Section 8
## Additional Clinical Applications of the T-Scan 10 System

# Chapter 22
# State-of-the-Art Diagnostic and Treatment Methods for the Successful Management of Hypersensitive Teeth

**Nick C. Yiannios**
*Private Practice, USA & Founder of The Center for Neural Occlusion, USA*

## ABSTRACT

*In dental literature, the clinically confounding association between the occlusion and hypersensitive teeth is poorly explained. Quantified occlusal contact force and timing parameters have been largely ignored in studies assessing hypersensitive teeth. This chapter introduces a novel occlusal concept; frictional dental hypersensitivity (FDH), after systemically simplifying the existing and often confusing terminology used in the literature over the past decades to describe the variant clinical presentations of the hypersensitive dentition. Clinical evidence from combining computerized occlusal analysis and electromyography is presented linking opposing posterior tooth friction and muscular hyperactivity to FDH. This chapter will outline how occlusion, many muscular TMD symptoms, and FDH are all interrelated. After the differences between dentinal hypersensitivity, cervical dentinal hypersensitivity, and frictional dental hypersensitivity and the myriad of etiologies and modern treatments available are explained, an effective treatment regimen combining the usage of Nd:YAG and Er:YAG lasers coupled with medical grade ozone as a first line diagnostic and treatment protocol for hypersensitive teeth of non-occlusal origins is discussed. Afterwards, both the original FDH Pilot Study and a 100 subject Cold Ice Water Swish follow-up FDH study are then presented that used a numerical Visual Analog Scale (NS/VAS) to quantify cold response dental hypersensitivity resolution observed in occlusally symptomatic patients that underwent the immediate complete anterior guidance development coronoplasty (ICAGD). This computer-guided occlusal adjustment procedure eliminated pretreatment FDH cold symptomatology, further supporting that dental hypersensitivity often has an occlusally-based, frictional etiology. Recent clinical research studies challenging the FDH theory are then presented, including a recent study that compared air indexing protocols to the cold ice water swish and the statistical correlation that was found between these two initiators of dental hypersensitivity following the ICAGD occlusal adjustment procedure. Additionally, consideration for the orthopedic influences that may directly affect the occlusion and neurology of the*

DOI: 10.4018/978-1-6684-9313-7.ch022

*system are outlined, as is the medical concept of dental allodynia which most dental practitioners are unaware of. Furthermore, trigeminal neurological influences are compared to autonomic sympathetic inputs in relation to the influence that they each have upon the hypersensitive dentition, because after all, pulpal neurology consists of not only trigeminal nerve fibers, but cervical sensory and sympathetic nerve fibers as well. Lastly, the greater auricular diagnostic nerve block is discussed, as is the influence that this nerve may have upon the hypersensitive mandibular posterior dentition.*

## INTRODUCTION

Dentin 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 has been generally promoted to occur in the cervical regions of teeth, as in the abfractive process or historically, on the occlusal surface where occlusal microtrauma, erosion, abrasion, and/or attrition has exposed dentinal tubules. Just before the turn of the 21st century, references began to differentiate DH from what has come to be known as Cervical Dentin Hypersensitivity (CDH); the former is currently used to describe short, dull and lingering hypersensitivity pain, whilst the latter is used to describe the fast, sharp and rapidly conductive pain associated with the hypersensitive dentition (Pashley, 1993; Coleman & Kinderknecht, 2000). Both hypersensitivity descriptors may occur in patients with or without open dentinal tubules in the vital dentition, and both are diagnosed through a process of exclusion.

- To date, the dental profession still lacks objective and scientifically validated information to diagnose and treat all cases of Dental Hypersensitivity (DTLH), but as this novel chapter will demonstrate, strides are underway to further elucidate the true genesis of the hypersensitive dentition.
- Within this chapter the term **Dental Hypersensitivity (abbreviated as DTLH***)*, will be referring to all forms of tooth hypersensitivity pains, such that DTLH encompasses DH, CDH, and Frictional Dental Hypersensitivity (FDH), described below.

Designed for practical clinical usage, this chapter will offer sensible alternatives to the confusing and often inaccurate descriptors used to both diagnose and describe the hypersensitive dentition, as well as explore how occlusion is often linked to the sharp, short duration, rapidly conductive acute pain consistent with the clinical diagnosis of Frictional *Dental* Hypersensitivity (FDH) which can occur *with or 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, resultant from prolonged frictional interactions between opposing teeth in excursive function and how the same occlusal surface friction can lead to hyperactive muscles which may over time cause abfractive events, exposed dentin, and patent dentin tubules. The new term of Frictional Dental Hypersensitivity (FDH) will be presented which describes *Dental Hypersensitivity (DTLH) of occlusal etiology irrespective of the presence or lack of exposed dentin*. The conditions known as Traumatic Occlusion (synonymous with occlusal microtrauma and hyperocclusion) and Cervical Dentin Hypersensitivity (CDH) 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 and CDH will also be reviewed, along with a discussion of the theories that potentially explain FDH events. A rational protocol for optimum