Chapter 9
Detecting and Quantifying Cervical Dentin Hypersensitivity Using Air Indexing Combined with the T-Scan System

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ABSTRACT

This chapter introduces the Air Indexing method for detecting and quantifying cervical dentin hypersensitivity as a companion to the T-Scan Occlusal Analysis System, which evaluates occlusal force and timing values of contacting teeth. The chapter discusses detection, diagnosis, and treatment of clinical signs and/or symptoms of Cervical Dentin Hypersensitivity (CDH). A 17-year-long retrospective study conducted between 1979 and 1996 is presented that illustrates the correlation between Cervical Dentin Hypersensitivity and its resolution following occlusal adjustment. Resulting stress from occlusal contact force is etiologic for non-carious cervical lesion formation and root degradation. This chapter details how biocorrosion and lost protective glycoproteins hasten the effects of applied force, creating CDH symptoms and cervical abfractions. Lastly, the Air Indexing method of CDH diagnosis is melded with T-Scan occlusal analysis to diagnose and treat CDH symptoms. Together, these two methods yield more CDH/occlusal insight than either method can alone.

INTRODUCTION

This chapter discusses detection, diagnosis, and treatment of the clinical signs and/or symptoms resultant from chronic dental microtrauma. The presence of Cervical Dentin Hypersensitivity (CDH) often indicates there is ongoing excessive stress being applied to the cervical regions of teeth, which occurs during both function and/or parafunction. There are two primary co-factors that initiate and promulgate the presence of CDH. They are "abfraction" and "biocorrosion", which occur simultaneously, making etiologic determination somewhat clinically complex. A physical science discussion of applied stress has been included within the chapter, to explain how

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cervical stress from occlusal microtrauma plays a role in creating non-carious cervical lesions (NCCLs) that are preceded by attaining a CDH threshold response. Other chapter sections will define Occlusal Disease (OD) as it relates to active or inactive findings of clinical pathology from microtrauma. The Air Indexing Method of CDH detection and quantification will also be described and illustrated. The last portion of the chapter discusses the combining of Air Indexing with the T-Scan III Occlusal Analysis System (Tekscan, Inc. S. Boston, MA, USA). When melded together, these two objective methods synergistically improve the clinician's ability to detect and treat active occlusal microtrauma with measured occlusal intervention, which can predictably lessen CDH symptomotology.

BACKGROUND

Occlusal stress may result from chronic *microtrauma* or *macrotrauma* (Speck et al., 1979). Chronic *microtrauma* is defined as the repetitive low force inter-arch contact of teeth over time. This type of trauma occurs during swallowing, habitual function, parafunction, or mastication (Dejak et al., 2003; Grippo, 1991; Grippo et al., 2004; Kydd, 1957; Shore, 1976; Straub, 1960). Microtrauma may exist with or without food bolus.

Microtrauma may be considered pathologic when it results in premature signs and/or symptoms of OD, by disrupting masticatory system health. The term Occlusal Disease, first appeared in the literature during 1990 (Lytle, 1990), was further modified 17 years later (Ruiz, 2007), and then redefined again, 1 year later (Ruiz & Coleman, 2008). Occlusal Disease includes clinical findings of occlusal wear, fractures of teeth or restorations, hypersensitivity of teeth during mastication, cervical dentin hypersensitivity (CDH) (Coleman et al., 2003; Coleman & Kinderknecht, 2000), tooth hypermobility (Harrel et al., 2006), fremitus (Harrel et al., 2006; Ruiz, 2007), abfractive stress (Coleman et al., 2003; Grippo, 1991; Ruiz, 2003, 2005, 2007), vertical bone loss or localized bone destruction (secondary to inflammatory and bacterial periodontal disease) (Harrel et al., 2006), and masticatory muscle or TMJ pain (Gremillion, 2006; Ruiz, 2005; Sipila et al., 2006). Originally published in Compendium.

In 1971, Ramfjord and Ash suggested that the Central Nervous System (CNS) reflex that stimulates bruxism, seems to emanate from the periodontal proprioceptors, specifically from within the Sharpey's fiber complex of the periodontal ligament (Ramfjord & Ash, 1971). This "nociceptive reflex" results from polysynaptic noxious stimuli (Okeson, 1998). It has been observed during the years when the dentition is transitional, but applies to all ages as a periodontal reflex response to the muscles of mastication resultant from dental mobility (Okeson, 1998).

Chronic microtrauma may be deemed physiologic or pathologic during bruxing episodes. Bruxism may be physiologic when it is observed among younger children (ages 7-14 years) who present with a transitional dentition, as deciduous teeth are chronologically lost. Wear patterns among children in this age group have been correlated to childhood bruxing tendencies (Ahmad, 1986). This bruxing seems to result from a periodontal nociceptive reflex response as deciduous roots resorb, leading to increased mobility. The mobility, or loss of deciduous canines in Angle's Class I or II occlusal schemes, appears to stimulate bruxing with the loss of cuspid rise (Williamson & Lundquist, 1983). Bruxing was found less frequently in the later teen years (ages 15-18) as canine protected guidance developed in Angle's Class I and/or II occlusal schemes (Ahmad, 1986). Since bruxism is observed less frequently during the later turbulent adolescent years, anxiety by itself does not appear to stimulate parafunction. The literature therefore suggests that bruxing among children is a physiologic nociceptive reflex