The Canary Dental Caries Detection System
www.thecanarysystem.com

Quantum Dental Technologies

Shining a new light on Dentistry

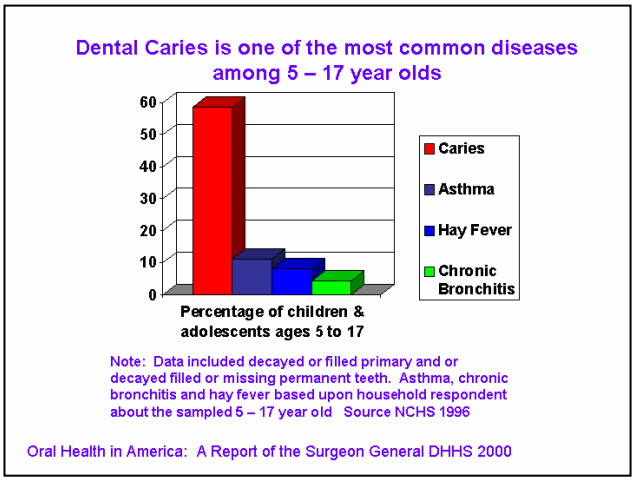
Stephen H. Abrams, DDS
748 Briar Hill Avenue
Toronto, Ontario
Canada M6B 1L3

Telephone: (416) 785 - 4547
Facsimile: (416) 785 - 7301
Facsimile: (416) 265 - 6795
E Mail: dr.abrams4cell@sympatico.ca

Sunday, November 09, 2008

Dental Caries and Remineralization; Does it Really Work?

Dental caries, according to the United States Surgeon General report on Oral Health in America, is one of the most common chronic diseases among five to seventeen year olds[1]. In their study, it was more common than asthma, hay fever or chronic bronchitis. Although we do not have this type of data available in Canada, one can assume that dental caries is extremely prevalent in the population. A great deal of general practitioner's time is spent treating dental caries. The dental profession understanding of caries and treatment approach has been evolving as new diagnostic devices and preventive techniques are introduced to our practices. In 2001, the National Institute of Health's (NIH) Consensus Conference on the Diagnosis and Management of Dental Caries throughout Life concluded:



"Dental caries is an infectious, communicable disease resulting in destruction of tooth structure by acid-forming bacteria found in dental plaque, an intraoral biofilm, in the presence of sugar. The infection results in the loss of tooth minerals that begins with the outer surface of the tooth and can progress through the dentin to the pulp, ultimately compromising the vitality of the tooth."[2]

This statement combines a number of new components from the traditional approach taught over the last twenty years in dental schools. Our patients assume that tooth decay is caused by eating sugary foods, not that dental caries is an infectious communicable disease caused by acid forming bacteria. Patients, along with us have the opportunity to look anew at how we diagnose, prevent and treat caries. The conference states that

> "In order to make continued progress in eliminating this common disease, new strategies will be required to provide enhanced access for those who suffer disproportionately from the disease; to provide improved detection, risk assessment, and diagnosis; and to create improved methods to arrest or reverse the noncavitated lesion while improving surgical management of the cavitated lesion."

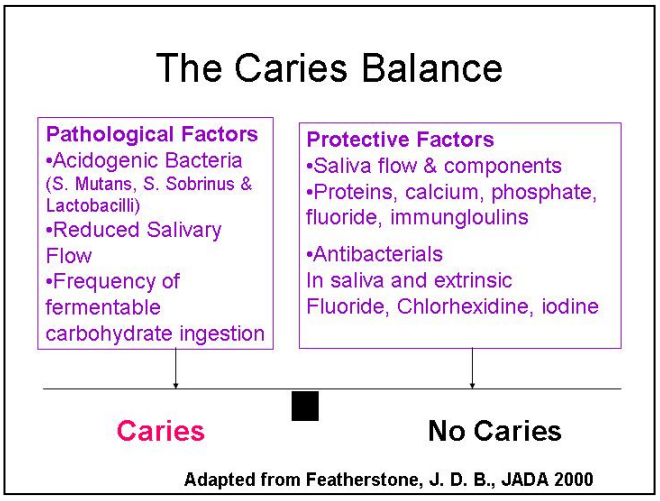
Dentistry is beginning to move from the surgical model for preventing tooth decay (placing restorations) to identification of early carious lesions and treating them with non-surgical methods including remineralization. One can place a number of restorations in a mouth, without treating the

underlying disease. The bacteria remain in the plaque biofilm on the remainder of the teeth capable of creating new areas of decalcification and cavitation. Patients are beginning to expect that we can treat this disease or at least provide them with a reason as to why they or their children continue to develop carious lesions.

Dental caries arises from an overgrowth of specific bacteria that can metabolize fermentable carbohydrates and generate acids as waste products of their metabolism. Streptococci mutans and Lactobacillus are the two principal species of bacteria involved in dental caries and are found in the plaque biofilm on the tooth surface [3 4 5]. When these bacteria produce acids, the acids diffuse into tooth enamel, cementum or dentin and dissolve or partially dissolve the mineral from crystals below the surface of the tooth. If the mineral dissolution is not halted or reversed, the early subsurface lesion becomes a "cavity". These early subsurface lesions are not detectable with our current technology.

These bacteria along with others colonize tooth surfaces as plaque which researchers have now come to recognize as a biofilm[6]. A biofilm is a well organized cooperating community of microorganisms[7]. Previously, bacteria had been studied as they grew in colonies in Petri dishes in a laboratory. With more sophisticated techniques, researchers have been able to study bacteria in their natural states[8]. Microorganisms in a biofilm are not evenly distributed. They are grouped in micro colonies surrounded by an enveloping intermicrobial matrix. Fluid channels move through this matrix carrying nutrients, waste products, metabolites, enzymes and oxygen. Each micro colony has its own environment with differing pH, nutrients, oxygen gradients etc. Bacteria in biofilms communicate with each other by sending out chemical signals that can trigger the release of proteins and enzymes. This type of environment may contribute to some of the resistance to anti-microbials[9]. One of the best methods for dealing with plaque biofilm is its meticulous removal by brushing or professional cleaning[10]. The use of some anti-plaque agents such as chlorhexidine, hexetidne amine fluoride / stannous fluoride, triclosan and others may inhibit biofilm development and maturation as well as affect bacterial metabolism and thus help in the prevention of caries and periodontal disease.[11].

The tooth surface undergoes demineralization and remineralization continuously, with some reversibility. When exposed to acids, the hydroxyapatite crystals dissolve to release calcium and phosphate into the solution between the crystals. These ions diffuse out of the tooth leading to the formation of the initial carious lesion. The reversal of this process is remineralization. Remineralization will occur if the acid in the plaque is buffered by saliva, allowing calcium and phosphate present primarily in saliva to flow back into the tooth and form new mineral on the partially dissolved subsurface crystal remnants[12]. The new "veneer" on the surface of the crystal is much more resistant to subsequent acid attack, especially if it is formed in the presence of sufficient fluoride. The balance between demineralization and remineralization is determined by a number of factors. Featherstone describes this as the "Caries Balance", or the balance between protective and pathological factors (see figure 1)[13].



The NIH Consensus Conference on Dental Caries concluded that dental caries is an infectious communicable disease. Detectable levels of Streptococcus mutans occur in children's mouths

only after the eruption of the first primary tooth. The source of infection appears to be the mother or caregiver[14]. A number of studies have found that mothers with high concentrations of salivary mutans streptococci tended to have highly infected children[15]. These children also had a greater risk of developing a large number of carious lesions in their primary teeth.[16] [17] Mothers with low levels of salivary Streptococci mutans had children with below threshold levels. Brambilla and others demonstrated that by using a mouth rinse protocol to reduce maternal mutans streptococci levels starting at six months of pregnancy up to delivery they were able to delay the colonization of bacteria in their children's mouths[18]. Using this evidence, we can now begin to help prevent or reduce the risk of caries in children.

One of the popular approaches to caries prevention is to develop a caries risk assessment approach for treating patients. This would involve identifying patients with an elevated series of risk factors for developing caries and providing them with more intensive preventive therapies. Risk assessment can be as simple as noting that a patient has developed one or more carious lesions within one year and then increasing recall frequency, reviewing home care etc. Understanding the "Caries Balance", as illustrated in figure 1, can facilitate the development of a more sophisticated approach to caries risk assessment. The following variables should be assessed in developing an overall assessment of caries risk:

- Number of decayed missing or filled teeth,
- Number of new carious lesions within the past year,
- Frequency and timing of ingestion of fermentable carbohydrates,
- Elevated levels of mutans streptococci and lactobacilli in saliva,
- Salivary flow rate,
- Lack of fluoride in the drinking water and use of fluoridated toothpastes,
- Poor oral hygiene at home care,
- Presence of heavy plaque on teeth,
- Presence of intraoral appliances,
- Presence of white spot lesions
- For infants, we need to assess these same factors in the mother and or caregiver.

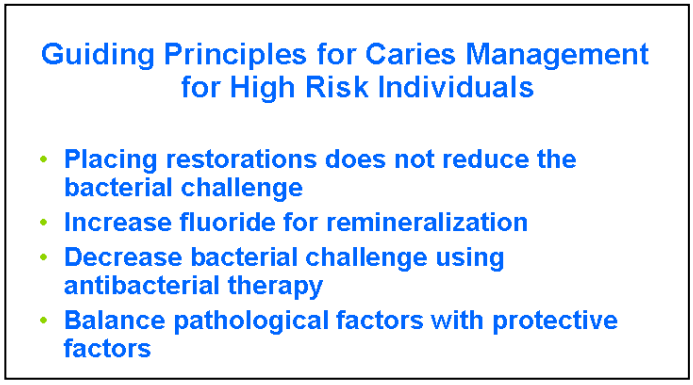
If a number of these factors are present, then considering how we can help our patients can reduce their risk for dental caries.

We must first assess which of these factors are significant in increasing our patient's risk for caries. In some instances, reduced salivary flow rates (such as Sjorgen's Syndrome) may dramatically increases the caries risk. In other situations, poor oral hygiene, poor diet and lack of fluoridated tooth pastes may increase risk. There is no correct ranking in order of importance for these risks but there are a number of papers on caries risk assessment that bear further review.[19] [20] [21] One of the most recent caries risk assessments were published in the Journal of the California Dental Association in 2003[22]. It provides both a template for caries risk assessment and some educational tools for patients. This can provide you with a starting place for creating your own series of tools and assessments for your practice.

There are a number of preventive tools we can be use. The most obvious is proper brushing using a fluoridated tooth paste. We can also:

- Modify diet,
- Use fluoride rinses and lozenges,
- Use high concentration fluoride tooth pastes or varnishes,
- Use of sugar free mints especially those containing Xylitol[23],
- More frequent recall appointments,
- Antibacterial mouth rinses including those containing chlorhexidine gluconate,
- For dry mouth use of baking soda containing tooth pastes or rinsing with a baking soda suspension.

These are the current set of tools that we can utilize. More tools are likely in the near future especially as technology for early detection improves. There is some discussion in the literature about the use of 10 percent povidone iodine which could be applied topically every two months to reduce the incidence of caries in high risk children[24]. Antimicrobial therapy may also help to reduce Streptococci mutans levels and thus reduce caries and the risk of transmission[25].



Caries Intervention in Dental Practice

The goal for the dental professional is to increase the protective factors and to disrupt the demineralization cycle. One must let patients know to decrease sugars, especially the sugars in soft drinks. Tell patients about soft drinks, and let them know about the website for the "Stop the Pop" campaign: www.modental.org. Diet analysis and diet control become a key issue in stopping demineralization. It not only involves eliminating foods that bacterial can metabolize but examining when the exposure to these foods occur. For example, a single can of diet pop consumed once a week is not as destructive as a single candy sucked slowly over one hour each day. It is both the composition and exposure time that makes foods cariogenic. Cariogenic snacks are much worse than cariogenic foods consumed during the course of a meal especially if the patient brushes after the meal.

Remineralization can be promoted through a variety of techniques[26 27 28 29]

Summary of Caries Process & Methods for Caries Intervention [30])

Parts of the Caries Process	Methods for Caries Intervention
Bacteria	Antibacterial therapy
Carbonated Hydroxyapatite	Make less soluble by transforming to other phases.
Fermentable Carbohydrates	Reduce the frequency of ingestion and or substitute with non-cariogenic sweeteners
Organic Acids	Neutralize the acid by providing extra buffering or enhancing saliva
Saliva	Enhance the saliva flow and function
Fluoride	Exploit the know effects on bacteria, inhibition of demineralization and enhancement of remineralization

Remineralization Agents (not a comprehensive list of all products)

Product	Dosage	Duration	Usage	Product Name
Chlorhexidine	.12% rinse	0.5 oz. twice a day for 14 days	Alone	Peridex Perioguard
Fluoride Varnish	5% NaF	2-4 times/year or for high caries risk 3 times in 10 days	Alone	Durafluor Duraphat (Medicom) FluorProtector (Ivoclar Vivadent) Cavity Shield (Omni)
Professional Topical Fluorides		1 - 4 minutes depending upon formulation	Applied by oral health professional	
Home Fluoride Dentifrice	1,000 ppm NaFl	One minute	Twice daily	Crest Total – Triclosan (Colgate)
Home Fluoride Dentifrice	5,000 ppm NaFl		Twice Daily	Prevident 5000 ControlRx (Omnii)
Over the Counter Fluoride Rinse	0.05% NaF	One minute	2-3 times/day	Reach Act
Oral Therapeutic Fluoride Rinse	1.1% NaFl 15,000 ppm	Five minutes	Once daily	Phos-Flur Rinse Colgate In-Office
Xylitol Gum	1- 2 pieces (needs to be 64% by wt.)	Five minutes	3 times/day	TheraGum,Spry, Wrigley, Trident, Orbit
Calcium Phosphate	?	?	?	Enamelon
CCP ACP		Once a day		GC Tooth Mousse Nova Min
Xylitol Rinse				Biotene

Incorporating Remineralization into Dental Practice

Recommend fluoride for *all* patients, not just children. Consider the benefits of a fluoride needs assessment. Oral-B offers a complete patient survey form to answer their fluoride exposure.
Fluoride recommendations should be customized for patients, depending on risk assessment. Fluoride has three main mechanisms of action: 1) inhibits metabolism of bacteria, such as *S mutans,* metabolism; 2) inhibits demineralization; and, 3) enhances remineralization[31]. Application choices may be topical or prescribed rinses (see chart above). Provide topical fluoride for patients on a regular basis customized to their particular caries risk.

One successful antibacterial therapy for cariogenic bacteria is **chlorhexidine rinses**. By prescribing a twice daily 2 week, 0.12% chlorhexidine rinse regimen, the cariogenic bacteria can be reduced significantly.

Fluoride varnish is yet another preventive tool. It is a concentrated fluoride dispensed in single-dose delivery or a tube. It has excellent adherence qualities to the tooth surface – whether applied wet or dry and is very effective, particularly around orthodontic brackets and erupting molars[32]. Clinical trials involving fluoride varnishes show caries incidence reduction ranging from 18% to 70% [33].

Saliva, especially the amount and flow are critical in the remineralization/demineralization balance. Saliva and its buffering contents deliver the needed minerals for the repair process of the tooth surface. An excellent mouthwash is Biotene. It contains no alcohol and has xylitol. Also, note that gum chewing – particularly brands containing **xylitol** will stimulate the flow of saliva.

Recommend toothpastes containing baking soda for the saliva deficient patient and, for their improved hydration, add 2 teaspoons of baking soda for each 8 ounces of water. Instruct them to swish frequently throughout the day. The baking soda will increase the available mineral content in saliva and thus help to promote remineralization[34].
Watch the medications that your patients are taking and monitor their medical health. A slight change in saliva production even induced by medication may be enough to increase their risk for caries.

Therapeutic gum chewing has been shown to be effective in reducing the transmissibility of the infectious bacteria that cause decay[35] [36], The five-carbon sugar substitute, xylitol is yet another protective factor, found in chewing gum and rinses. **Xylitol** benefits include:
1) reduced caries and plaque;
2) decreased cariogenic bacteria[37];
3) enhanced remineralization and,
4) increased salivary flow[38].
Patients will receive xylitol's benefit by chewing gum (64% by weight) three times per day for a minimum of five minutes. Obviously, brands that contain xylitol at the top of their ingredient listing are more efficatious.

Summary

Dental caries is an infectious communicable disease caused by a variety of acid forming micro-organisms. In the presence of fermentable carbohydrates these organisms will produce acids which will cause the destruction of the tooth. The tooth surface, both enamel and root surface undergo periods of demineralization and remineralization during the day depending upon the conditions in the oral cavity and the biofilm on the tooth surface. Dentistry, in the past, has waited until cavitation into the outer ½ of the enamel shell has occurred before initiating any invasive treatment. Fluorides were one of the first remineralization agents used by oral health professionals for remineralizing early lesions and making the tooth surface more resistant to destruction by acids. As new techniques for detection and remineralization develop, they have become incorporated into clinical practice for the early, non-invasive treatment of carious lesions.

Stephen Abrams, DDS

---

[1] Department of Health and Human Services, *"Oral Health in America: A Report of the Surgeon General"*, National Institute of Dental and Craniofacial Research, 2000, page 63

[2] *"NIH Consensus Development Conference on Diagnosis and Management of Dental Caries Throughout Life March 26 – 28 2001*", Journal of Dental Education, Volume 65, # 10, 2001, page 1162

[3] Van Houte, J., "*Bacterial specificity in the etiology of dental caries*", Int. Dent. J., 1980, Volume 30, pages 305 – 326

[4] Van Houte, J., "*Role of Microorganism in the caries etiology*", J. Dent. Res., 1994, Volume 73, pages 672-681

[5] Featherstone, J. D. B., "*The Caries Balance: Contributing Factors and Early Detection*", CDA Journal, 2003, Volume 13 # 2 pages 129 – 133

[6] Bernimoulin, J. P., "*Recent concepts in plaque formation",* J. Clin Periodontology, 2003, Volume 30, Supplement # 5 pages 7 – 9

[7] Marsh, P. D., Bradshaw, D. J. *"Dental Plaque as a biofilm",* J. Industrial Microbiology, 1995, Volume 14, pages 169 – 175

[8] Overman, P. R., "*Biofilm: A New View of Plaque*", J Contemporary Dent Practice, 2000, Volume 1, # 3 page 1 – 12

[9] Marsh, P. D., Bradshaw, D. J., "*Physiological approaches to the control of oral biofilms*", Adv. Dent. Res., 1997, Volume 11, # 1, pages 176 – 185

[10] Haffajee, A. D., Arguello, E. I., Ximenez-Fyvie., Socransky, S. S., "*Controlling the plaque biofilm*", Int. Dent. J., 2003, # 53 Supplement # 3, pages 191 – 199

[11] Baehni, P. C., Takeuchi, Y., *"Anti-plaque agents in the prevention of biofilm – associated oral diseases*", Oral Diseases, 2003, Volume 9, Supplement, # 1 pages 23 – 29

[12] Melberg, J. R., "*Remineralization: A status report for the American Journal of Dentistry, Part 1*, Am J. Dent., 1988, Volume 1, # 1, pages 39 – 43

[13] Featherstone, J. D. B., "*The Science and Practice of Caries Prevention*", JADA, 2000, Volume 131, pages 887 – 899

[14] Berkowitz, R. J., *"Acquisition and transmission of Mutans Streptococci*", CDA Journal, 2003, Volume 31, # 2, pages 135 – 138

[15] Caulfield, P. W., "*The fidelity of initial acquisition of mutans streptococci by infants from their mothers"*, J. Dent. Res., 1995, Volume 74, pages 681 – 685

[16] Kohler, B., Andreen, I., Jonsson, B., *"The earlier the colonization by mutans streptococci, the higher the caries prevalence at 4 years of age*", Oral Microbiol. Immunol., 1988, Volume 3, pages 14 – 17

[17] Caulfield, P.W., Cutter, G. R., Dasanayake, A. P., "*Initial acquisition of mutans streptococci by infants: evidence for a discrete window of infectivity"*, J. Dent. Res., 1994, Volume 72, # 1, pages 37 – 45

[18] Brambilla, E., et al., "*Caries Prevention During Pregnancy: Results of a 30-Month Study*", JADA, 1998, Volume 129, pages 871 – 877

[19] Krasse, B., "*Caries risk: A practical guide for assessment and control*", Chicago, Quintessence Books, 1985:7

[20] Anusavice, K., "*Clinical Decision-Making for Coronal Caries Management in the Permanent Dentition",* J. Dent. Edu., 2001, Volume 65, # 10, pages 1143 – 1146

[21] Tinanoff, N., Douglass, J. M., *"Clinical Decision –Making for Caries Management in Primary Teeth"*, J. Dent. Edu., 2001, Volume 65, # 10, pages 1133 – 1142

[22] Featherstone, J.D. B., et al., "*Caries Management by Risk Assessment: Consensus Statement, April 2002*, CDA Journal, 2003, Volume 31, # 3, pages 257 – 269

[23] Kakuta, H., Iwani, Y., Mayanagi, H., Takahashi, N., *"Xylitol Inhibition of Acid Production and Growth of Mutans Streptococci in the Presence of various Dietary Sugars under Strictly Anaerobic Conditions*", Caries Research, 2003, Volume 37, # 6 pages 404 – 409

[24] Den Besten, P. K., Berkowitz, R. J., "*Early childhood caries: An overview with reference to our experience in California"*, J Cal Dent Assoc., 2003, Volume 31, # 2, pages 139 – 143

[25] Slavkin, H. C., "*Streptococcus Mutans, Early Childhood Caries and New Opportunities*", JADA, 1999, Volume 130, pages 1787 – 1792

[26] Nyvad B, Machiulskiene V, Baelum V. *Reliability of a New Caries Diagnostic System Differentiating Between Active and Inactive Caries Lesions.* Caries Res 1999; 33:252-260.

[27] Rozier RG, *Effectiveness of Methods Used by Dental Professionals for the Primary Prevention of Dental Carries.* J Dent Educ 2001; 65:1063-1072

[28] .Bader JD, et al. *Diagnosis and management of Dental Caries.* Evidence Report. Chapel Hill: University of North Carolina; 2000

[29] Anusavice KJ, *Efficacy of Nonsurgical Management of the Initial Caries Lesion.* J Dent Educ 1997; 61:895-905.

[30] Featherstone, J. D. B., "Innovative Methods for Early Caries Intervention", In Early Detection of Dental Caries II Proceedings fo the 4th Annual Indiana Conference Indiana University School of Dentistry, Stookey, G., editor, 1999

[31] Anderson MH, Bales DJ, Omnell KA. Modern management of dental caries: the cutting edge is not the dental burr. J Am Dent Assoc 1993;124:36-44

[32] Hamilton IR, Bowden GHW. Fluoride effects on oral bacteria. In: Fluoride in Dentistry (eds. Fejerskov O, Ekstrand J, Burt BA), pp230-251. Mucksgaard, Copenhagen, 1996

[33] Strohmenger, L., Brambilla, E., "The use of fluoride varnishes in the prevention of dental caries: Short Review", Oral Diseases, 2001, Volume 7, pages 71 – 80

[34] . Research on the Impact of Sodium Bicarbonate on Various Aspects of Oral Health. Compendium of Continuing Education in Dentistry, 2001, Volume 8, Supplement 21.

[35] Isokangas P, Soderling E, Pienihakkinen K, Alanen P. *Occurrence of Dental Decay in Children After Maternal Consumption of Xylitol Chewing Gum, A Follow Up Study from 0 to 5 Years of Age.* J Dent Res 2000; 79:1885-1889

[36] Solderling, E., Isokangas, P., Pienihakkinen, K., Tenovuo, J., Alanen, P., "Influence of maternal xylitol consumption on mother-child transmission of mutans streptococci: 6 year follow up", Caries Research, 2001, Volume 35, pages 173 – 177

[37] Kakuta, H., Iwani, Yl, Mayangi, H., Takahashi, N., "Xylitol inhibition of acid production and growth of Mutans Streptococci in the presence of various dietary sugars under strictly anaerobic conditions", Caries Research, 2003, Volume 37, pags 404 - 409

[38] Scheinin A, Makinen KK, Tammisalo E, Rekola M. Turku sugar studies XVIII. Incidence of dental caries in relation to 1-year consumption of xylitol chewing gum. Acta Odontol Scand 1975;33:269-278