Caries Process and Prevention Strategies: Demineralization/Remineralization

The following is a transcription that has been taken verbatim from the presenter's audio. No Edits have been made.

Video Transcript

Hello and welcome to dentalcare.com's cariology course. This section focuses on remineralization and demineralization. This is part five of a 10 part series entitled Caries Process and Prevention Strategies. Caries is a chemical dissolution of the hard two structures, enamel and dentin, by the acid created as the bacteria in dental plaque ferment carbohydrates. The development of caries is dependent on the interplay between processes that cause demineralization of tooth enamel and those which cause remineralization. Only when factors favor the high acidity that leads to demineralization, does caries occur.

In this course, the dynamic processes of demineralization and remineralization are discussed, paying particular attention to tooth hard tissue structure, the role of acid production by cariogenic bacteria and the critical pH that which tooth enamel beings to dissolve. The role of acid reducing bacteria, saliva and fluoride in tooth hard tissue remineralization will also be explained.

First we wanted to talk about a couple of clinical significant snapshots or questions that you might be faced within your own dental practice. For one, how does understanding the demineralization and remineralizing cycle help me prevent, uh, or arrest the caries process in my patients? While the de-min, re-min cycle is like the ebb and flow of money in a checking account. If too many withdrawals are made and too few credits received, then the account becomes overdrawn. Credits that match or exceed the debits leads to healthy financial situation. The same applies to calcium ions entering and exiting the tooth.

Some loss of calcium inevitably occurs at meal times as the cariogenic bacteria in the biofilm, on the surface of the tooth, metabolize the sugars in the diet through the process of glycolysis. This creates a low pH, or acidic environment that is capable of driving the demineralization process. Between meals, the saliva brings a pH back to safe levels or above five point five and the calcium ions can return to the tooth or remineralization can occur. If there's not enough time for sufficient remineralization, then there's an overall loss of calcium from the tooth. The subsurface lesion may develop. Bacteria enter the tooth material and the capitation process commences.

To prevent the occurrence of caries in your patients, it's important to include information about foods that lead to demineralization in oral health counseling. And note that saliva needs time between food intakes to restore any loss of calcium by sugar containing foods and beverages. Foods rich in calcium and that stimulate saliva flow are very beneficial at the end of any meal. Examples would be yogurt, cheese or milk, which is super saturated with calcium or sugar fee chewing gum, which stimulates saliva.

Another question, why is the use of fluoride agents so prominent in the prevention of dental caries? Hydroxyapatite crystals in enamel are impure due to the presence of carbonate ions. Carbonate ions make the

carbon hydroxyapatite or the carbonated hydroxyapatite weak and much more easily dissolved by acids. Fluoride ions can replace some of the carbonate and hydroxyl ions to create fluorapatite. Fluorapatite is physically much stronger than carbon hydroxyapatites and more resistant to acid dissolution. Essentially, fluoride tips the de-min, re-min balance in favor of the re-min.

Fluoride should be applied daily in low concentration via the use of toothpaste that has been proven with bio-tail-, bio availability of fluoride. In other words, have the ADA Seal of Acceptance. Additional forms of fluoride application should be considered for patients more at risk of caries due to frequent consumption of sugars or poor saliva flow. These would include fluoride rinses, daily or weekly, depending on the strength and professional application of gels or foams that are rich in fluorides.

Our expectation is that upon completion of this course, the dental professional should be able to discuss the differences in how tooth enamel and dentin structure are affected by demineralization, to be able to explain the role of bacterial acid reduction in demineralization, to understand the relationship between the critical pH and demineralization, to identify the five zones of caries dentin in the advanced lesion, to be able to describe how demineralization affects young children, the elderly and other special populations and finally, to be familiar with the factors that promote remineralization.

Tooth enamel demineralization, triggered by an increase in the acidity of bacterial plaque, is the initiation of the caries process. In any discussion of the caries process, particular attention is paid to the enamel, the hard outer most layer. Because it is the primary contact of cariogenic bacteria and where the demineralization process, that can lead to caries, first begins. It's also the only tissue, the tooth, that does not have the ability to grow or repair itself after maturation… Making it even more crucial that its demineralization is prevented.

Caries can also develop in dentin, the hard layer under the enamel. So, understanding the chemical composition of this layer and how it is affected by demineralization is also important… The process of the remineralization or the replacement of lost minerals that hard dental tissues, can halt, slow down and in some cases, reverse, the caries process. Saliva and fluoride are two key players in remineralization. Healthy saliva contains healthy amounts of calcium and phosphate ions that can replenish lost minerals in the hard tooth structure. The fluoride can be incorporated into the tooth structure to strengthen.

For caries prevention, factors in the oral cavity must be highly favorable for remineralization to occur so that this process can be effective. If the environment is more favorable for demineralization, the remineralization process may have little or no influence or not occur at all and caries will develop. There are three main factors of interest in our discussion of hard tissue structuring composition. These include the enamel, enamel formation and dentin. The enamel is the most mineralized tissue of the body, forming a very hard, then, translucent layer of calcified tissue that covers the entire anatomic crown of the tooth. Enamel is so hard because it is composed primarily of inorganic materials. Roughly 95 percent of enamel is calcium and phosphate ions, combined to make up strong hydroxyapatite crystals.

Approximately one to two percent of enamel is made up of organic materials. Particularly enamel specific proteins called enamelins. Which have a high affinity for binding the hydroxyapatite crystals. Water makes up the remainder of the enamel, but counting for about four percent of its composition.

Hydroxyapatite crystals contain calcium and phosphate ions in the following proportions: Ca-10, Po4-6 and OH-2. Hydroxyapatite readily incorporates trace minerals into its crystal lattice. These ions can be negatively charged, such as fluoride bicarbonate or positively charged such as sodium, zinc, strontium or potassium. The concentrations in these trace minerals change the solubility of enamel. For example, the presence of fluoride in the crystal structure strengthens the structure and decreases the solubility. While carbonate

incorporation increases solubility. It's been found that hydroxyapatite crystals have more fluoride and less carbonate than crystals in the interior, making the outer surface less soluble than deeper layers of the enamel.

The inorganic, organic and water components of enamel are highly organized. Millions of carbonated hydroxyapatite crystals are arranged in long thin structures called rods, that are four to eight microns in diameter. Feuding cross sections, these rods appear key holed shaped structures. It's estimated that the number of rods in a tooth ranges from five million in the lower lateral incisor to 12 million in the upper first molar.

In general, rods extend at right angles from the dental enamel junction, the junction between the enamel and the layer below it called dentin, to the tooth surface. Surrounding each rod is a rod sheath made up of a protein matrix of enamelins. The area in between these rods is referred to as interrod enamel or interrod cement.

While it has the same crystal composition, crystal orientation is different, distinguishing rods in the interrod enamel. Minute spaces exist where crystals do not form between the rods, typically called pores. They contribute to enamel's permeability. Which allows fluid movement and diffusion to occur. But they also cause variations in density and hardness in the tooth. Which can create spots that are more prone to demineralization, the loss of calcium and phosphate ions, when the oral pH becomes too acidic.

Enamel is formed by an epithelial cells called ameloblasts. Just before a tooth erupts from the gums, the ameloblasts are broken down, removing enamel's ability to regenerate or repair itself. This means that when enamel is damaged by injury or decay, it cannot restore itself. When a tooth erupts, it is also not fully mineralized. To completely mineralize the tooth, calcium, phosphorus and fluoride ions are taken up from saliva to add a layer, up between 10 and 100 microns of enamel over time.

Dentin is a hard, light yellow, porous layer of tissue directly underneath enamel and cementum. Dentin constitutes the largest portion of the tooth and consists of approximately 70 percent in inorganic matter and 30 percent organic matter and water. Its organic matter is calcium and phosphate ions that form hydroxyapatite crystals as an enamel. But crystals are 30 times smaller, making dentin somewhat softer than enamel. Unlike enamel, dentin is a living tissue with the ability for constant growth and repair. This is made possible by the presence of odontoblasts. Which are cells on the outer layer of the pulp whose biological function is the creation of new dentin.

Tiny dentinal tubules that run between the cementoenamel junction of the interface of crown enamel and the tooth roots cementum and the pulp layer beneath it assist in this regenerate-, regeneration process. Odontoblasts processes in the pulp layer region of the tubules, creating new dentin and mineralize it. Nerves also fast release dentinal tubules, allowing dentin to transmit pain, unlike the enamel… Demineralization consists of bacterial acid production, acid and hydroxy, hydroxyapatite solubility and the role of critical pH.

Bacteria aggregate in dental plaque on the outer surface of teeth. Bacteria convert glucose, fructose and sucrose into acids through a process called glycolysis. Which is the main energy generating pathway in all bacteria, including caries associated streptococcus mutans. In the figure below, monosaccharides glucose, galactose and fructose can enter the glycolysis pathway at the point shown in the diagram. The dotted lines in the pathways indicate that there are additional intermediate steps. Streptococcus mutans is capable of metabolizing pyruvate or pyruvic acid further to generate yet more energy and more acid by products. When excess sugars are available, they favor the lactate dehydrogenase pathway to produce lactic acid. This causes the pH in the immediate environment of the tooth to decrease rapidly, making saliva and the interbacterial fluid in dental plaque more acidic.

How quickly acid is produced is due in part to the microbial composition of the dental plaque. In general, the more acidogenic aciduric bacteria, such as strep mutans, are present in plaque, the faster the acid is produced. The rate of acid production is also depended on the

speed at which plaque bacteria are able to metabolize the dietary carbohydrate. While sucrose is metabolized quickly, prompting a rapid decrease in pH, a large molecule, such as starch, diffuses into plaque more slowly because of a need to be broken down before it can be assimilated by the plaque microbes.

The rate of acid production is also influenced by the density of plaque. Less dense bacteria plaque, which can be penetrated by buffering saliva and oxygen, produces less acid and a very dense plaque. Which cannot be assessed or accessed by saliva and oxygen. When saliva is... or when sugars are not available, typically between meals, bacteria use their energy reserves and for formic and acetic acids in the process. These are weaker acids that are not associated with damage to the tooth structure.

The solubility of hydroxyapatite is greatly affected by the pH of all fluids. In general, a more acidic environment causes hydroxyapatite to become more soluble. While a less acidic environment makes hydroxyapatite less soluble. In a healthy oral environment that is not undergoing an acidic challenge due to dietary, gastric or medicinal related acids, plaque fluid and saliva are super saturated with calcium, phosphate and hydroxyl ion, preventing the dissolution of tooth enamel.

Despite the super saturation however, hydroxyapatite crystals do not continuously grow on the tooth surface. This is because saliva contains protein inhibitors, hydroxyapatite crystal growth, such as [statherin] as well as proline-rich proteins that coat the enamel surface and prevent seeding by exposed crystals. If an acid challenge crosses plaque fluid in saliva to become increasingly more acidic, calcium phosphate and hydroxyl ions combine with hydrogen, removing these ions from the solution. The solution, therefore, becomes undersaturated with respect to hydroxyapatite and tooth hard structure dissolves. The more undersaturated plaque fluid and saliva is, the greater the amount of dissolution.

In general, the solubility of dental hard tissues increases by a factor of 10 with a drop of every single pH unit. Dissolution continues until saturation is established once again. Because dental plaque is in close proximity to the tooth and generally prevents access of saliva to enamel, more attention is paid to the level of supersaturation in the enterobacterial fluid in dental plaque. This fluid loses at two percent saturation very quickly in a response to exposure to sucrose and becomes more unsaturated as a concentration of sucrose increases.

Frequent sucrose exposes-, exposures the cause the pH of dental plaque fluid cycle up and down repeated and quickly, have been found to deplete calcium reservoirs in plaque. This promotes pH induced under saturation. Which increases the cariogenic potential of plaque fluid. This is why the frequency of sugar intake is considered more harmful that total sugar intake when it comes to caries. Critical p.h. is a term given to the highest pH which there is a net loss of minerals from tooth enamel. This is the pH at which saliva and plaque fluids cease to saturated with calcium and phosphate. Thereby permitting hydroxyapatite to dissolve. Critical pH is generally accepted to be around five point five. But it can be a little higher or lower, depending on individual factors. During the demineralization process, acids defuse between the rods and reach deep areas of the enamel and into dentin, where carbonated hydroxyapatite crystals are more susceptible to demineralization.

The calcium and phosphate ions that are lost from the tooth defuse out into the dental plaque fluid and saliva. If the acid attack is chronic and prolonged, progressively greater amounts of calcium and phosphate minerals defuse out of the tooth, causing the crystal and structure of the tooth to shrink in size, while pores in large. Eventually a cries lesion develops. It's rate of development is a function of the degree of under saturation of fluid in its environment and rates of diffusion of ion into and out of the enamel.

The initial stages of the carious lesion are characterized by a partial dissolution of the tissue, leaving a two to 50 micron thick mineralized surface layer and a subsurface

lesion with a mineral loss of 30 to 50 percent extending into enamel and dentin. In a clinical examination, the lesion will appear chalky white and softened. In practice, the goal is to stop the process at the white spot lesion stage. While intervention can still be a non-surgical.

If the lesion advances, the outer enamel layer can eventually become capitated. At this point, the lesion is not reversible and requires operative intervention. Besides, um, observing an obvious hole in the tooth during a clinical examination, the dental professional might also notice that an advanced lesion will fill sticky or soft when gently touched with a dental probe.

Demineralization in special populations. Early childhood caries or ECC effects primary, maxillary in tee... interior teeth. It occurs when sugared liquids, including milk, lay against the anterior region of the mouth for prolonged periods, such as when a child is allowed to fall asleep with a bottle or while nursing. This is why ECC is also termed Nursing Bottle Caries, Baby Bottle Caries or Nursing Caries.

According to epidemiological data presented at the most recent national health and nutrition examination survey or NHANES, ECC affects one percent of children by the age of 12 to 24 months and five percent of children by 35 months. The condition is more prevalent in poor people and in people of minority races, with as many as 80 percent of children under five in the Native American population have ECC.

With age, the gingival tissues recede below the edge of the enamel, exposing the dentin or cementum. These layers are much more soluble than enamel and therefore, are more susceptible to acid attack. This is why root caries are more prevalent in geriatric patients. In this population, decreased salivary flow due to age or medications and a change in diet to softer foods, can make root caries difficult to manage. The following table summarizes the differences between enamel caries and root caries. And the differences are quite marked. So, as you can see from, from the, the list in the table.

There are also conditions can affect the formation of enamel. And thus, increase the risk of demineralization. These include the genetic disorder amelogenesis imperfecta. In which enamel is never completely mineralized and flakes off easily, exposing softer dentin to cariogenic bacteria. Other conditions are linked with increased enamel demineralization, such as gastroesophageal reflux disease or GERD and eating disorder bulimia, because stomach acids in the oral cavity is highly acidic.

Other special needs patients include those with an inability to remove plaque because of a mental or physical limitation. In patients with xerostomia or dry mouth due to certain medications, cancer therapies or conditions such as Sjogren's syndrome. Here's a short video by Dr. George Stookey on diseases that affect salivary flow in caries.

While one could list a, a group of diseases that, that affect dental caries or one could simply categorize the diseases into a single category, that involves simply those that, either through direct, uh, involvement or through treatment, affect salivary function and salivary flow. Anything that reduces salivary flow will surly increase dental caries. Caries attack rates and the absence of saliva can be, uh 10 to 20 times as great as, uh, as a normal individual.

An example of a condition like that could be, for example, uh, cancer of, uh, say the parotid gland or, or in the head and neck, uh, area. Where radiation treatments are used to, uh, to treat the disease. Radiation treatments will, uh, destroy, uh, the salivary glands and their ability to function. And so, you inevitably have reduced salivary flow and, and enormous cries attack rates. Uh. In one month equivalent to what would normally be seen in, in perhaps two years in, in an individual. Other diseases could be those that, um, that effect renal function and, and, uh, diabetes, uh, anything that causes a perception of dry mouth or the medications that, that, uh, are used to treat any disease. Um.

They may also reduce salivary flow and it's recognized that the majority of medications that are prescribed for, for, for medical reasons, um, have the, the ability to reduce or the, side effect of reducing salivary flow. So, patients on medications are, are almost certainly at risk for increased caries.

A key step in the remineralization process is recovery of plaque pH to a level that is higher than critical pH. The factors that affect this include the buffering capacity of saliva, whether fermentable carbohydrate remains in the mouth and the diffusion of acids from plaque with the saliva or teeth. It's also includes by the production of bases in plaque. Anomia from the deamination of amino acids and the breakdown of the urea and saliva are examples of reactions that make plaque pH less acidic. These bases are important to neutralize acid when carbohydrate intact is moderate.

The rise in pH to a less acidic level may also be assisted my the removal of acids by bacteria. For example, the bacterial genus Veillonella use lactate as a sub-straight, metabolizing its less acidic products, such as propionic acid, and acetic acid. Here's another short, but excellent, video with Dr. George Stookey on the topic of remineralization and fluoride.

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Remineralization. When plaque pH rises above five point five, remineralization can start to occur. Above this pH, interbacterial plaque fluid and saliva return to be saturated and then supersaturated with respect to the hydroxyapatite. Remineralization of dental lesions requires the presence of partially demineralized crystals that can grow to their original size when exposed to fluid that is supersaturated with respect to the hydroxyapatite minerals.

Because the carious lesion contains partially demineralized crystals, it's possible for it to become remineralized. Considerable remineralization of the surface of carious lesions has been observed. However, due to slow diffusion, it's difficult to maintain a high level of supersaturation in deeper layers of the enamel. So, remineralization of the lesion body can be quote slow, if it occurs at all. The surface layer of the lesion that has been remineralized therefore prevents the lesion body from being further demineralized. But it also inhibits it's remineralization.

The process by which demineralized crystals grow to become remineralized is quite complicated. During the period of supersaturation, crystal growth is possible as demineralized crystals grow to become remineralized is quite complicated. During the period of supersaturation, crystal growth is possible as demineralized crystals see new crystals from solution.

But crystal growth is susceptible to poisoning by foreign substances and hydroxyapatite growth inhibitors, present in saliva, can interfere with the process. Therefore, newly precipitated crystals, are usually very small and contain many defects, such as mission ions, which make it more soluble. Crystals that are predominately bathed in a large valuable solution, saturated with respect to

hydroxyapatite will tend to perfect themselves as thy become remineralized. The soluble parts will reform and crystals will grow to reach their maximum natural size in a process called Ostwald ripening.

In a clinical examination, a remineralized carious lesion will initially appear as a white scar under a shiny hard surface. This is because the surface layer of the lesion is remineralized. But the lesion body is not, as explained previously. The fate of white spot lesion is worth noting.

Because of its water content and flow rate, saliva physically cleanses the oral cavity of food and debris, removing sources that promote acidity as well as dilutes and removes organic acids from dental plaque. Saliva also contains a number of electrolytes and organic molecules that minimizes decreases in local pH, creating an environment that favors remineralization. For example, sodium bicarbonate and phosphates, along with other salivary components, act as buffers or neutralizing agents in saliva. In addition, one salivary protein called sialin, tends to raise salivary pH to neutral levels. Saliva is also supersaturated with calcium and phosphate ions, increasing the likely hood of remineralization.

Key factors that promote remineralization. The primary one, of course, is fluoride. This mineral primarily exerts it's well known anticaries effects by reducing demineralization and enhancing remineralization. When fluoride is present in low concentrations in saliva and plaque fluid, fluoride ions are likely to be incorporated into the remineralizing surface of the lesion, making the repaired section higher in fluoride than it originally was.

The material formed on the surface of the legion is more accurately called fluorapatite, a more stable and less soluble mineral that has protected the lesion body underneath. It also binds firmly with calcium, making it less likely that calcium ions are pulled out of the tooth and into the solution. When saliva and plaque fluid is supersaturated, with respect to fluoride, and when fluorapatite is formed, it has been found the damage to tooth structure does not start occurring at a pH of five point five. But rather at a more acidic four point five, emphasizing fluoride's protective effect. The overall effect is reduced dental demineralization as a result of the protective outer layer of fluorapatite. If fluoride is not available, the oral environment begins to favor demineralization.

In the United States, fluoride is most commonly delivered systemically via the water supply or topically in the form of over the counter or prescription fluoride dentifrices and mouth washes. In more serious cases, professionally applied fluoride varnishes, gels, forms or slow release applications may be necessary… Here's a graphic that shows important factors related to the remineralization process. Conditions in both the biofilm and in the enamel itself, will help dictate whether or not local conditions are suitable for the remineralization process to take place.

So, in conclusion, demineralization and remineralization are dynamic processes of mineral loss from the hard tissue of the tooth and of its repair. These are not distinct processes. Rather, both occur, to some extent, on tooth surfaces at any given time. Which process wins is dependent on several factors in the oral environment, such as frequency of sucrose consumptions and the status of saliva and whether these factors create an environment that favors demineralization or remineralization. Clearly, the goal in dental practice is to help the patient maintain an oral environment that both prevents demineralization and enhances remineralization to prevent the formation of caries.

Just as a quick reference guide, here is a series of seven figures that provide a brief overview of the various stages of lesion initiation and progression. These may prove useful to you while describing the lesion formation and reversal process to patients… Let's conclude this section by discussing how this information can help you in your practice. First, fully understanding the processes of remineralization and demineralization will help you clearly identify evidence based and

scientifically supported interventions to reduce subsurface mineral loss in making decisions regarding your patient's at home care and reduction of caries risk.

Second, information about remineralization and demineralization when communicated at the level of the patient, can be a powerful tool when driving compliance and overall adherence to your at home oral care recommendations. Describing how caries develop and making the connection to your specific recommendation instills a strong sense of trust and confidence in patients and can be far more powerful than simply instructing patients to brush their teeth more often. Thank you very much.