Attrition, abrasion, corrosion and abfraction revisited

A new perspective on tooth surface lesions

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ABSTRACT

Overview. The authors propose updated and revised nomenclature, definitions and classification for tooth surface lesions. Their objective is standardization, clarity and clinical utility for the dental practitioner. The article presents a schema of the pathodynamic mechanisms in the formation of tooth surface lesions—three basic physical and chemical mechanisms, their interactions and their dental manifestations.

Conclusions and Clinical Implications. The use of precise definitions will assist the practitioner in determining the etiology of various tooth surface lesions. Understanding the pathodynamic mechanisms and their many possible interactions, as set forth in the schema, will enable the practitioner to make an accurate differential diagnosis and to provide effective prevention and treatment. It also will assist dentists in communicating more effectively with their colleagues as well as with their patients. In addition, the schema helps identify areas in which future research is indicated.

Since the publication of one of the first textbooks for dentistry by English anatomist and physiologist John Hunter in 1778,[1] the definitions and classification of the terms "attrition," "abrasion" and "erosion" have been in a state of confusion. Furthermore, the more recent introduction of the terms "abfraction," to designate stress-induced noncarious lesions,[2] and "corrosion,"[3] to designate chemical degradation have not resolved this dilemma fully. During the two centuries that have ensued since Hunter published his work, a succession of investigators[4-14] have unsuccessfully approached the problem of tooth surface denudation or wasting, using terms and definitions that were mutually contradictory.

Understanding the pathodynamic mechanisms of tooth surface lesions and their many possible interactions will enable the practitioner to make an accurate differential diagnosis and to provide effective prevention and treatment.

In the 1960s, investigations into the etiology of tooth surface lesions by German investigators[15-18] generated renewed interest in these pathological lesions. More recently, American, Australian, English and Japanese investigators[19-32] also have addressed the mechanism of stress concentration in the cervical area. They indicated that occlusal loading forces result in tooth flexure, causing mechanical microfractures and tooth substance loss in the cervical area. These stress-induced lesions are termed "abfractions."[2] Other studies[24,26,27,29] have concluded that acid in areas of stress concentration results in either static stress corrosion or cyclic (fatigue) stress corrosion, both of which should be considered in the etiology of noncarious cervical lesions, or NCCLs. These coactive mechanisms also may produce lesions in other areas of the crowns of teeth, including proximal areas, where stress is concentrated.[31,32] Many of these investigators have supported the conclusion that the etiology of tooth surface lesions generally is a multifactorial event.

DEFINITIONS OF CAUSES OF TOOTH SURFACE LESIONS

The causes of tooth surface lesions henceforth proposed are classified as attrition, abrasion, corrosion and abfraction.

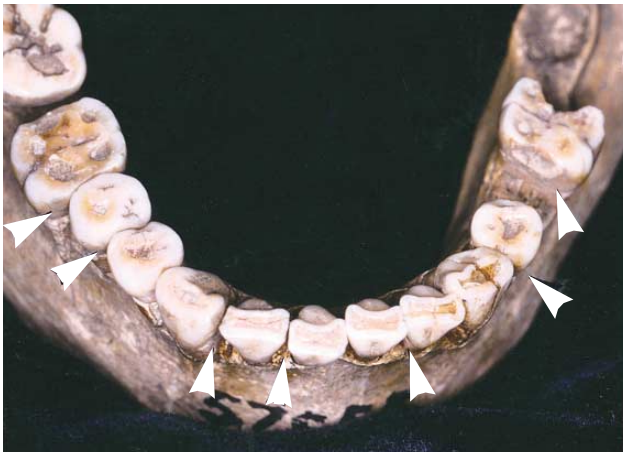

Figure 1. Occlusal abrasion and proximal incisal attrition dating before 1000 B.C. in the mandible of a Pit House American Indian man from the Ohio Valley. Arrows indicate flattened areas of proximal attrition. (Reprinted with permission of the Peabody Museum of Archeology and Ethnology, Harvard University, Cambridge, Mass. Copyright President and Fellows of Harvard University.)

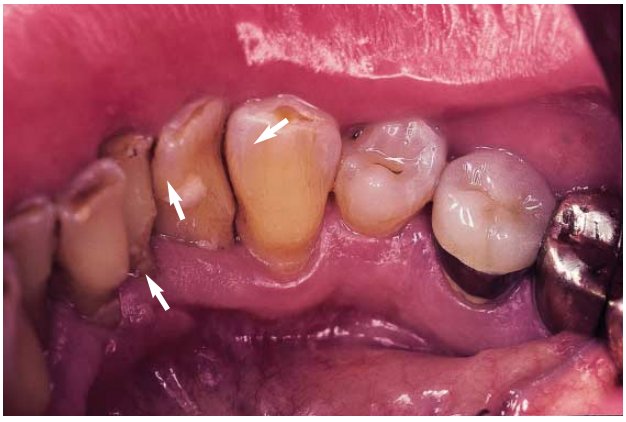

Figure 2. Proximal attrition at the mesial aspect of tooth no. 27 (arrow) against the cingulum of the rotated lateral incisor. Multifactorial lesions are present; lingual abfractions at the cervical areas of teeth nos. 24 and 25 resulting from fingernail biting, as evidenced by the incisal abrasion combined with attrition (bruxism). Consumption of sour candies reveals that corrosion was a cofactor as indicated by the incisal invagination (Class VI) and extent of the lingual corrosion-abfractions.

Attrition. Tooth-to-tooth friction causes the form of wear called "attrition." Occlusal and incisal attrition can occur during deglutition and clenching[32-35]; however, wear becomes most severe during bruxism, as evidenced by the advanced and often rapid wear of the teeth seen in that condition. Proximal attrition (which occurs at contact areas) can cause a reduction of the dental arch[36] (Figures 1 and 2).

Abrasion. Friction between a tooth and an exogenous agent causes wear called "abrasion." If teeth are worn on their occlusal surfaces, incisal surfaces or both by friction from the food bolus, this wear is termed "masticatory abrasion" (Figure 1). The differential wear rate between dentin and enamel occurring in areas of exposed dentin may be a cofactor in the formation of some Class VI lesions.[13] Masticatory abrasion also can occur on the facial and lingual aspects of teeth as coarse food is forced against these surfaces by the tongue, lips and cheeks during mastication.

Abrasion can occur as a result of overzealous toothbrushing, improper use of dental floss and toothpicks, or detrimental oral habits such as chewing tobacco; biting on hard objects such as pens, pencils or pipe stems; opening hair pins with teeth; and biting fingernails. Abrasion also can be produced by the clasps of partial dentures. Occupational abrasion may occur among tailors or seamstresses who sever thread with their teeth, shoemakers and upholsterers who hold nails between their teeth, glassblowers, and musicians who play wind instruments.

Corrosion. Tooth surface loss caused by chemical or electrochemical action is termed "corrosion." There are both endogenous and exogenous sources of corrosion.

Endogenous sources of corrosion. Bulimia produces a unique pattern of enamel loss. The corrosion, called "perimolysis," is most marked on the palatal surfaces of maxillary anterior teeth and, in more severe cases, on the buccal surfaces of posterior teeth. This pattern is consistent with the head's position while vomiting. The forcefully directed movement of the vomitus, which has a mean pH of 3.8,[37] determines the site and extent of dental corrosion.[38]

As first reported by Howden,[39] a special pattern of surface loss also is observed in patients with gastroesophageal reflux disease, or GERD. However, the movement of acid gastric juice in GERD as compared with that in bulimia is slower, less forced, more prolonged, more pervasive and more likely to intermingle the acid with food, especially when the condition is "silent" and unknown to the patient. The enamel appears thin and translucent; enamel is lost on the posterior occlusal and anterior palatal surfaces; depressions or concavities occur at the cervical areas of upper anterior teeth. "Cupped" or invaginated areas develop where dentin has been exposed on the occlusal surfaces of posterior teeth because of wear. This dentinal cupping results from the joint digestive action of hydrochloric acid and the proteolytic enzyme pepsin that is contained in gastric

juice.[40,41] Atypical sites of corrosion may occur at locations where the gastric reflux fluid pools, especially while the patient is asleep.[42-44] When the dentist finds evidence of gastric reflux, referral to a gastroenterologist for evaluation and control is indicated.

Gingival crevicular fluid, as suggested by Bodecker,[45] has been shown to be acidic and may be corrosive when in contact with teeth in the cervical region.

Biocorrosion, or caries, is the loss of hard tissue tooth substance caused by corrodents that are produced by resident bacterial plaque. The etiology of caries is a process that generally is accepted as involving both bacterial acidogenic and proteolytic mechanisms.[11]

Exogenous sources of corrosion. It has been reported that any food substance with a critical pH value of less than 5.5 can become a corrodent and demineralize teeth.[46-48] This may occur as a result of consuming and/or mulling highly acidic foods and beverages such as mangoes and other citrus fruits, drinking carbonated soft drinks and sucking sour candies. Acidic mouthwashes also may be implicated. Acidulated carbonated soft drinks have become a major component of many diets, particularly among adolescents and young children.[49] In 2000, the per capita consumption of these beverages in the United States was 53 gallons.[50]

The citrate ion may be particularly destructive because of its binding or chelating action on calcium. Although carbonated beverages frequently are cited in the literature as a cause of tooth decalcification, their corrosive effect results more from added citric and phosphoric acids than from the carbon dioxide they contain.[42,51-53] Carbonated mineral water was found to increase in pH by almost one-half a unit as it is poured; its corrosive effect on enamel was shown to be minimal.[54] As reported by Lussi,[52] the corrosive potential of an acidic drink does not depend exclusively on its pH value, but also is strongly influenced by its buffering capacity, the chelation properties of the acid and by the frequency and duration of ingestion.

There are many substances that can corrode teeth. As reported by Verrett,[38] chewable vitamin C tablets,[55] aspirin tablets,[56] aspirin powders[57] and the use of the amphetamine drug Ecstasy[58] have been associated with corrosion on the occlusal surfaces of posterior teeth.

Topical application of cocaine to the oral mucosa has been reported to produce cervical corrosion on the facial surfaces of maxillary anterior and first premolar teeth.[59] Raw cocaine that has been cut with confectioner's sugar or cream of tartar, both highly cariogenic, are the diluters of choice.[60] Alcohol abuse has been reported to cause a high incidence of corrosion, owing to the chronic regurgitation and vomiting that stems from the gastritis associated with alcohol abuse.[61,62]

Occupational tooth corrosion can occur during exposure to industrial gases that contain hydrochloric or sulfuric acid, as well as acids used in plating and galvanizing and in the manufacture of batteries, munitions and soft drinks.[11,42]

The term 'erosion' should be deleted from the dental lexicon and supplanted by the term 'corrosion' to denote chemical dissolution of teeth.

Erosion, as defined by the American Society for Testing and Materials Committee on Standards,[63] is "the progressive loss of a material from a solid surface due to mechanical interaction between that surface and a fluid, a multicomponent fluid, impinging liquid or solid particles." This can be observed as a shoreline is eroded by the pounding surf, or bridge supports are eroded by the rush of river waters around them. No such powerful flow of fluids occurs in the human mouth to affect teeth. Therefore, erosion, as defined here, has no significant effect on teeth. The term "erosion" should be deleted from the dental lexicon and supplanted by the term "corrosion" to denote chemical dissolution of teeth.

Abfraction. Abfraction is the microstructural loss of tooth substance in areas of stress concentration. This occurs most commonly in the cervical region of teeth, where flexure may lead to a breaking away of the extremely thin layer of enamel rods, as well as microfracture of cementum and dentin.[21,22] These lesions, which appear to result from occlusal loading forces, frequently have a crescent form along the cervical line, where this brittle and fragile enamel layer exists[21-23] (Figures 2-5).

Palamara and colleagues[29] used scanning electron microscopy and profilometry to assess enamel loss in the cervical area of extracted teeth under cyclic occlusal loading, within the range of

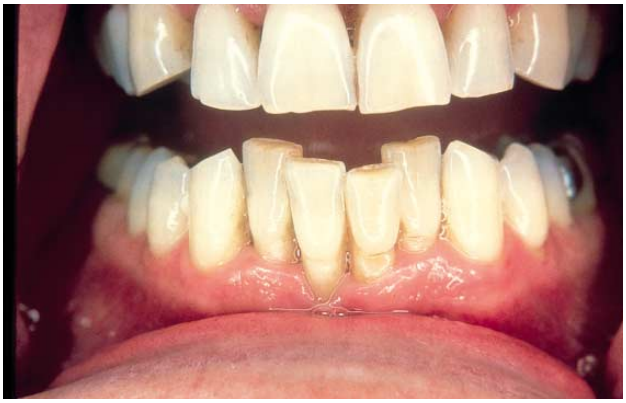

Figure 3. Cervical abfraction on the mandibular left incisor. Of the two mandibular central incisors, this tooth has experienced the greater incisal wear (attrition) indicating the greatest occlusal stress, owing to the malposition, occlusal interference and bruxism. Although the root of tooth no. 25 is more labially positioned, has the greater recession and would be more vulnerable to toothbrush/dentifrice abrasion, it has developed only a minimal lesion. Multifactorial lesion of the anterior teeth manifested as a dentinal invagination on the incisal edge (Class VI lesion) of the mandibular left central incisor. Etiologic factors included bruxism, a coarse diet and consumption of acidic fruits.

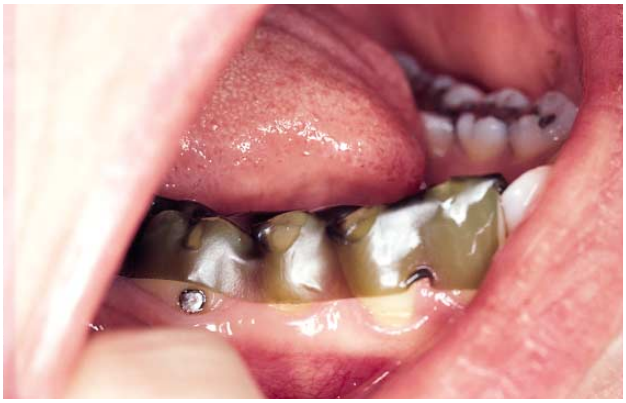

Figure 5. The abfractive lesions depicted in Figure 4, caused by the mechanism of stress from eccentric loading as a cofactor, and verified by the use of occlusal indicator wax (Kerr Dental, Orange, Calif.)

loads encountered in normal function. A tooth submerged in water showed enamel fractures at the cementoenamel junction, or CEJ, after only 200,000 cycles, equivalent to about 2.5 months of normal chewing. This increased after 500,000 cycles. One small area of enamel was completely chipped away from the CEJ.

In a similar study, Hanaoka and colleagues[25] reported development of a crack network about 1.2 millimeters in width on the superficial cementum surface along the CEJ, which increased as the number of cycles increased. They stated that "mechanical microcracks on cementum and dentin … may act as the initial

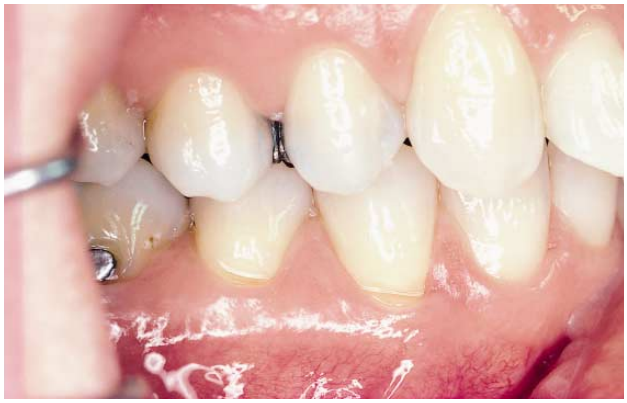

Figure 4. Abfractions, in the form of wedge-shaped lesions, starting in the cervical enamel of the two premolars caused by eccentric loading. The loss of tooth structure surrounding the molar amalgam appears to be caused by stress (abfraction) and toothbrush/dentifrice abrasion.

contributor to the formation of cervical defects. Abfraction has a possibility of being the initial factor and the dominant progressive modifying factor in producing cervical lesions."[25]

Occlusal loading forces applied to the teeth are transmitted through them to the periodontal supporting structures, which may cushion and dissipate the resultant stresses. Thus, mobile teeth are less likely to develop the stress concentration that can produce abfraction. In their studies, Kuroe and colleagues[64,65] indicate a positive correlation of cervical tooth surface lesions with tooth stability and periodontal support.

Stresses that concentrate to produce abfractions in teeth usually are transmitted by occlusal loading forces.[15-28] Occlusal interferences, premature contacts, habits of bruxism and clenching all may act as stressors.[33] Tooth contact during swallowing occurs 1,500 times daily according to Shore[33] and 2,400 times daily according to Straub[34] and Kydd.[35] These repetitive static and cyclic occlusal loads also could contribute to the formation of abfractions; however, when in combination with a corrodent, an abrasive or both, the odontolytic effect may become highly significant.

The presence of occlusal wear facets may be related to the formation of cervical lesions. Indeed, this has been the finding of many investigations.[22,26,28,66-68] Kornfeld[68] indicated that the cervical surface lesions tended to occur on the part of the tooth opposing the side that had developed an occlusal wear facet caused by attrition.[67] In other words, if the attrition facet was found toward the mesio-occlusal edge of a tooth, the abfraction would tend to occur toward the distal cervical

region, where flexure would tend to concentrate the stress. Grippo and Simring[3] reported a case with a disto-occlusal attrition facet and a mesiogingival biocorrosion (caries) abfraction lesion.

COMBINED MECHANISMS OF TOOTH WEAR

Although some of the aforementioned individual mechanisms may act independently, combined mechanisms occur frequently during the dynamics of interocclusal activity. From a bioengineering perspective, many additive or synergistic combinations of mechanisms may occur simultaneously, sequentially or alternately, thus explaining the loss of dental hard tissue.

Attrition-abfraction. Attrition-abfraction is the joint action of stress and friction when teeth are in tooth-to-tooth contact, as in bruxism or repetitive clenching (Figure 3).

Abrasion-abfraction. Abrasion-abfraction is the loss of tooth substance caused by friction from an external material on an area in which stress concentration due to loading forces may cause tooth substance to break away. Such a synergistic tooth-destructive effect may be observed cervically when toothbrushing abrasion exacerbates abfraction to produce wedge-shaped lesions (Figures 4 and 5). The critical roles of both toothbrushing abrasion and occlusal loading of an anatomically vulnerable zone may be one reason why such lesions are limited almost exclusively to the buccal and labial cervical areas of teeth.

Corrosion-abfraction. Corrosion-abfraction is the loss of tooth substance due to the synergistic action of a chemical corrodent on areas of stress concentration. This physicochemical mechanism may occur as a result of either sustained or cyclic loading and leads to static stress corrosion or cyclic stress corrosion.

Static stress corrosion. Static stress corrosion is the loss of tooth structure owing to the action of a corrodent on an area of sustained stress. This may occur during clenching. Static stress corrosion may be observed as demineralization that occurs around orthodontic appliances in the presence of a corrodent (Figure 6).

Cyclic (fatigue) stress corrosion. Cyclic (fatigue) stress corrosion is the loss of tooth structure due to the action of a corrodent in an area of concentrated stress during cyclic loading. This combination of mechanisms could occur during mastication, as seen among patients who engage in fruit mulling as dentinal invaginations, but is seen most strikingly among patients who brux in the presence of endogenous (for example, GERD) or exogenous (carbonated soft drinks) corrodents. In such situations, tooth substance may be lost rapidly and extensively.[28]

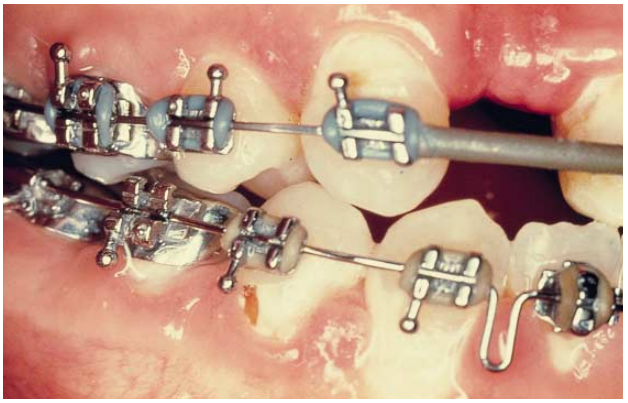

Figure 6. Corrosion (demineralization) on teeth nos. 6 and 26 through 28 and biocorrosion (caries) on the facial aspect of teeth nos. 6 and 28 accelerated by stress (abfraction) in the cervical areas in which stress is concentrated by orthodontic appliances, and exacerbated by poor oral hygiene.

In addition to the acidic nature of bacterial plaque, it has been shown that gingival crevicular fluid also is acidic.[42] Thus, the occasional finding of subgingival cervical lesions may well be examples of a corrosion-abfraction process.

Furthermore, Palamara and colleagues[29] found that when cyclic loading was combined with immersion in 1 percent lactic acid, buffered to pH 4.5 (mimicking the weak acid of dental plaque), exaggerated effects of tensile stress resulted. Regardless of the presence or absence of load, during acid dissolution (corrosion), greater volume loss occurred in the cervical area than in the middle one-third of the teeth. However, loaded teeth showed a loss of enamel 10 times greater than that of unloaded teeth.

Attrition-corrosion. Attrition-corrosion is the loss of tooth substance due to the action of a corrodent in areas in which tooth-to-tooth wear occurs. This process may lead to a loss of vertical dimension, especially in patients with GERD or gastric regurgitation. An occlusal or incisal pattern of wear develops (see description in the next paragraph).

Abrasion-corrosion. Abrasion-corrosion is the synergistic activity of corrosion and friction from an external material. This could occur from the frictional effects of a toothbrush on the superficially softened surface of a tooth that has been demineralized by a corrosive agent. Teeth that

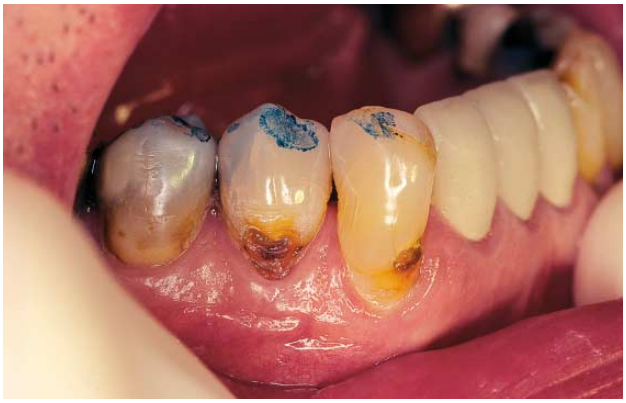

Figure 7. Biocorrosion (caries)-abfraction: articulating paper markings indicate eccentric loading, which induced stress concentration in the cervical region (abfraction) and may have exacerbated the caries (biocorrosion).

BOX

PATHODYNAMIC MECHANISMS OF TOOTH SURFACE LESIONS.

INDIVIDUAL

- Friction (resulting in wear): attrition or abrasion
- Corrosion (resulting in chemical degradation)
- Stress (resulting in microfracture and abfraction)

COMBINED

- Attrition-abfraction
- Abrasion-abfraction
- Corrosion-abfraction
- Static stress corrosion
- Cyclic (fatigue) stress corrosion
- Attrition-corrosion
- Abrasion-corrosion
- Biocorrosion (caries)-abfraction

MULTIFACTORIAL

- Combinations of friction, corrosion and stress

are out of occlusion could be affected by this mechanism and develop cervical lesions, since they frequently extrude, thus exposing the vulnerable dentin. Similarly, gingival recession may expose the cementum and dentin to this odontolytic process.

Abrasion-corrosion also is observed frequently on occlusal surfaces. Simple abrasion or attrition, in the absence of corrosion, results in broad, flat occlusal facets or tables with enamel and dentin fairly evenly worn. However, when the cuspal enamel has worn down to the level where dentin becomes exposed, a unique pattern of occlusal wear develops. "Islands" of superficially softened dentin, each within a rim of the still-hard enamel, become "cupped" or invaginated owing to the joint digestive action of the proteolytic enzyme pepsin and the hydrochloric acid in gastric juice.[40,41] The occlusal pattern of dentin cupping or invagination also is seen in people who eat large quantities of acidic foods, such as mangoes, citrus or similar fruits and plants such as sorrel (Figure 3). In such cases, the more rapid wear of softened dentin leading to occlusal invagination results from differential abrasion-corrosion, which in turn results from friction by the acidic food bolus (as described first by Simring[69] and subsequently by Eccles[70]).

Biocorrosion (caries)-abfraction. Biocorrosion (caries)-abfraction is the pathological loss of tooth structure associated with the caries process, where an area is micromechanically and physicochemically breaking away due to stress concentration. A common site for this synergistic activity is the cervical area of the tooth, where it may be manifested as root or radicular caries. The combined mechanisms of static stress corrosion and cyclic (fatigue) stress corrosion can account for the rapid odontolytic progression of these types of carious lesions. We postulate that these two mechanisms frequently may be considered as cofactors in the etiology and progression of caries, particularly root caries (Figures 6 and 7). Lehman and Meyers[32] published a noteworthy treatise on this subject, asserting (on the basis of evidence deduced from photoelastic studies) that caries occurs in areas of stress concentration. An important distinction from other dental hard-tissue lesions is that bacterial plaque produces the corrodents. In vitro studies have demonstrated that stress concentration in the presence of acid should be considered in the etiology of NCCLs.[24,27,29] Therefore, we may extrapolate that the same combined mechanisms, at times, may apply to caries formation and progression—especially in root caries, which occurs in an area in which stress concentrates intensely. This is an area warranting further research.

Orthodontic forces, which cause stress concentration, appear to contribute to the etiology of biocorrosion (caries), usually in the cervical facial region of teeth and associated with the presence of bacterial plaque in patients who have poor oral hygiene habits (Figure 6).

MULTIFACTORIAL MECHANISMS

Frequently, more than two mechanisms may be involved in the etiology of tooth surface lesions (Box; Table). For example, a corrosive cervical lesion could be exacerbated by toothbrushing

abrasion. When to these two mechanisms are added the effect of stress (abfraction) resulting from bruxism or occlusal interference, these lesions then become corrosive-abrasive-abfractive in nature (Figures 2, 3 and 8). These various mechanisms can occur either synergistically, sequentially or alternately. The schema described in the following section provides a means for the clinician to identify the existing mechanisms and their interactions in any given situation.

TABLE

ETIOLOGY OF TOOTH SURFACE LESIONS.

PATHODYNAMIC MECHANISMS	ETIOLOGIC FACTORS
Stress (Microfracture/ Abfraction)	
Endogenous	Parafunction (such as bruxism, clenching) Occlusion: premature contacts, eccentric loading Deglutition
Exogenous	Mastication of hard, resistant foods Habits: biting foreign objects such as pencils, pipe stems, fingernails Occupational behaviors: playing wind instruments, using teeth to hold foreign objects Dental appliances: orthodontic, removable denture clasps and rests
Corrosion (Chemical Degradation)	
Endogenous	Plaque: acidogenic and proteolytic bacteria Gingival crevicular fluid Gastric juice in patients with gastroesophageal reflux disease, bulimia, eructation
Exogenous	Consumption of acidic beverages, citrus fruits and juices Occupational exposures to acidic industrial gases and other environmental factors
Friction (Wear)	
Endogenous (attrition)	Parafunction (such as bruxism, clenching) Deglutition
Exogenous (abrasion)	Mastication of coarse foods Inappropriate or overzealous use of dental hygiene instruments: toothbrush, dentifrice, dental floss, toothpicks, interdental cleaners Detrimental oral habits: fingernail biting, pipe smoking, tobacco chewing, hair pin opening Occupational behaviors: severing thread with teeth, blowing glass, playing wind instruments Dental appliances: removable denture clasps and rests Ritual behaviors: mutilation of teeth

SCHEMA OF PATHODYNAMIC MECHANISMS FOR TOOTH SURFACE LESIONS

It now becomes apparent that there are three basic physical and chemical mechanisms involved in the etiology of tooth surface lesions (Box; Figure 9). The various types of dental lesions are the result of these mechanisms acting either alone or in combination. The mechanisms are

- friction, including abrasion (which is exogenous) and attrition (which is endogenous), leading to the dental manifestation of wear;
- corrosion, leading to the dental manifestation of chemical or electrochemical degradation;
- stress, which results in compression, flexure and tension, leading to the dental manifestations of microfracture and abfraction.

These three basic mechanisms and their areas of overlap and interaction, as indicated in the schema in the form of a Venn diagram, are the initiating and perpetuating etiologic factors in producing tooth surface lesions (Figure 10). Certainly, the structure and composition of teeth as well as their environment are additional determinants of the dental lesions. Frequently, one mechanism will predominate; however, other mechanisms may be involved, thus making the etiology

Figure 8. Abfraction-attrition-corrosion-abrasion: multifactorial lesions are depicted as the progressive loss of the tooth surface on the incisal and facial surfaces of teeth (nos. 22-25) owing to the pathodynamic mechanisms of stress, corrosion and friction. The lesions at these locations suggests that the manifestations are attrition and abfraction from occlusal stress, owing to bruxism, as well as corrosion, indicated by the incisal "cuppings" (Class VI lesions) and smooth, rounded margins of the lesions. Toothbrush/dentifrice abrasion also may be a cofactor.

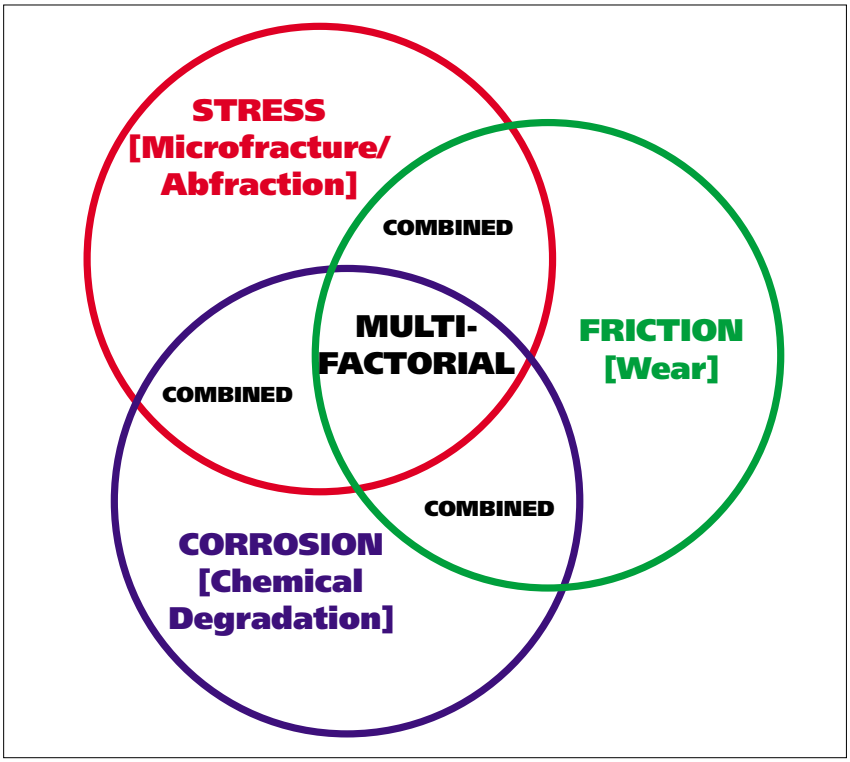

Figure 9. Schema of pathodynamic mechanisms of tooth surface lesions shows the three physical and chemical mechanisms within each circle. Under each mechanism, in brackets, is the resultant dental manifestation. Where any two circles overlap, combined mechanisms are active. In the center, all three mechanisms overlap, indicating a multifactorial etiology with many possibilities of varying degrees, sequences of input by each pathodynamic mechanism, or both.

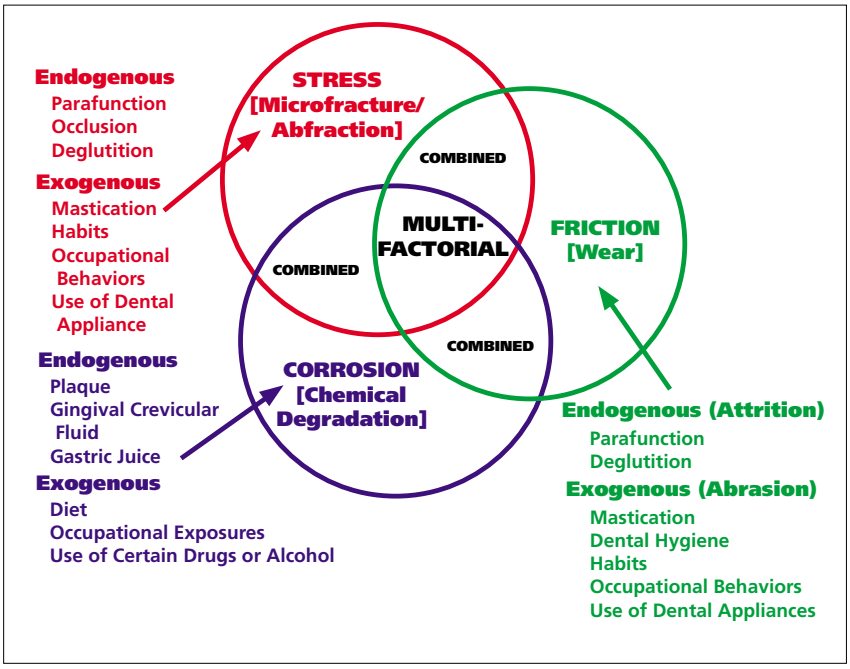

Figure 10. Expanded schema of pathodynamic mechanisms, showing some of the etiologic factors (endogenous and exogenous) that produce the mechanisms.

multifactorial in nature. Attempts at "differential diagnosis," which gives primacy to a single mechanism, are likely to fail because they miss the interactive synergy of the various coactive mechanisms. Understanding the multifactorial nature of these lesions will assist the clinician in developing a multifaceted approach to their prevention, diagnosis, treatment and control. From a heuristic standpoint, we hope that use of the schema will lead to research that can pinpoint precisely which etiologic factors are active in any given lesion and the extent or significance of the specific factors involved (Table).

CONCLUSION

We feel that our proposed definitions of the mechanisms responsible for tooth surface lesions will simplify and clarify dentistry's understanding of the etiology of both carious and noncarious lesions. In light of evidence gleaned from recent scientific studies, it is incumbent that dentistry should achieve a common language with our sister sciences—especially the rapidly emerging fields of biomedical and biodental engineering—and use more precise definitions and terminology.

The mechanisms of stress, corrosion and friction appear to be critical factors in the etiology and progression of tooth surface lesions. The pathodynamic schema we present here can become an effective guide for the clinician in evaluating the many clinical situations encountered. It also may guide the researcher to elucidate the impact of individual mechanisms and their many possible interactions. Understanding these mechanisms, their interactions and

their dental manifestations will enable dentists to diagnose the complex etiology of previously enigmatic tooth surface lesions in a differential manner. Furthermore, the schema will help dentists institute proper prevention and treatment methods and communicate more effectively with their patients. A successful diagnosis and treatment plan requires keen observation, a thorough patient history and careful evaluation. ■

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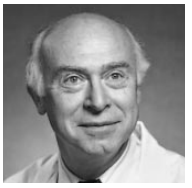

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