Chapter 8
Occlusal Considerations in the Hypersensitive Dentition

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ABSTRACT

In the literature, Dentinal Hypersensitivity (DH) is considered to arise from exposed dentin and patent dentinal tubules. However, clinical observation of recurrent DH sensitivity indicates it can occur in the presence or absence of exposed dentin. Quantified occlusal contact force and timing parameters have been ignored in studies assessing hypersensitive teeth. This chapter introduces a novel occlusal concept: Frictional Dental Hypersensitivity (FDH). Clinical evidence from combining computerized occlusal analysis and electromyography is presented linking opposing posterior tooth friction and muscular hyperactivity to Dentin Hypersensitivity. This chapter proffers how occlusion, muscular TMD symptoms, and frictional Dentin Hypersensitivity are all related. Lastly, a Pilot Study is presented that used a Visual Numerical Analog scale to quantify Dentin Hypersensitivity resolution observed in symptomatic patients who underwent the Immediate Complete Anterior Guidance Development (ICAGD) coronoplasty. This computer-guided occlusal adjustment eliminated pretreatment FDH symptomatology, further supporting that Dentinal Hypersensitivity has an occlusally-based, frictional etiology.

INTRODUCTION

Dentinal Hypersensitivity (DH) is classically described in the dental literature as a sharp, acute pain of short duration, arising from open dentinal tubules in vital teeth, which is diagnosed through a process of exclusion with a thorough dental screening, examination, and history (Porto, Andrade, & Montes, 2009). DH is generally occurs in the cervical regions of teeth, as in the abfractive lesion, or on the occlusal surface where occlusal microtrauma, erosion, abrasion, or attrition, has exposed dentinal tubules.

This chapter will explore how occlusion is frequently linked to the sharp, short duration acute pain, consistent with the clinical diagnosis of Dentinal Hypersensitivity, which can occur without exposed dentinal tubules. The included literature will detail how dental occlusion can be one of the primary causative factors in the development of hypersensitive dentitions, re-
sultant from prolonged frictional interactions between opposing teeth in function, and how the same occlusal surface friction can lead to hyperactive muscles, which may over time, cause abfraactive events, exposed dentin, and patent dentinal tubules. The new term of Frictional Dental Hypersensitivity (FDH) will be presented, which describes DH of frictional occlusal etiology, with or without exposed dentin. The condition known as Traumatic Occlusion (synonymous with occlusal microtrauma and hyperocclusion) will also be defined, compared and contrasted to FDH, as well as to classical DH that involves exposed dentin. The myriad of scientific theories attempting to explain the causation of DH will also be reviewed, along with a discussion of the mechanisms that explain Frictional Dental Hypersensitivity events. A rational protocol for optimum treatment using occlusal adjustment to treat FDH, and the clinical factors that can identify FDH patients whom could benefit from computer-guided occlusal treatment, are detailed, as well.

Proposed Mechanism of Frictional Dental Hypersensitivity

A tooth’s pain response from FDH may be indicative of a significant muscular occlusal overload, applied to specific teeth within the functional range of motion. The nociceptive response, which may be inflammatory in origin, and occurs within the pulpal and/or periodontal interface of the overloaded teeth, leads to frequent short duration pain episodes.

It has been documented that excess frictional engagement of the posterior teeth, over time (Disclusion Time > 0.5 seconds) is known to promote hyperactivity of the muscles of mastication (Kerstein & Wright, 1991; Kerstein, 1993; Kerstein & Radke, 2006; Kerstein & Radke, 2012). Hypothetically, if excess muscular hyperactivity could be linked to the nociceptive response that manifested clinically as FDH, then the occlusal adjustment procedure known as Immediate Complete Anterior Guidance Development (ICAGD), could be used to decrease the Disclusion Time to less than 0.5 seconds (Kerstein 1992), and quiesce the hyperactive musculature thereby indirectly eliminating the FDH symptomotology.

ICAGD is a precise and conservative, measurement-driven coronoplasty, which involves a subtractive removal phase and occasionally an additive phase (when orthodontic movements are not an option), to optimize a physiologic change in both occlusal force and timing endpoints. ICAGD has been shown through research and case reports to result in a clinical minimization of periodontal ligament compression time, which indirectly results in the reduction of hyperactive elevator muscle activity down to a more normal functional baseline (Kerstein & Wright, 1991; Kerstein, 1993; Kerstein & Radke, 2006; Kerstein & Radke, 2012).

This chapter will define Dentinal Hypersensitivity (DH), describe theories of its etiology, and discuss the research that points both away from, and towards, a mechanically activated occlusal etiology. It is important to note that the traditional DH concept involves exposed dentinal tubules, while the novel FDH concept does not require or necessarily involve exposed dentinal tubules, FDH will be implicated as a precursor to the genesis of DH episodes. Additional chapter content will highlight clinical patient diagnostic procedures, as well as the traditional treatments available for Dentinal Hypersensitivity. Lastly, this chapter will proffer how occlusion, muscular TMD dysfunctional conditions, and frictional DH are all intricately related. A pilot study will be presented that indicates predictable resolution of DH can be achieved following the ICAGD coronoplasty, further supporting the concept of frictionally caused, Dentinal Hypersensitivity (FDH).
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