FG. Structural features of salivary function. Crit Rev Oral Biol Med 1993;4:251-259
28. Silverstone LM: The structure of carious enamel, including the early lesion. Oral Sci Rev 1973;3:100-160
29. Ten Cate JM, Duijsters PPE: Alternating demineralisation and remineralisation of artificialenamel lesions. Caries Res 1982;16:201-210
30. Featherstone JDB (ed): Clinical Aspects of De/Remineralization of Teeth: Advances in Dental Research (Vol 9). Newbury Park, CA, Sage, 1995, pp. 1-340
31. Katz S: The use of fluoride and chlorhexidine for the prevention of radiation caries. J Am Dent Assoc 1982;104:164-170
32. Featherstone JDB, Glena R, Shariati M, et al: Dependence of *in vitro* demineralization and remineralization of dental enamel on fluoride concentration. J Dent Res 1990;69:620-625
33. ten Cate JM, Featherstone JDB: Mechanistic aspects of the interactions between fluoride and dental enamel. Critl Rev Oral Biol 1991;2:283-296
34. Hamilton IR, Bowden GHW: Fluoride effects on oral bacteria. In: Fejerskov O, Ekstrand J, Burt BA (eds): Fluoride in Dentistry. Copenhagen, Munksgaard, 1996, pp. 230-251
35. Featherstone JDB: Prevention and reversal of dental caries: role of low level fluoride. Community Dent Oral Epidemiol 1999;27:31-40
36. Young DA, Featherstone JD, Roth JR, et al: Caries management by risk assessment: implementation guidelines. J Calif Dent Assoc 2007;35:799-805
37. Burke FJ, Liebler M, Eliades G, et al: Ease of use versus clinical effectiveness of restorative materials. Quintessence Int 2001;32:239-242
38. Mount GJ, Ngo H: Minimal intervention: early lesions. Quintessence Int 2000;31:535-546
39. Wright JT, Cutter GR, Dasanayake AP, et al: Effect of conventional dental restorative treatment on bacteria in saliva. Community Dent Oral Epidemiol 1992;20:138-143
40. Anusavice KJ: Efficacy of nonsurgical management of the initial caries lesion. J Dent Educ 1997;61:895-905
41. Anderson MH, Bales DJ, Omnell K-A: Modern management of dental caries: the cutting edge is not the dental burr. J Am Dent Assoc 1993;124:37-44
42. Guzman-Armstrong S, Warren JJ: Management of high caries risk and high caries activity patients: rampant caries control program (RCCP). J Dent Educ 2007;71:767-775
43. Zero DT, Fontana M, Lennon AM: Clinical applications and outcomes of using indicators of risk in caries management. J Dent Educ 2001;65:1126-1132
44. Brennan DS, Spencer AJ, Roberts-Thomson KF: Caries experience among 45-54 year olds in Adelaide, South Australia. Aust Dent J 2007;52:122-127
45. Hopcraft MS, Morgan MV: Exposure to fluoridated drinking water and dental caries experience in Australian army recruits, 1996. Community Dent Oral Epidemiol 2003;31:68-74
46. Leone CW, Oppenheim FG: Physical and chemical aspects of saliva as indicators of risk for dental caries in humans. J Dent Educ 2001;65:1054-1062

47. Roberts-Thomson K, Stewart JF: Risk indicators of caries experience among young adults. Aust Dent J 2008;53:122-127; quiz 186
48. Isokangas P, Alanen P, Tiekso J: The clinician's ability to identify caries risk subjects without saliva tests–a pilot study. Community Dent Oral Epidemiol 1993;21:8-10
49. Disney JA, Graves RC, Stamm JW, et al: The University of North Carolina Caries Risk Assessment Study. II. Baseline caries prevalence. J Public Health Dent 1990;50:178-85
50. Klock B, Krasse B: A comparison between different methods for prediction of caries activity. Scand J Dent Res 1979;87: 129-139
51. Petersson LG, Twetman S, Dahlgren H, et al: Professional fluoride varnish treatment for caries control: a systematic review of clinical trials. Acta Odontol Scand 2004;62:170-176
52. Twetman S, Axelsson S, Dahlgren H, et al: Caries-preventive effect of fluoride toothpaste: a systematic review. Acta Odontol Scand 2003;61:347-355
53. Ammari AB, Bloch-Zupan A, Ashley PF: Systematic review of studies comparing the anti-caries efficacy of children's toothpaste containg 600 ppm of fluoride or less with high fluoride toothpastes of 1,000 ppm or above. Caries Res 2003;37:85-92
54. Marinho VC, Higgins JP, Sheiham A, et al: Combinations of topical fluoride (toothpastes, mouthrinses, gels, varnishes) versus single topical fluoride for preventing dental caries in children and adolescents. Cochrane Database Syst Rev 2004;(1):CD002781
55. Loe H, Schiott CR, Karring G, et al: Two years oral use of chlorhexidine in man. I. General design and clinical effects. J Periodontal Res 1976;11:135-144
56. Greenstein G, Berman C, Jaffin R: Chlorhexidine. An adjunct to periodontal therapy. J Periodontol 1986;57:370-377
57. Anderson MH: A review of the efficacy of chlorhexidine on dental caries and the caries infection. J Calif Dent Assoc 2003;31:211-214
58. Simons D, Brailsford S, Kidd EA, et al: The effect of chlorhexidine acetate/xylitol chewing gum on the plaque and gingival indices of elderly occupants in residential homes. J Clin Periodontol 2001;28:1010-1015
59. Simons D, Brailsford SR, Kidd EA, et al: The effect of medicated chewing gums on oral health in frail older people: a 1-year clinical trial. J Am Geriatr Soc 2002;50:1348-1353
60. Autio-Gold J: The role of chlorhexidine in caries prevention. Oper Dent 2008;33:710-716
61. Bader JD, Shugars DA, Bonito AJ: Systematic reviews of selected dental caries diagnostic and management methods. J Dent Educ 2001;65:960-968
62. Anderson MH: Chlorhexidine: how useful is it in combatting the bacterial challenge and dental caries? J Calif Dent Assoc 2003;31:211-216
63. Spolsky VW, Black BP, Jenson L: Products–old, new, and emerging. J Calif Dent Assoc 2007;35:724-737
64. Deshpande A, Jadad AR: The impact of polyol-containing chewing gums on dental caries: a systematic review of original randomized controlled trials and observational studies. J Am Dent Assoc 2008;139:1602-1614
65. Lee YE, Choi YH, Jeong SH, et al: Morphological changes in *Streptococcus mutans* after chewing gum containing xylitol for twelve months. Curr Microbiol 2009;58:332-337
66. Trahan L: Xylitol: a review of its action on mutans streptococci and dental plaque–its clinical significance. Int Dent J 1995;45:77-92
67. Azarpazhooh A, Limeback H: Clinical efficacy of casein derivatives: a systematic review of the literature. J Am Dent Assoc 2008;139:915-924; quiz 994-995
68. Navazesh M, Christensen C, Brightman V: Clinical criteria for the diagnosis of salivary gland hypofunction. J Dent Res 1992;71:1363-1369
69. Narhi TO, Kurki N, Ainamo A: Saliva, salivary micro-organisms, and oral health in the home-dwelling old elderly–a five-year longitudinal study. J Dent Res 1999;78:1640-1646
70. Turner M, Jahangiri L, Ship JA: Hyposalivation, xerostomia and the complete denture: a systematic review. J Am Dent Assoc 2008;139:146-150
71. Abelson DC, Barton J, Mandel ID: The effect of chewing sorbitol-sweetened gum on salivary flow and cemental plaque pH in subjects with low salivary flow. J Clin Dent 1990;2:3-5
72. Radford DR, Sweet SP, Challacombe SJ, et al: Adherence of *Candida albicans* to denture-base materials with different surface finishes. J Dent 1998;26:577-583
73. Turner MD, Ship JA: Dry mouth and its effects on the oral health of elderly people. J Am Dent Assoc 2007;138(Suppl):15S-20S
74. Sanchez-Garcia S, Gutierrez-Venegas G, Juarez-Cedillo T, et al: A simplified caries risk test in stimulated saliva from elderly patients. Gerodontology 2008;25:26-33
75. Wilson RF, Ashley FP: Identification of caries risk in school children: salivary buffering capacity and bacterial counts, sugar intake and caries experience as predictors of 2-year and 3-year caries increment. Br Dent J 1989;167:99-102