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CERVICAL LESIONS

Origin and Development of Cervical Dentin Hypersensitivity and Noncarious Cervical Lesions: Literature Review

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Abstract: This article reports on published literature for causative factors of cervical dentin hypersensitivity and noncarious cervical lesions. The author conducted an exhaustive literary review of both conditions to examine etiologic cofactors involved for these dental findings. Previous literature found that these two conditions arise from combinations of dental stress distant from occlusal contacts, biocorrosion, and possibly friction. It is impossible to separate these three factors for the modern dentate human due to masticatory function and tooth contact when swallowing. The author concludes that in vivo study is needed to clarify the roles that etiologic factors play in the development of this type of dental pain and/or noncarious lesions. The clinical significance of this review is that a dental and medical history and active etiologic factors need to be uncovered for cervical dentin hypersensitivity with resulting noncarious cervical lesions. The successful clinician needs to determine causative factors, if possible, prior to treatment.

LEARNING OBJECTIVES

- Identify causative factors of cervical dentin hypersensitivity and noncarious cervical lesions
- Describe how combinations of dental stress distant from occlusal contacts, biocorrosion, and friction can affect these two conditions
- Explain the abfraction concept and the development of the term

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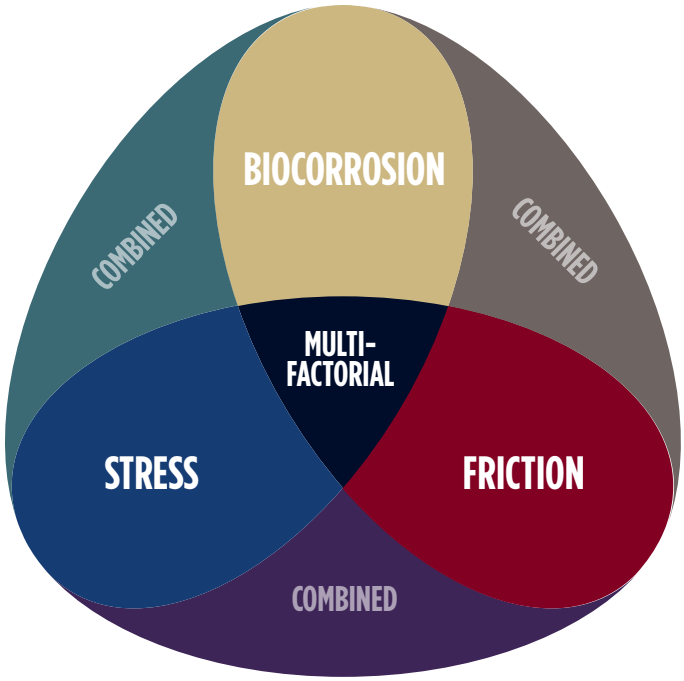
The loss of dental tissue in the cervical region of the tooth is an increasingly common finding in clinical practice, with prevalence rates of up to 85% in some populations.[1-4] Despite noncarious cervical lesions (NCCLs) affecting a majority of the populations and requiring dentists to regularly contend with this common pathology in patients, there remains disagreement over the mechanisms and factors involved in the etiology and progression of these lesions.[5-11] Moreover, concern exists regarding the durability of restorative materials and successful control and treatment of this pathology.[12-14]

Prior to the concepts of modern dentistry, which have embraced theory and clinical guidelines based on scientific evidence, the etiology of the loss of enamel and dentin located near the cementoenamel junction (CEJ) was focused solely on friction caused by excessive toothbrush/dentifrice abrasion on teeth.[15,16] Some clinicians attributed NCCLs exclusively to the degradation of tooth structure as a result of acids derived from exogenous and endogenous sources.[17,18]

Zsigmondy in 1894 first described NCCLs as "keilformige defekte" (wedge-shaped lesions),[19] and in 1932 Kornfeld referred to these lesions as "cervical erosions."[20] The term "noncarious cervical lesion" seems to have first appeared in Shore's book, *Temporomandibular Joint Dysfunction and Occlusal Equilibration*, published in 1976.[21] As for the significance of stress in the etiology of NCCLs, Korber in 1962 described and computed the elastic deformation of teeth.[22] He stated that horizontal forces applied to teeth give rise to flexion-causing tension and compression in the cervical region. Lukas and Spranger investigated horizontal loading of teeth during lateral movements of the mandible and, like Korber, found that both torsion and translation (twisting and straight-line movement) occurred at the cervical area.[23] Brady and Woody did an exhaustive electron microscopic investigation of NCCLs.[24] McCoy in 1982 and shortly thereafter Lee and Eakle were the first Americans to publish and lecture on the significance of stress occurring in the cervical area.[25,26]

In 1991, Grippo coined the term "abfraction," which designated the loss of tooth substance in areas of stress concentration promoted by dental flexure.[27] Abfraction in the formation of NCCLs is due to the stresses resulting from biomechanical loading forces exerted on teeth (static, as in deglutition and clenching, or cyclic, as in mastication or parafunction) that can cause enamel, dentin, and cementum to break away.[6,8,26,27] The use of the term "abfraction" to describe the manifestations of stress in areas of stress concentration prompted the publishing of numerous articles that created contention by refuting the role of stress in the etiology of NCCLs.[6,10,11,27]



Stress *(Abfraction)*	**Biocorrosion** *(Chemical, Biochemical, and Electrochemical Degradation)*	**Friction** *(Wear)*
Types of stress Tension Compression Shear Flexion Torsion	Endogenous (acid) Plaque (caries) Gingival crevicular fluid Gastric hydrochloric acid	**Endogenous (attrition)** Parafunction Deglutition
Motions of stress Static Fatigue (cyclic) Dental appliances	Exogenous (acid) Diet Occupations Miscellaneous	**Endogenous (abrasion)** Mastication Tongue action
Endogenous Occlusion Mastication Deglutition Parafunction Tongue action	Proteolysis Enzymatic lysis (caries) Proteases (pepsin and trypsin) Collagenases	**Exogenous (abrasion)** Dental hygiene Habits Occupations Dental appliances
Exogenous Habits Occupations Dental appliances	Electrochemical (piezoelectric effects on dentin)	**Erosion (flow of liquids)**

Fig 1. Diagram showing the multifactorial nature of stress, biocorrosion, and friction for cervical dentin hypersensitivity (CDH) and noncarious cervical lesions (NCCLs). It lists the initiating and perpetuating etiological mechanisms and agents that cause CDH and NCCLs. Mechanisms/agents from any ("combined") or all ("multifactorial") of the three columns typically overlap. (Diagram adapted with permission from Grippo JO, Coleman TA, Messina AM, Oh DS. *J Esthet Restor Dent*. 2018;30[3]:187-192.)

While the origin and development of NCCLs has been vigorously debated, it appears these anomalies are related to three distinct and fundamental etiological mechanisms, namely stress, biocorrosion, and friction (Figure 1),[10,28,29] which are defined as follows:

- Stress—manifests as abfraction caused by stress-strain concentration from pathologic occlusion and parafunction
- Biocorrosion—the chemical, biochemical, and electrochemical degradation of tooth substance caused by endogenous and exogenous acids, proteolytic agents, and piezoelectric effects on dentin
- Friction—tooth substance wear caused by toothbrush/dentifrice abrasion

Thus, this article aims to contribute to the theoretical knowledge base of the etiology of the stress-strain mechanism, explaining how this process influences the origin and development of NCCLs. It will also discuss the significance of stress acting in concert with biocorrosion and friction as cofactors in the etiology of NCCLs.

The Abfraction Concept and Development of the Term

As mentioned, abfraction is the pathologic loss of tooth tissue microstructure in areas of stress concentration caused by eccentric occlusal loading forces. A subset designation of NCCLs, these lesions occur mostly at the CEJ, wherein flexure can lead to a disruption of the extremely thin layer of enamel prisms and cause microfracture of the cementum and dentin.[6,8,9] NCCLs are related to the direction, magnitude, frequency, duration, and location of the occlusal load resulting in varying shapes of lesions in the cervical region.[8] Abfractive lesions have been said to be due to flexure and ultimate fatigue of enamel and dentin at a location away from the point of loading when supportive alveolar bone exists.[27]

American scholars have shown an interest in this pathology since the 1970s and 1980s.[24-29] According to Grippo's aforementioned 1991 publication, abfraction occurs when excessive nonaxial or eccentric occlusal forces are applied to teeth, promoting cusp flexion and resultant stress concentration in the cervical area, which causes ultimate material fatigue to susceptible teeth at locations away from the point of loading.[27] In addition to their varying shapes,[27] a common feature of many abfractive lesions is the morphology of wedge-shaped lesions that have well-defined, angled flat walls and can also occur in the subgingival region.[8,28,30-32]

The occlusal forces of 66.5 pounds (30 kg) during swallowing and 58.7 pounds (27 kg) during chewing represent averages of only 41% and 36%, respectively, of the average maximum biting strength of 162 pounds (73 kg).[33] Furthermore, the length of time in which the teeth remain in contact during intercuspation is only about 194 milliseconds during chewing and a surprisingly much higher 683 milliseconds during swallowing.[34] It is thought that magnitudes of forces during bruxism are much higher than those loads found during normal functional activity.[35] Thus, one may presume that occlusal parafunction is more prone to promote tooth substance loss in the cervical region than physiological processes.[6,28] Waugh reported Eskimos have above-average bite strength and recorded one individual having a bite strength of 348 pounds (158 kg).[36]

Dentin has varying micro- and macrostructure and can support major stress concentration more so than enamel. This assertion was a main point of criticism represented in the abfraction concept.[37] Neither Michael et al[31] nor other critical authors ever considered that abfractions commonly occur when a biocorrodent is working synergistically with effects from stress.[9,38-43]

Frictional effects from toothbrushing and dentifrice use have been proposed as contributory to NCCL development and/or maturation. The present author could not find any in vivo studies to support or refute the view that modern soft-bristle toothbrushes with low abrasive index dentifrices contribute to abfraction/NCCL development or maturation. However, in a population with Hansen's disease who did not brush their teeth, NCCLs were present.[2] This population group regularly consumed acids in their diet, which are noted in Figure 1 as an example of exogenous biocorrosion. Also, a study of a Mexican population from the late 19th century, which pre-dates toothbrushing or dentifrices for personal oral hygiene, found prominent NCCLs that were probably related in their etiologies more so to stress conditions rather than biocorrosion or friction.[44]

A subset designation of noncarious cervical lesions (NCCLs), [abfraction] lesions occur mostly at the cementoenamel junction, wherein flexure can lead to a disruption of the extremely thin layer of enamel prisms and cause microfracture of the cementum and dentin.

One could presume that with good personal dental hygiene efforts, vigorous toothbrushing would remove softened dentin or fractured enamel hydroxyapatite. It must be understood that soft-tissue damage from abrasion precedes hard-tissue loss in a given location. Effects from abrasion on exposed surfaces vary depending on the integrity/resistance of the surface, the frequency of abrasion, and the direction of force application. A more traditional series of abfractions from chronic occlusal sequelae without the influence of abrasion from toothbrushing is illustrated in Figure 2.

Abfraction advancement can be affected by altered friction occurring from toothbrush/dentifrice abrasion, acids in one's diet, reflux conditions, or stress in occlusal contact.[45] All of these incidents have an effect on teeth and in the formation of NCCLs resulting from stress and strain. However, NCCLs can be regarded as unavoidable for many, if not most, people.

Gastroesophageal reflux (GER) disease (GERD), silent GERD, and other anorexic conditions produce loss of hard tissue on lingual surfaces due to acidic or protease challenges over time. GER applies to conditions when an incidental ocassion of reflux action occurs, such as drinking too much alcohol, or an event of undiagiosed food allergy. It is of interest that Fauchaud in the 1700s did not

appreciate the differences between the chemical effects of biocorrosion and the effects of friction, which is a physical mechanism.[28,46] He was attempting as a physician to explain oral or systemic conditions using terms available at that time. Chronicity is a key factor separating a pathologic state from more normal health. Figure 3 illustrates a patient with anorexia nervosa, which is an example of chronic endogenous biocorrosion.

Cervical Dentin Hypersensitivity

It is clinically notable that the terms "dentin hypersensitivity" and "cervical dentin hypersensitivity" (CDH) do not apply to the same dental pulp pain. Dentin hypersensitivity is self-limiting over a week or two following tooth preparation, crown cementation, or restoration placement from a transient pulpitis. CDH, however, will remain active for a long period of time unless occlusal therapy is provided and/or biocorrosive effects are reduced.[47-49] CDH commonly occurs before or during the development of visible/detectable NCCLs. The "hydrodynamic theory" for open dentin tubules proposed by Brännström in the early 1960s is what led to the development of desensitizing materials that are commonly promoted in current-day dentifrices.[50] It is the present author's view that etiologic conditions must be evaluated/determined prior to treatment.

Results of the Review

Multifactorial etiologic conditions for NCCLs have been reported, primarily from occlusal stress and biocorrosive conditions. The presence of CDH, a pulp pain, must not be ignored as it could be a precursor to development of NCCLs, yet it does not present during all formations of NCCLs. Nonvital or minimized dentin tubule openings into the oral environment will produce different responses to indices (stimuli) of air, cold, tactile stimulation, electrical stimulation, acid exposure, and combinations thereof. Therefore, CDH is not always present during the process of the formation of abfractive lesions, which are a subset of NCCLs. Dental physicians must determine etiologic factors prior to treatment, in the author's opinion.

Treatment Options

Treatment options are best selected with informed consent of the patient and with the clinician following secure identification of causative conditions. Treatments may include direct composites, direct-indirect restorations, veneers, and full- or partial-coverage prostheses. Glass-ionomers may be used as a temporary measure to block the sharp pain of CDH, but their esthetic shortcomings relegate their use as a temporary measure.

Conclusions

Abfractions are a dental flexure subset of NCCLs, also resulting from biocorrosive conditions and manifested as hard-tissue loss. CDH often precedes the visual presence of NCCLs and is primarily a result of chronic occlusal loading and biocorrosion. In vivo research is indicated to determine if frictional contributions from toothbrushing and/or dentifrice abrasion play a role in the development of CDH and/or NCCLs.

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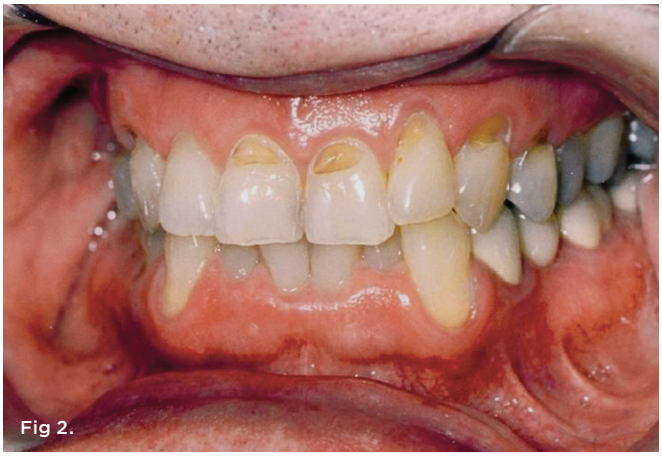

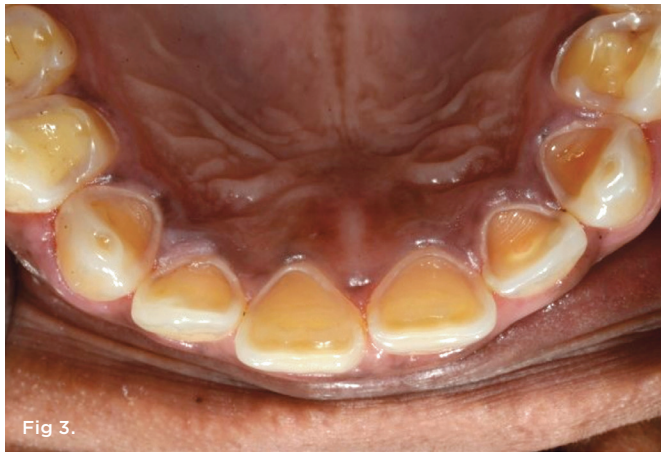

Fig 2. Photograph of a 75-year-old male patient of the author. This patient's wife had reported him bruxing at night, which was seemingly related to the round-the-clock wear of a Nesbit appliance on the lower left region of tooth No. 20 that was replaced by a fixed bridge, Nos. 19 through 21. The wife reported a week later that her husband stopped nighttime bruxing behavior within a few days. This photograph was taken at a periodontal recall visit 2 years after placement of the lower left fixed bridge. There was still no evidence of direct or indirect bruxing behavior.
Fig 3. A clinical example of severe chronic endogenous biocorrosion effects from anorexia nervosa. This patient was diagnosed and treated by a physician for this condition, which left lingual maxillary enamel almost free of this hard tissue. (Photograph is courtesy of Ali Tunkiwala, MDS, used with his permission.)

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