A Clinician's Guide to CAMBRA: A Simple Approach

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Abstract: Caries risk assessment identifies those factors that are pathologic and suggests ways an astute clinician can implement protective strategies that can prevent progression and/or return the patient to health. Caries management by risk assessment (CAMBRA) focuses on treating and preventing the cause of the disease at an early stage, rather than waiting until it causes damage to tooth structure. This article summarizes and simplifies information previously published about CAMBRA implementation from the perspective of today's practicing clinician. The most recent science on prevention, remineralization, antimicrobials, and pH, as well as the use of fluoride, xylitol, and casein phosphopeptide-amorphous calcium phosphate (CCP-ACP) is also discussed.

BACKGROUND

Dental caries is a transmissible and infectious bacterial disease. It is the most common disease among US children.[1] Specifically, dental caries is prevalent in underprivileged children, and low socioeconomic status is recognized as an inherent risk for caries. Further, caries continues to be a significant problem for adolescents as well as adults. Dental caries can become a chronic disease that affects individuals throughout their lifetimes. The Third National Health and Nutrition Examination Survey (NHANES III)-Phase 1 collected data from 1988 to 1994 that indicated approximately 25% of children and adolescents in the 5- to 17-year age range accounted for 80% of the caries in the permanent teeth. These data indicate dental caries continues to be a major oral health concern in children in the US and worldwide.[2] This suggests the population of individuals susceptible to dental decay continues to expand with increased age. It is evident from numerous other studies that dental caries continues to affect individuals throughout childhood and beyond.[4]

This disease is caused by a pathologic biofilm dominated by acidogenic and aciduric bacteria that metabolize sugars and other fermentable carbohydrates, resulting in the production of small molecular weight acids as metabolic byproducts. These small-chain organic acids diffuse into the subsurface enamel and begin demineralizing it, releasing calcium and phosphate ions into the biofilm. Ultimately, a white spot lesion develops at the site. If this process is allowed to progress, the intact enamel surface will collapse and cavitate, requiring surgical restorative repair. The key to caries treatment and disease prevention is to focus on modifying and correcting the biofilm disease component and modifying oral factors to favor health. This can be accomplished with strategies to decrease disease-causing risk factors and to increase health-promoting and protective factors for each patient.

THE CHANGING FACE OF CARIES MANAGEMENT

The practice of dentistry historically has used a surgical-restorative model to deal with the damage (decay) caused by the disease of dental caries. Restoration and repair of the teeth is an important part of any comprehensive treatment for a patient with dental caries, but emphasis also needs to be placed on assessing, diagnosing, and treating the biofilm disease component. Traditionally, patients could return for evaluation year after year and, regrettably, learn of the need for more fillings, more crowns, root canals, and, in the end stages, extractions caused by dental caries. This disease became, in essence, a lifelong illness. In fact, 71% of all

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restorative treatments are performed on previously restored teeth, with recurrent decay as a predominant cause.[3] At issue, however, is the fact that the clinical decision of when restoration is indicated is not consistent among practicing clinicians. The concepts of cavitated vs noncavitated lesions and restoration vs remineralization continue to evolve and are subject to interpretation. Patients who have had extensive treatment may have had continuous years of low risk and little, if any, recurrent decay. However, when a new risk factor develops, for example medication-induced xerostomia, a dramatic change in the oral biofilm can cause dental caries and extensive decay, leading to rapid breakdown of the teeth. Figure 1 illustrates the worst possible outcome of this change.

Historically, the concept of enamel demineralization by bacterial acids explained the process of cavitation. Undisturbed, this process led to bacterial invasion of the dentin and further demineralization and destruction. Researchers began to examine how the enamel surface could be influenced by adding fluoride to the oral environment to reduce the effects of the acid produced by the bacteria. Armed with this evidence, dentists promoted community water fluoridation and used topical fluoride applications in their offices. The first clinical trials using fluoridated dentifrice were done in 1950, and by 1980 almost all dentifrices contained fluoride. However, even with the new approach to protect the enamel against cavitation, there was little focus on the actual cause of the disease—the microbial component, the pathogenic biofilm. Directing treatment at controlling the biofilm has been termed the *medical model* of caries management.

Early research identified mutans streptococci (MS) and lactobacillus bacteria (LB) in the biofilm as the primary causative agents in dental caries,[4,5,6] and many studies demonstrated a relationship of these bacteria to production of decay.[7] Acid production from fermentation of carbohydrates by MS and LB was theorized to play a major role in the demineralization of enamel and dentin.[6] However, as many as 28 different species have also been implicated and may play a role in the caries process[8-11](Table 1). Recently, there is a respectful appreciation of the complexity of the diverse biofilms involved with low pH being the selection factor favoring these acidogenic, aciduric, and cariogenic bacteria.[7] Further research is needed to clarify the extent to which these other species contribute to the caries process. The medical management of caries requires a more thorough understanding of the role and nature of the dental biofilm and the interaction of these bacterial species in both diseased and healthy states. Furthermore, the biofilm on the teeth is unique in the body and substantially different from other tissue surfaces because teeth are a nonshedding surface. Researchers are examining in detail the biofilm and bacterial profiles. With this growing body of scientific evidence, a new strategy of diagnosis, prevention, restoration, and maintenance has been developed and validated.[12-14]

PREVENTION

Classically, dental professionals have taught patients that prevention focuses on brushing, flossing, and avoiding sweets. Risk assessment and chemical remineralization has not been part of this more traditional discipline of prevention. With today's knowledge, prevention has become tied to risk factors. Some patients can maintain lifetime low-risk levels. However, caries risk can change with time depending on factors, such as age, diet, medications, and salivary physiology/pathology. Caries management is related to a patient's current risk factors along with state-of-the-art diagnostic and early lesion-detection techniques.

RISK ASSESSMENT MADE EASY

The purpose of a caries risk assessment is to identify patients most at risk for future decay so they may receive appropriate treatment interventions based on their risk, rather than treating everyone the same. If a patient is healthy or at low risk, then the focus should shift from treatment to prevention and health maintenance. The result is a determination of whether the patient has no risk for the disease and can visit annually or biannually for routine

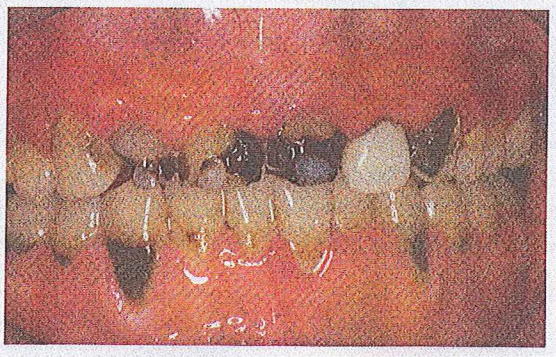


Figure 1 An example of the extreme decay pattern from the lack of buffering capacity, low resting pH, and a selective shift to an acidogenic biofilm caused by xerostomia.

Table 1: Published Studies Implicating Specific Bacteria Associated with Dental Caries

Bacterial Species	Authors										
	Beighton[9]	Acaveda[46]	van Houte et al[10]	Becker et al[8]	Loesche[4]	Nikawa[47]	Kleinberg[48]	Yip[49]	Tanner[50]	Hoshino[51]	Sissons[52]
Streptococcus salivarius	x			x							
Streptococcus parasanguinis				x							x
Streptococcus constellatus				x							
Streptococcus mutans		x	x	x	x	x	x				x
Streptococcus sobrinus			x		x	x	x				
Streptococcus oralis	x			x				x			x
Streptococcus milleri			x								
Streptococcus mitis			x								x
Streptococcus gordonii			x								
Streptococcus anginosus			x								
Streptococcus cricetus							x				
Streptococcus intermedius									x		x
Streptococcus vestibularis											x
Lactobacillus fermentum				x							x
Lactobacillus plantarum										x	x
Lactobacillus acidophilus							x	x			
Lactobacilllus casei							x				
Candida albicans						x					x
Actinomyces israelii	x										x
Actinomyces gerensceriae	x			x							
Actinomyces naeslundii	x										
Veillonella				x							
Veillonella parvula							x				
Bifidobacterium			x	x							
Neisseria sicca							x				
Fusobacterium nucleatum subsp *animalis*				x							
Capnocytophaga gingivalis											x

prevention recommendations and reassessment, or the patient is at continued risk for the disease and needs a more aggressive approach to treating the biofilm disease and specifically targeted prevention protocols, in addition to any restorative needs.

A risk assessment can be conducted by the dental hygienist. A simple caries risk assessment form that identifies the patient's risk factors is essential. There are many caries risk assessment forms readily available. These focus on disease indicators, risk factors, and protective factors. Patients can be stratified into high, moderate, or low risk. Identifying whether the caries is active or inactive is also essential. The most important decision in the treatment room is whether the patient is at risk for dental caries and continued or future lesion development.

This assessment process should differentiate patients into one of two groups: healthy and at low risk for future disease or active dental caries. In addition to categorizing patients quickly and accurately, the risk assessment also should identify each patient's specific risk factors that potentially are contributing to the biofilm disease process. This patient-specific information is then used for treatment recommendations. Many times the risk factors are modifiable, for example, diet, frequency of snacking, and home care. Some risk factors are not modifiable, such as age, amount and quality of saliva, and medication-induced xerostomia. These require counterbalance in the treatment/prevention protocol to compensate for the risk factors. This process needs to be conducted quickly and efficiently to be incorporated into an active dental practice.

DENTAL CARIES TREATMENT STRATEGIES

For all patients, in addition to any needed restorative treatment, the chemistry of demineralization and remineralization that is determined by the biofilm make-up and oral environment must be addressed. Restoration of the defects may return the teeth to function but have little to do with correcting the dental caries biofilm disease. There are many options available for treating the biofilm disease process. A comprehensive approach to treating a patient with a high dental caries risk involves addressing every aspect of the disease.

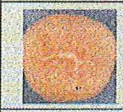
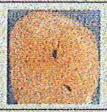
Most treatment planning begins with identifying the reparative procedures required to correct the physical damage to the teeth. However, this should also include remineralization of lesions that have an intact enamel surface and are not yet cavitated and the use of minimally invasive restorative strategies with biomimetic materials on those lesions that have surface cavitation and bacterial decay into the dentin. Next come strategies focused on the therapeutic approach to correcting.the bacterial biofilm component of the disease. These procedures include antimicrobial agents, pH corrections, and metabolic agents, such as xylitol. Additional strategies include making behavioral changes to improve the oral environment to favor a healthy biofilm. Typically, these involve oral hygiene instruction for improved home care and plaque control and dietary counseling. Last are the nonmodifiable factors, including special needs, xerostomia, and medication-induced xerostomia, that may need to be accounted for and addressed by adding more protective factors. These strategies can be broken down into major categories.

REMINERALIZATION THERAPY

Remineralization historically has involved the use of topical fluoride.[15,6] Fluoride can be applied using different methods: 1-ppm public water fluoridation, 1,100-ppm fluoride dentifrice, 5,000-ppm fluoride gels and foams, 223-ppm fluoride rinse, and 23,000-ppm fluoride varnish. Fluoride's basic mode of action is to enhance remineralization and inhibit demineralization.[6] Fluoride ions incorporate into remineralizing enamel/dentin, changing carbonated apatite to a fluoroapatitelike form that is more acid tolerant and makes the hard tissues more acid resistant. Fluoride also inhibits bacterial intracellular enzymes.

More recently, casein phosphopeptide-amorphous calcium phosphate (CCP-ACP) has made calcium and phosphate ions bioavailable to aid in the remineralization process.[16] The concept is best understood in simple terms: Acid demineralization removes calcium and phosphate ions from tooth mineral, and remineralization places these minerals back into the tooth. Fluoride enhances remineralization but will not occur without adequate amounts of calcium and phosphate ions. These ions usually come from adequate amounts of healthy saliva; however, CCP-ACP products ensure adequate levels of these required ions. The benefits of providing additional sources of these ions are not yet clear. It is quite logical to supplement sources of calcium and phosphate in patients with xerostomia where these molecules could be in short supply. However, some studies suggest there is added benefit to increasing the availability of calcium and phosphate in patients at high risk for caries rather than rely solely on the calcium and

Table 2: Occlusal Protocol[13]

ICDAS code	0	1	2	3	4	5	6
							
Definitions	Sound tooth surface; no caries change after air drying (5 sec); or hypoplasia, wear, erosion, and other noncaries phenomena	First visual change in enamel; seen only after air drying, or colored change "thin" limited to the confines of the pit and fissure area	Distinct visual change in enamel; seen when wet, white or colored, "wider" than the fissure/fossa	Localized enamel breakdown with no visible dentin or underlying shadow; discontinuity of surface enamel, widening of fissure	Underlying dark shadow from dentin, with or without localized enamel breakdown	Distinct cavity with visible dentin; frank cavitation involving less than half of a tooth surface	Extensive distinct cavity with dentin; cavity is deep and wide involving more than half of the tooth
Histologic depth		Lesion depth in P/F was 90% in the outer enamel with only 10% into dentin	Lesion depth in P/F was 50% inner enamel and 50% into the outer 1/3 dentin)	Lesion depth in P/F with 77% in dentin	Lesion depth in P/F with 88% into dentin	Lesion depth in P/F with 100% in dentin	Lesion depth in P/F 100% reaching inner 1/3 dentin
Sealant/restoration Recommendation for low risk	Sealant optional DIAGNOdent may be helpful	Sealant optional DIAGNOdent may be helpful	Sealant optional or caries biopsy if DIAGNOdent is 20-30	Sealant or minimally invasive restoration needed	Minimally invasive restoration	Minimally invasive restoration	Minimally invasive restoration
Sealant/restoration Recommendation for moderate risk	Sealant optional DIAGNOdent may be helpful	Sealant recommended DIAGNOdent may be helpful	Sealant recommended or caries biopsy if DIAGNOdent is 20-30	Sealant or minimally invasive restoration needed	Minimally invasive restoration	Minimally invasive restoration	Minimally invasive restoration
Sealant/restoration Recommendation for high risk *	Sealant recommended DIAGNOdent may be helpful	Sealant recommended DIAGNOdent may be helpful	Sealant recommended or caries biopsy if DIAGNOdent is 20-30	Sealant or minimally invasive restoration needed	Minimally invasive restoration	Minimally invasive restoration	Minimally invasive restoration
Sealant/restoration Recommendation for extreme risk **	Sealant recommended DIAGNOdent may be helpful	Sealant recommended DIAGNOdent may be helpful	Sealant recommended or caries biopsy if DIAGNOdent is 20-30	Sealant or minimally invasive restoration needed	Minimally invasive restoration	Minimally invasive restoration	Minimally invasive restoration

* Patients with one (or more) cavitated lesion(s) are high-risk patients. ** Patients with one (or more) cavitated lesion(s) and xerostomia are extreme-risk patients.

*** All sealants and restorations to be done with a minimally invasive philosophy in mind. Sealants are defined as confined to enamel. Restoration is defined as in dentin. A two-surface restoration is defined as a preparation that has one part of the preparation in dentin and the preparation extends to a second surface (note: the second surface does not have to be in dentin). A sealant can be either resin-based or glass ionomer. Resin-based sealants should have the most conservatively prepared fissures for proper bonding. Glass ionomer should be considered where the enamel is immature, or where fissure preparation is not desired, or where rubber dam isolation is not possible. Patients should be given a choice in material selection.

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phosphate in saliva.[16] More studies are needed to answer this question. CCP-ACP, known commercially as Recaldent® (Bonlac Foods Ltd, Melbourne, Australia), is available in MI Paste (GC America, Inc, Alsip, IL) and Trident® Xtra Care™ (Cadbury Adams USA, LLC, Parsippany, NJ).[17] There is a possibility that the positively charged calcium in these calcium-phosphate products can bind with the negatively charged fluoride ion when mixed together or used in succession. Therefore, a dentist should consider instructing patients to use these products at different times of the day or at least separate them with time. Although the length of time separation has not been determined scientifically, some experts say at least an hour or two should be sufficient.

RESTORATIVE STRATEGIES WITH MINIMALLY INVASIVE DENTISTRY

Dental caries can be site, tooth, patient, and population specific. Ideally, successful caries prevention implies there will be no irreversible changes to any tooth site or surface (occlusal, proximal, smooth, or root surface). If prevention fails at any site, early lesion detection should trigger protocols for chemical remineralization and interventions to arrest and reverse early damage caused by demineralization before surface cavitation occurs. With CAMBRA, whether the enamel surface is cavitated is the determining factor in deciding to remineralize a lesion chemically. If the enamel surface is still intact, the bacteria are physically too big to diffuse through the enamel surface to infect the dentin; therefore, they can be repaired successfully with remineralization protocols.[13] Based on scientific evidence, current recommendations are to intervene surgically only on the proximal smooth surface if the bitewing radiograph shows a solid enamel radiolucency going from the surface through the enamel and penetrating the dentin.[13,18,19] Therefore, with CAMBRA, surgical restoration should be performed only after surface cavitation develops. The restoration procedure should use the most minimally invasive approach possible to maintain the maximum amount of healthy tissue and

the structural integrity of the tooth. Also, the restoration should be completed with the dental restorative material best suited for that patient's lesion.

Traditionally, cavitated lesions were identified using a sharp explorer tip, a visual examination, and radiographs. Explorers can vary in sharpness; therefore, lesions have been detected in various states of cavitation. However, numerous studies have reported the use of a dental explorer is not adequate for detecting early occlusal caries[20-23] and may lead to a significant number of undetected lesions,[20,22,24] including some false positives but also, in some cases, traumatic surface defects.[25] Radiographs also are not useful for detecting early occlusal lesions because of the masking effect of the facial and lingual enamel. New research has suggested that dentists should use the visual International Caries Detection and Assessment System (ICDAS) code[26] system. This system can be thought of as a code, 0 to 6, that correlates what is seen clinically with a definition and what research has reported histologically.[26] Recently Jenson et al[13] published a table that describes different protocols based on ICDAS code and caries risk (Table 2). Included in this table is how one would use detection technologies, specifically the DIAGNOdent (KaVo, Lake Zurich, IL), in the decision-making process.

The current state of lesion detection leaves behind the dental explorer and brings a more scientific purview with the use of ICDAS codes, digital radiographs, and some new technologies based on light transmission and reflection, such as the DIFOTI® (Electro-Optical Sciences, Irvington, NY) or Midwest Caries I.D.™ (DENTSPLY Professional, York, PA). These technologies detect differences in light transmission/reflection in demineralized enamel compared with that of normal enamel, allowing detection of demineralization long before it reaches the cavitation stage (ie, white spot lesions).

One detection approach, QLF (QLF™, Inspektor Dental Care, Amsterdam, The Netherlands), measures the degree of demineralization, using quantitative light-induced fluorescence, which is based on the natural fluorescence of teeth. With this computer-assisted technology, white spot lesions can be monitored over time to determine if the lesion is progressing or remineralizing.[27] Simply put, healthy enamel structure has different optical properties than decalcified enamel; it has a different optical signature. The fluorescent signal reflected from the decalcification (white spot lesion) of the enamel is captured by a fiber-optic sensor and interpreted through a computer-based algorithm to determine the amount of demineralization.

Digital radiographs provide not only less radiation exposure, but also the ability to enhance and enlarge the images easily, enabling better detection and monitoring of early lesions. Computer-aided detection tools, such as Logicon Caries Detector™ Software (Kodak Dental Systems, Atlanta, GA), can be useful in this process. Enhanced detection does not mean more aggressive restoration. Radiographic lesions that do not penetrate the dentin are not cavitated[18] and can be chemically remineralized.

Visual inspection remains an excellent method to detect lesions on the tooth root simply because lesions on the facial and lingual are easily seen without the need of other technologies.[28] The chemistry of remineralization is the same on the root (cementum) as it is for enamel.[29,30] However, very early lesion detection in this location is difficult because, theoretically, no visible change will be present in an early root lesion. Early demineralization occurs at the molecular level. Because no enamel is covering the root surface, there will be no visible white spot formation. Many individuals have proposed that any exposed root is a root at risk[31] because of its lower mineral content and vulnerability to acid and enzyme dissolution. Careful monitoring of remineralization of these early lesions is recommended because of the more porous nature of cementum and dentin (less mineral compared with enamel) and the close proximity to the dental pulp in deeper root lesions. If restoration is needed on a root surface, glass ionomer restorative materials should be considered, especially when a rubber dam is not feasible, because these materials adhere chemically and release fluoride. Glass ionomers also have the unique ability to continually replenish the fluoride loss from its surface every time the surface is exposed to topical fluoride from other sources; this is sometimes described as "charging and recharging."[32]

THERAPEUTIC CARIES STRATEGIES

Chlorhexidine has been used as a first-defense antimicrobial in treating dental caries, and it is somewhat effective in attacking MS,[33] although it has little effect on lactobacilli, a major bacterial group involved in the caries process. Ethyl alcohol and essential oils have been used in the past and, more recently, there have been studies[34,35] and review papers[36] on the use of 10% povidone iodine and recommendations for 0.10% sodium hypochlorite as antimicrobial rinses.[17] Products from the chlorehexidine category include Peridex® (Zila, Inc, Phoenix, AZ) and PerioGard® (Colgate-Palmolive Co, New York, NY).[17]

Raising the pH or buffering the oral environment to promote remineralization has been used as the main pH strategies. The concept behind this is based on the chemistry of demineralization and remineralization. The demineralization/remineralization continuum for enamel peaks at roughly a pH of 5.5. Below that, pH demineralization occurs, while above it, remineralization occurs. During demineralization, minerals diffuse out of the tooth by passive diffusion. Unless the pH is first neutralized, thus stopping the demineralization and outward diffusion of minerals, remineralization will not be possible.[37] There is, however, a second benefit of avoiding acidic pH in the mouth; prolonged periods with a low pH in the mouth is the selection pressure for the oral biofilm that favors the acidogenic, aciduric, and cariogenic bacteria.[7,38-40] Avoiding the acidic pH range helps encourage repopulation of commensal bacteria. pH strategies can comprise an important component of the overall plan for patients with high caries risk.[7,39,40] Products for pH strategies include the Arm and Hammer® baking soda line (Church & Dwight Co, Inc) and CariFree® line (Oral BioTech, Albany, OR). These products are available as gums, dentifrices, oral neutralizing gels and sprays, and rinses.[17] These products neutralize acid and encourage a nonacidic environment to help the chemistry of remineralizaton and to avoid acidogenic and aciduric biofilms, as previously explained.

Xylitol is a naturally occurring alcohol sugar that is not metabolized by MS. This effective anticaries agent inhibits the attachment of the biofilm and interferes with intracellular metabolism. MS cannot use or break down xylitol and, therefore, expend energy to expel it from the cell.[41] Xylitol is available in many forms: gum, lozenges, mints, sprays, rinses, pastes, and a baking substitute for sugar or other sweeteners.[17] Xylitol is low in calories and does not stimulate insulin production in individuals with diabetes. Studies indicate a dose of 6 grams to 10 grams per day will significantly reduce levels of MS.[42] Patients should be cautioned that xylitol can create gastrointestinal distress at high levels of consumption. It also can be toxic for dogs, so pet owners need to be aware of this complication. There are a multitude of xylitol products made by various companies, including Omni™ Preventive Care, a 3M ESPE company (St. Paul, MN), Epic Dental (Provo, UT), Xlear Inc (Orem, UT), and Oral BioTech.[17]

MODIFIABLE CARIES RISK FACTORS

A significant modifiable risk factor for dental caries is dietary habits.[6,19,43] Scientific studies clearly demonstrated it is the pH drop from dietary sugars that initiates dental caries.[7,38-40] Furthermore, the most important factor is not the amount of sugar consumed by the patient, but the frequency of intake during the day. Frequent between-meal snacking leads to prolonged periods of low pH in the mouth. Based on the traditional Stephan Curve, with frequent snacking, the saliva never has the opportunity to buffer the low pH and return the environment to one of remineralization. This favors the cariogenic bacteria and increases the risk for dental caries. Total bacterial numbers also are important in the caries process, and heavy plaque has been predictive of high caries risk.[44] Therefore good oral hygiene instruction and improving a patient's skill to regulate plaque should not be abandoned.

NONMODIFIABLE CARIES RISK FACTORS

As previously discussed, some patients may have risk factors that cannot be changed. Patients with special needs may not be able to develop adequate plaque control or improve dietary habits, and they may have limited access to care. Patients with xerostomia do not have adequate saliva to buffer and protect the teeth. Additional strategies will need to be developed for each of these patients based on his or her specific condition to compensate for the lack of saliva and keep a healthy biofilm and caries balance in place. Although this may include cholinergic agonists in some instances when appropriate, it does not appear that this strategy is commonly used in practice today. Most clinicians rely on products to attempt to supplement or replace missing items when saliva is inadequate. Saliva has so many important functions that this often gets complex and may involve greater use of antimicrobial therapy, calcium and phosphate supplementation or fluoride on a more frequent basis, or even more frequent pH correction to support the normal functions of healthy saliva.

CONCLUSION

CAMBRA is a growing philosophy designed to identify, diagnose, and correct the dental caries biofilm component of the caries disease process, in addition to restoring the teeth to function. This rapidly emerging trend is being adopted by most dental schools[45] and its use is growing in private practices. The goal is quick, simple, and accurate identification of those patients at most risk for the disease as well as their accompanying risk factors, so that an appropriate course of corrective action can be implemented.

The value of this philosophy is to create current good health and prevent future disease. Incorporating these new protocols into private practice can be challenging for the dental team. The best success will come from developing a simplified, systematic, and personalized approach for all patients. The science of CAMBRA can be thought of in simple terms and need not be complex or intimidating. With CAMBRA, the dental team can apply the best available science to help their patients achieve the highest levels of oral health.

DISCLOSURE

Dr. Young and Dr. Whitehouse are stockholders of Oral BioTech. Dr. Kutsch is an owner of Oral BioTech.

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