

DENTAL 'EROSION' REVISITED

JOHN O. GRIPPO, D.D.S.; MARVIN SIMRING, D.D.S.

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."² Shafer, 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 para-function 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.

serves only to confuse the recognition of the etiology and to delay institution of proper treatment."³ Unfortunately, the cause for this consternation prevails to the present day.^{4,5}

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.⁶

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.⁶ "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.⁶

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.^{7,8} 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.⁹⁻¹¹ 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,¹²⁻¹⁷ 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,¹⁸⁻²³ dentistry likewise could adopt this term.

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

NON-METALLIC CORROSION

"Perry's Chemical Engineers' Handbook"²⁴ describes both metallic and non-metallic corrosion. Metallic corrosion is defined as the degradation of a metal by chemical or electro-

chemical 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 ABRASION

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).^{24,25} Piezoelectricity refers to the small electric charge that is generated when teeth are mechanically deformed.

The dynamic stresses that occur 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 flexure.²⁶⁻²⁸ 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.^{28,30} 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 most flexed zone, the cervical surface layer, be it enamel, cementum or dentin.³⁶⁻³⁸ In an *in vitro* stress corrosion study, a greater amount of tooth substance was lost in areas of tension than in the areas of compression.²⁵ (A similar effect of the back 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 intense occlusal loading.)

Many investigators hypothesize that these interocclusal forces create physical microfractures, or abfractions, at the cervical region, which in turn re-



Figure 1. Experimental teeth demonstrate the effects of stress and corrosion termed "abfraction." The tooth on the left is unstressed, while the tooth on the right shows the effects of a horizontal static load (150 pounds) resulting in stress corrosion. Both teeth were immersed for 96 hours in citric acid (pH 3.5). (Photograph courtesy of Steven Masl and Chris Nesman, Western New England College).



Figure 2. A subgingival abfractional lesion on the upper right first molar, inaccessible to the toothbrush, probably caused by the flexural effect of cyclic occlusal loading.

mesial 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

posed 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. Intraoral chemical influences and toothbrush/dentifrice 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.²⁵⁻²⁶ The brittleness of tooth structure (especially the extremely thin layer of enamel near the cervical line) is an additional factor in determining the abfractional response to loading forces. Based on many decades of observation, the authors, as well as other investigators, believe that many factors combine to produce regressive cervical lesions.^{11,17,19,24}

Further evidence against the solely chemical explanation for some of these so-called "erosive" lesions comes from a study conducted by Rost and Brodie,²⁷ who reported similar degradation on plastic teeth and denture base materials. Sognnaes, Wolcott and Xhonga¹ 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 wasting is greater on the enamel than on the restorative material, although the restoration has a higher wear rate.²⁸ Furthermore, these lesions may develop subgingivally, at sites inaccessible to toothbrush abrasion (Figure 2).^{4,19,20}

sult in cervical lesions.²⁹⁻³¹

It seems logical and appropriate to adopt a terminology that would most accurately describe the etiology of such lesions, namely, "stress corrosion," which also can result in "stress corrosion cracking."¹¹

Based on this reasoning, Grippo suggested that lesions that result from the micro-cracking of tooth substance (and the process of physical or physicochemical wasting) be referred to as "abfraction," meaning "breaking away," since this term describes so graphically the process and the resulting effect.³⁰

ROLE OF FUNCTION AND PARAFUNCTION

Sognnaes²⁹ cites the work of Bird³¹ and Kornfeld,³² 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

by Sognnaes,²⁹ 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.³³ In addition, Kristen and Rothe found that 97 percent of patients who had wedge-shaped cervical lesions also had parafunctional disorders.³⁴ Furthermore, Graehn, Berndt and Staeger studied a random group of 915 patients and concluded that "a causal relation appears to exist between wedge-shaped defects, occlusal disharmony, parafunctions and strong psychological tension of patients."³⁵

Stress resulting from occlusal force had been recognized as an factor in the development of cervical lesions more than half a century ago.^{31,32} Nevertheless, no one has published research on the role that occlusal therapy (for example, orthodontics, supragingival occlusal splints, coronoplasty, occlusal adjustment and restoration) might play in the prevention or arrest of such lesions. Hopefully, adoption of the more accurate terminology pro-

The concept of abfraction 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, silicate and composite. Also, after treating cervical lesions with a variety of Class V restorations, researchers found that the rate of progress of the destruction decreased from an average of 7 microns to 2 microns a week.³³

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 abfraction.
- The restoration and its adjacent enamel could have been more resistant to abrasion and corrosion than the exposed dentin at the surface of an unrepaired abfraction; thus, restoration of cervical lesions provided both protective and retardant values.
- The restoration, while subject to abrasion and flexural abfraction, may not have been subjected to significant intraoral stress corrosion (chemical effects), whereas the tooth was. Thus, the

wasting 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 pro-

pose 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

those of the Odontoceti, or sperm whales. Their teeth appear to wear away from mechanical erosion as a result of the huge volume of water flowing over them.³⁴

In Sognnaes' 1963 presentation on "Dental Hard Tissue Destruction with Special Refer-

ence to Idiopathic Erosion," the role of function was casually mentioned but not discussed.³⁵ 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-abfraction;
- biocorrosion-abfraction (caries plus flexural stress).

Although controlled clinical studies have not yet conclusive-



Figure 3. Frequently seen combined lesions termed "abrasion-abfraction" result from stress and corrosion and are further enhanced by toothbrush abrasion.



Figure 4. A stone cast reveals the unusual patterns of abrasion-abfraction as observed in Figure 3. Note the three-dimensional nature of these multifactorial lesions.

that the amounts 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

ly proven 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.^{36,40} The wasting of teeth as seen in bulimia, termed "perimyololysis," is another example of the combined activities of erosion and corrosion occurring during the flow of regurgitated acidic stomach contents over the teeth.^{7,41} Schachtele has reported that any food substance with a critical pH value of less than 5.5 can demineralize (corrode) the hard tissues of teeth.⁴² 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 teeth; however, the major activity is one of corrosion or chemical dissolution. The dynamic movement of the corrosive material by the contaminated air mechanically imparts the effects of erosion.⁷

Abrasion-corrosion.

Should a tooth surface be demineralized by some exogenous or endogenous acidic agent, then the frictional effects of the toothbrush would easily brush the surface away. Such "abrasion-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 "abrasion-corrosion." Such frequently seen combined lesions have formerly been erroneously referred to as "erosion-abrasion."⁴³ Bodecker suggests the gingival crevicular fluid as the source of the acid and provides convincing evidence to support this concept.⁴⁴

Abrasion-abfraction. The pathologic loss of tooth substance caused by frictional effects on an area of abfraction (that is, a surface that has been weakened by loading forces) can be termed "abrasion-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 abfractioning.^{45,47} These lesions would be a



Figure 5. Abrasion-abfractions on the premolars are smooth and caries-free where they have been polished by the toothbrush/dentifrice and/or the food bolus. Caries (biocorrosion-abfraction) has developed on the molar due to its mesial tilt. The tilt created a niche where plaque could accumulate. An attrition facet can be noted on the disto-occlusal surface of the molar.

result of the combined effects of both abrasion and abfraction.

Biocorrosion-abfraction.

The combined effect of biocorrosion (caries) and abfraction is the pathologic loss of tooth substance associated with caries (biocorrosion) 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.⁴⁸ Furthermore, as the depth of the lesion enlarges through degradation, the degree of stress concentration and thus of biocorrosion-abfraction increases. This may well explain the rapid progression of radicular caries.

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



Dr. Grippio is in private practice and is senior lecturer, Bio-engineering Program, School of Engineering, Western New England College, Springfield, Mass. Address reprint requests to Dr. Grippio, 123 Dwight Rd., Langmeadow, Mass. 01106.



Dr. Simring is retired; he was formerly clinical professor and director, Postdoctoral Clinic, Department of Periodontics, New York University College of Dentistry, New York; and associate professor, Department of Periodontology, University of Florida, Gainesville.

surface above the general occlusal plane of the dentition. This discrepancy in occlusal pattern may well have produced functional interference, 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 induced marked stress corrosion at the mesiobuccal 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 mesiobuccal confirm the findings of Kornfeld regarding the relationship of occlusal attrition facets and cervical lesions.³²

SUMMARY

Due to the confusion that exists about the application of the term "erosion," 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 "combined 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 or implied are strictly those of the authors and do not necessarily reflect the opinions or official policies of the American Dental Association or its subsidiaries.

The authors thank all of the following for their counsel: Dr. Nicholas A. DiSalvo, professor emeritus, chairman, Department of Orthodontics, School of Dental and Oral Surgery, Columbia University, New York; Dr. John E. Fitzgerald, assistant professor, Department of Periodontology, University of Connecticut Health Center, Farmington, Conn.; Dr. John J. Lucca, professor emeritus, chairman, Department of Prosthodontics, School of Dental and Oral Surgery, Columbia University, New York; James V. Masi, Ph.D., professor, Material Sciences, and chairman, Bioengineering, Western New England College, Springfield, Mass.; John E. Ritter, Ph.D., professor, Mechanical Engineering, University of Massachusetts, Amherst, Mass.

1. Black GV. Vol. 1. A work on operative dentistry. Pathology of the hard tissues of the teeth. 1st ed. Chicago: Medico-Dental; 1908:39-59.
2. Black GV. Vol. 1. Operative dentistry. Pathology of the hard tissues of the teeth—oral diagnosis. Revised by Robert E. Blackwell, 9th ed. South Milwaukee, Wis.: Medico-Dental; 1955:166-70.
3. Shafer WG, Hine MK, Levy BM. A Textbook of oral pathology. 4th ed. Philadelphia: Saunders; 1983:318-23.
4. Sognnaes RF, Wolcott RB, Xhonga FA. Dental erosion I. Erosion-like patterns occurring in association with other dental conditions. JADA 1972;84(3):571-6.
5. Bader JD, Levitch LC, Shugars DA, Heymann HO, McClure F. How dentists classified and treated non-carious cervical lesions. JADA 1993;124(5):46-54.
6. Glossary of metallurgical terms and tables. ASM Handbook Committee, Materials Park, Ohio: American Society for Metals; 1979.
7. Findborg JJ. Pathology of the dental hard tissues. 1st ed. Philadelphia: Saunders; 1970:294-321.
8. Mohl ND, Zarb GA, Carlson GE, Rugh JD. A textbook of occlusion. 1st ed. Chicago: Quintessence; 1988:209-12.
9. Perry BH, Green D. Perry's Chemical engineers' handbook. 8th ed. New York: McGraw-Hill; 1984:Section 23;1-66.
10. Fontana MG, Greene ND. Corrosion engineering. New York: McGraw-Hill; 1978:1-14.
11. Jones RH. Stress corrosion cracking. Materials Park, Ohio: ASM International; 1992:1-40.
12. Jones DW, Sutow EJ. Stress corrosion failure of dental porcelain. Transaction 1987;86(2):40-3.
13. Chen K-J, Ko Y-C. Slow crack growth in

- silica, high alumina, alumina-chromia, and zircon brick. Am Ceram Soc Bull 1988;67(7):1,228-34.
14. Fraker AC, Eichmiller FC. Corrosion principles in dental implantology. In: Clark's clinical dentistry. Philadelphia: Lippincott; 1990;Vol. 5:1-10.
15. Johnson LB, Lawless KR. Corrosion under stress of materials composing dental amalgam. J Biomed Mater Res 1975;9:255.
16. Ritter JE. Crack propagation in ceramics. In: Engineered materials handbook: ceramics and glass. Materials Park, Ohio: ASM International; 1992;Vol. 4:694-9.
17. Anusavice KJ, Soderholm K-J, Grossman DG. Implications of amalgam and ceramic degradation in the oral environment. Mat Res Bull 1993;18(9):64-72.
18. Anderson DJ. Measurement of stress in mastication I and II. J Dent Res 1956;35(5):664-73.
19. Neumann HH, DiSalvo NA. Compression of teeth under load of chewing. J Dent Res 1957;36(2):286-90.
20. Tyldesley WR. The mechanical properties of human enamel and dentine. Br Dent J 1959;106(8):286-90.
21. Bowen RL, Rodrigues MS. Tensile strength and modulus of elasticity of tooth structure and several restorative materials. JADA 1962; 64(3):378-87.
22. Haines DJ, Berry DC, Poole DFG. Behavior of tooth enamel under load. J Dent Res 1963;42(3):883-8.
23. Lehman ML, Meyer ML. Relationship of dental caries and stress concentrations in teeth as revealed by photoelastic tests. J Dent Res 1966;45(Nov./Dec.):1,706-14.
24. Spranger H, Haim G. Zur Analyse hochfrequenter Schwingungen in der Hartsubstanz menschlicher Zahne. Stoma (Heidelberg) 1969;Vol. 22:145.
25. Grippo JD, Masi JV. The role of bio-dental engineering factors (BEF) in the etiology of root caries. J Esthet Dent 1991;39(2):71-6.
26. Korber KH. Die elastische Deformierung menschlicher Zahne. Dtsch Zahnartzl Z 1962;17:691.
27. Grosskopf G. Untersuchungen zur Entstehung der sogenannten keilförmigen Defekte am organum dentale. Med Diss Frankfurt/Main, Germany 1967.
28. Lukas D, Spranger H. Untersuchungen über die Horizontalbelastung des Zahnes bei definierten Unterkieferlateralbewegungen. Dtsch Zahnartzl Z 1973;28:250.
29. Gordon JE. The science of structure and materials. New York: Scientific American Books; 1988.
30. Lukas D, Spranger H. Experimentelle Untersuchungen über die Auswirkungen unterschiedlich gemessener Gelenkbahn und Binnwinkel auf die Horizontalbelastung des Zahnes. Dtsch Zahnartzl Z 1973;28:755-8.
31. Klahn KH, Kohler KU, Kreter F, Mutsch A. Spannungsoptische Untersuchungen zur Entstehung der sogenannten keilförmigen Defekte an Organum dentale. Dtsch Zahnartzl Z 1974;9:923-7.
32. Seloia LG, Shillingburg HT, Jr, Kerr PA. Finite element analysis of dental structures—axisymmetric and plane stress idealizations. J Biomed Mater Res 1975;9:237-54.
33. Yettram AL, Wright KWL, Pickard HM. Finite element stress analysis of the crowns of normal and restored teeth. J Dent Res 1976;55(6):1,004-11.
34. Rubin C, Krishnamurthy N, Capilouto E, Yi H. Stress analysis of the human tooth using a three-dimensional finite element

model. *J Dent Res* 1983;62(2):82-6.

35. Goel VK, Khara SC, Ralston JL, Chang KH. Stresses at the dentinoenamel junction of human teeth—A finite element investigation. *J Prosthet Dent* 1991;66(4):451-9.

36. Hood JAA. Experimental studies on tooth deformation: stress distribution in class V restorations. *N Z Dent J* 1972;68(Apr):116-31.

37. Powers JM, Craig RG, Ludema KC. Frictional behavior and surface failure of human enamel. *J Dent Res* 1972;52(6):1,327-31.

38. Thresher RW, Saito GE. The stress analysis of human teeth. *J Biomech* 1973;6:443-9.

39. Le Bau GI. The primary cause and prevention of dental caries. *Bull Union Cty Dent Soc* 1968;47(5):11-3.

40. Le Bau GI. The primary cause and prevention of dental caries. *Bull Union Cty Dent Soc* 1968;47(6):13-6.

41. Spranger H, Weber G, Kung YS. Kariesprophylaxe und Kariestherapie. Untersuchungen über die Ätiologie, Pathogenese und Therapeikonsequenzen der zervikalen Zahnschmelzverluste. *Der Hessische Zahnartz Separatum Otto-Loos-Preis der Lan-*

deszahnarztekkammer Hessen 1973;328-41.

42. Brady JM, Woody RD. Scanning microscopy of cervical erosion. *JADA* 1977;94(4):726-9.

43. McCoy G. On the longevity of teeth. *Oral Implantol* 1983; 11(2):248-67.

44. Lee WC, Eakle WS. Possible role of tensile stress in the etiology of cervical erosive lesions of teeth. *J Prosthet Dent* 1984; 52(3):374-80.

45. Heymann HO, Sturdevant JR, Bayne SC, Wilder AD, Sluder TB, Brunson WD. Examining tooth flexure effects. *JADA* 1991;122(5):41-7.

46. Braem M, Lambrechts P, Vanherle G. Stress-induced cervical lesions. *J Prosthet Dent* 1992;67(5):718-22.

47. Grippo JO. Non-carious cervical lesions: the decision to ignore or to restore. *J Esthet Dent* 1992;4(Supplement):55-64.

48. Levitch LC, Bader JD, Shugars DA, Heymann HO. Non-carious cervical lesions: a review. *J Dent* 1994;22(4):195-207.

49. Grippo JO. Abfractions: A new classification of hard tissue lesions of teeth. *J Esthet Dent* 1991;3:14-9.

50. Sognaes RF. Mechanisms of hard tissue destruction. Washington, D.C.: Am Assoc for the Advancement of Science, 1963; publication no. 75:91-153.

51. Bird CK. Erosion and abrasion of natural teeth: The remedy or correlation of these conditions. *Dent Cosmos* 1931;7:3:1,204-8.

52. Kornfeld B. Preliminary report of clinical observations of cervical erosions, a suggested analysis of the cause and the treatment for its relief. *Dent Items of Interest* 1932;54(12):905-9.

53. Xhonga FA, Wolcott RB, Sognaes RF. Dental erosion II. Clinical measurements of dental erosion progress. *JADA* 1972;84(3):577-82.

54. Kristen T, Rothe S. Keilförmige Defekte und ihre Beziehung zu oralen Parafunktionen. *Med Diplomarbeit* 1989 Berlin.

55. Graehn G, Berndt C, Staeger B. Zur Epidemiologie keilförmiger Defekte. *Dtsch Stomatol* 1991;41(Mar):210-3.

56. Bodecker CF. Local acidity: a cause of local erosion—abrasion. *Ann Dent* 1945;4(6):50-5.

57. Rost T, Brodie AG. Possible etiologic factors in dental erosion. *J Dent Res* 1961;40(2):385.

58. Klevezal GA, Kleinenberg SE. Age determination of mammals from annual layers in the teeth and bones. Academy of Sciences of the USSR, Moscow; Severtsov Institute of Animal Morphology; 1967. Translated from Russian by the Israel Program for Scientific/Translations; Jerusalem, 1969.

59. Stafne EC, Lovestadt SA. Dissolution of tooth substance by lemon juice, acid beverages and acids from some other sources. *JADA* 1947;34(9):586-92.

60. Smith BGN, Robb ND. Dental erosion in patients with chronic alcoholism. *J Dent* 1989;17(5):219-21.

61. Holst JJ, Lange F. Perimylolysis. A contribution toward the genesis of tooth wasting from non-mechanical causes. *Acta Odont Scand* 1939;1:36-48.

62. Schachtele C. Nutrition news. Dec. 1982;45(4).

63. Jørgensen KD, Matono R, Shimokobe H. Deformation of cavities and resin fillings in loaded teeth. *Scand J Dent Res* 1976;84(1):46-50.

IMPLANT DENTISTRY TRAINING PROGRAM 1995

June 1995 - April 1996

Course Director: Arun K. Garg, D.M.D.
*Associate Professor of Surgery
Director, Center for Dental Implants
University of Miami School of Medicine*

General practitioners and specialists alike, who wish to expand their practices to include implant dentistry, will find this program to be perfectly suited to their needs and to their busy professional schedules. The program will deliver state-of-the-art education required to become knowledgeable on the latest theories, surgical techniques, prosthetic techniques, and innovations. This program will allow significant one-on-one interaction between participants and faculty.

This course is designed for General Dentists, Oral Surgeons, Periodontists and Prosthodontists. This is an eleven month program with a two-day session, meeting on a Saturday and Sunday of each month. This program is scheduled to begin on June 10, 1995.

Tuition: \$7850.00

Fee includes course materials, continental breakfast, refreshment breaks, lunch prosthetic model and handbooks.

For registration information contact:
(MECC) PO Box 430376, Miami, FL 33243-0376.
Telephone 305/663-1628 FAX 305/663-1644

ered to be necessary for credit, then a method should be devised where more articles are offered; partial credit given; or a greater span of issues should be considered. This current sort of CE (like much if not most of current CE) has little practicality.

The \$10 price is right but my time is much too valuable to waste on reading articles that have no interest or practicality for me.

Whoever thought of this program should rethink it or content himself with believing that CE—any CE—is of value to the professional.

Kevin W. Toal
St. Louis

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. Grippo and Simring¹ are to be commended for their article "Dental 'Erosion' Revisited" (May), reviewing the mechanisms for the development of non-carious cervical lesions. While we agree with Drs. Grippo 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-carious cervical lesions was indeed a source of confusion.²

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-

"Allow Us To Be Direct..."



Because at Panoramic Corporation, being direct is what we do best. Here's our offer...

We manufacture and sell the PC-1000 panoramic X-ray machine "directly-to-the-doctor" from our Fort Wayne, Indiana plant. Our direct sales method allows you to purchase the PC-1000 for thousands of dollars less than competitive machines offered through dealers. That's why the PC-1000 is the #1 selling panoramic X-ray machine in the United States.

Our direct price includes:

- All shipping expenses;
- Installation by trained technicians;
- Introductory staff training at installation;
- Two year parts and labor warranty —
the best standard warranty in the industry;



The PC-1000 is also available as a panoramic/cephalometric combination. Both machines are high quality, user-friendly and shipped largely assembled for quick and easy installation by our trained technicians.

We invite you to join the thousands of dentists throughout the United States who have discovered the savings and diagnostic benefits of purchasing a PC-1000 direct from Panoramic Corporation.

Call us at **1-800-654-2027** for more information.



Panoramic Corporation

4321 Goshen Road • Fort Wayne, Indiana 46818 • (219) 489-2291
(800) 654-2027 • FAX (219) 489-5683



ogy is understood:

■ **Attrition**—the physiologic loss of tooth structure from tooth-to-tooth contact, as in mastication and bruxism.

■ **Abrasion**—the loss of tooth structure from repetitive frictional processes such as tooth brushing.

■ **Erosion**—the loss of tooth structure from chemicals such as dietary, gastric or environmental acids.

Stress-induced cervical lesions (SICL) (or simply, stress lesions)—the loss of tooth structure due to tensile stress damage from tooth flexure during masticatory and parafunctional (for example, bruxing) movements.¹

These terms describe the vast majority of the non-carious lesions encountered. Combination of these terms can be used for lesions of multiple causes—for example, SICL/abrasion. For lesions of uncertain etiology, the term idiopathic is still appropriate.

The definitions are based on etiology for good reason. Studies of Heymann and others^{2,3} of North Carolina and Van Meerbeek and others⁴ of the Netherlands demonstrate that retentive failure rates of restoration of stress-induced cervical lesions are dependent on the ability of the restorative material to accommodate movement without debonding during tooth flex-

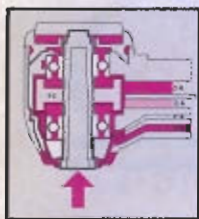
ure. Restorative material for the treatment of the other type of lesions (attrition, abrasion, erosion) do not require such flexibility. Thus, understanding the etiology, making the proper diagnosis and selecting the appropriate restorative material will have significant impact on the long-term success of the restorative treatment.

William C. Lee, D.D.S.
W. Stephan Eakle, D.D.S.
Department of
Restorative Dentistry
University of California
San Francisco

1. Grippio JO, Simring M. Dental "erosion" revisited. JADA 1995;126:619-30.

2. Bader JD, Levitch LC, Shugart DA, Heymann HO, McClure P. How dentists classified and treated non-carious cervical le-

Only NSK handpieces control patient vector cross-infection at the source...



FACT: All conventional dental handpieces draw fluids and contaminants into heads, couplings, hoses and dental units.

Infective materials may enter the water tubing beyond the handpiece only to be blown back into the mouth of a

following patient, thus nullifying the most rigorous autoclaving of the handpiece.

ANSWER: NSK invented the Clean Head System to prevent such cross-infection.

It consists of a labyrinth of air passages at the **CLEANHEAD** head of the handpiece and a duckbill valve in the water spray tube of the handpiece.

Research has confirmed the effectiveness of the NSK Clean Head System.⁵

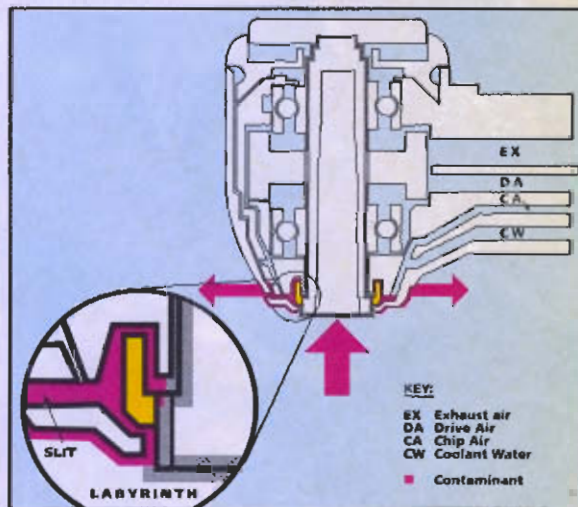
The labyrinth prevents the sucking back of materials, including viral matter, into

the turbine chamber and the duckbill prevents flowback of contaminated water from the handpiece into the water supply tube.

The NSK Clean Head System is also available on NSK low speed handpieces, attachments and clinical motors. Every NSK clinical product is autoclavable.

The NSK Clean Head System takes your professional precautions against cross-infection another step forward.

Copies of the University Studies on the NSK Clean Head System are available on request from your nearest NSK distributor.



NSK's Clean Head system limits internal contamination to the turbine labyrinth, expelling contaminated fluids immediately they enter the head. NSK's anti-water-retraction valve prevents contaminants from entering the water lines and dental unit.⁶

NSK NAKANISHI DENTAL MFG. CO., LTD.

Distributed in America by:
NSK AMERICA CORPORATION
709B Cooper Court, Schaumburg, Illinois 60173 USA
Telephone: 708-843-7664 Facsimile: 708-843-7622

sions. *JADA* 1993;124:46-54.

3. Lee WC, Eakle WS. Possible role of tensile stress in the etiology of cervical erosive lesions of teeth. *J Prosthet Dent* 1984;52:374-80.

4. Heymann HO, Sturdevant JR, Brunson WD, Wilder AD, Sluder TB, Bayne SC. Twelve-month clinical study of dentinal adhesives in class V cervical lesions. *JADA* 1988;12:53-78.

5. Heymann HO, Sturdevant JR, Bayne SC, Wilder AD, Sluder TB, Brunson WD. Tooth flexure: effects on cervical restorations: a two-year clinical study. *JADA* 1991;122:41-7.

6. Van Meerbeek B, Peumans M, Verschueren M, Clady S, Braem M, Lambrechts P, Vanherle G. Clinical status of ten dentin adhesive systems. *J Dent Res* 1994;73:1690-702.

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 bioengineering 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-carious 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 959 dentists surveyed were unfamiliar with the bioengineering 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, as Bader stated, 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 bioengineering since teeth undergo many dynamic interactions. As stated in Caputo and Standlee's "Biomechanics in Clinical Dentistry," "all dental tissues and structures follow the same laws of physics as any other material."¹ 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."² These axioms form the basis of much of the bioengineering that is involved with the tooth flexure theory which Drs. Lee and Eakle so lucidly 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,³ Kohler⁴ and Grosskopf⁵ in the '60s. All suggested that eccentric loading and flexure appear to play a role in the etiology of non-carious 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.⁶ Drs. Spranger and Haim also suggested that piezoelectricity may play a role in the genesis of cervical lesion.⁷ Spranger, Weber and Kung (1973) described the genesis of cervical lesions as being a multifactorial event.⁸ 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 ad-

vances 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 "ahfractions." 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-carious 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 outmoded classification and lead to widespread therapeutic advances.

The excellent recommendation by Drs. Lee and Eakle 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.

John O. Grippo, D.D.S.
Longmeadow, Mass.
Marvin Simring, D.D.S.
New York

1. Caputo AA, Standlee JP. Biomechanics in clinical dentistry. 1st Ed., Chicago: Quintessence; 1987:8.

2. Bayne SC, Taylor DF, Zardiackas LD. Biomaterials science. 6th Ed., Chapel Hill, NC: Brightstar; 1992:Forward.

3. Korber KH. Die elastische Deformation menschlicher Zähne. Dtsch Zahnärztl Z 1962;17:691-8.

4. Köhler E. Experimentelle Untersuchung über die Ausbreitung von Fremdstoffen in den menschlichen Zahnartgeweben. Dtsch Zahnärztl Z 1969;20:721-36.

5. Grosskopf G. Untersuchungen zur Entstehung der sogenannten keilförmigen Defekte am organum dentale. Med Diss:Frankfurt/Main 1967.

6. Lukas D, Spranger H. Experimentelle Untersuchungen über die horizontal Belastung des Zahnes bei definierten Unterkieferlateralebewegungen. Dtsch Zahnärztl Z 1973;28:280-9.

7. Spranger H, Haim G. Zur Analyse hochfrequenter Schwingungen in der Hartsubstanz Menschlicher Zähne. Stoma Heidelberg 1969;22:145-52.

8. Spranger H, Weber G, Kung Y-S. Kariesprophylaxe und Karstherapie. Untersuchungen über die Ätiologie Pathogenese und Therapi Konsequenzen der

zervikalen Zahnhartsubstanzverluste (sog. keilförmige Defekte) Der Hessische Zahnarzt Separatum Otto-Laos-Pries. 1973;328-41.

DENTAL 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 forces 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 sup-

port 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 doctors 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 counseling 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 implant dentistry 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.

Donald H. Masters, D.D.S.
President
Hilt Tatum Jr., D.D.S.
Immediate Past-President
American Academy of
Implant Dentistry
Chicago