DENTAL 'EROSION' REVISITED

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Since its publication in 1908, G.V. Black's "A Work on Operative Dentistry" has served as the definitive text in most dental schools and has influenced the thinking of essentially all present-day dentists. Unfortunately, Dr. Black's use of the term "erosion" has led to much ambiguity in the dental community since he did not distinguish between etiologies and the various lesions which they cause. His terminology lacked specificity. Further, his use of the term was misleading, as many etiologies of dental hard tissue lesions are in conflict with the precise meaning of "erosion." G.V. Black established misuse of the term by stating that toothbrush abrasion was a cause of erosion. The ninth edition of Black's book listed 11 causes for the development of erosive lesions—among which were "friction, most generally of the toothbrush carrying some abrasive tooth powder." Shaffer, Hine and Levy, however, asserted that "attrition, abrasion and erosion are three separate and distinct processes, each of which results in loss of tooth substance. The terms are frequently used interchangeably, but such careless terminology

ABSTRACT

The term "erosion," as currently used in dentistry, is not only a misnomer but also is misleading, according to the authors. The authors highlight the critical roles of occlusal function and parafunction in the multifactorial etiology of cervical lesions and relate their significance to clinical practice. They propose a revised nomenclature for and classification of dental hard tissue lesions.

Erosion serves only to confuse the recognition of the etiology and to delay institution of proper treatment." Unfortunately, the cause for this constellation prevails to the present day.

GIVING 'EROSION' A UNIFORM MEANING

Dental professionals currently use the term "erosion" to describe a loss of tooth substance by a chemical process that does not involve known bacterial action. The "Glossary of Metallurgical Terms and Tables," endorsed by the American Society for Metals, defines the term "erosion" as the abrasive destruction of materials by the movement of liquids or gas, with or without solid particles. Corrosion, as defined by the same text, is the physical deterioration of a material by chemical or electrochemical attack—which describes more precisely the process dentists identify as dental erosion.

Based on these definitions, the material loss that occurs during erosion, as described by engineers, results from mechanical degradation rather than a chemical breakdown. Current dental textbooks, however, state that erosion is chemically induced. In contradistinction, the chemically induced loss of a material, as defined in the field of engineering, is termed "corrosion."

Since other scientific disciplines define the term "erosion" with consistency, dentistry should consider adopting a vocabulary that conforms with that of the rest of the scientific community.

Furthermore, engineers use the term "stress corrosion" whenever stresses are concentrated in the presence of a corrosive substance. This pro-
cess, though well recognized in the engineering community, has not received adequate attention in dentistry. A search through the dental literature shows that numerous studies have been conducted on stress corrosion failure in metal and ceramics, but none with regard to its effects on the hard tissue of teeth per se. Since teeth exist in a dynamic and complex ecosystem involving changes in chemistry, forces, stresses and strains, dentistry likewise could adapt this term.

Chemical effects on the hard tissue of the teeth, which until now have been called "tension" by dentists, would be classified more appropriately as corrosion or stress corrosion, and not corrosion at all.

NON-METALLIC CORROSION

"Perry's Chemical Engineers' Handbook" describes both metallic and non-metallic corrosion. Metallic corrosion is defined as the degradation of a metal by chemical or electrochemical attack in a particular environment. The deterioration of plastics and other non-metallics, which are susceptible to swelling, crazing, softening and so forth, is essentially physicochemical rather than electrochemical in nature. Teeth, which are non-metallic and crystalline, fall into this latter category. Most dentists associate the term "corrosion" with metals exclusively, not realizing that non-metals also are susceptible to corrosion. Non-metallic corrosive effects are seen on tooth enamel, cementum and dentin.

STRESS CORROSION AND ABRACTION

The physicochemical degradation of tooth substance is a significant event that may occur during interocclusal activity. This most notably involves the cervical region of the teeth and takes place in the presence of acidic substances. This process, which is a synergistic interaction of mechanical stress caused by loading forces and chemical corrosive reactions, is properly termed "stress corrosion." In addition to this physicochemical activity, the effect of piezoelectricity may contribute to tooth substance loss (Figure 1). Piezoelectricity refers to the small electric charge that is generated when teeth are mechanically deformed.

The dynamic stress that occurs in the mouth during interocclusal activity such as chewing or bruxing significantly influence the breakdown of tooth structure. Engineering studies have demonstrated that when teeth are loaded in a horizontal direction, the effect of stress becomes concentrated in the cervical region, causing fracture. The cyclic tension and compression that occur in this area can reach a fatigue limit and result in cracking or breakage of the tooth structure. These horizontal loading forces cause a microscopic bending of the anatomical crown of the tooth, with resulting tensile stress concentration in the cervical region on the side of the tooth from which the force is directed. At the same time, the opposite region is under compressive stress. When the direction of the force changes (as in bruxism), the tooth bends in the opposite direction and the stresses correspondingly reverse at this cervical area. This bending of the tooth from side to side results in fatigue and fracture of the root in the most flexed zone, the cervical surface layer, be it enamel, cementum or dentin. In an in vivo stress corrosion study, a greater amount of tooth substance was lost in areas of tension than in the areas of compression. A similar effect of the hook and forth micro-bending of the crown, which is firmly held by its root, is root fracture of a tooth restored with a post-and-core, especially when such a tooth is subjected to interocclusal loading.

Many investigators hypothesize that these interocclusal forces create physical microfractures, or abrasions, at the cervical region, which in turn re-
result in cervical lesions. It seems logical and appropriate to adopt a terminology that would most accurately describe the etiology of such lesions, namely, “stressed corrosion cracking,” which also can result in “stress corrosion cracking.” Based on this reasoning, Grippo suggested that lesions that result from the micro-cracking of tooth substance and the process of physical or physiochemical weakening be referred to as “abfraction,” meaning “breaking away,” since this term describes graphically the process and the resulting effect.

ROLE OF FUNCTION AND PAREFUNCTION

Sognnaes criticizes the work of Bird and Kornfeld, who independently described the presence of abnormal excessive occlusal wear on the teeth among patients exhibiting dental erosion. Kornfeld even went so far as to relate the wearing facets to precise parts of the teeth, claiming that “erosion” tended to occur on the part of the tooth opposite to the side that was subject to abnormal wear. In other words, if the wearing facets were found toward the mental edge of a tooth, the “erosion” would tend to occur toward the distal portion of the labial or buccal surface. This mechanism is quite understandable in terms of the process of abfraction. The mechanisms of pulpal or salivary effects, suggested by Sognnaes, have not been satisfactorily substantiated.

In a study that related various factors (bruxism, horizontal brushing, use of acidic juices, use of alcohol and smoking) to the development of cervical dental lesions, only bruxism showed a strong positive correlation. In addition, Kristen and Rothe found that 97 percent of patients who had wedge-shaped cervical lesions also had parafunstional disorders. Furthermore, Grahn, Berndt and Stange studied a random group of 975 patients and concluded that “a causal relation appears to exist between wedge-shaped defects, occlusal disharmony, parafunstions and strong psychological tension of patients.”

Stress resulting from occlusal force has been recognized as an factor in the development of cervical lesions more than half a century ago. Nevertheless, no one has published research on the role that occlusal therapy (for example, orthodontics, supragingival occlusal splints, compensatory occlusal adjustment and restoration) might play in the prevention or arrest of such lesions. Hopefully, adoption of the more accurate terminology proposed in this article will foster research along these lines.

EVIDENCE FOR COMPLEX ETIOLOGY

Although they play a role, physical loading forces are unlikely to be entirely responsible for the formation of cervical lesions. Intracoronal chemical influences and toothbrush-induced abrasion combined with the dynamics of interocclusal activity (for example, chewing, swallowing and parafunction) lead to stress corrosion and contribute to development of cervical lesions. The brittleness of tooth structure (especially the extremely thin layer of enamel near the cervical line) is an additional factor in determining the abfraiding response to loading forces. Based on many decades of observation, the authors, as well as other investigators, believe that many factors combine to produce progressive cervical lesions.

Further evidence against the solely chemical explanation for some of these so-called “erosive” lesions comes from a study conducted by Rust and Brocks, who reported similar degradation on plastic teeth and denture base materials. Sognnaes, Wolcott and Xiong also reported the progress of such defects in chemically inert filling materials such as gold foil. Abrasion alone can be ruled out as the critical factor in the genesis of “erosive” lesions by noting that when lesions develop on restored teeth, the wearing is greater on the enamel than on the restorative material, although the restoration has a higher wear rate. Furthermore, these lesions may develop subgingivally, at sites inaccessible to toothbrush abrasion (Figure 2).
The concept of abrasion readily explains these findings, especially when considered in conjunction with abrasion and stress corrosion, which would differentially affect all of the materials involved: tooth, amalgam, gold, acrylic, ceramic, alloy, and composite. Also, after treating cervical lesions with a variety of Class V restorations, researchers found that the rate of progress of the destruction increased from an average of 7 microns to 2 microns a week.

At least three mechanisms probably are involved here:

- The restoration may have acted as a strut to support the tooth and, thus, minimized flexure and abrasion.
- The restoration and its adjacent enamel could have been more resistant to abrasion and corrosion than the exposed dentin at the surface of an unrestored abrasion; thus, restoration of cervical lesions provided both protective and retardant values.
- The restoration, while subject to abrasion and flexural abrasion, may not have been subjected to significant intrasurface stress corrosion (chemical effects), whereas the tooth was. Thus, the wearing process, while distinctly retarded, was greater on the enamel than on the restorative material.

**"EROSION" RE-EXAMINED**

In light of the mechanisms that occur during the process of so-called "dental erosion," we propose that the concept as used in dentistry be re-examined, since it plays at best minor and secondary roles in the genesis of hard-tissue lesions. Pure erosion (in the sense of wearing away by fluid flow) probably plays an insignificant role in human dentistry. It is unlikely that the amount of water consumed by humans or the flow of other non-corrosive materials would ever cause any significant loss of human tooth hard tissues. (The only lesions that have been examined by the authors that appeared to have resulted from pure erosion are those of the Okjotoyoi, or sperm whales. Their teeth appear to wear away from mechanical erosion as a result of the huge volume of water flowing over them.)

In Sognnaes' 1963 presentation on "Dental Hard Tissue Destruction with Special Reference to Idiopathic Erosion," the role of function was casually mentioned but not discussed. The significance of stress corrosion as a factor was never considered, since in all probability it was not recognized at that time. The factors of stress and stress corrosion very well could have been the missing links in his investigation into the etiology of these enigmatic lesions, which he called "idiopathic" for lack of his finding an adequate etiology or pathogenesis.

**COMBINED LESIONS**

Because of the diverse dynamics that take place in the oral cavity, particularly during interocclusal contact, most of the lesions that we see result from a combination of two or more processes. From an engineering perspective, four combined, or synergistic, events may explain the loss of dental hard tissue:

- erosion-corrosion;
- abrasion-corrosion;
- abrasion-abrasion;
- biocorrosion-abrasion (caries plus flexural stress).

Although controlled clinical studies have not yet conclusive
ly proves that combined lesions are formed by these mechanisms, the concept that a combination of factors causes them makes engineering sense.

Erosion-corrosion. Erosion-corrosion may result from consumption of highly acidic wine, citrus juices or carbonated beverages. The wearing of teeth as seen in bulimia, termed "peritymolysis," is another example of the combined activities of erosion and corrosion occurring during the flow of regurgitated acidic stomach contents over the teeth. Schachtel has reported that any food substance with a critical pH value of less than 5.5 can demineralize (corrode) the hard tissues of teeth. People who are exposed to a flow of occupational corrosive chemicals, such as hydrochloric and sulfuric acid fumes, also may experience erosion-corrosion.

In each of these instances, mechanical flow (erosion) by a corrosive material causes the combined degradation of the tooth; however, the major activity is one of corrosion or chemical dissolution. The dynamic movement of the corrosive material by the contaminated air mechanically impacts the effects of erosion.

Abraision-corrosion. Should a tooth surface be demineralized by some exogenous or endogenous abrasive agent, then the frictional effects of the toothbrush would easily brush the surface away. Such "abraision-corrosion" could occur if a person were to brush his or her teeth immediately after drinking an acidic beverage such as wine or citrus fruit juice (Figure 3). The resulting tooth substance loss from toothbrush abrasion on the corroded tooth surface should be termed "abraision-corrosion." Such frequently seen combined lesions have formerly been erroneously referred to as "erosion-abraision." Beddoer suggests the gingival crevicular fluid as the source of the acid and provides convincing evidence to support this concept.

Abrasion-abfraction. The pathologic loss of tooth substance caused by frictional effects on an area of abrasion (that is, a surface that has been weakened by biting forces) can be termed "abraision-abfraction." A good example of this would be the effect of toothbrushing on cervical areas that are subject to flexure and where tooth substance weakened physically or physicochemically (by stress corrosion) is abfraacting.

These lesions would be a result of the combined effects of both abrasion and abfraction.

Bicorrosion-abfraction. The combined effect of bicorrosion (caries) and abfraction is the pathologic loss of tooth substance associated with caries (bicorrosion) where plaque adheres preferentially to the tooth surface roughened by flexural microfracture (abfraction). This would be particularly notable in abfractions that occur in areas difficult to access for effective oral hygiene (Figure 4).

One study has suggested that root caries could be potentiated by the presence of plaque in areas of stress concentration resulting in stress corrosion. Furthermore, as the depth of the lesion enlarges through degradation, the degree of stress concentration and thus of bicorrosion-abfraction increases. This may well explain the rapid progression of radicular caries.

Figure 5 illustrates the effect of severely tilted teeth on bicorrosion-abfraction. The mesial tilt of the molar has resulted in elevation of its distal occlusal.

Dr. Ronald J. Saupa, who is postdoctoral resident in pediatric dentistry, University of Washington, Seattle, addressed inquiries to Dr. Drager, 125 Dwight Pl., Longmont, CO 80501.
surface above the general occlusal plane of the dentition. This discrepancy in occlusal pattern may well have produced functional interferences, as evidenced by the flat attrition facets and loss of original cusp morphology at the distal portion of the molar's occlusal surface. The resultant stress due to occlusal interference, may have initiated necrotic stress decorative on the mesioocclusal gingival aspect of this tooth and accelerated the observed biocorrosion (caries). The location of the attrition facets at the distal occlusal aspect and the location of the cervical lesion at the mesioocclusal confirm the findings of Kornfeld regarding the relationship of occlusal attrition facets and cervical lesions.

**SUMMARY**

Due to the confusion that exists about the application of the term "eruption," it is timely and fundamental that a uniform definition be established among the sciences. Adoption of the terms "corrosion," "stress corrosion," the new term "abfraction" and "enamel lesions" in the dental literature in referring to tooth lesions will allow more accurate descriptions of the dynamic events that are now recognized in the mouth. Such terminology would recognize the critical role of occlusal function and parafunction in the multifactorial etiology of dental hard tissue lesions. Use of a common, precise and specific language will improve communication, and should simplify and promote research in this area of bioengineering. This approach will assist in determining the etiology and making a differential diagnosis of these various hard tissue lesions. It will enable the clinician to institute more predictable preventive and therapeutic measures, such as orthodontics, coronoplasty, occlusal adjustment, bite guard appliances and restorative dentistry.

The opinions expressed are solely those of the authors and do not necessarily reflect the opinion of policymakers of the American Dental Association or its subsidiaries.

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Editor's note: We appreciate receiving feedback from readers on the journal's CE program, whether it be compliments or criticism. Dr. Toal raises a valid point, and the editors will take it into consideration.

DENTAL EROSION

Drs. Grippi and Simring are to be commended for their article "Dental Erosion: Revisited" (May), reviewing the mechanisms for the development of non-caries cervical lesions. While we agree with Drs. Grippi and Simring that there is some confusion with the term erosion, we believe that much of the confusion lies not in the terminology, but in understanding of the etiology. Dr. Bader and associates found in a study of North Carolina dentists that the etiology of non-caries cervical lesions was indeed a source of confusion. While many engineering principles are applicable to dentistry, dentistry is not simply an engineering science. It makes no more sense for dentistry to adopt engineering terminology than for engineers to adopt dental definitions. The introduction of a set of new terms will not alleviate the confusion. We suggest that the existing terminology found in the literature is adequate once the etiol-
Only NSK handpieces control patient vector cross-infection at the source...
Authors' response: The intent of our paper was to propose a more therapeutically useful nomenclature for dental hard tissue lesions. Furthermore, we contend that an awareness of biogenic engineering factors may shed light upon the etiology of these lesions.

The recent study by Bader and others at the University of North Carolina (1993), which was cited in the letter, confirms our opinion that confusion exists regarding the etiology of non-cervical lesions. In addition, they stated: "Classification and treatment were also areas of professional uncertainty... Part of this problem may be the result of the terms available for use." It is likely that most of the 859 dentists surveyed were unfamiliar with the biogenic engineering mechanisms set forth in our paper. An understanding of these mechanisms and a knowledge of more precise terms might have guided them to determine the etiology of most of the cervical lesions. Unfortunately, without this information, the problem may be the result of the terms presently used.

To determine the etiology of any pathologic lesion, it is necessary to consider and investigate every factor that may contribute to its genesis. The science of teeth, considered as biomaterial, involves extensive biogenic engineering since teeth undergo many dynamic interactions. As stated in Caputo and Standbee's "Bioengineering in Clinical Dentistry," "all dental tissues and structures follow the same laws of physics as any other material." According to Bayne and others, "the biology of a cell and the actions of tissues follow the same principles of physical chemistry that are the basis for all materials science." These axioms form the basis of much of the biogenic engineering that is involved with the tooth flexure theory which Drs. Lee and Eddie so boldly espoused in their paper.

Naturally, biologic principles must be integrated with the engineering concepts in any living system. The concept of tooth flexure and tooth degradation was first proposed in the outstanding early engineering studies of Korber, Kohler, and Grosskopf in the 50's. All suggested that eccentric loading and flexure appear to play a role in the etiology of non-cervical lesions. Lukas and Spranger published their investigations into the horizontal loading of teeth demonstrating that both torsion and translation take place at the cervix. Drs. Spranger and Haim also suggested that piezoelectricity may play a role in the genesis of cervical lesions. Spranger, Weber, and Kung (1973) described the genesis of cervical lesions as being a multifactorial event. Unfortunately, most of their noteworthy studies have not been previously cited, nor recognized in the American dental literature. Their fine work should be re-examined in light of modern technological advances as there are strong indications that these factors are missing links in our understanding of the genesis of cervical lesions.

We adhere to our position that much can be gained by having a precise and uniform language so as to improve communications with our sister sciences. For example, a more descriptive definition of the term "erosion" as traditionally defined would be "erosion/corrosion." This designation more precisely describes the dynamics of flow (erosion) and the biochemical acidic (corrosion) degradation of hard tissue tooth substance. Thus we denote that two activities occur during this event.

We concur with the fourth category referred to as "stress lesions," but differ by naming them "abfractions." A variety of physical stresses in addition to tensile stress occur in the cervical region during the dynamics of function and parafunction—namely, torsional, compressive, shear and flexural. Furthermore, we suggested in our paper that "stress corrosion" is exacerbated in the area of stress concentration, especially if the toothbrush is abrading and inducing an added stress raiser component.

Ultimately, time will provide the final test of whether to retain the old catchall misnomer "erosion" to describe all non-cervical hard tissue lesions or whether it should be consigned to the limited and specific use indicated in our paper. If its former use is indeed a misnomer (as we have indicated), then it will soon become archaic and finally obsolete.

Furthermore, our suggested specific functional terms will
Hopefully supplant the extemaded classification and lead to widespread therapeutic advances.

The excellent recommendation by Dr. Lee and Eckle to utilize a more flexible material when restoring abfraction lesions is a logical outcome of the new concepts proposed in our revisited terminology. Another valuable outgrowth of this new approach will be the inclusion of occlusal adjustment, coronal reshaping and biteguard construction in the therapy of stress-associated lesions (abfractions) of teeth.

Surely, it is not easy to drop our overly conservative adherence to old terms, which we have always used, even when it becomes evident that they were based on now-antiquated concepts; but progress demands that we have the courage of our convictions to change in the light of new data.

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DELTAL IMPLANTS

We were certainly pleased to see that the March issue of JADA focused on dental implants, but it was an embarrassment to us as implant professionals that a notable and respected organization such as the ADA would be so blatant as to print a glaring example of inappropriate diagnosis and implant treatment on the cover of The Journal.

The cover graphic portrayed four root implants placed in the lower jaw supporting fourteen artificial teeth. We submit that it is completely inadequate support and anchorage for this type of implant prosthodontics. Even though the cover is a graphic, this treatment suggests potential for success where in actuality it has an extremely high probability of failure. There are many predictable and potential flaws acting upon the relatively small bone-implant interface supporting these fourteen teeth that would encourage a failure of the interface and possible breakage of the prosthesis. If such failure were to occur, which is quite likely, it would be very difficult to defend in a malpractice suit. Consequently, the American Dental Association is, by omission or commission, a party to providing highly questionable information to its readership via this cover.

Unfortunately, many of the dentists who are placing implants, after having attended a two-day company-sponsored course, may not have any appreciation for the physics involved in a proper implant support system. This is precisely why the American Academy of Implant Dentistry (AAID) is trying to promote the pursuit of comprehensive dental implant education to qualify practitioners to perform not only the placement of the implants and construction of the prosthesis, but the proper diagnosis that precedes the placement and the ability to manage the case if indeed there are problems which ensue. We have championed the cause of recognized, bona fide credentials for dentists performing these important procedures because we know from experience that there are many factors in the implant reconstructive process that can invite failure; only very few that promote success.

This example makes us wonder why the ADA did not utilize consultants who are properly trained in this field to assist in advising the editorial staff when such an undertaking is planned.

In conclusion, the AAID will be most happy to provide counsel for any aspect of implant dentistry that is contemplated by the ADA editorial staff in the future. We are happy that the editor has seen fit to highlight this implant deglupty in The Journal and would hope the issue being raised here can be properly handled by appropriate depiction of potentially successful implant cases done correctly.

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