Tooth Wear: When to Treat, Why and How

By

Thomas D. Larson DDS, MSD
Associate Professor
Department of Restorative Sciences
Division of Operative Dentistry
University of Minnesota School of Dentistry
8-450 Moos Tower
515 Delaware St SE
Minneapolis, MN 55455
612-624-5998
larso004@umn.edu

Abstract
Tooth wear has been described in the literature as physiologic- that is normal, expected over the life span of an individual, and not creating a pathologic condition. It has also been described in pathologic terms caused by stress, corrosion and friction, utilizing a variety of mechanisms and affected by a host of endogenous and exogenous factors. From a clinician’s point of view, when should we decide to restore a tooth or change the conditions in the mouth to protect the teeth; and what should we consider using to either prevent or restore abnormal i.e. pathologic tooth wear.

This review in Part I will look at what is normal, non-pathologic tooth wear; and etiologies associated with all forms of tooth wear; and in Part II the effects of tooth wear in enamel and dentin; when it may be advisable to intervene in the wear processes diagnosed on specific patients; and what methods of prevention and restoration can be utilized to restore or maintain the dentition. This review will not look at the need for full mouth reconstruction due to wear.

Introduction
The literature on tooth wear is extensive and various descriptions of normal i.e. physiologic tooth wear as well as pathologic tooth wear has evolved specific terminology descriptive of the etiologies, mechanisms and locations of tooth wear. Grippo suggested the latest taxonomy in 2004. In a thorough review of attrition, abrasion, corrosion and abfraction, Grippo describes the etiologies of tooth wear as coming from stress, corrosion and friction and the possibility of combining one or more of these etiologies and mechanisms. He defines attrition as tooth-to-tooth friction occurring on the incisal, occlusal or proximal surfaces of teeth. Friction through attrition is endogenous caused by parafunction and deglutition or exogenous caused by mastication, dental hygiene, habits, and occupational behaviors or through the use of dental appliances. Abrasion is defined as the friction between teeth and some external substance that causes friction. Gritty food substances, abrasive tooth polishes, or inappropriate e.g. horizontal, tooth brushing with tooth paste may cause abrasion. Abfraction is a microstructural loss of tooth material in areas of greatest stress concentration from occlusal contact, and is found in the cervical regions of teeth. Suggesting multifactorial etiologies, Grippo describes stress caused wear as microfracture/ abfraction. Abfraction from exogenous wear can be caused by mastication, habits, occupational behaviors and the use of dental appliances. Endogenous sources of stress come from parafunction, occlusion and deglutition as etiologies for the abfraction lesion. Grippo describes corrosion as the chemical degradation of tooth structure that can be endogenous from plaque, gingival crevicular fluids or gastric juices; or exogenous from diet, occupational exposures and through the use of certain drugs or alcohol.

Normal Tooth Wear
Lambrechts has studied normal in vivo tooth wear of enamel and reported that attrition, i.e. normal occlusal contact wear of human enamel was found to be 29 micron on molars and 15 micron on premolars per year. In a different clinical study that measured the wear of all teeth using digitized study models, the average tooth wear loss over one year was measured as 0.04mm³ by volume or 10.7 micron by depth, which approximately
doubled by year two. Examination of five hundred patients over an eighteen-year period, in which study models were compared, led to the conclusion that non-pathologic tooth wear is a slow minimally progressive process.

In a study of Swedish children from ages 3-20 years, it was found that as children age from 3 to 5 years there is increased attrition on deciduous teeth. Sixty-three percent of the three year olds had no incisal wear. Whereas in the five year olds only 19% had no incisal wear. In the permanent dentition, this same study reported that the ten, fifteen and twenty year olds had no incisal wear in 78%, 51%, and 35% patients respectively. Another longitudinal clinical study confirms the finding that as children age they tend to demonstrate more incisal wear. In a clinical study this same author confirms that as children and adolescents get more permanent teeth, there is more incisal wear.

Dental arch shape has been reported to affect tooth wear rates. In an anthropological study, casts of Aboriginals born between 1900 and 1940 were examined. Hypsiloid or U-shaped maxillas have more buccally directed wear whereas parabolic or hyperbolic maxillas exhibit much heavier wear in the lingual aspects of the occlusal surfaces. The authors contrast this “as steep oblique wear vs. flat horizontal planes”. It has been reported that tooth wear may be a functional compensation for modifications in the anterior dentoalveolar complex. In this study the changes that were described included lingual tipping of maxillary incisors with a decrease in the horizontal overlap between the maxillary and mandibular teeth. This was accompanied by a more edge-to-edge bite and incisal attrition.

Adult tooth wear is related to the craniofacial morphology of the patient as a child with the cephalometric measurements of ABN, ramal height and sex being significant, Craniofacial morphology can play a small but significant relationship to adult tooth wear.

Normal tooth wear may occur as a result of the speech envelope. In a clinical study that investigated subjects with incisal tooth wear versus those with no incisal wear; in subjects with incisal tooth wear had a smaller speech envelope. The study did not clearly elucidate whether the increased wear caused the smaller speech envelope or a smaller speech envelope contributed to the wear.

An anthropological study compared tooth wear between modern and medieval subjects. It was assumed that medieval subjects had an abrasive diet, whereas the modern subjects had either an acidic diet or an average softer Western diet. The abrasive diet caused more substance loss of tooth structure on the occlusal surfaces and occlusal cupping was common. The acidic diet produced less occlusal cupping than the abrasive diet but more than the Western diet. There were more buccal lesions (63%) found in subjects on the acidic diet than either of the other two diets (0% and 8% respectively). The authors suggest that the finding of concavities on smooth surfaces is indicative of erosion (corrosion).
In a clinical study of five hundred and eighty-six subjects, researchers from Wales assessed tooth wear in dentate individuals using a Tooth Wear Index and noted that there was increased wear on the occlusal and incisal surfaces as age increased. Except for the lingual surfaces of maxillary incisors, the lingual and facial surfaces showed no significant variations in wear with aging. Males demonstrated the greatest amounts of wear compared to females.\textsuperscript{13}

A clinical study of thirty patients compared the tooth wear of those patients with a chewing pattern described more as chopping, vertical chewing to those patients described as grinding, with more lateral movement to their chewing pattern. The grinding type of chewing pattern resulted in significantly more occlusal wear of posterior tooth segments compared to the chopping type.\textsuperscript{14}

In a clinical study from Australia, it was reported that in those children who received optimally fluoridated water or regularly used a fluoride supplement, there was less wear noted in all sextants compared to those who did not ingest fluoride. Except in the fluoridated patients, the mandibular molar sextants did not seem protected against occlusal erosion.\textsuperscript{15}

\textbf{Etiology of Tooth Wear}

As described previously by the taxonomy used to describe tooth wear, there appears to be three mechanisms and various combinations of these mechanisms by which tooth wear occurs. Friction, corrosion and stress can all contribute singly or in combinations to create the types of wear reported in the literature and found in dental practices treating dentate populations. In mathematically describing wear, the equation states that wear is proportional to the normal force times the distance, so movement becomes another factor to consider in determining etiology.

\textbf{A. Corrosion}

Erosive wear, now called corrosion, is typically seen in young adolescents due to diet. Ingestion of large quantities of acidic soft drinks, many with a pH of 2.3, are capable of demineralizing teeth.\textsuperscript{16} While saliva can provide some buffering capacity for such acidic drinks, in large quantities the acid will have an effect on tooth enamel. Exercise dehydration will also decrease the salivary protection and increase tooth wear.\textsuperscript{17} The use of a sports drink will not itself cause significant erosion as found in clinical observational studies.\textsuperscript{18,19} Over consumption of acidic soft drinks in young children can clearly lead to faster erosion of deciduous teeth than permanent teeth.\textsuperscript{20} In a British study of 14-year-old students where consumption of carbonated drinks was from 1-3 cans per day, it was found that the tooth wear as measured by a tooth wear index had no correlation to salivary flow rate or buffering capacity. There was a correlation with acid regurgitation.\textsuperscript{21}

Sites of erosive tooth wear may be saliva-dependent, as was found in a survey of 450 patients with tooth wear. In this study, non-carious cervical lesions were associated with occlusal attrition in 27.71\% of the facial sites compared to 2.61\% of the lingual sites. The most common site for non-carious cervical lesions was in the maxillary incisors. The least common was in mandibular molars.\textsuperscript{22}
Liquid flow rates affect the severity of dental erosion. In a study that measured flow, *in vitro*, it was found that erosive depth increased with increased time of exposure, total volume, increased flow rate, and decreased outlet diameter. Drinking acidic drinks through a straw directed at the teeth increases the erosive depth. Tooth surface pH is affected by drinking method. Holding an acidic drink orally shows the greatest pH drop, followed by a long-sipping method, followed by gulping, which showed the least drop in pH. After acids have demineralized enamel, licking with the tongue could have an ability to remove more enamel than if no tongue licking is present.

Corrosion can be caused by a variety of medical conditions. Gastro-esophageal reflux (GERD) patients commonly show marked erosion of the lingual surfaces of the maxillary anterior teeth, incisal surfaces of maxillary and mandibular teeth and the occlusal surfaces of mandibular molars. (See Figure 1) Asthma has been shown to cause corrosion (erosion) on the occlusal surfaces and less on the lingual surfaces as seen in GERD. Chronic alcoholism is expected to cause a corrosive (erosive) type of wear associated with bruxing and clenching. It is expected to see more rapid cupping of the occlusal cups tips and general occlusal wear. Bulimia and other eating disorders also cause increased corrosive wear of the teeth because of the stomach acid affecting the teeth. (Figure 2)

Methamphetamine and Ecstasy (methylenedioxymethamphetamine) use also results in increased tooth wear. Snorting of methamphetamine leads to higher tooth wear in the maxillary anterior teeth. Whereas continued methamphetamine use can lead to over consumption of carbonated drinks, clenching and grinding. So the effects of corrosion, attrition and stress abfractions can all be found. These drug users also typically have extensive smooth surface caries.

Syndromes in which xerostomia occur demonstrate excessive tooth wear. In a case report, it was reported that in four subjects with congenital dysfunction of the major salivary glands, excessive tooth wear was present. Syndromes included Sjogren’s, Prader-Willi, and congenital rubella. Patients with medication induced xerostomia also demonstrated increase tooth wear. Normal salivation provides many protections to the teeth including lubrication, buffering capacity, clearance of acidic substances by swallowing, pellicle formation; and the capacity for remineralization of demineralized tooth substance.

Corrosion and attrition are often combined in mouths of bruxers. Simultaneous erosion and abrasion results in 50% more wear than by alternating erosion and abrasion singly. In a six-year clinical study in which erosion and wedge-shaped defects presumably caused by toothbrush abrasion were followed, there was a definite progression of the lesions despite dietary and hygiene counseling. Using multiple linear regression statistics, the authors found that nutritional acids and age were significant risk factors and explained 28% of the variability. Using the same statistical technique the authors reported that tooth brushing and age contributed to 21% of the variability found in the progression of wedge-shaped defects. Extrinsic factors that are environmental have been suggested as causing erosive tooth loss. The exposure to acid fumes, swimming in pools with low
pH, and dietary contributions have been described as contributing to corrosion.\textsuperscript{44} Endogenous factors are also described as causing corrosion. Erosion from dietary acids or gastric acids is described as smooth lesions appearing cupped on the occlusal or incisal surfaces and concave on facial or lingual surfaces.\textsuperscript{45} Progression of these lesions is stated to be intermittent, with periods of activity and inactivity. Prevalence has been reported as occurring in 80\% of children and 43\% of adults.\textsuperscript{46}

**B. Friction**

Tooth wear because of attrition or abrasion is frequently also seen with some corrosion.\textsuperscript{47} (See Figure 3) In a clinical study of one hundred patients, 98\% had evidence of erosion and attrition while 82\% had signs of abrasion as well.

A reported *in vitro* study showed that both the degree of load, the time of applied stress and the use of an acid environment altered the wear found on premolars. The lower the load the lower the frictional loss of tooth structure. The greater the amount of time the force was applied, the greater the tooth loss. The citric acid medium actually decreased the loss comparing the same time and load. The authors described the outcome as simulating a three-body wear in neutral pH conditions, whereas two-body wear in the acidic environment due to increased polishing.\textsuperscript{48}

In a clinical study with a sample size of seven, bite force and dento-facial morphology were described in patients exhibiting severe dental attrition. The patients were all male. They had high bite force levels in the incisal regions of 63\% of the molar bite force and all had a rectangular facial morphology. The maxillary dental arch also was described as more rectangular than normal.\textsuperscript{49} In another Swedish study with a sample size of fifty-four adults, with advanced occlusal wear, it was reported that the level of maximum bite force was high compared to normal populations. The craniofacial structure is characterized by “deviation in the vertical direction, a small angle between the mandibular-palatal planes and a small gonial angle” compared to Swedish norms.\textsuperscript{50}

Many studies have been completed regarding occlusal forces, but the degree of sophistication in measurement has changed significantly over time. An initial finding was that “The functional chewing forces are small compared to static isometric closing forces that the stomatognathic system can exert.”\textsuperscript{51} Anderson first reported masticatory force in 1956 using four subjects. He reported that normal force varied by the consistency of the food being chewed (carrot versus meat versus biscuit) between about 16-32 lbs (71-142N).\textsuperscript{52} More recently it also has been reported that the magnitude of masticatory forces ranges from 2-40 pounds (9-180N) with a duration of from 0.25-0.33 seconds.\textsuperscript{53} Maximal biting force has been measured in young subjects and falls between 115-120 lbs (516-532N).\textsuperscript{54} The presence of restorations did not affect the bite force. Gender differences do occur. The mean maximal bite force for men has been measured as 190 lbs (847N) versus 134 lbs (597N) for women.\textsuperscript{55} In patients that brux, maximal bite force has been measured at 205 lbs (911N) in the molar region of men versus 128 lbs (569N) in the incisor region of men.\textsuperscript{56} This difference between anterior and posterior biting force has been measured as less than these reported values elsewhere with 79-82 lbs (353-365N) in the molar region and 12-14 lbs (56-64N) in the premolar region and 14 lbs (65N) at the
incisors.\textsuperscript{57} Whatever the actual values, it is apparent that the most extreme forces are in the most posterior teeth. When these forces are calculated as a force per area and then converted to international units, a force of 205 pounds affecting a point of contact 1/32 inch square places 45.23 MPa of force. Normal chewing force using the same area of contact results in a force of 8.826 KPa, well below the ultimate tensile strength of 42.1 MPa of enamel and 61.6 MPa of superficial dentin.\textsuperscript{58}

When posterior teeth are lost and the proprioception is altered, the maximal bite force also goes down.\textsuperscript{59,60} Nocturnal bite force of bruxing is different than daytime maximum bite force. In a study that measured those differences, nocturnal bruxing forces were reported as a mean of 49 lbs (220N) and a maximum force of 93 lbs (415N) versus a voluntary daytime force of 174 lbs (775N).\textsuperscript{61} However nocturnal bruxing forces were timed as duration of 7.1 seconds versus a normal chewing duration of 0.25-0.33 seconds. This means that the longer duration of bruxing with greater force than used for chewing will cause greater damage and wear to teeth because the longer time of force application causes a longer contact path on the tooth.

Clenching force on one tooth is reported to be up to ten times greater on the canine, than maximum biting forces distributed in a balanced way.\textsuperscript{62,63} Maximum biting forces exerted by the muscles are exerted in the maximum intercuspal position and are distributed according to distance from the condyles, with the second molar taking 55\% of the maximum force and the incisors taking 20\% of the force.\textsuperscript{64} Bruxing and clenching forces can reduce the protection afforded by salivary lubrication by reducing the film thickness sufficiently to increase frictional contact and increase the wear of the opposing surfaces.\textsuperscript{110} Clenching causes very little if any wear, but undoubtedly contributes to stress. Wear requires movement. Damage can occur only if the force exceeds the strength of the material or through a fatigue process. (See Figure 4)

What effects do these forces; whether from normal chewing, single tooth bruxing, multiple tooth bruxing or clenching do to the teeth? In a photo elastic study using about 4.4 lbs (2kg) of force on a tooth in a vertical load it was found that distal incline planes or slopes of cusps and lingual incline planes or slopes of the buccal cusps received the greatest force on mandibular molars. The magnitude of the stress is increased considerably when the occlusion was flat plane.\textsuperscript{65} The total force is the same; the distribution of this force changes. A vertical force contacting an inclined surface is resolved into a force normal to the surface and one parallel to the surface. As the incline becomes horizontal, the normal force increases and the parallel force decreases, but the total force is the same.

Using extracted teeth prepared for endodontic access with MOD cavity preparations, teeth were stressed with a load, either continuously or cyclically. Continuous loading resulted in progressive cuspal displacement both time- and load-dependent. It took twenty minutes for the tooth to recover from the deformation. Cyclic loading resulted in cumulative increase in cusp displacement but only to a very small extent $\approx 1$ micron. The conclusion is that continuous loading as in clenching can be more destructive than cyclic
loading as in chewing because of an increase in the fatigue of the stressed teeth over time.66

An interesting anthropological study did a finite-element analysis of 29 intact molars wherein a cleavage type load was applied. They reported that first maxillary molar functional and non-functional cusps dissipate loads equally well. In maxillary second and third molars the non-functional cusps resist loads better. Mandibular molar functional cusps all dissipate force equally well.67

C. Stress
In 1984 Lee and Eakle proposed a theory that tensile stresses from occlusal forces caused cervical erosive lesions (also called non-curious cervical lesions, NCCL).68 Grippo later called such lesions “abfraction” (taken from engineering vocabulary). Abfraction occurs from the loading forces placed on teeth during static events such as swallowing or clenching and cyclic events such as chewing or bruxing. He states that the flexing and fatiguing of the weakest point in the distribution of the stress along the enamel-dentin boundaries, at the cervical margin, cause this type of lesion.69 (See Figure 5)

In a clinical study that examined the performance of dentinal adhesives in non-curious cervical lesions, Heymann et al. agreed that the results showing failures of Class V composites in part are explained by the flexure of the teeth resulting in restoration loss.70 In subsequent publication, other authors supported the concept of “abfraction” by reporting development of subgingival non-curious cervical lesions.71 There are those who disagree with the concept of abfraction stating that there is no correlation between occlusal/ incisal wear and development of NCCL;72 that stresses may be dissipated by periodontal ligament and alveolar bone;73 that using a more sophisticated model of enamel prism anisotropy (orientation) in stress concentration alters stress distribution to the cervical in mathematical modeling;74 and that in vitro simulation using tooth brushing with and without non-axial forces resulted in no difference in tooth wear in the cervical margins.75 Bader et al. provided evidence in a case-controlled clinical study, that development and progression of non-curious cervical lesions was multifactorial and that multiple mechanisms may initiate and allow progression of these lesions.76 Previous in vitro studies cited here have shown that NCCL progress in an acidic environment worsen when greater stress is introduced.42 While no direct evidence can be presented, abfraction lesions theoretically may not progress except in acidic conditions. It is also reasonable to conclude that abfraction lesions may progress due to toothpaste abuse (horizontal tooth brushing with any abrasive toothpaste) as it has been shown to do in vitro.77,78

The finite element models studying this concept demonstrate that there is adequate force transmitted through the cusps or cusp inclines to the CEJ to exceed the known strength of enamel.79 If a restoration is present (Occ, MO, DO, MOD) and depending on the width and depth of the restoration it can increase the cervical stresses exceeding the tensile and shear stresses of enamel.80 A finite element study correlated to the strains seen in human teeth showed that strains were concentrated near the CEJ regardless of load direction. As measured in this study, the asymmetrical strains developed in the cervical regions of teeth
in response to oblique occlusal forces are consistent with asymmetric non-carious cervical lesions.\textsuperscript{81}

In a clinical study of fourteen years, the progression of non-carious cervical lesions was positively correlated ($r^2 = 0.98$) to occlusal volume loss and was significant. The volume loss reported over fourteen years was 0.9-11.5 mm$^3$ for cervical lesions and between 0.39-7.79 mm$^3$ for occlusal wear.\textsuperscript{82} A recent clinical study showed a significant correlation between the presence of non-carious cervical lesions and occlusal attrition.\textsuperscript{83}

Multifactorial etiologies have been accepted to explain the development and progression of abfraction lesions.\textsuperscript{84,85,86} Other etiologies most frequently cited are corrosion\textsuperscript{77}, and toothbrush (toothpaste) abrasion.\textsuperscript{87} The lesions were characterized in a clinical study using 171 teeth from 57 patients. Ninety-one percent of the lesions occur with axial depths between 1-2 mm; 49% had occluso-gingival widths of 1-2 mm; 74% had angular shape of 45-135 degrees. Commonly (76%) the lesions had sclerotic dentin, and 73% had little to no sensitivity. Seventy-five percent of the patients presented with an Angle Class I occlusion on the involved side with 60% group function on excursive movement. Eighty-two percent of the teeth had wear facets, and 99% had little to no mobility.\textsuperscript{88} The lack of mobility has been confirmed by another study that looked at periodontal support. In this study the authors reported that abfractions are less likely to occur in patients that are periodontally compromised due to tooth movement.\textsuperscript{89} While abfraction cannot be totally discounted, it is probably by itself a small instigating mechanism requiring other mechanisms such as corrosion or friction to advance in size.

Part II
Mechanisms of wear
This section will describe the oral mechanisms that protect or breakdown or are overwhelmed by the various etiologies that effect tooth wear as well as the mechanisms of the etiology and how it affects teeth.

A. Corrosion mechanisms
Saliva provides numerous protective functions for the teeth. Saliva provides normal hydration to the tooth, lubricates the food bolus enabling easier chewing and swallowing. Saliva also provides some bacterial protection and helps to control the demineralization/remineralization of tooth surfaces. Salivary proteins such as acidic proline-rich proteins, statherins, histatins and cystatins have affinity for mineral surfaces, inhibit calcium phosphate precipitation and protect the integrity of the enamel surfaces.\textsuperscript{90,91} Any disease process, or acidic challenge that overwhelms these defenses will result in a corrosive loss of tooth structure. Salivary pellicle proteins, as well as the salivary calcium and phosphate and the plaque fluid retard demineralization of tooth surfaces. The bicarbonate/carbonate buffer system in the saliva neutralizes acids.\textsuperscript{92}

Salivary pellicle will protect the enamel from short-term erosion of organic acids\textsuperscript{93} and can form within three minutes. However, dietary acids such as citric acid are not inhibited from dissolution of the enamel.\textsuperscript{94}
In an *in vitro* study, challenges to enamel were made with an acid below pH 5.0 for 10 and 20 minutes and for three or six exposures each time frame. The net mineral loss was the same. However the increased frequency of acid exposure increased the total demineralization results. The conclusion was that frequent acid attacks could overwhelm the ability of the saliva to remineralize the enamel. Erosive depth clearly depends on pH values of the acid and the time of exposure. Once enamel has been softened by acid attack, oral soft tissues can abrade the enamel more easily. However, when low pH acids are used together with attrition, the enamel wear is lower than in neutral pH situations due to a smoothing effect of erosion of contacting surfaces. The greater the load and the more time acid is applied to enamel, the greater the wear.

In an analysis of cementum with surface root caries that had been exposed to fluoride, it was reported that fluoride was detected in the outer 25 micron of the cementum. However the fluoride did not penetrate into the dentinal structure. Cemental structure participates in the remineralization process.

When dentin is exposed to the oral environment, it has been found to be susceptible to acid erosion even with relatively high pH values and it showed little likelihood of remineralization. The acid removes the dentinal smear layer and opens the dentinal tubules. Increasing the temperature, concentration and exposure times of acids increases the erosion of enamel and dentin. Dentin rate of wear depends on the density and tubule orientation, with peritubular dentin and better-mineralized zones of dentin more resistant to wear and erosion.

The type of acid affects the erosive capability, with citric acid being more erosive than hydrochloric acid and hydrochloric acid more erosive than phosphoric acid. Removal of the outer fluoride rich layer of enamel results in a greater erosive loss of enamel when exposed to citric acid. *In vitro* testing of four different soft drinks showed that the pH, phosphate and fluoride concentrations of the beverages correlated with the demineralization of the enamel. Calcium concentration and titratable acidity were correlated with the demineralization of the dentin.

The tooth responses to tooth wear; whether from corrosion or friction include the formation of reactionary and reparative dentin and obduration of dentinal tubules. The pulp also responds by forming calcific mineralized tissues, decreasing the pulpal blood supply. More rarely, pulpal sequelae can result from corrosion. In a clinical study 11% of tooth wear patients were found to experience dental pulp irreversible degeneration as a result of late stage erosion. (See Figure 6)

### B. Friction mechanisms

When considering the affects of friction on enamel and dentin, one should consider the physical properties of these substances to understand the mechanisms by which friction affect teeth. Enamel is a hydroxyapatite crystalline structure and has higher hardness and elastic modulus than human dentin. It is more brittle. Primary deciduous human enamel has a hardness of 4.88 GPa compared to a hardness of between 3.3 and 3.9 GPa in permanent tooth enamel, with the lower value being perpendicular to the enamel rods.
Primary deciduous enamel has a modulus of 80.35 GPa compared to adult enamel between 87.5 GPa and 72.2 GPa, with the lower value being perpendicular to the enamel rods. Enamel is anisotropic related to the alignment of fiber-like apatite crystals. Enamel tufts wear more rapidly than surrounding well-mineralized areas of enamel. Orientation of enamel prisms affects the wear with prisms more parallel to the surface more easily cleaved by the wear process, than prisms oriented perpendicular to the surface. Because the enamel is hard and brittle, and because the enamel prisms are organized perpendicular to the external surface, the physical properties are maximized against opposing surfaces. As a result enamel is built to withstand a lifetime of normal forces without being breached. Hypo mineralization of permanent human enamel lowers both the hardness and the modulus of elasticity significantly from normal human enamel.

One of the more interesting findings about permanent human molar enamel is that in a maxillary second molar, the range of hardness and modulus of elasticity varies considerably with the higher hardness and the higher elasticity found at the occlusal surface of the molar, and lower values found at the DEJ. This was correlated to the differences in the different chemistry of the hydroxyapatite. The P$_2$O$_5$ and the CaO were highest at the occlusal surface and lower at the DEJ whereas Na$_2$O and MgO showed the opposite trend. Because of these differences the occlusal enamel on the lingual side is stiffer than on the buccal side. Conversely the inner enamel shows the opposite. It is postulated that these differences in chemistry may be related to different function. Human enamel structure where inter-rod spaces are minimized shows ability for molar cusps to withstand greater horizontal tensile stress.

Hardness of dentin is considered to be dependent on mineral concentration. Mineral concentration of the dentin and its hardness has been correlated to the location on the tooth. In adult teeth, mantle dentin that is immediately adjacent to the dentin-enamel junction is softer than primary dentin underlying it. Primary adult dentin has decreasing hardness as the pulp is approached probably due to a decrease in the hardness of the intertubular dentin matrix caused by less mineralization. The hardness and modulus of elasticity of dentin decreases as the pulp is approached in deciduous teeth as well.

Tensile strength is a critical mechanical property for a tooth because most of the destructive occlusal forces placed on teeth are tensional or bending causing tensile and compressive strains. The ultimate tensile strength (UTS) of dentin depends on the dentinal tubule orientation. The UTS is lowest for tensile force that is parallel to the tubule orientation, i.e. at right angles to the collagen fibers in dentin (54Mpa), and greatest at 90 degrees to the tubule orientation (92Mpa) (fracture parallel to the tubule direction). Testing for tensile strength in dentin inherently shows wide standard deviations in the sample, suggestive that tensile strength is controlled by the distribution of flaws in the dentin specimens.
When considering strength properties of the dentin, one must look at the various dentinal structures separately to determine the weakest structures, and their orientation in order to predict wear patterns and the effects of destructive behaviors on tooth wear. Using an atomic force microscope to determine Young’s modulus of elasticity, it has been found that the modulus of peritubular dentin is 30 GPa and that of intertubular dentin is 15 GPa. The hardness of the dentin varies greatly being 2.3 GPa for peritubular dentin and 0.5 GPa for intertubular dentin. The AFM also showed that the peritubular dentin was spatially homogeneous whereas the intertubular dentin showed considerable spatial variation in its elasticity. These properties will affect how the dentin wears and fractures and how a crack can propagate. The structure of the dentin contains a hydrated matrix of collagen fibrils that is reinforced with a nanocrystalline, carbonated apatite that is called the intertubular dentin. The peritubular dentin is a matrix composed of type I collagen fibers with a carbonate rich apatite mineral phase. The mineralized fibers are isotropic by the pulp but become anisotropic in mid-dentin areas. Near the pulp the mineral phase is needle like but changes to plate like near the DEJ. Peritubular dentin is a hypermineralized wall of the dentinal tubules and is roughly 0.5-1.5 µm in thickness. The collagen fibers are cross-linked by covalent bonds providing the dentin matrix with stability and improved tensile strength. In this same study the findings indicate that the amount of collagen cross-linking varied by tooth type with the molars having the most followed by premolars and canines. Incisors had the least collagen cross-linking. The authors felt that this suggests the dentin may be functionally adaptive to the stress placed against it.

Dentin hydration can dramatically affect the physical properties. Dehydrated human dentin demonstrates lower strain values at fracture i.e. is a more brittle material. Hydrated or rehydrated dentin (dentin that became dehydrated but is exposed to moisture to rehydrate) requires significantly greater strain at fracture. Dehydrated dentin also shows a significantly reduced stress relaxation response when a predetermined compressive load is applied to a maximum value and released. This partially explains the increased wear in xerostomic patients.

Dentin because of the aforementioned properties is an excellent supporting structure for the enamel, being tough yet resilient. Dentin, when exposed however cannot overcome wear. Once the enamel has been breached by wear, the dentin will wear more rapidly than the enamel. This exposes the enamel rods, unsupported and more susceptible to fracture. The lesion will progress faster given the same forces and duration once dentin is exposed.

When considering wear process, two (opposing teeth e.g.) and three-body (opposing teeth with an abrasive slurry between them e.g.) wear testing is used to describe the wear process. Two-body wear with opposing teeth of flat and pointed specimens were tested and the loss of tooth substance was much greater in two-body versus three-body specimens. The wear was also significantly greater on flat specimens compared to pointed specimens. This finding would support that normal chewing of food will be less destructive to the teeth than will bruxing or clenching, something that is clinically self-evident.
Human enamel will wear more under dry, unlubricated conditions than under a lubricated condition, though when the load increases sufficiently, the wear will be greater.\textsuperscript{135} Enamel prisms and specifically enamel crystals wear by development of striations in their surface. The width of the wear striation is not related to particle size of the material being chewed because of the orientation variables of the prisms, with differential wear noted in functionally different areas of the tooth enamel.\textsuperscript{136} Introducing abrasives from toothpastes for example causes small surface scratches on the enamel surface to disappear and larger scratches to appear and expand. When brushing with out dentifrice, the surface of the enamel is protected by a salivary pellicle.\textsuperscript{137}

Tooth brushing can create tooth wear. Hard brushes have been found to create 3.6 times as much wear as soft toothbrushes.\textsuperscript{138} Saliva has no effect on reducing eroded tooth wear from tooth brushing.\textsuperscript{139} Mean enamel wear of different dentifrices was between 0.05 to 0.40 micron after one hour of brushing with 375 g. of force. The more abrasive the dentifrice, the greater the wear with a significant correlation made between abrasivity and wear.\textsuperscript{140} Similar ranges of enamel loss were found in an \textit{in situ} clinical study.\textsuperscript{141}

Acid erosion increases the susceptibility to abrasion, with dentin affected more than enamel. Dentin loss can be correlated to the abrasivity (RDA) of toothpastes used.\textsuperscript{142}

\textbf{C. Stress mechanisms}

In an \textit{in vitro} study where the teeth were exposed to lactic acid pH 4.5 with and without cyclic tensile stress, it was found that the cyclic stress resulted in greater tooth substance loss in the cervical third of the tooth and on the mesio-buccal side (under tension) than on the disto-buccal side (under compression).\textsuperscript{143} Tensile stress in the enamel increases with an increase in the coefficient of friction.\textsuperscript{144} Teeth wear faster in the mouths of bruxers versus non-bruxers because of the increased time of force application.\textsuperscript{145} Increasing load also increases dentin loss.\textsuperscript{146} Tooth-wear on abraded occlusal surfaces displays greater mean depth/breadth ratio of scooped dentin than samples taken from teeth undergoing erosion.\textsuperscript{147} Wear under stress causes microscopic pitting of the enamel related in part to the particle size of the material creating friction on opposing surfaces.\textsuperscript{148}

\textbf{Interventions for Prevention and Restoration of Tooth Wear}

To judge the progression of tooth wear in an individual it is wise to make study casts to compare the various sites of tooth wear from year to year. This is helpful in deciding when to intervene. A tooth wear index (TWI) as suggested in the literature may also be used to chart existing tooth wear and track it over time. Smith and Knight have suggested an index often cited in the literature that can show progression of lesions over time.\textsuperscript{149} The index is described by scores of 0-4 in Table 1.

\begin{table}
\centering
\begin{tabular}{|c|c|p{10cm}|}
\hline
Score & Surfaces & Criteria \\
\hline
0 & B/L/O/I & No loss of enamel surface characteristics \\
\hline
\end{tabular}
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<tbody>
<tr>
<td></td>
<td>C</td>
<td></td>
<td>No change in contour</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>B/L/O/IC</td>
<td>Loss of enamel surface characteristics</td>
<td>Minimal loss of contour</td>
<td></td>
<td></td>
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<tr>
<td>2</td>
<td>B/L/OICI</td>
<td>Loss of enamel exposing dentin for less than 1/3 of the surface</td>
<td>Loss of enamel just exposing dentin</td>
<td>Defect less than 1 mm deep</td>
<td></td>
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<tr>
<td>3</td>
<td>B/L/OICI</td>
<td>Loss of enamel exposing dentin for more than 1/3 of the surface</td>
<td>Loss of the enamel and substantial loss of dentin but not exposing the pulp or secondary dentin</td>
<td>Defect 1-2 mm deep</td>
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<tr>
<td>4</td>
<td>B/L/OICI</td>
<td>Complete loss of enamel, or pulp exposure or exposure of secondary dentin</td>
<td>Pulp exposure or exposure of secondary dentin</td>
<td>Defect more than 2 mm deep, or pulp exposure or exposure of secondary dentin</td>
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A raw score has no meaning without understanding the age of the patient and what a normal pattern of wear would look like over time. The wear normally expected in a 60-year-old patient would be abnormal if seen in a 20-year-old. There are indices that include age however, they are complicated mathematical models used to predict what wear should be. One paper does describe an average of tooth wear using this index on tables for each tooth for 45-54 year olds, 55-64 year olds, 65-74 year olds, 75 plus year olds, male subjects and female subjects. These averages may be useful in comparing tooth wear index scores among individual patients. Once the assessment of tooth wear is made, a diagnosis of pathology including probable etiologies of the tooth wear should be recorded. If the wear is normal for the age of the patient, no further action beyond recording is necessary.

If the wear is considered pathologic, some form of intervention is recommended to prevent further wear, minimize damage or protect the teeth. Interventions depend on the etiology of the wear and because wear can be multifactorial, a range of interventions may be necessary. Abrahamsen has described a methodology used to determine which etiologies may be causing the tooth wear. He suggests hand held study casts are necessary to make the determination so that the occlusal aspects can be clearly seen.

1. If the wear is greater in the anterior than the posterior and the wear facets match up on opposing casts the wear is from bruxism.
2. Where there is more anterior than posterior wear and the lingual surfaces of the maxillary anterior teeth are worn smoothly from gingival tissue; and the lingual surfaces of maxillary posterior teeth seem affected, the cause is from acid regurgitation. In this situation the worn surfaces of the casts will not match up.
3. If the posterior teeth have greater wear than the anterior teeth and cupping or cratering is present with the mandibular first molar most severely affected, the cause is from swishing of acidic drinks. In this instance the worn surfaces of the casts do not coincide and the edges of the enamel look sharp.
4. If the posterior teeth have greater wear than the anterior teeth and cupping or cratering is present but there is even posterior wear on all of the upper and lower teeth, the wear is created by fruit mulling where acidic fruits are kept in contact with the teeth for extended periods while chewing.

5. If the anatomic details on the teeth appear faded with a sandblasted appearance or the facial surfaces of the lower canines and premolars have cervical notches, the cause is from toothbrush/dentifrice abrasion. In this case the worn surfaces of the casts will not match up.

6. This author also suggests a miscellaneous category but does not enumerate it. Abfraction lesions and other forms of tooth wear not described above would fit into this ill-defined category. Since abfraction is caused in part by occlusal stress, the tooth with the facial NCCL would also have an occlusal wear facet that would fit the opposing teeth.

**Recommendations for Treatment**

**A. Corrosion**

After an acid attack, saliva can remineralize dentin and takes approximately thirty minutes to do so.\(^1\) Erosion and abrasion of enamel surfaces exposing dentin increases susceptibility to both erosion and abrasion alone or in combination and can be correlated to the abrasivity of the dentifrice used.\(^2\) A mild toothpaste with low abrasivity is recommended together with a soft bristle brush. Horizontal tooth brushing must be eliminated.

Diet plays a critical role in corrosion. The chemical factors that affect the corrosion of the tooth are the pKa values of the acid, the adhesion and chelating properties, calcium, phosphate and fluoride content. The behavioral factors of eating habits, life style choices and excessive consumption of acids affect corrosion. The biological factors of flow rate, buffering capacity of the saliva, salivary composition, pellicle formation, tooth composition, fluoride exposure, dental and soft tissue anatomy all can affect corrosion. Reducing the frequency and contact of acids can be used to control corrosion.\(^3\) As can the addition of protective agents such as fluoride,\(^4\) calcium phosphates and regular oral hygiene practices used to promote health by plaque removal, and control of the oral micro biota. Fluoride used in toothpaste (1450 ppm NaF) is able to remineralize enamel acid dissolution from carbohydrates applied for 30 seconds each for 1, 3 or 5 times a day.\(^5\)

Patients most at risk for corrosion in combination with frictional wear suffer from dehydration, caffeine addiction, GERD, asthma, diabetes mellitus, hypertension and other conditions leading to xerostomia. Routine examination is recommended for the salivary glands, oral mucosa, skin and eyes for evidence of salivary hypo function. A self-management plan should be developed to control or alleviate the xerostomia limiting the damage of erosion and abrasion of the teeth.\(^6\) Common aids include the use of remineralising materials including fluorides in toothpastes, gels, and varnishes; and calcium phosphates in solution (Recaldent, Bonlac Bioscience Int. Pty Ltd, Melbourne,
Australia, sold as MI paste for professional use), and also in sugarless chewing gums (Trident with Recaldent, Cadbury Adams USA LLC, Parsippany N.J.); xylitol chewing gums; use of non-alcohol rinses (Oral Balance, Laclede Inc., Rancho Dominguez, Calif.); wetting agents including artificial saliva (Moi-Stir, Kingswood laboratories, Indianapolis, Ind; MouthKote, Parnell Pharmaceuticals Inc, San Rafael, Calif.; Xerolube, Colgate Oral Pharmaceuticals, Canton Mass.).

Fluoride, barium sulfate, silver nitrate, and especially oxalate cause a precipitation in the dentinal tubules occluding them and preventing fluid flow. All of these agents present in a variety of products can be used to desensitize teeth, where exposed dentin is present.

When corrosion lesions are sensitive and the lesion depth is minimal, dentin bonding agents have been applied to protect the area from further acid erosion and will provide some level of protection, though for how long is unknown.

Clinically it may be difficult to know a NCCL (non-carious cervical lesion) is from erosion only. The clinician may look at the exposed enamel and notice a matte finish to the surface, indicative of acid demineralization, but not in all cases will erosion appear this way because of subsequent abrasion.

Some erosive lesions will be very sensitive to touch or air. These lesions probably do not have a smear layer and probably do not have sclerotic dentin. Other lesions may have sclerotic dentin. This is significant if the depth of the lesion is at or beyond an ideal cavity depth for the restorative material that would be used. Once the progression of a corrosive lesion has shown consistent progression and dietary counseling has not changed the pattern of wear, it is time to intervene with a restoration to protect the tooth from additional corrosion that may threaten the pulp. If no sclerotic dentin is present, and there is no abfraction component from occlusal stress, the lesion may be bonded utilizing a three-part etch, rinse, prime and bond system followed by composite resin. (See Figure 7, the restored patient seen in Figure 3) If amalgam is preferred a cavity preparation with 90° cavosurface margins may be prepared. Bonding the amalgam utilizing a three step etch, rinse, prime and bond system together with a dual or chemically cured resin cement is recommended to decrease microleakage and post-operative discomfort. (See Figure 8) In some patients where the corrosion process continues unabated, the loss of tooth substance around the restoration will become evident and additional protection may be required. Extreme cases of corrosion may require a crown or veneer to protect the teeth. (See Figure 9, the restoration of the patient in Figure 2)

**B. Friction**

In a sampling of white subjects, worn anterior teeth and premolars were compared to unworn teeth. It was found that there was a 1.01mm difference in length between the worn and unworn central incisors; a 0.93mm difference between worn and unworn canines; 0.31mm between worn and unworn lateral incisors. The study showed a clear loss of tooth structure from attrition of significant magnitude to consider restoration. Esthetic restoration of worn anterior teeth is often considered to improve the length lost to attrition. Anterior tooth attrition is frequently due to bruxing, clenching and
paranormal functional habits, as well as habits such as biting thread or fishing line, chewing fingernails, chewing on objects such as pencils, rather than from normal chewing. Some patients will exhibit a pattern of wear caused by lower anterior teeth fitting into the wear pattern of upper teeth in latero-protrusive motion. This can be caused by constant light rubbing over many hours of the day, creating a keyhole effect. Worn incisal tips on canines are a common finding as people with canine guidance age. (See Figure 4)

Restoration of these anterior defects, when not associated with significant posterior tooth wear, may be required to break the patient of the habits creating the wear, thereby protecting the teeth from further wear. The least invasive method to restore these defects is by application of bonded composite resin. Because many of the exposed dentinal defects will have sclerotic dentin, bonding to the dentin can be complicated by the significant mineralization of the dentin. Increased etching time as well as preparation to expose fresh dentin has been suggested to improve retention. Overlap of the restoration on enamel surfaces is also suggested to improve retention and improve esthetics. In the studies cited anterior guidance was altered to achieve posterior disclusion.

Restoration of teeth affected by bruxing, where canine guidance is restored can be seen in the following four figures. The patient wanted the midline diastema closed. To do so and maintain appropriate tooth proportionality, the centrals and laterals had to be lengthened. By altering the lateral guidance the additions (in the form of veneers) were durable enough to protect the teeth and maintain the integrity of the restoration. (See Figures 10-13)

Bruxism is thought to affect 5-20% of a normal population. Normal loss of enamel due to natural wear is estimated to be about 10-20 micron per year averaged. Bruxers exhibit three to four times the normal wear. Interocclusal appliances are recommended to limit wear especially in the mouths of nocturnal bruxers. Severe occlusal wear seen in posterior teeth and anterior teeth may require protection from fracture, restoration of lost vertical dimension, improvement in occlusal guidance by restoration of all the teeth with combinations of crowns and/or onlays, or veneers to restore appropriate function, esthetics and guidance, lessening the stress on the individual teeth and the periodontal support mechanisms.

Localized posterior wear creating sensitivity, and/or fremitus (i.e. visible or palpable tooth mobility due to occlusal pressure) can be adjusted to help resolve the tooth sensitivity.

C. Stress
Abfraction lesions created by occlusal stress and in combination with either toothbrush abrasion, and/or corrosion may or may not be sensitive. Localized occlusal adjustment is recommended to decrease the effect of the stress (especially lateral occlusal stress) which may be the etiology creating the NCCL, thereby removing the flexure of the tooth from creating greater concentrations of stress. If the lesion depth has reached an ideal cavity
depth of 1-1.5mm, it may be time to consider restoration of the defect to protect the pulp, after the occlusion has been adjusted to remove lateral stress on the tooth.

These lesions are frequently restored with bonded composite resin restorations. However the loss of this restoration is high if the clinician relies solely on bonding to retain the restoration. Many authors recommend some form of mechanical undercut, as well as bonding. In addition, layering of the initial composite material solely on dentin, then covering with a second or third layer that covers the enamel may limit the effect of the polymerization shrinkage of the composite. This will improve longevity, limit post-operative sensitivity and microleakage. Layering composite material over a resin modified glass ionomer also seems to improve the retention. Use of a three step etch prime and bond is recommended to improve retention as well. As mentioned above, a bonded amalgam restoration can also be placed after preparation of ninety-degree cavosurface margins and some convergence of occlusal and gingival walls.

If the lesion appears to have sclerotic dentin, it is recommended to double the etching time to improve the bond to this hyper mineralized dentin. The different bonding systems have bond strengths significantly different; with the three-step etch, prime and bond exhibiting the best retentive value to sclerotic dentin.

**Conclusions**

Dentists should monitor tooth wear in individual patients especially when they perceive some unusual wear patterns. When tooth wear does not seem to fit the age and occlusion of the patient, its progression should be monitored. Once a pattern of pathologic tooth wear has been identified, attempts should be made to intervene and disrupt the suspected etiologies, whether from corrosion, friction or stress. Year to year comparisons of study casts can be used to assist in the diagnosis and determination of etiology. Prevention and preservation of existing tooth structure is the goal using interventions with the least invasiveness; while also restoring tooth contours as needed to protect the teeth from further pathologic wear and ultimately to prevent fracture of the tooth.
Bibliography


Miller WD. Experiments and observations on the wasting of tooth tissue variously designated as erosion, abrasion, chemical abrasion, denudation, etc. Dental Cosmos 1907; 49:1-234, 109-124, 225-247.


Dunbar UR, Hemmings KW. Treatment of localized tooth wear with composite restoration at an increased vertical dimension. Dent Update. 1997;24:72-75.


Brackett WW, Dib A, Brackett MG, Reyes AA, Estrada BE. Two-year clinical


