Caries Process, Prevention, and Management: Erosion

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- Dr. Amaechi reports no conflicts of interest associated with this course. He has no relevant financial relationships to disclose.

Caries Process, Prevention and Management consists of a series of ten continuing education courses that may be taken individually or as a complete series. This 2025 newly updated series was first developed in conjunction with the American Dental Education Association in 2008 and has now been comprehensively revised by authors who are members of the board of directors of the American Academy of Cariology (AAC) to reflect the current knowledge and best practices and quality depth of knowledge on the caries process, prevention, and management. Procter & Gamble is delighted to provide this resource enabling high quality educational experiences and an up-to-date understanding of both the science and its relevance to all clinicians in practice and we thank the authors for their contributions. Click *here* to learn more about the AAC.

Course Contents

Overview

This is part 7 of a 10-part series entitled Caries Process and Prevention Strategies. This course establishes the concept of erosive tooth wear (ETW) as a condition that is distinct from caries, and as a public health issue with increasing prevalence in people of all ages. The mineralized tooth substance (dental enamel, dentine and cementum) can be worn through either physical processes, such as attrition and abrasion or chemical processes triggered by the exposure to acids not derived from oral bacteria. These three processes are discussed, as well as the chemical, biological, and behavioral factors that increase or reduce risk of tooth surface loss. In addition, diagnosis, monitoring methods, prevention measures, and ways of protecting erosion-affected surfaces from further erosive damage are introduced.

Learning Objectives

Upon the completion of this course, the dental professional should be able to:

- Describe why tooth wear poses a serious public health issue.
- Discuss the difference between physical and chemical wear on hard dental tissues
- Describe the process by which erosion can occur.
- Be familiar with how to measure and monitor Erosive Tooth Wear.
- Discuss the factors that may predispose an individual to the risk of Erosive tooth wear.
- Be familiar with how to diagnose Erosive Tooth Wear.
- Describe factors that may influence or modify the occurrence of Erosive Tooth Wear.
- Advise the patient on the diet, behavioral, and medical factors that can reduce dental Erosive Tooth Wear.
- Describe the possible ways you may protect affected tooth tissue from further erosive damage.

Glossary

Buffering capacity – Buffering properties of an aqueous solution are a measure of resistance to pH change, and can be represented by titratable acidity, which is the amount of base, given in mmol/l, needed to raise the pH to a defined level (normally 7.0). And buffering capacity is the buffering at the pH of the investigated solution.

Chelation – Chelation is the ability of a molecule to form complex with an ion. As it applies to the oral cavity, chelation is the process whereby citric acid has the ability to demineralize enamel to a much greater degree than its pH can explain. Through its chelating properties, citric acid removes calcium from the enamel surface, and through chelation forms a compound from which the calcium cannot be released. Therefore, the calcium is not available to diffuse back into the tooth. Citric acid also has the ability to chelate calcium in saliva, reducing the remineralizing effect.

Demineralization – The chemical process by which minerals (mainly calcium) are removed from the mineralized tooth substance – enamel, dentin, and cementum. The chemical process occurs through dissolution by acids or by chelation, and the rate of demineralization will vary due to the degree of supersaturation of the immediate environment of the tooth and the presence of fluoride. In optimal circumstances, the minerals may be replaced through the process of remineralization.

Dental Abrasion - Dental abrasion is the physical loss of mineralized tooth substance caused by objects other than teeth.

Dental Attrition - Dental attrition is the physical loss of mineralized tooth substance caused by tooth-to-tooth contact.

Dentin hypersensitivity – Tooth pain that is characterized by brief, sharp, well-localized pain in response to thermal, evaporative, tactile, osmotic, or chemical stimuli that cannot be ascribed to any other dental disease or condition. Exposed dentin is a feature, and therefore the condition is associated with enamel wear (usually erosion) or gingival recession.

Developed countries – A term not frequently used today in classifying countries, as no definitive definition exists. The term is used to describe countries with industrialized economies and higher levels of gross domestic product. Developed countries are able to spend more on health systems. These systems are typically treatment-oriented and focus services on the needs of the individual rather than the community.

Erosive tooth wear – Erosive tooth wear is the chemical loss of mineralized tooth substance caused by the exposure to acids not derived from oral bacteria and/or by chelation.

Fluorapatite – A crystal structure in tooth mineral (Ca10 (PO4)6 F2) resulting from the replacement of hydroxyl ions (OH-) in the hydroxyapatite structure with fluoride ions (F-). Fluorapatite (also commonly referred to as fluoroapatite, fluorhydroxyapatite or fluorohydroxyapatite) is stronger and more acid resistant than hydroxyapatite.

GERD – Gastroesophageal reflux disease; the reflux of hydrochloric acid generated in the stomach that travels to the mouth. Erosive tooth wear will occur upon the frequent contact of the acid with enamel surfaces.

Hydroxyapatite – Crystals of calcium phosphate – (Ca10 (PO4)6 OH2) that form the mineral structure of teeth and bone. Enamel comprises approximately 98% hydroxyapatite (by weight). Much of the hydroxyapatite in enamel, however, is a calcium-deficient carbonated hydroxyapatite, the crystals of which are readily dissolved by acids. The addition of fluoride creates fluorapatite, which is less soluble and more acid-resistant.

Ions - Atoms or molecules that carry either a positive or a negative electric charge in a solution. For example, sodium chloride (NaCl, common table salt) in water dissociates into Na+ and Cl– ions.

Prevalent – Widespread; widely or commonly occurring.

Remineralization – The chemical process by which minerals (mainly calcium) are replaced into the substance of the mineralized tooth substance – enamel, dentin and cementum. The process requires an ideal environment that includes supersaturation with calcium and phosphate ions, and adequate buffering. In the presence of fluoride, remineralization is enhanced.

Tooth wear - The cumulative surface loss of mineralized tooth substance due to physical or chemo-physical processes (dental erosion, attrition, abrasion). Does not include surface loss due to dental caries, resorption or trauma.

Xerostomia – A subjective assessment of mouth dryness, usually but not always associated with low levels of saliva production. Inadequate production of saliva occurs for many reasons, most commonly as an unwanted effect of many prescription and over-the-counter medicines. Saliva is necessary for maintaining a healthy mouth, and, in relation to dental caries, is essential for remineralization.

Introduction

Large epidemiological data has shown a considerable percentage of the general population to be affected by or at least to be at risk of developing Erosive tooth wear (ETW).[1-3] This is not surprising considering that the acids leading to ETW can have multiple sources, including different health conditions, dietary habits as well as specific occupations or activities. Clinical data suggest that ETW leads to substantial and irreversible loss of tooth structure, affecting dental functions and esthetics, and may potentially lead to dentin hypersensitivity, all of which affect the quality of life of the affected individuals. As high as 97.6% of ETW prevalence has been reported in European adults[1] and 30-50% in children,[2-3] while the limited ETW data available for the United States, derived from a national survey conducted more than 20 years ago (2003-2004 NHANES), had also shown alarmingly high prevalence among teenagers (46%)[4] and adults (80%).[5] These figures have strongly suggested that ETW is a serious public health issue that is highly prevalent in developed countries. It is now necessary, regarding this increasing report of the incidence and prevalence of ETW, that dental health professionals should be familiar with the etiological and predisposing factors of ETW as well as its prevention and management.

Clinical Significance Snapshots

Is ETW really a concern for me and my patients?

Erosive tooth wear (ETW) is a public health issue with increasing prevalence in people of all ages. ETW affects a considerable part of the general population, and can be found among all categories of patients, depending on exposure to the risk factors. However, because in ETW the teeth are practically rinsed with acids, it is difficult for plaque microorganisms to tolerate the low pH level that causes ETW. For this reason, ETW-affected tooth surfaces are plaque-free, unlike caries that occur on plaque-covered surfaces, and as such, it can occur in people that maintain good oral hygiene. Furthermore, as life expectancy and dental awareness increase, people's teeth last longer and must work harder. Wear and tears are only natural, but teeth today have more work to do over a longer period than previous generations that had teeth extracted and wore dentures. Nevertheless, there are categories of individuals that are more prone to ETW.

The people adopting healthier diets that include more fruits and vegetables, as well as their juices, many of which are acidic, are putting themselves at an increased risk of ETW. In addition, increased consumption of carbonated beverages, with sugar, or sugar-free, and a concurrent decrease in milk consumption has led to increased acid intake and a reduced calcium intake.

Saliva is one of our main defenses against acid attack. Yet more patients are taking an increased number of prescriptions and over-the-counter medications, more than 85% of which can reduce saliva flow, and thus decrease this line of defense. Reduced saliva flow during sleep when saliva flow rate is very low may exacerbate ETW in patients suffering from gastro-esophageal reflux disease (GERD) due to passive regurgitation causing repeated direct contact of teeth with gastric contents, the pH of which can be as low as 1, resulting to acidic dissolution of the mineralized tooth substance.

Early and minor ETW has a smooth and silky-shiny appearance without no noticeable signs or symptoms, thus patients can hardly detect it. Even when detected, patients hardly seek treatment until it gets to an advanced stage when it either becomes symptomatic (dentin hypersensitivity) or affects the esthetics of their teeth. For this reason, dentists and dental hygienists may be the first to detect it on routine dental examination. However, early or minor ETW is a call for ETW risk assessment to reveal the cause of the problem. Other indicators of ETW are yellowing and loss of whiteness, change in the surface texture, absence of Perikymata, changes in shape, and loss of occlusal contact and/or occlusal height when advanced in relation to the patient's age.

Is erosive tooth wear the most common form of tooth wear?

Tooth wear is the cumulative surface loss of mineralized tooth substance due to physical or chemo-physical processes. Evidence of ETW can be found in almost every mouth and may co-exist with the other physical forms of tooth wear (ETW, attrition, abrasion, Non-carious Cervical Lesions). Although dental abrasion, which is the physical loss of mineralized tooth substance caused by objects other than teeth, can be distinguished from ETW, which is the chemical loss of mineralized tooth substance caused by frequent contact between acids and the mineralized tooth substance, ETW hardly occurs alone. The acidic erosive agents soften the mineralized tooth substance and decrease its wear resistance, thus rendering it more susceptible to abrasion from either the surrounding oral soft tissues, through food mastication or inappropriate use of toothbrushing/toothpaste, particularly if used immediately after an acid attack. In this case, the clinical signs often lead to an incorrect diagnosis of "toothbrush abrasion." Abrasion is commonly associated with cervical region (gingival margin). This association has been linked to Non-Carious Cervical Lesion (NCCL), which are V-shape cavities seen running horizontally along the gingival margin of some teeth in some patients. NCCL has been demonstrated to be created with horizontal toothbrushing with toothpaste. Toothbrushes without toothpaste do not create these lesions, and no correlation between firmness of toothbrush or abrasive index and the size of the NCCL. With similar process, dental attrition, which is the physical loss of mineralized tooth substance caused by tooth-to-tooth contact, as in bruxism, can be exacerbated by softening of the mineralized tooth substance by the acidic erosive agents, thus reducing its wear resistance.

It is imperative to assess erosive risk factors in all patients demonstrating signs or symptoms of tooth wear, no matter how obvious the diagnosis may be. Seldom does any one element of tooth wear occur alone.

How is dental erosion linked to my patient's oral hygiene?

Following the exposure of enamel to acid (of dietary or gastric source), the surface layers of enamel is softened, making it susceptible to physical loss until it is remineralized and rehardened by the natural processes of saliva. If the oral hygiene procedure of toothbrushing with toothpaste is performed before this remineralization, the oral hygiene procedure may lead to physical removal of some of the softened material, which leads to irreversible loss of the tooth substance. With this in mind, the common practice among individuals of toothbrushing with toothpaste as either a means of refreshing their mouth after exposure to erosive agent (vomiting or regurgitation as the case with an eating disorder) should be discouraged. Routine morning oral hygiene of toothbrushing with toothpaste by patients suffering from GERD, should also be discouraged. Instead, patients should be advised to immediately use a remineralizing mouthrinse to enhance rapid remineralization of the softened tooth surface as well as serve as a mouth refresher. The use of time-delay technique, such as allowing at least 60 minutes before brushing as advised by some practitioners, to achieve remineralization by natural saliva alone may not be the best, considering it has been demonstrated that the softened tooth tissue may be worn by abrasion from the surrounding oral soft tissues and/or mastication before it can be remineralized by the slow process of saliva remineralization. Moreover, it is not feasible to obtain patients' compliance with a time-delay technique without provision of an alternative mouth refresher.

Tooth Wear: Interaction between Physical and Chemical Tooth Substance Loss

While ETW is the physical result of frequent contact between acids and the tooth surface, abrasion is a non-physiological wearing away of the mineralized tooth substance through mechanical processes involving substances repeatedly contacting the teeth. However, ETW hardly occurs alone; softening (demineralization) of the mineralized tooth substance by acidic challenge (erosive agent) decreases the wear resistance of the surface, thus rendering it more susceptible to abrasion from either the surrounding oral soft tissues, through food mastication or tooth-brushing.

Physical Wear – Abrasion, Attrition, Non-Carious Cervical Lesion

Abrasion is commonly associated with cervical region (gingival margin). This association has been linked to Non-Carious Cervical Lesion (NCCL), which are V-shape cavities seen running horizontally along the gingival margin of some teeth in some patients. Studies have shown that NCCL is created with horizontal brushing with commercial toothpastes.[6] Toothbrushes without toothpaste do not create these lesions. There is no correlation between firmness of toothbrush or abrasive index and the size of the NCCL; however, the various shapes of NCCL are due to toothbrush filament deflection.[6] The amount and direction of filament deflection is affected by stiffness, juxtaposition of teeth, contours of gingival and teeth, and pressure. However, NCCL can be exacerbated by demineralization by erosive agent. Tooth wear by attrition is the physical loss of mineralized tooth substance caused by tooth-to-tooth contact without the influence of any other factor, as can be seen in bruxism. This can also be exaggerated by demineralization from erosive agents. Thus, the four tooth wear types are interrelated in the mechanism of formation.

Figure 1. Abrasion

Figure 2. Attrition

Chemical Wear (Erosive Tooth Wear)

The chemical dissolution of mineralized tooth substance can be caused by acid originating from either intrinsic or extrinsic sources. Acids of intrinsic source, coming from hydrochloric acid produced by the parietal cells in the stomach, cause ETW by gastric acid reaching the oral cavity and the teeth, and acting regularly on the mineralized tooth substance over a period of time. This may be the result of chronic vomiting, persistent regurgitation, or rumination. The acidity of the gastric juice ranges from pH 1 to 3, so it is conceivable that regurgitation or vomiting into the mouth might result in marked tooth destruction in the form of ETW. Conditions which are associated with chronic vomiting or regurgitation and therefore can predispose an individual to the risk of erosion are (a) certain medical conditions, such as gastro-esophageal reflux disease (GERD), bulimia nervosa, anorexia nervosa, cyclic vomiting syndrome (CVS), psychogenic vomiting syndrome, pregnancy-induced vomiting and (b) certain lifestyles, such as Chronic alcoholism and Binge drinking. Acids of extrinsic sources come from (a) items that are ingested, such as acidic food and beverages, (b) occupational related, (c) medications, and (d) certain lifestyles.

However, regardless of the origin of the acid, the effect is the same: a low pH environment in the oral cavity. The initial reaction is that enamel first undergoes softening, the loss of mineral from a layer extending a few micrometers below the surface. As softening progresses over time, dissolution can completely remove portions of enamel, or the whole enamel layer, exposing the dentin underneath. When dentin is exposed to acid, first there is dissolution at the junction of the peritubular and intertubular dentin. Next, there is loss of the peritubular dentin and widening of the tubule lumina. Finally, there is the formation of a demineralized collagenous mix that provides some protection of the underlying tissue. However, this layer is also vulnerable to damage and can ultimately be eroded away as well.[8]

Factors that may Predispose an Individual to the Risk of Erosive Tooth Wear

An individual can be rendered susceptible to the risk of ETW by acid-related or non-acid-related factors. Acids that could cause ETW have been described as originating from gastric, dietary or environmental sources.

Acid-related Risk Factors

Medical conditions: Some medical conditions expose the teeth to frequent and repeated direct contact with gastric contents, the pH of which can be as low as 1, so its destructive capabilities are especially severe. Conditions such as chronic vomiting in eating disorders such as anorexia and bulimia nervosa, passive regurgitation in gastro-esophageal reflux disease (GERD) and chronic alcoholism and binge drinking, have all been associated with ETW.[9] GERD are likely to be more destructive due to decrease in salivary flow during sleep.[10]

Misuse of acidic dietary products: Frequent and prolonged ingestion of acidic fruit juices and acidic beverages (alcohol, herbal teas, energy drinks) as observed in situations like habitual intake, dieting with citrus fruits and fruit juices, and drinking during strenuous sporting activities, have been associated with ETW.[11] These practices would lower the pH of the oral fluids for a prolonged period, thus exposing the teeth to prolonged period of acidic challenge with consequent dissolution of the mineralized tooth tissue. Especially problematic is the habit of swishing soda in the mouth to prevent the uncomfortable sensation of carbonation in the throat. This habit enhances the dissolution process because the solution on the surface layer adjacent to tooth mineral will be readily renewed.

Use of acidic medicaments and illegal drugs: Some medicaments such as acetylsalicylic acid, ascorbic acid, liquid hydrochloric acid, iron tonics, and acidic saliva stimulants/substitutes prescribed for frequent intake for long periods of time have the potential to soften dental hard tissues, and as such have high erosive potentials.[12,13] Addictive use of certain illegal drugs like cocaine and ecstasy have the side-effects of dehydration and hyposalivation, leading excessive consumption of acidic beverages by the users, thus predisposing these individuals to the risk of ETW.[13]

Low pH Toothpastes and Mouthrinses: Some oral care products such as toothpastes and mouthrinses exhibit pH values as low as 3.4. The acidic nature of these products is intended to enhance the chemical stability of some compounds but may also enhance the incorporation of fluoride ions in tooth mineral forms, as well as the precipitation of calcium fluoride on the tooth surface.[13] However, when tested under more clinically relevant conditions, all these products caused progressive enamel surface loss over time, similar to that of an orange juice, thus indicating these oral care products to be a risk factor for ETW.[14]

Occupation: Certain industrial processing procedures expose the workers to acidic fumes or aerosols as observed in battery and fertilizer factories, professional swimming in improperly pH-regulated swimming pools, and professional wine tasting, have all been linked to ETW.[12,15-17]

Non-acid Risk Factors

The presence of acid is not the only way by which ETW can occur. The **non-acid factors** that may predispose a person to ETW are as follows.

Excessive oral hygiene procedure: Used as directed, most toothpastes are safe. However, if used aggressively or abusively – too much force, too much paste, too frequently – the detergents and abrasive particles essential for cleaning under normal circumstances have the potential to increase physical loss of the mineralized tooth substance. Thus, frequent tooth brushing with abrasive dentifrice as practiced by some health/aesthetic-conscious individuals may render the tooth surface more susceptible to ETW due to removal of the more protective highly mineralized outer layer of tooth surface.[18] Furthermore, frequent toothbrushing may cause reduction of the thickness of the acquired salivary pellicle, which would adversely affect its established protective role against ETW.[19]

Chelating agents: Chelation is the process by which certain agents form complex with calcium to remove it from the mineralized tooth substance or complex with calcium in saliva to reduce saliva's supersaturation, thus triggering demineralization that leads to ETW. Products with chelating ability include mouthrinses that contain the ingredient EDTA, and food and beverages that contain citric acid. Up to 32% of the calcium in saliva can be complexed by citrate at concentrations common in fruit juices, reducing the supersaturation of saliva and driving the equation to dissolution of tooth mineral.[20]

Factors that Influence the Severity of Erosive Tooth Wear

Certain factors can determine the occurrence and severity of ETW among individuals as well as the erosive potential of some erosive agents. These factors can be biological, chemical, or behavioral.

Biological Factors

Oral Biofilm (acquired salivary pellicle): Acquired salivary Pellicle is a layer of structureless, homogeneous biofilm formed post-eruptively, on the surface of mineralized tooth surfaces, by the selective adsorption of hydroxyapatite-reactive salivary glycoprotein.[27] This layer forms within minutes on the surface of a tooth surface after its removal by toothbrushing, chemical dissolution, or prophylaxis.[28-30] This biofilm has been shown to protect the tooth surface against ETW by serving as a barrier that prevents the direct contact of an acid and the tooth's surface through its diffusion-limiting properties (semi-permeability) as well as serving as a reservoir of remineralizing electrolytes.[19] Figure 3 shows a scanning electron microscopy images of 2-hour formed acquired salivary pellicle before and after 10-minute exposure to orange juice where it was able to reduce ETW by the juice.[19] This ability to protect against ETW depends on its thickness and enzymatic composition.[28-30] The enzymatic composition of the pellicle also plays an important role: The presence of the enzyme carbonic anhydrase VI in the pellicle may protect against tooth erosion because it speeds the neutralization of demineralizing hydrogen ions on the tooth's surface.[30]

Physiological movement of oral soft tissues: It has been demonstrated that enamel surface softened by an erosive agent may be worn by abrasion from the surrounding oral soft tissues, such as the keratinized surface of the tongue, before it can be remineralized by saliva, with consequent loss of mineralized tooth substance and manifestation of ETW.[31-33] The most severe erosive lesions are typically found in the palatal surfaces of the upper teeth (Figure 4), because of the abrasive effect of the tongue. It has been shown that the tongue is able to remove already softened enamel and dentin.[31-33]

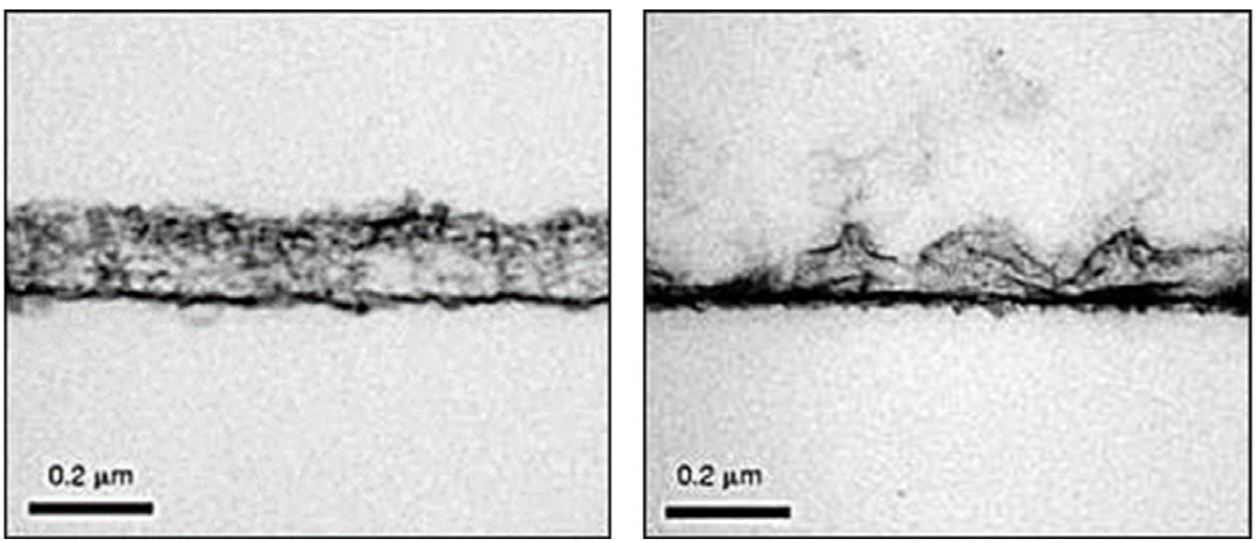

Figure 3.(A) Attrition Transmission electron microscopy image of the 2-h in situ formed pellicle on enamel surface. **(B)** The 2-h pellicle after 10 min of erosive challenge in situ by orange juice.

Adapted from: Lussi A. From diagnosis to therapy. Basil, Switzerland: Karger; 2006. Monographs in Oral Science Series. Vol 20.

Figure 4. Example of ETW on the palatal surface of upper teeth.

Figure 5. ETW on facial surface of upper incisors.

Tooth Position: The position of teeth determines their susceptibility to ETW because different sites in the mouth are affected by variations in salivary flow and composition. As such, facial surfaces of upper incisors have higher susceptibility to erosion because the exposure to saliva is lower (figure 5), while lingual surfaces of lower teeth have lower erosion susceptibility because the exposure to protective saliva is higher.[34]

Tooth Structure and Composition: Differences in the structure and chemical composition between various type of enamel (human permanent and deciduous) has been shown to play role in the development and progression of ETW.[35] Erosive lesion progressed 1·5 more rapidly in human deciduous than in human permanent enamel.[35] The fluoride and the calcium phosphate concentrations in deciduous enamel are lower than in permanent enamel formed under the same condition, with a slightly higher proportion of organic matter in deciduous enamel than in permanent enamel, indicating a lower degree of mineralization in deciduous than in permanent enamel.[36-38] Furthermore, variation in chemical composition of different areas of the enamel of deciduous and permanent teeth has long been established.[37] These variations may determine the variation in severity of ETW among individuals, different teeth within same individual, and different areas of the same tooth.

Saliva and its properties: Saliva is the most important biological factor in the prevention of ETW.[39-42] It starts acting even before the acid attack with an increase in salivary flow in response to visual or olfactory stimuli or to chewing, increasing the buffering system and diluting and clearing acids on tooth surfaces during the erosive challenge.[39] The chemical composition, pH, flow Rate, buffering capacity as well as the remineralization potential of saliva varies among individuals, and determine their susceptibility to ETW.[39-42] A low salivary flow rate due to xerostomia, dehydration, use of certain medications, salivary gland pathology, or when there are no stimuli to trigger a protective salivary response (such as when a patient is suffering with GERD) means that teeth are less protected during an acid attack. A high salivary flow rate, on the other hand, has a protective effect against acid, particularly because it has the ability to clear acids from teeth surfaces.[39,42] Effect of saliva flow rate with respect to ETW is more devastating in GERD patients because of the low saliva flow rate during sleeping and in Xerostomic patients. A higher hydrogen bicarbonate content increases the capacity of saliva in neutralizing and buffering acids to protect from ETW, while a low buffering capacity is strongly associated with increased erosion. Furthermore, saliva that is supersaturated with calcium and phosphate ions is more effective at maintaining the integrity of teeth by remineralizing the hydroxyapatite in enamel, while saliva that is undersaturated with calcium and phosphate cannot replenish enamel's mineral content.[34,39] The degree of supersaturation of hydroxyapatite, fluorapatite and calcium fluoride also increases as saliva flow is stimulated and increases. It is also important to note that sites poorly bathed by saliva or mainly bathed with mucous saliva (which typically contains fewer mineralizing ions) are more likely to show ETW when compared to sites protected by saliva that is serous in nature.[34,43]

Chemical Factors

The chemical factors involved in the development of ETW relate to the parameters inherent to erosive beverages, food, or other products, such as their pH, buffering capacity, acid type, as well as the calcium, phosphate, and fluoride concentration.

pH and Buffering Capacity: With regards to the pH and buffering capacity of the products, the greater the buffering capacity of an edible item, the longer it will take for saliva to neutralize the product's acid. So, a beverage with a higher buffering capacity will be more erosive than others within the same pH class. Even if a product is at a low pH, it is possible that other factors are strong enough to prevent ETW. Similarly, it is also possible that a less acidic product can cause ETW because it has the capacity to complex calcium, pulling the mineral out of the tooth surface to cause demineralization. While pH is an important factor, there is no specific pH of a product below which damage will occur.[11,44]

Acid Type: The erosive character of lactic and citric acid in products is higher than that of acetic, maleic, phosphoric, and tartaric acids.[11,45]

Calcium, Phosphate, and Fluoride Concentration: Solutions oversaturated with respect to dental hard tissue will protect against dental surface softening.[11,46,47] A low degree of undersaturation with respect to enamel or dentin leads to a very initial surface demineralization which is followed by a local rise in pH and increased mineral content in the liquid surface layer adjacent to the tooth surface. This layer will then become saturated with respect to enamel and will not demineralize further. A high degree of undersaturation with respect to dental tissue will demineralize the tooth surface considerably more.[11,46,47]

Consistency of the product: The stickiness of the product being consumed, with more sticky products generally being linked to higher erosion risk.[11]

Behavioral Factors

The manner in which dietary acids are introduced into the mouth, such as sipping, gulping, swishing, or using a straw, affects how long the teeth are in contact with the erosive challenge, and how much ETW experienced by the individual.[11] Temperature of acidic drinks and beverages influences the erosive potential of the product. Cold drinks have been demonstrated to have less erosive potential than a warm drink.[35] Frequent and prolonged consumption of acidic drinks and beverages results in ETW.[35] Even cosmetic procedures may pose a problem: Some whitening gels have been found to soften and alter enamel, suggesting an increased susceptibility to dental erosion.[48]

How to diagnose Erosive Tooth Wear

Erosive tooth wear is a surface phenomenon, and therefore the diagnosis can be easily made by visual inspection.[21-24] In initial stages, the normal surface texture and lustre of enamel is lost with the loss of Perikymata, making the enamel surface to exhibit a silky-shiny appearance. With continued acid impacts, convex tooth structures flatten, and distinct defects develop, with erosive lesions on smooth surfaces, which are mainly located coronal to the enamel-cementum junction, being flat, dull, with an intact enamel rim along the gingival margin, and exhibiting shallow concavity that the width clearly exceeds its depth. On occlusal and incisal surfaces, the early erosive lesions are characterized by flattened and cupped cusps and restorations may stand proud of the surface. In advanced stages, the occlusal morphology may completely disappear, with involvement of dentin.[21-24]

Erosive Tooth Wear Risk Assessment

An important step towards prevention of ETW should be the identification of those individuals who are at risk of ETW through risk assessment. Risk assessment is the clinical process of identifying and estimating the probability of an individual developing new erosive lesions or progression of existing lesions during a specified period.[25] This would enable the identification of the individual etiologic factors contributing to the patient's existing ETW, which would provide the basis to explain to the patients how to prevent their ETW, and to communicate and facilitate needed change in behavior to prevent the problem. Risk assessment can be performed using an established ETW risk assessment tool, which guides the user through the most common risk factors associated with dental erosive wear, and when combined with the Basic Erosive Wear Examination (BEWE)[26] or other similar scoring system, leads to a final decision on patient's risk status.[25]

Qualitative and Quantitative Measuring and Monitoring

Once detected and diagnosed, the severity and extent of an erosive tooth wear must be recorded to establish the clinical baseline, so that progression can be detected. Longitudinal monitoring of the erosive process would enable the outcomes of the preventive and therapeutic strategies to be assessed either qualitatively or quantitatively at the recall and review visits. There are several qualitative and quantitative ways to measure and monitor ETW in clinical practice. The established techniques include, but are not limited to:

Basic Erosive Wear Examination (BEWE): This is a management-based index in which the severity level of a lesion is scored on a four-grade level based on its extent on the tooth surface; (0) No erosive tooth wear, (1) Initial loss of surface texture, (2) Distinct defect, hard tissue loss <50% of the surface area, and may involve dentin, (3) Hard tissue loss ≥50% of the surface area, and may involve dentin.[26,49] In BEWE, the dentition is divided into sextant and the buccal, occlusal and lingual surfaces of every tooth in each sextant is examined for ETW, awarded a score value between 0 and 3, and the highest score in each sextant is recorded. The sum of the scores from the sextants constitutes the BEWE score for the patient. The BEWE for can be used to determine the ETW risk status of the patient and the clinical management, and for longitudinal monitoring of the lesion progress. [26,49] This has the problem of inability to detect slight change in lesion severity, particularly if the progression is in depth and not in area of the lesion.

Intraoral scanners (IOS): The recent advances of digital dentistry, with the development of high-resolution tools for 3D dental imaging, and their growing access to dental practitioners have created opportunities for enhancing the detection and differentiation of dental hard-tissue conditions, including ETW lesions.[50] IOS can generate high-resolution 3D digital images of the teeth, including morphological features and photographic (colored) images. IOS can be used to capture 3D digital dental images of the teeth and then use BEWE to perform ETW assessments on the generated images.[50] This also has the problem of inability to detect slight change in lesion severity, particularly if the progression is in depth and not in area of the lesion.

Tooth Wear Index (TWI): The TWI records the degree of wear on all visible surfaces of each tooth. The severity of an erosive lesion is scored by estimating the percentage of the entire surface affected by the condition and by whether dentine is exposed or not. Unlike the BEWE, the scores from all tooth surfaces are recorded, and as such it is useful for detection and longitudinal monitoring of erosive lesion.

Silicone Matrix: This is a simple and more reliable objective and quantitative clinical method of monitoring the progression status of an erosive lesion over time. With this system, a silicone putty impression of the teeth is taken, when removed from the tray, and get sliced into sections through the centers of the erosive lesions. When a section is replaced on tooth surface, it is a perfect fit to the tooth surface, if the erosive lesion progresses, on the next review visit a gap will become visible between the silicone and the lesion surface, and the depth of the gap can be measured in millimeters (mm) using the calibrated periodontal probe (explorer). If no gap, then the lesion is arrested.[49]

Clinical photographs: Clinical photographs are simple useful, but subjective, method of monitoring erosive tooth wear. However, the dexterity of the photographer and the ambient conditions such as light reflections affect the quality of the product.[49]

Prevention and Treatment Strategies

It is pertinent to mention that it may be difficult to gain the compliance of the affected individuals to eliminate the implicated risk factors considering that such persons may have psychological or professional inclination to the factors. However, the following recommendations, if implemented in a preventive program, may prevent occurrence, limit the damage, modify habit or protect the remaining affected tooth tissue.

Early Diagnosis and Monitoring

Early detection and diagnosis would pave way for early management to prevent further progression. It is important to bear in mind that patients can barely detect early enamel erosion due to its smooth and shiny appearance, and even when detected patients hardly seek treatment until when advanced and complicated by either dentin hypersensitivity or affecting patient's esthetics. However, once diagnosed, risk assessment should be performed to discover the responsible risk factors, then the lesion should be measured, and treatment implemented, followed by monitoring on recall visits.[51]

Preventive Strategies

Referral for treatment of any implicated medical conditions: Patients that have underlying medical conditions (GERD, bulimia, substance abuse, etc) that predispose them to the risk of ETW should be referred to the medical practitioner responsible for treatment while implementing dental preventive measures.[51]

Use of patient-applied remineralizing agent: Patients should be advised to use a remineralizing oral care product, rather than toothbrushing, immediately following the intake of an erosive product. This will enhance the saliva remineralizing capacity to speed up the remineralization of the softened mineralized tooth substance, prior to toothbrushing. Although some practitioners recommend 60 minutes delay before toothbrushing, the saliva remineralizing rate is not fast enough to accomplish the rehardening of the tooth tissue within this period. Remineralization can be accomplished by using fluoride mouthrinses (or dairy products in absence of a fluoride mouthrinse) or fluoride gels.[51,52]

Use of professionally applied remineralizing agent: Further progression of the ETW can be arrested by periodic professional application of fluoride varnishes or gels to increase the resistance of the affected tooth tissue to further erosive attack.[51,53]

Condition/method of drinking: Taking an erosive drink ice-cold reduces its erosive effect,[35] while the use of a straw would reduce the contact of the teeth with the erosive agent as well as enhance the rate of clearance of the agent from oral cavity.[54] The drink should be swallowed quickly and not sipped slowly or 'swished' around the mouth.[35,51,54]

Use of modified products: Studies have shown that a drink which contains citric acid that was supplemented with calcium, phosphate, and fluoride reduced the erosive potential of the solution.[44,51,55-57] The same was true when acidic carbonated drinks were modified with these three minerals.[44,51,58] Yogurt, which is acidic with a pH of 4 hardly has any erosive effect due to its high calcium and phosphate content, which makes it supersaturated with respect to the hydroxyapatite in enamel.[44,51,58]

Use of protective devices: A close-fitting mouth guard with a neutralizing agent, such as antacid suspension, applied to the fitting surface, may protect those who are at high risk of ETW due to their occupation (while on duty) or medical condition (e.g. GERD during sleep, anorexia/bulimia during vomiting).[51,59,60]

Health Education

ETW should be prevented and managed through interprofessional (Doctor, Clinical psychologist, Pharmacist, Dietician) counseling based on the outcome of the risk assessment and the implicated risk factors. The advice should be individualized and relate to the observed etiological factor.[51,61-63]

Protecting Affected Tooth Surface from Further Erosive Damage

While the patient is undergoing treatment for the existing ETW has been instituted, one of the following treatment modalities may be considered for protection of the remaining tooth tissue from further erosive damage; dentine bonding agents, adhesively retained composite resin, porcelain veneers, or fluoridated sealants.[51,53,63] In advanced cases, the worn dentition should be managed by restorative treatment.[63]

Establishing Continue Care through Recall Visits

Following counseling, it is important to establish recall visits for continue care to prevent relapses of the condition. The recall care regime should be matched to the patient's requirements to check patient compliance, monitor wear, reinforce advice, and encourage maintenance of changed behavior.[51,63,65]

Conclusion

Erosive tooth wear is becoming increasingly prevalent, and its damaging effect is emerging as a serious public health issue. There is no doubt that mineralized tooth substance loss by erosion can become pathological, leading to complications such as pain, dentin hypersensitivity, and pulpal inflammation, if measures are not taken to prevent the loss of the mineralized tissue. Knowing the factors that promote ETW as well as preventive strategies can go a long way in averting erosion or significantly slowing its progression. The use of a remineralizing mouthrinse or lozenge specific for ETW would be particularly useful to facilitate rapid rehardening of the softened tooth tissue prior to toothbrushing as well as for effective prevention of ETW. There is a need to develop a health education program targeted towards the reduction of the prevalence of ETW through interprofessional management involving all healthcare personnel. The qualities of acidic dietary products should be modified thereby reducing their erosive potential. Becoming familiar with how to assess the level of damage and treat it to prevent the onset of related complications.

Course Test Preview

To receive Continuing Education credit for this course, you must complete the online test. Please go to: www.dentalcare.com/en-us/ce-courses/ce716/start-test

1. Which of the following statements about dental erosion is true?

A. Dental erosion is not a serious public health issue.
B. Dental erosion is caused by bacteria.
C. Dental erosion is non-bacterial chronic loss of dental tissues.
D. Dental erosion is only prevalent in less developed countries.

2. Why is dental erosion of particular concern to dentists?

A. When reparative processes can no longer protect teeth, complications can include pain, dentin hypersensitivity, and pulpal inflammation.
B. It can lead to oral cancer.
C. Dental erosion is not a serious concern for dentists.
D. It can be an indicator of future caries.

3. Which of the following is correct about attrition?

A. Attrition is only caused by pathological behavior, like tooth grinding.
B. Attrition can be physiological when it is due to normal wear or pathological when caused by certain habits of the patient, like tooth grinding.
C. Attrition only damages the premolars and molars.
D. Attrition is the wearing away of dental tissue by foreign objects in the mouth.

4. Which of the following is correct about abrasion?

A. Abrasion is the wearing away of dental tissue due to tooth-to-tooth contact.
B. Abrasion is only caused by pathological behavior.
C. Abrasion causes include oral habits like horizontal brushing with commercial toothpastes.
D. Abrasion only damages the premolars and molars.

5. What is the first step of tissue loss during the dental erosion process?

A. Enamel exposed to acid first undergoes softening, and as softening progresses over time, dissolution can remove portions of enamel or the whole enamel layer.
B. There is dissolution at the junction of the peritubular and intertubular dentin.
C. There is formation of a demineralized collagenous mix.
D. There is the widening of tubule lumina.

6. Which factor is NOT a major causes of dental erosion?

A. Chronic vomiting/regurgitation
B. Consumption of carbonated beverages
C. Consumption of acidic foods and beverages
D. High pH environment

7. Which is NOT an example of an extrinsic source of erosive acid?

A. Chewable vitamin C
B. Gastric acid
C. Mouth rinses that contain sodium chloride
D. Herbal teas

8. What percent of calcium in saliva can be complexed by citrate in fruit juices?

A. 12%
B. 47%
C. 32%
D. 81%

9. Which of the following is true about the calcium, phosphate, and fluoride concentration of a beverage?

A. Solutions oversaturated in calcium, phosphate, and fluoride with respect to dental tissue will protect against dental surface softening.
B. A low degree of undersaturation does not impact enamel at all.
C. A high degree of undersaturation will only cause an initial surface demineralization.
D. Supplementing a solution with calcium, phosphate, and fluoride does not affect its erosive potential.

10. What helps the salivary pellicle have a protective effect?

A. It is a source of remineralizing electrolytes.
B. It cannot be removed once fully formed.
C. It contains acid-neutralizing enzymes, like carbonic anhydrase VI.
D. A and C

11. On which teeth surfaces are the most serious dental erosions typically found?

A. Facial surfaces of the upper incisors
B. Lingual surfaces of the lower teeth
C. Palatal surfaces of the upper teeth
D. All tooth surfaces.

12. What is NOT a technique used to measure and monitor ETW in clinical practice?

A. Basic Erosive Wear Examination (BEWE):
B. Tooth Wear Index (TWI):
C. Silicone Matrix
D. Longitudinal Wear Index (LWI)

13. What qualities describe the appearance of enamel in early erosion?

A. Silky-shiny
B. Bumpy, dull, yellow
C. There are no detectable changes in early erosion
D. Thick, bumpy

References / Additional Resources

1. West NX, Davies M, Sculean A, Jepsen S, Faria-Almeida R, Harding M, Graziani F, Newcombe RG, Creeth JE, Herrera D. Prevalence of dentine hypersensitivity, erosive tooth wear, gingival recession and periodontal health in seven European countries. J Dent. 2024 Nov;150:105364. doi: 10.1016/j.jdent.2024.105364. Epub 2024 Sep 22. Erratum in: J Dent. 2025 Jan;152:105452. doi: 10.1016/j.jdent.2024.105452. PMID: 39317300.
2. K. Yip, P. P. Y. Lam, and C. K. Y. Yiu, 'Prevalence and Associated Factors of Erosive Tooth Wear among Preschool Children-A Systematic Review and Meta-Analysis', Healthcare (Basel), vol. 10, no. 3, p. 491, Mar. 2022, doi: 10.3390/healthcare10030491.
3. F. Marschner, P. Kanzow, and A. Wiegand, 'Systematic review and meta-analysis on prevalence and anamnestic risk factors for erosive tooth wear in the primary dentition', Int J Paediatr Dent, Jul. 2024, doi: 10.1111/ipd.13250.
4. McGuire J, Szabo A, Jackson S, Bradley TG, Okunseri C. Erosive tooth wear among children in the United States: relationship to race/ethnicity and obesity. Int J Paediatr Dent. 2009 Mar;19(2):91-8. doi: 10.1111/j.1365-263X.2008.00952.x. Erratum in: Int J Paediatr Dent. 2009 May;19(3):222. PMID: 19250393.
5. Okunseri C, Wong MC, Yau DT, McGrath C, Szabo A. The relationship between consumption of beverages and tooth wear among adults in the United States. J Public Health Dent. 2015 Fall;75(4):274-81. doi: 10.1111/jphd.12096. Epub 2015 Apr 28. PMID: 25919191.
6. Dzakovich JJ, Oslak RR. In vitro reproduction of noncarious cervical lesions. J Prosth Dent. 2008;100 (1):1-10.
7. Bartlett DW, Coward PY. Comparison of the erosive potential of gastric juice and a carbonated drink in vitro. J Oral Rehabil. 2001 Nov;28(11):1045-7.
8. Larsen MJ. Chemical events during tooth dissolution. J Dent Res. 1990 Feb;69 Spec No:575-80; discussion 634-6. doi: 10.1177/00220345900690S114.
9. Scaramucci, T., Carvalho, J.C., Hara, A.T., Zero, D.T. (2015). Causes of Dental Erosion: Intrinsic Factors. In: Amaechi, B. (eds) Dental Erosion and Its Clinical Management. Springer, Cham.
10. Ortiz AC, Fideles SOM, Pomini KT, Buchaim RL. Updates in association of gastroesophageal reflux disease and dental erosion: systematic review. Expert Rev Gastroenterol Hepatol. 2021;15(9):1037-1046. doi:10.1080/17474124.2021.1890030
11. Chan AS, Tran TTK, Hsu YH, Liu SYS, Kroon J. A systematic review of dietary acids and habits on dental erosion in adolescents. Int J Paediatr Dent. 2020;30(6):713-733. doi:10.1111/ipd.12643
12. Zero DT, Lussi A. Etiology of enamel erosion intrinsic and extrinsic factors. In Addy M, Embery G, Edgar WM, Orchardson R, eds. Tooth Wear and Sensitivity: Clinical Advances in Restorative Dentistry. London: Martin Dunitz; 2000:121-139.
13. Hellwig E, Lussi A. Oral hygiene products, medications and drugs - hidden aetiological factors for dental erosion. Monogr Oral Sci. 2014;25:155-62. doi: 10.1159/000359942.
14. Pontefract H, Hughes J, Kemp K, Yates R, Newcombe RG, Addy M. The erosive effects of some mouthrinses on enamel. A study in situ. J Clin Periodontol 2001; 28:319-324.
15. Petersen PE, Gormsen C. Oral conditions among German battery factory workers. Community Dental Oral Epidemiology 1991;19: 104-16.
16. Chikte UM, Josie-Perez AM, Cohen TL. A rapid epidemiological assessment of dental erosion to assist in settling an industrial dispute. Journal Dental Association of South Africa 1998;53:7-12.
17. Centerwall BS, Armstrong CW, Funkhouser GS, Elzay RP. Erosion of dental enamel among competitive swimmers at a gas-chlorinated swimming pool. American Journal of Epidemiology 1986;123: 641-647.
18. Hunter ML, West NX. Mechanical tooth wear: the role of individual toothbrushing variables and toothpaste abrasivity. In: Addy M, Embery G, Edgar WM, Orchardson R, editors. Tooth wear and sensitivity: Clinical Advances in Restorative Dentistry. 1st ed. London: Martin Dunitz; 2000. p. 161-169.

19. Vukosavljevic D, Custodio W, Buzalaf MA, Hara AT, Siqueira WL. Acquired pellicle as a modulator for dental erosion. Arch Oral Biol. 2014 Jun;59(6):631-8. doi: 10.1016/j.archoralbio.2014.02.002.
20. Meurman JH, ten Cate JM. Pathogenesis and modifying factors of dental erosion. Eur J Oral Sci. 1996 Apr;104(2 (Pt 2)):199-206.
21. Ganss C, Lussi A, Schlueter N. The Histological Features and Physical Properties of Eroded Dental Hard Tissues. Monogr Oral Sci. 2025;33:72-80. doi: 10.1159/000543884.
22. Ganss C, Jung K, Lussi A. How to Diagnose and Monitor Erosive Tooth Wear and When Is It an Oral Disease? Monogr Oral Sci. 2025;33:1-18. doi: 10.1159/000543573.
23. Vach K, Ganss C, Schlueter N, Vach W. Identifying clusters of raters with a common notion of diagnosing erosive tooth wear: a step towards improving the accuracy of diagnostic procedures. Eur J Med Res. 2025 Jan 9;30(1):15. doi: 10.1186/s40001-024-02260-1.
24. Ganss, C., Schlueter, N. (2015). Diagnosis of Dental Erosion. In: Amaechi, B. (eds) Dental Erosion and Its Clinical Management. Springer, Cham.
25. Young, A., Stenhagen, K.R., Mulic, A., Amaechi, B.T. (2015). Dental Erosive Wear Risk Assessment. In: Amaechi, B. (eds) Dental Erosion and Its Clinical Management. Springer, Cham. https://doi.org/10.1007/978-3-319-13993-7_7.
26. D Bartlett, C Ganss, A Lussi. Basic Erosive Wear Examination (BEWE): a new scoring system for scientific and clinical needs. Clin Oral Investig. 2008 Jan 29;12(Suppl 1):65–68. doi: 10.1007/s00784-007-0181-5.
27. Amaechi BT, Higham SM, Edgar WM, et al. Thickness of acquired salivary pellicle as a determinant of the sites of dental erosion. J Dent Res. 1999 Dec;78(12):1821-8. doi: 10.1177/00220345990780120901.
28. Hannig M, Fiebiger M, Güntzer M, et al. Protective effect of the in situ formed short-term salivary pellicle. Arch Oral Biol. 2004 Nov;49(11):903-10. doi: 10.1016/j.archoralbio.2004.05.008.
29. Hannig M, Balz M. Influence of in vivo formed salivary pellicle on enamel erosion. Caries Res. 1999 Sep-Oct;33(5):372-9. doi: 10.1159/000016536.
30. Hannig C, Hannig M, Attin T. Enzymes in the acquired enamel pellicle. Eur J Oral Sci. 2005 Feb;113(1):2-13. doi: 10.1111/j.1600-0722.2004.00180.x.
31. Amaechi BT, Higham SM, Edgar WM. Influence of abrasion on the clinical manifestation of human dental erosion. Journal of Oral Rehabilitation 2003;30:407-413.
32. Amaechi BT, Higham SM, Edgar WM. Development of an in situ model to study dental erosion. In: Addy M, Embery G, Edgar WM, Orchardson R, editors. Tooth wear and sensitivity: Clinical Advances in Restorative Dentistry. 1st ed. London: Martin Dunitz; 2000. p. 141-152.
33. Gregg T, Mace S, West NX, et al. A study in vitro of the abrasive effect of the tongue on enamel and dentine softened by acid erosion. Caries Res. 2004 Nov-Dec;38(6):557-60. doi: 10.1159/000080586.
34. Amaechi BT, Higham SM. Eroded enamel lesion remineralization by saliva as a possible factor in the site-specificity of human dental erosion. Arch Oral Biol . 2001 Aug;46(8):697-703. doi: 10.1016/s0003-9969(01)00034-6.
35. Amaechi BT, Higham SM, Edgar WM. Factors influencing the development of dental erosion in vitro: enamel type, temperature and exposure time. J Oral Rehabil. 1999 Aug;26(8):624-30. doi: 10.1046/j.1365-2842.1999.00433.x.
36. JENKINS, G.N. (1978) The Physiology and Biochemistry of the Mouth, 4th edn, pp. 54–112. Blackwell Science, Oxford.
37. Naujoks R, Schade H, Zelinka F. Chemical composition of different areas of the enamel of deciduous and permanent teeth. (The content of Ca, P, CO2, Na and N2). Caries Res. 1967;1(2):137-143. doi:10.1159/000259508
38. STACK MV. Variation in the organic content of deciduous enamel and dentine. Biochem J. 1953;54(2):xv.
39. Carvalho TS, Niemeyer SH, Young A, Lussi A. Factors Related to Erosive Tooth Wear throughout a Lifetime. Monogr Oral Sci. 2025;33:149-177. doi: 10.1159/000543570. Epub 2025 May 28.

40. Hannig C, Hannig M. Interaction between Saliva, Pellicle, and Dental Erosion. Monogr Oral Sci. 2025;33:128-148. doi: 10.1159/000543558.
41. Nobre M, Almeida L, Magalhães M, Carvalho R, Rua J, Proença L, Vieira AM. Tooth Wear and Salivary Factors: Insights from a Cohort of Dental Students. J Clin Med. 2025 Mar 13;14(6):1936. doi: 10.3390/jcm14061936.
42. Madariaga VI, Pereira-Cenci T, van Gennip LLA, van Leeuwen SJM, Walboomers XF, Loomans BAC. Exploring the relationship of salivary pH and flow rate with tooth wear severity: A cross-sectional study. J Dent. 2025 Jan;152:105499. doi: 10.1016/j.jdent.2024.105499.
43. Young WG, Khan F. Sites of dental erosion are saliva-dependent. J Oral Rehabil. 2002 Jan;29(1):35-43.
44. Hara, A.T., Carvalho, J.C., Zero, D.T. (2015). Causes of Dental Erosion: Extrinsic Factors. In: Amaechi, B. (eds) Dental Erosion and Its Clinical Management. Springer, Cham.
45. Hannig C, Hamkens A, Becker K, et al. Erosive effects of different acids on bovine enamel: release of calcium and phosphate in vitro. Arch Oral Biol. 2005 Jun;50(6):541-52. Epub 2005 Jan 7. doi: 10.1016/j.archoralbio.2004.11.002.
46. Larsen MJ. Dissolution of enamel. Scand J Dent Res. 1973;81(7):518-522. doi:10.1111/j.1600-0722.1973.tb00358.x
47. Larsen MJ, Nyvad B. Enamel erosion by some soft drinks and orange juices relative to their pH, buffering effect and contents of calcium phosphate. Caries Res. 1999;33(1):81-87. doi:10.1159/000016499
48. de Freitas PM, Turssi CP, Hara AT, et al. Monitoring of demineralized dentin microhardness throughout and after bleaching. Am J Dent. 2004 Oct;17(5):342-6.
49. Amaechi, B.T. (2015). Assessment and Monitoring of Dental Erosion. In: Amaechi, B. (eds) Dental Erosion and Its Clinical Management. Springer, Cham.
50. Machado AC, Phillips TS, Zimmerman R, Scaramucci T, Amaechi BT. Monitoring erosive tooth wear with intraoral 3D scanner: A feasibility study. Am J Dent. 2022 Feb;35(1):49-54.
51. Amaechi BT, Higham SM. Dental erosion: possible approaches to prevention and control. J Dent. 2005 Mar;33(3):243-52. Epub 2004 Nov 26. doi: 10.1016/j.jdent.2004.10.014.
52. Buzalaf, M.A.R., Cardoso, C.d.A.B., Magalhães, A.C., Amaechi, B.T. (2015). Prevention and Control of Dental Erosion: Patient Self-Care. In: Amaechi, B. (eds) Dental Erosion and Its Clinical Management. Springer, Cham.
53. Kaidonis, J.A., Anastassiadis, P.M., Lekkas, D., Ranjitkar, S., Amaechi, B.T., Townsend, G.C. (2015). Prevention and Control of Dental Erosion: Professional Clinic Care. In: Amaechi, B. (eds) Dental Erosion and Its Clinical Management. Springer, Cham.
54. Smith AJ, Shaw L. Comparison of rates of clearance of glucose from various sites following drinking with a glass feeder cup and straw. Medical Science Research, 1993;21:617-619.
55. Scaramucci T, Sobral MA, Eckert GJ, Zero DT, Hara AT. In situ evaluation of the erosive potential of orange juice modified by food additives. Caries Res. 2012;46(1):55-61. doi: 10.1159/000335572.
56. Hughes JA, West NX, Parker DM, Newcombe RG, Addy M. Development and evaluation of a low erosive blackcurrant juice drink 3. Final drink and concentrate, formulae comparisons in situ and overview of the concept. Journal of Dentistry 1999;27: 345-350.
57. Attin T, Meyer K, Hellwig E, et al. Effect of mineral supplements to citric acid on enamel erosion. Arch Oral Biol. 2003 Nov;48(11):753-9.
58. Attin T, Weiss K, Becker K, et al. Impact of modified acidic soft drinks on enamel erosion. Oral Dis. 2005 Jan;11(1):7-12. doi: 10.1111/j.1601-0825.2004.01056.x.
59. Kleier DJ, Aragon SB, Averbach RE. Dental management of the chronic vomiting patient. Journal of American Dental Association 1984;108: 618-621.
60. Turssi CP, Vianna LM, Hara AT, do Amaral FL, França FM, Basting RT. Counteractive effect of antacid suspensions on intrinsic dental erosion. Eur J Oral Sci. 2012 Aug;120(4):349-52. doi: 10.1111/j.1600-0722.2012.00972.x.

61. Gross, G.S., Amaechi, B.T. (2015). Prevention and Control of Dental Erosion: Dietary Management. In: Amaechi, B. (eds) Dental Erosion and Its Clinical Management. Springer, Cham.
62. Leung, G., Amaechi, B.T. (2015). Prevention and Control of Dental Erosion: Psychological Management. In: Amaechi, B. (eds) Dental Erosion and Its Clinical Management. Springer, Cham.
63. Amaechi BT. Dental Erosion and Its Clinical Management. Berlin, Germany: Springer; 2015. 329 p.
64. King, P.A. (2015). Restoration of the Worn Dentition. In: Amaechi, B. (eds) Dental Erosion and Its Clinical Management. Springer, Cham.
65. Amaechi, B.T. (2015). Recall, Maintenance Care Cycle, and Outcomes Assessment. In: Amaechi, B. (eds) Dental Erosion and Its Clinical Management. Springer, Cham.

Additional Resources

- No Additional Resources Available

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