Abfraction, Abrasion, Biocorrosion, and the Enigma of Noncarious Cervical Lesions: A 20-Year Perspective

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ABSTRACT

Hitherto, noncarious cervical lesions (NCCLs) of teeth have been generally ascribed to either toothbrush—dentifrice abrasion or acid "erosion."
The last two decades have provided a plethora of new studies concerning such lesions. The most significant studies are reviewed and integrated into a practical approach to the understanding and designation of these lesions. A paradigm shift is suggested regarding use of the term "biocorrosion" to supplant "erosion" as it continues to be misused in the United States and many other countries of the world. Biocorrosion embraces the chemical, biochemical, and electrochemical degradation of tooth substance caused by endogenous and exogenous acids, proteolytic agents, as well as the piezoelectric effects only on dentin. Abfraction, representing the microstructural loss of tooth substance in areas of stress concentration, should not be used to designate all NCCLs because these lesions are commonly multifactorial in origin. Appropriate designation of a particular NCCL depends upon the interplay of the specific combination of three major mechanisms: stress, friction, and biocorrosion, unique to that individual case. Modifying factors, such as saliva, tongue action, and tooth form, composition, microstructure, mobility, and positional prominence are elucidated.

CLINICAL SIGNIFICANCE

By performing a comprehensive medical and dental history, using precise terms and concepts, and utilizing the Revised Schema of Pathodynamic Mechanisms, the dentist may successfully identify and treat the etiology of root surface lesions. Preventive measures may be instituted if the causative factors are detected and their modifying factors are considered.

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INTRODUCTION

Since the dawn of modern dentistry, the etiology of noncarious cervical lesions (NCCLs) has been ascribed by some dentists to toothbrush/dentifrice abrasion alone.[1–20] Others have asserted that these lesions are mainly caused by acids and termed "erosion,"[21–24] more appropriately termed "biocorrosion," which embraces all forms of chemical, biochemical, and electrochemical degradation. Following the introduction of the term abfraction by Grippo in 1991 and amended in 2004, to represent the microfracture of tooth substance in areas of stress concentration, the term remains misconstrued and misused.[25–27] Published studies have demonstrated the effects of stress combined with acids[28–31] and enzymatic proteases as being factors in the genesis of NCCLs.[32–34] Piezoelectric effects on dentin have also been reported.[35–40] Studies also suggest that stress may be a cofactor in the etiology of caries, especially of cervical or root caries.[26,27,40] Unfortunately, the term abfraction has become a "buzzword," implying a single etiology, and is frequently used erroneously to designate

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Schema of Pathodynamic Mechanisms of Tooth Surface Lesions

1. Endogenous
  a. Parafunction
  b. Occlusion
  c. Deglutition
2. Exogenous
  a. Mastication
  b. Habits
  c. Occupations
  d. Dental appliances
3. Types of stress
  a. Static
  b. Fatigue (cyclic)

STRESS (abfraction)

FRICTION (wear)

COMBINED

MULTI-FACTORIAL

COMBINED

COMBINED

1. Endogenous (acid)
  a. Plaque (caries)
  b. Gingival crevicular fluid
  c. Gastric H Cl
2. Exogenous (acid)
  a. Diet
  b. Occupations
  c. Miscellaneous
3. Proteolysis
  a. Enzymatic lysis (caries)
  b. Proteases (pepsin and trypsin)
  c. Crevicular fluid
4. Electrochemical (piezoelectric effect on dentin)

BIOCORROSION (chemical, biochemical and electrochemical degradation)

1. Endogenous (Attrition)
  a. Parafunction
  b. Deglutition
2. Endgenous (Abrasion)
  a. Mastication
  b. Action of the tongue
3. Exogenous (Abrasion)
  a. Dental hygiene
  b. Habits
  c. Occupations
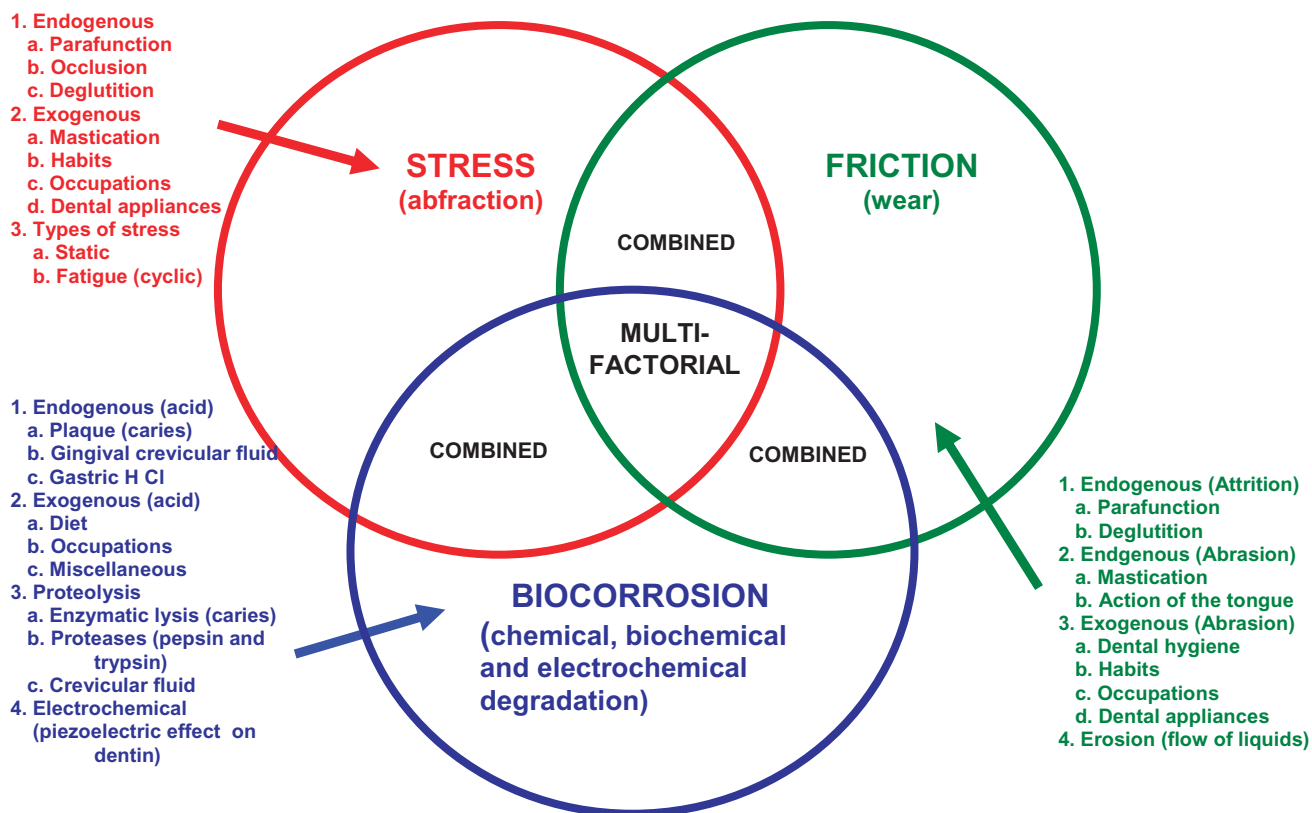 **d. Dental appliances**
4. Erosion (flow of liquids)

FIGURE 1. Revised schema of pathodynamic mechanisms. This schema indicates the initiating and perpetuating etiologic factors that produce tooth surface lesions.

all NCCLs. Because of the complex interaction of these various mechanisms—corrosion (causing chemical degradation), stress (manifested by abfraction), and friction (from toothbrush/dentifrice abrasion)—it is generally incorrect to designate all NCCLs as being caused by only one mechanism (Figure 1, Table 1). The clinician should consider all etiologic and modifying factors before completing the diagnosis or initiating treatment if indicated.

Stress concentration resulting from occlusal loading forces can occur at various locations in teeth during interocclusal contact.[41] The modes of force application that apply to dentistry are compression, tension, flexion, and shear. Occlusal loading forces resulting in stress, especially during parafunction, causes fatigue (subsurface damage) of the tooth substance and occurs immediately below the zone of contact; but in the case of NCCLs it is distant (Lawrence H. Mair, University of

TABLE 1. Etiology of tooth surface lesions

Pathodynamic mechamisms	Etiologic factors
Stress (abfraction) (see red circle in **Figure 1**)	
Endogenous	Parafunction: bruxism, clenching
	Occlusion: premature contacts or eccentric loading
	Deglutition
Exogenous	Mastication of hard and resistant foods
	Habits: biting objects such as pencils, pipe stems, and fingernails
	Occupations: holding nails with teeth, playing wind instruments
	Dental appliances: orthodontic appliances, partial denture clasps and rests
Biocorrosion (see blue circle in **Figure 1**)	

TABLE 1. Continued

Pathodynamic mechamisms	Etiologic factors
Endogenous (acid)	Plaque: acidogenic bacteria
	Gingival crevicular fluid
	Gastric juice in patients with GERD, bulimia
Exogenous (acid)	Consumption of acidic beverages, citrus fruits and juices
	Occupational exposures to acidic industrial gases and other environmental factors
Proteolysis	Enzymatic lysis (caries)
	Proteases (pepsin and trypsin)
	Crevicular fluid
Electrochemical	Piezoelectric effect on dentin
Friction (see green circle in **Figure 1**)	
Endogenous (attrition)	Parafunction: bruxism, clenching
	Deglutition
Exogenous (abrasion)	Mastication of coarse foods
	Dental hygiene: overzealous brushing, misuse of dental floss, toothpicks and interdental cleaners
	Habits: fingernail biting, opening bobby pins, biting pipe stems
	Occupational behaviors: severing thread with teeth, blowing glass, playing wind instruments
	Dental appliances: orthodontic appliances, partial denture, clasps and rests
	Ritual behaviors: mutilation of teeth
Erosion	Flow of liquids

TABLE 2. Modifying factors in the etiology of tooth surface lesions

Saliva
1. Buffering capacity
2. Composition
3. Flow rate
4. pH
5. Viscosity
Teeth
1. Composition
2. Form
3. Structure
4. Mobility
5. Remineralization
6. Dental arch form
Positional prominence or deficiency
1. Facial
2. Lingual
3. Occlusal
Noxious habits
Diet
1. Composition
2. Frequency
3. Acid beverages
Medical and general health issues
Modes of application of force
1. Magnitude
2. Direction
3. Frequency
4. Site
5. Duration

Central Lancashire, personal communication, 2007). Resultant stresses within the teeth are dependent upon the magnitude, direction, frequency, site of application, and duration of force[40] in addition to its orientation with respect to the principal axes of the teeth, as well as the form, composition, and stability of the teeth[42,43] (Table 2). Considering these factors, stress concentration can act synergistically as a cofactor with either microbial or nonmicrobial corrodents, as well as abrasives, to induce carious and/or noncarious lesions.

Tribology, according Mair "investigates the relationship between lubrication, friction and wear. In tribology, the

fundamental wear processes are: abrasive (rubbing) wear, adhesive (pulling) wear, wear due to fatigue (subcritical cracking), fretting (dragging) wear, erosive (liquid flow) wear and corrosive (dissolution) wear."[44,45] If one of the surfaces is a liquid or a gas then the process is termed erosion.[46] "Friction is the microdeformation of the surface atoms as they absorb the kinetic energy of movement. As the molecules spring back to their original position they release the newly stored energy as heat. Hopefully, this heat is removed by the lubricant that is the third factor in the tribos of tribology—lubrication, friction, and wear. If the heat is not removed then failure occurs, resulting in tooth wear or fracture" (Lawrence H. Mair, University of Central Lancashire, personal communication, 2007). The aforementioned statements explain the role of abrasion as a cofactor in the etiology of NCCLs.

BIOCORROSION VERSUS EROSION

Current dental literature in many countries frequently states that "erosion" is the loss of enamel and dentin caused by the action of acids unrelated to bacterial action. This definition of "erosion" fails to recognize, or account for proteolysis and piezoelectric effects which respectively are also involved in the biochemical and the electrochemical degradation of tooth substance. The authors contend that, "Biocorrosion which is the chemical, biochemical or electrochemical action which causes the molecular degradation of the essential properties in a living tissue" is a more precise term than erosion. Biocorrosion to teeth can occur by means of chemical exogenous and biochemical endogenous acids,[21–24,26,27] by biochemical proteolytic enzymes,[32–34] and also piezoelectric effects[35–40] acting upon the organic matrix of dentin, composed mainly of collagen (**Figure 1**). Consequently, the all-encompassing term biocorrosion should supplant the use of the term "erosion." Erosion is not a chemical mechanism; however, a physical mechanism causing wear by friction from the movement of liquids.

As reported by Lussi, enamel is 85% inorganic,[47] composed mainly of hydroxyapatite, and is readily disintegrated by acid. Dentin being 33% organic[47] is readily degraded by proteolytic enzymes.[32–34] Sources of

these proteolytic enzymes (proteases) can be produced by plaque microorganisms, and come from the gingival crevicular fluid.[33,34] While acid alone can demineralize the dentinal surface layer, the dentin organic matrix is not water soluble. Thus, the demineralized surface area can act as a diffusion barrier to limit the progression of demineralization and hard tissue loss.[33,34]

In an in vitro study, Schlueter and colleagues have shown that proteolytic enzymes from the stomach (pepsin) and pancreas (trypsin) can degrade the demineralized dentinal organic matrix.[32] The action of both enzymes was significantly greater than either enzyme alone. These proteolytic enzymes may enter the mouth during such conditions as gastroesophageal reflux disease (GERD), habitual regurgitation, or bulimia nervosa characterized by self-induced vomiting.

It has been found that in hiatal hernias, which are common in people over age 50, the esophagus tends to become shorter; thus, bringing the stomach up into the thorax. This increases the likelihood that gastric juice, digestive enzymes from the pancreas, and bile proteases may enter the mouth as in GERD (William F. Erber, Gastroenterologist, Brooklyn, NY, personal communication, 2010). With an increasingly aging American population we may anticipate an epidemiologic rise in the prevalence of dental biocorrosion.

Caries or microbial biocorrosion occurs when microorganisms grow as biofilms of plaque, usually chemoautotrophs, which act on teeth by acidogenesis, as in the formation of caries.[48,49] "Dental caries is initiated by acid decalcification of hydroxyapatite, the inorganic component of enamel. This is followed by enzymatic degradation of the relatively small amount of enamel protein (proteolysis.) Next, cariogenic bacteria invade the tooth and continue to undermine and destroy the enamel and dentin, which results in cavitation."[50] Thus, these mechanisms of acidogenesis (chemical action) and proteolysis (biochemical action) may appropriately be termed acts of "biocorrosion," or simply caries as is commonly used.[27,51]

Studies have shown that the electrochemical action of the piezoelectric effects on dentin does occur.[35–39] Surprisingly,

enamel does not have piezoelectric properties. It has been demonstrated in a study that these effects are capable of removing calcium ions from teeth.[38,40]

COMBINED MECHANISMS

Notwithstanding the paucity of scientific studies regarding static stress biocorrosion and fatigue (cyclic) stress biocorrosion in teeth,[28–31] clinical manifestations of NCCLs strongly suggest that these combined mechanisms (**Figures 2**) do occur. Static stress-biocorrosion results when a corrodent is present on the surface of teeth, which are subjected to sustained loading forces, as in prolonged clenching, deglutition, or during active orthodontic treatment.[27] Cyclic (fatigue) stress-biocorrosion results when, in the presence of a corrodent, an intermittent load is applied, as in bruxing, parafunctional occlusal tapping, clenching, or mastication. The principles of thermodynamics indicate that chemical and biochemical activity (biocorrosion) is accelerated in the presence of stress.

FIGURE 3. Advanced Biocorrosion/*ab*fractions in #5, 6, 11–14, 27, 28, and 29 caused by both static stress and fatigue (cyclic) stress, and the consumption of a highly acidic beverage called bissap (sorrel) in a patient with Hansen's disease (leprosy). Photograph courtesy of Babacar Faye, DDS, Asst. Professor of Operative Dentistry, School of Odontology and Medicine, University of Dakar, Dakar, Senegal, Africa. These patients do not brush their teeth and in most instances do not have fingers to hold a toothbrush.

FIGURE 2. An admitted intense clencher with Sjogren's syndrome who used citric acid lozenges to stimulate salivary flow. The occlusal attrition, which has worn away the buccal cusp of the second premolar, suggests bruxism, along an eccentric path, as a significant source of the resultant stress from occlusal loading force. The varied geometry, entirely within the enamel, of these combined cervical lesions was caused mainly by acid in addition to static and fatigue stress. However, they are termed biocorrosive/abrasive/abfractions since toothbrush/dentifrice abrasion plays a minor codestructive role in their genesis.

ADDITIONAL COFACTORS

Abrasion/biocorrosion takes place whenever the surfaces of teeth are covered with an acidic or proteolytic corrodent and then are abraded by friction. This would occur when teeth are brushed with a dentifrice immediately after drinking something acidic, or after regurgitating. Simply stated, the biocorrosion that occurs at a microstructural level acts upon the tooth surfaces which are then abraded by the toothbrush/dentifrice.[3–20]

It would also follow that if bacterial plaque were present, producing acid and proteases, it would then act upon the tooth surface, especially the cervical dentin. This biofilm can be removed by the abrasion (friction) from the toothbrush and erosion (flow) by rinsing as when using a mouth rinse. Both of these actions would eliminate the microstructural loss of softened tooth substance, thus making the dentin of the cervical area a NCCL as a result of abrasion and erosion (flow) acting as cofactors.

The erosive effect of the flow of water on teeth, which involves movement, is insignificant. However, when the erosive effect of an acid occurs, as when a person swishes with a carbonated drink or during vomiting, erosion/biocorrosion would result in the loss of tooth substance.[19–21] It has been reported that erosion/biocorrosion can also occur by the frequent use of acidic mouth rinses.[52]

MULTIFACTORIAL ORIGIN OF NONCARIOUS CERVICAL LESIONS

Prior to the introduction of the term abfraction by Grippo in 1991,[25] numerous papers were published on tooth deformation, stress distribution in teeth, as well as loss of tooth substance resulting from loading forces.[53–66] Following the publication of the hypothesis, by McCoy [64] as well as Lee and Eakle,[65] that "tensile" stresses were responsible for the loss of enamel in the cervical region, many have focused their attention on this specific type of stress. Photoelastic techniques and finite element analysis (FEA) have verified that the cervical region is the zone of maximum stress concentration.[33,60–63]

Lucas and Spranger in 1973 published investigations of the horizontal loading of teeth during lateral movements of the mandible.[58] They demonstrated both torsion and translation taking place in the cervical region of teeth. In the same year, Spranger and colleagues described the genesis of cervical lesions as a multifactorial event involving stress, biocorrosion, and friction.[59]

In 1985, Ott and Proschel reported the in vitro development of surface lesions of the teeth, which they interpreted as early wedge-shaped defects.[66] Their study correlated the defects with the occurrence of occlusal dysfunction.

Shortly thereafter Grippo and Masi verified flexion by using a strain gauge on a tooth mounted in a loading frame.[40] They also reported the first studies of stress

biocorrosion in teeth, wherein accelerated biocorrosion rates of enamel loss occurred when teeth were subjected to a static load in an acid environment.[26,40] Unfortunately, the loss of tooth substance in dentin was not quantified at that time. They also reported piezoelectric effects in teeth that were loaded both statically and cyclically.[38,40] These piezoelectric effects (in excess of 10^{-14} coulombs/Newton) were sufficient to transport calcium ions, thus serving as a cofactor in the demineralization of teeth. Further study is indicated in order to verify these seminal observations.

Since the early 1990s, numerous publications have emerged with conflicting views on the genesis of NCCLs.[67–94] Most of the contention centered on the significance of occlusion, the biomechanics of occlusal force and its resultant stress and strain. More recent FEA studies have supported the significance of stress in the cervical region as the zone of maximum stress concentration.[72–76] Hopefully, ongoing research, using technological advances, will definitively resolve this dilemma.

Palamara and colleagues, used 1% lactic acid (pH 4.5) to simulate the conditions of dental plaque under a repeated load.[28] Their study demonstrated that when cyclic loading was combined with immersion in this acid, tensile stress effects were observed on enamel in the cervical region. These results are consistent with clinical observations and support the concept of static stress-biocorrosion and fatigue (cyclic) stress-biocorrosion as cofactors in the formation of NCCLs. These mechanisms may also cause lesions entirely within the enamel, with varying locations and geometry as in Figure 2.

In 2005, Staninec and colleagues were the first to report a series of in vitro fatigue-cycling experiments on human dentin cantilever beams in two different environments.[29] They revealed that both mechanical stress and lower pH values accelerated material loss of the dentin surfaces. Their results demonstrated the mechanism of cyclic physical fatigue failure (stress) combined with biocorrosion.

More recently Mishra and colleagues concluded, in a study of a beam of bovine dentin, that stress from static loading combined with a low pH is associated with increased subsurface demineralization at the fixed end of the beam.[30] In a similar study, they concluded that combined stress and lower pH increase surface loss at the fixed end of the beam, which in a tooth represents the cervical region, the site of stress concentration.[31]

Noma and colleagues showed that cementum cracks initiated in the cervix, after repeated compressive loadings, extended toward the root apex. They concluded that the effects of stress from occlusal cyclic loading could induce fatigue fracture on the root surface.[77] Their findings support the contention that NCCLs may begin when molecular bonds are broken and microfracture (abfraction) occurs in areas of stress concentration.[27]

Occlusal force and its resultant stress come into play during the dynamics of interocclusal activity whenever teeth and restorations fracture. Furthermore, stress and tooth flexure can also cause composite and amalgam restorations in the cervical area to avulse after repeated loading.[95–98] In addition, it appears that occlusal stress affects the surface of materials such as gold foil by changes in contour following excessive and repeated loading (Figure 4A), analogous to the process of abfraction.[99] As stated by Caputo and Standlee, "All dental tissues and structures follow the same laws of physics as any other material and structure."[100]

Static stress-biocorrosion and fatigue (cyclic) stress-biocorrosion occur most frequently in the cervical region and appear as NCCLs if these areas are kept free of plaque (Figures 2, and 4A–5B). In contradistinction, root caries (bacterial biocorrosion) occur in these same areas if oral hygiene is neglected, because a correlation exists in the etiology of these two lesions.[27]
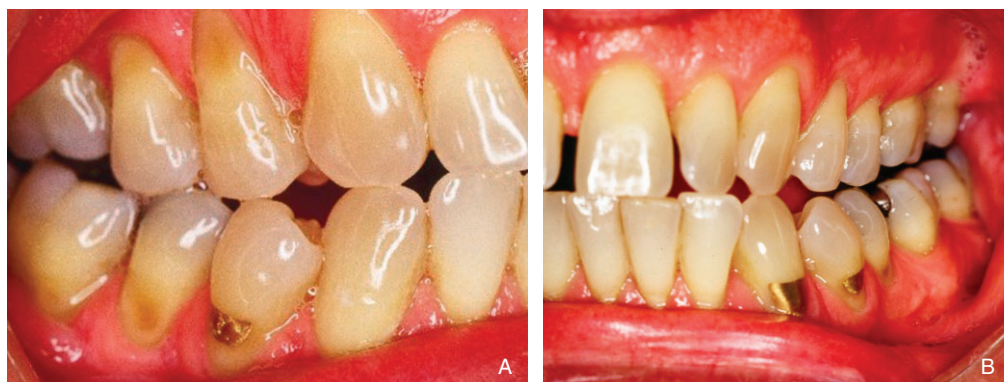

FIGURE 4. A, Multifactorial noncarious cervical lesions in both upper and lower premolars indicate the effects of fatigue (cyclic) stress, biocorrosion, and friction from the toothbrush/dentifrice. Excessive loading on the premolars due to the lack of anterior guidance (cuspid) appears to have also affected the gold foil whose surface has changed. B, Patient depicted in Figure 3A in left lateral excursion. Gold foils were all placed by the same operator who was a Professor of Operative Dentistry. Posterior teeth are being discluded by cuspid rise, thus minimizing the effects of stress. The foils are not affected by stress (abfraction), toothbrush/dentifrice (abrasion), nor (biocorrosion) from acids.
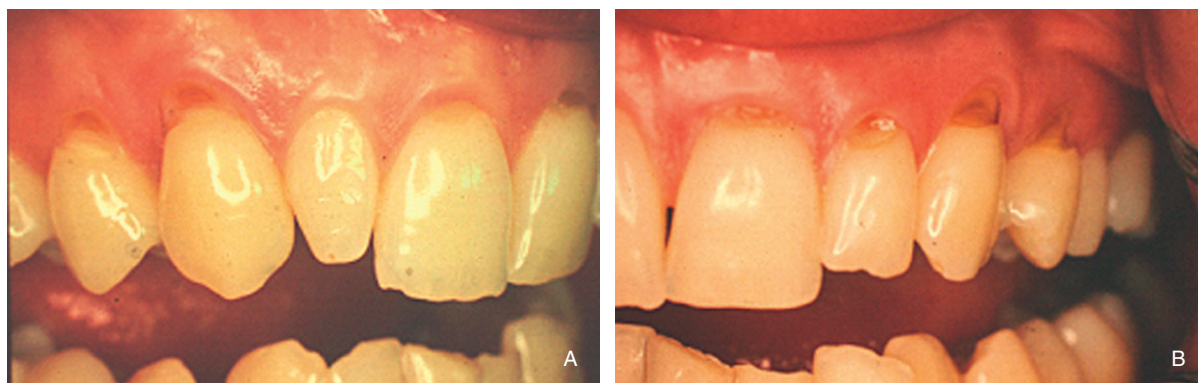
FIGURE 5. A, Multifactorial noncarious cervical lesions on #5, 6, and 8 designated as biocorrosive/abrasive/abfractions caused by the mechanisms of stress (abfraction), biocorrosion (acid and proteolysis), and friction (toothbrush/dentifrice abrasion). Tooth #7 is unscathed since it is subjected to less fatigue (cyclic) stress and because it is out of the occlusal plane of the adjacent teeth that absorb the occlusal load. B, Note that tooth #10, in contradistinction to #7, has a wear facet on the mesial incisal edge, indicating increased loading with resultant stress concentration in the cervical area. The lack of saliva in the vestibule of these areas (#5, 6, 8–12) in addition to acidic as well as proteases regurgitating from this suspected bulimic, would permit fatigue (cyclic) biocorrosion to occur, thus causing the rapid destruction of cervical tooth structure.

It has been estimated that during mastication and deglutition, teeth cycle or make contact approximately one million times per year.[101,102] Shore stated that teeth contact 1,500 times daily when swallowing.[103] According to Gibbs and colleagues, the jaw remains closed during swallowing for an average of 683 milliseconds, which is three times longer than the 194 milliseconds of occlusal contact during chewing.[104] Their study also disclosed that when using an average biting force of 66.5 pounds it persisted for an average of 522 milliseconds of the total 683 milliseconds in the closed inter-cuspal position. Furthermore, their study found that this average swallowing force of 66.5 pounds (296 N) is greater than the chewing force of 58.7 pounds (261 N). If a premature contact occurs on a tooth then the stress induced by cyclic loading, over time, can cause the tooth substance to degrade. This degradation results when stress works in concert with a corrodent whether an acid or a protease.

Occlusal stress must be considered on a molecular level in order to appreciate the effects that occur. It is understood that stress can act synergistically with a corrodent to cause either static stress biocorrosion or fatigue (cyclic) stress biocorrosion on tooth substance.[26–31,40] Bonds between molecules can be broken individually by the mechanisms of stress,

friction, or biocorrosion or by any combination of these factors acting together in the destruction of susceptible materials, including teeth. The dynamics of occlusal contact are very complex, as are the following modifying factors: salivary buffering capacity, composition, flow rate, pH, and viscosity, as well as tooth composition, form, structure, mobility, positional prominence, and dental arch form, in addition to tongue action, noxious habits, medical and general health issues, remineralization of both enamel and dentin, dietary intake, composition and frequency of food and beverage consumption (Table 2). Thus, it often becomes a daunting and frequently futile task to ascribe a single mechanism as the primary or sole cause of NCCLs. This concept was organized and presented in the Schema of Pathodynamic Mechanisms of Tooth Surface Lesions developed by Grippo, Simring, and Schreiner (JADA).[27] The present authors Grippo, Simring, and Coleman have updated and revised "The Schema" in light of new developments (Figure 1, Table 1 and 2).

MODIFYING FACTORS

In addition to the varying composition of teeth, the clinician should also consider their form and

structure.[105,106] The cuspal inclines of teeth, which provide an efficient means of mastication, become stressed when a steep, nonaxial contact force occurs during tooth to tooth contact. If these contacts are premature and eccentric, the stress to the cervical region intensifies, with resultant greater stress concentration in that area.

When occlusal surfaces are worn flat, occlusal forces are dissipated fairly evenly over the opposing surfaces and directed axially, thus decreasing flexure and stress concentration in the cervical area. Ritter and colleagues noted this to be a common finding among primitive groups and may explain why NCCLs do not generally occur in those dentitions.[107]

Young and Khan stated that there is little evidence that strains in lingual enamel and dentin would be any different from those that occur at the buccal sites during function.[108] However, the architectural arch form counteracts inward forces, that could collapse the arch, by distributing the forces among all the components of the arch. Analogously, the dental arch form mitigates lingually directed forces. Thus, the dental arch inhibits lingual flexure of teeth, but readily permits facially directed forces to flex teeth and result in stress concentration at the cervices of the teeth.[108]

The cushioning effect of the periodontal ligament (PDL) is another modifying factor. It has been shown that there exists a negative correlation between tooth mobility and NCCLs.[27,42] A mobile tooth, whether it is the result of a wide PDL and/or a short root and/or a low bone level, will tilt and distribute stress to the supporting PDL and alveolar bone. A stable tooth, when stressed laterally, will flex in the cervical area and result in stress concentration in that area.

Occlusal positional prominence of the teeth is also an important factor in determining possible overstress and trauma. Where an individual tooth or tooth segment extends occlusally beyond the occlusal plane, some people are provoked to extend their mandibles into atypical paths or positions in order to contact these positionally prominent teeth. Thus they achieve intense force that these eccentric isolated contacts provide for

parafunctional activity. Close examination for unusual wear facets and wear patterns can provide clues to such sources of overload. Wear facets indicate sites of initial contact with sufficient force to wear down the enamel at that location, along mandibular excursive pathways, especially during parafunction (**Figures 2 and 4A**).[42]

Facial positional prominence is also significant because it predisposes to toothbrush/dentifrice abrasion, especially with excessive cross-brushing. Conversely, a tooth or teeth in a recessed buccal or labial bay, protected by adjacent facially prominent teeth, would be shielded from the onslaught of abrasion.[109] The development of NCCLs depends ultimately on whether or not the confluence of pathodynamic factors exceeds the odontolytic threshold for a tooth in its oral environment.

Since the 1960s, the role of stress has been cited by some as the primary cause of these enigmatic lesions.[41,53–57] Davis stated that toothbrush-dentifrice acts as a stress raiser by creating an area of stress concentration in the cervical region of teeth resulting from frictional abrasion when brushing with a dentifrice.[110] This seems to be plausible because of the prevalence of NCCLs on the facial surfaces of teeth today in contradistinction to the teeth of primitive groups who did not brush.[111] Davis's theory, however, does not fully explain the presence of lesions on teeth, with their adjoining ones unscathed, as in **Figure 5A**, or lesions extending beneath the finishing margins of crowns and the gingiva.[25–27]

The cleansing action of the tongue by friction is another factor that protects lingual surfaces against the formation of NCCLs. Lingual surfaces are also more difficult to reach, especially for cross-brushing. In addition, people are less motivated to brush the lingual surfaces because they are not seen by others. The complicating factor of biocorrosion due to the ever greater consumption of acidic beverages should be considered in the growing prevalence of NCCLs.

A unique study was conducted by Faye and colleagues on a nontoothbrushing population with Hansens's disease (leprosy). Their preliminary study demonstrated

that toothbrush/dentifrice use was not a factor in the etiology of NCCLs, which existed in 48 (47%) of the 102 Senegalese subjects. They concluded that occlusal stress and incisal stress combined with the consumption of highly acidic beverages causing biocorrosion were the etiologic mechanisms of the NCCLs. This group was selected because they had deformed hands that precluded them from using a toothbrush (**Figure 3**).[89]

A most important factor to be considered concerning the location and etiology of NCCLs is the modifying effect of the flow rate, buffering capacity, pH, viscosity, and composition of saliva. Kleinberg stated that there is five times more saliva on the lingual surfaces than in the vestibule (Israel Kleinberg, SUNY Stony Brook of NY, personal communication, 2006). That observation is also supported by Jenkins.[112] These reputable sources of information support the contention that saliva, particularly lingual serous saliva, which has a high flow rate and buffering capacity from bicarbonates, accounts for the paucity of lingual NCCLs. In contradistinction, NCCLs are most commonly found on the facial surfaces where the mucous saliva is present and lacks these buffering effects.[113,114] Xerostomia, or dry mouth syndrome, is caused by systemic disease, can be medicinally induced, or due to aging. Mouth breathing may complicate this effect by fostering evaporation of the saliva, especially in the anterior labial area.

SUMMARY

In view of the resistance to change for the past 100 years, the authors contend that it is time for a paradigm shift, utilizing updated terminology and concepts to designate the mechanisms involved in tooth surface lesions. As a consequence, this will improve communication with our related sciences, primarily in biomedical engineering. The term "biocorrosion" should be accepted to supplant the use of the term "erosion," previously referred to as chemical degradation, because both exogenous and endogenous acids, proteolysis and electrochemical action can be embraced by this more precise term. Abfraction, representing the mechanism of stress,

as the loss of tooth substance in areas of stress concentration, should not be used to designate all NCCLs because these lesions are commonly multifactorial in origin. These lesions are caused by acids, proteases, and piezoelectric effects acting on the dentin which is 33% organic in composition.

In order to achieve a more accurate differential diagnosis of the etiology of NCCLs, before designating a single mechanism, the clinician must take a comprehensive medical and dental history, perform an occlusal examination, inventory the diet, and review oral hygiene practices. The buffering capacity, composition, flow rate, pH, and viscosity of saliva as well as differences between lingual and vestibular saliva are important modifying factors in the genesis of NCCLs. A tooth's positional prominence or lack thereof, either occlusally, facially, or lingually, should be evaluated in determining the effects of these factors. By addressing the interactive synergy of the various coactive mechanisms, stress, friction, and biocorrosion, and their modifying factors, the clinician can then identify the complex etiology of these multifactorial lesions.

The use of the Revised Schema of the Pathodynamic Mechanisms of Tooth Surface Lesions (**Figure 1**) with (**Table 1**) and Modifying Factors (**Table 2**) provide a convenient and practical approach in determining the etiology and designation of NCCLs.

Further studies are suggested in order to elucidate the cofactors of static stress biocorrosion and fatigue (cyclic) stress biocorrosion, as well as the piezoelectric effects on dentin in the etiology of NCCLs.

DISCLOSURE AND ACKNOWLEDGEMENTS

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