Dentinal Hypersensitivity: Etiology, Diagnosis and Management

A Peer-Reviewed Publication
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Educational Objectives
The overall goal of this course is to provide dental professionals with information on the etiology, diagnosis and treatment of dentinal hypersensitivity. Upon completion of this course, the participant will be able to do the following:
1. Know the incidence of dentinal hypersensitivity and risk factors for this condition
2. Know the anatomical and physiological features, and the accepted theory, associated with dentinal hypersensitivity
3. Understand the need for screening and diagnosis by exclusion for dentinal hypersensitivity
4. Know the treatment options available for dentinal hypersensitivity and considerations in selecting these.

Abstract
Dentinal hypersensitivity has been referred to as one of the most painful and chronic dental conditions, with a reported prevalence of between 4% and 57% in the general population and a higher prevalence in periodontal patients. It may also occur as a result of, or during, dental treatment. Clinicians must screen for dentinal hypersensitivity and diagnose by exclusion, determine appropriate treatment, and provide treatment and preventive recommendations. Consideration should also be given to treating dentinal hypersensitivity associated with dental treatment. Traditional treatments have included adhesive resins, fluoride varnishes, HEMA, iontophoresis, gingival grafts and desensitizing dentifrices. Other technologies include the use of bioglass particles, ACP, as well as 8% arginine and calcium carbonate paste.

Introduction
During routine dental examinations, our patients frequently inquire about dentinal hypersensitivity that was one episode or is chronic and recurring due to a given action, e.g., drinking cold beverages, eating hot foods, breathing in and out. This common complaint is defined as dentinal hypersensitivity, but it is also known as root sensitivity, or just sensitivity. Patients describe this phenomenon as sharp, short-lasting tooth pain, irrespective of the stimulus.1 Holland et al. described dentinal hypersensitivity as “characterized by short, sharp pain arising from exposed dentin in response to stimuli typically thermal, evaporative, tactile, osmotic or chemical and which cannot be ascribed to any other form of dental defect of pathology.”2

The prevalence of dentinal hypersensitivity has been reported to be between 4% and 57% in the general population.3-10 Among periodontal patients, its frequency is considerably higher (60%-98%).11,12 This hypersensitivity may be due to cementum removal during root instrumentation. Dentinal hypersensitivity has been described as generally occurring in patients 30 to 40 years old,11 but it can occur in patients significantly younger or older. Women may be affected more often than men.14 Dentinal hypersensitivity affects incisors, canines, premolars and molars, with canines and premolars reported to be affected most often.15,16 Patients with dentinal hypersensitivity may not specifically seek treatment, because they do not view it as a significant dental health problem, but will mention it at a routine dental appointment.17 At other times, patients will seek treatment recommendations from their dental professionals. Some patients are concerned whenever there is dental pain,18 and for some the first time they experience dentinal hypersensitivity creates fear that there is something more serious occurring. The authors of this course have had patients report sensitivity who believe that it may be a toothache that requires immediate attention so that the pain does not get worse. Patients can identify areas of dentinal hypersensitivity before a clinical exam is performed. This may be chronic, or unpredictable and cause intermittent discomfort that is difficult to pinpoint.19,20 Other patients cannot distinguish between dentinal sensitivity and gingival sensitivity. Interestingly, in some developing countries, patients think that sensitivity means that a tooth is bad and they would like to have it removed. Patients may also experience dentinal hypersensitivity as a result of treatment such as scaling and root planing or during routine and normal actions associated with treatment, such as when a tooth is dried using an air spray or scratched with the tip of an explorer. Dental treatment can also exacerbate pre-existing sensitivity.

Dentinal hypersensitivity has all the criteria to be considered a true pain syndrome.21 It is important to distinguish sensitivity pain, that of short duration, from pain of longer duration not treatable with desensitizing agents. A painful response that lingers or that wakens the person from a sound sleep may be the result of pulpal inflammation. A diagnosis by the dentist is necessary to establish a cause and effect, and a diagnosis by exclusion must be made for dentinal hypersensitivity, ruling out other conditions requiring different treatment. After the diagnosis of dentinal hypersensitivity has been made, depending on the etiology, recommendations can be made for effective treatment. Calvo noted in 1884: “There is great need of a medicament, which while lessening the sensitivity of dentin, will not impair the vitality of the pulp.”22 Recommendations can include in-office, at-home professionally dispensed or over-the-counter treatments.23-26 Regardless of which treatment recommendations are made and provided, it is important to follow up with the patient to evaluate the therapeutic results.

Etiology and Physiology of Dentinal Hypersensitivity
Dentinal hypersensitivity can have multiple etiologies. It is important that the patient’s medical and social history, lifestyle, medications and supplements being taken, diet and food habits, and oral hygiene be thoroughly reviewed. Before making a diagnosis of dentinal hypersensitivity,
other oral conditions must be ruled out, including occlusal trauma, caries, defective restorations, fractured or cracked teeth, potential reversible or irreversible pulpal pathology, or gingival conditions. For instance, pain during chewing may be due to a fractured and mobile restoration that is rubbing against the dentin or diagnostic for a cracked tooth.

Dentin is sensitive due to its anatomy and physiology. It is a porous, mineralized connective tissue with an organic matrix of collagenous proteins and an inorganic component, hydroxyapatite. Dentinal tubules are micro-canals that radiate outward through the dentin from the pulp cavity to the dentinal surface, with different configurations and diameters in different teeth. For human dentin, one square millimeter can contain 30,000 tubules, depending on depth. Each tubule contains a Tomes fiber (cytoplasmic cell process) and an odontoblast that communicates with the pulp. Within the dentinal tubules there are two types of nerve fibers, myelinated (A-fibers) and unmyelinated (C-fibers). The A-fibers are responsible for the sensation of dentinal hypersensitivity, perceived as pain in response to all stimuli.

The most widely accepted mechanism of dentin sensitivity is the hydrodynamic theory, first described by Brånstrom. In this model, the aspiration of odontoblasts into the dentinal tubules, as an immediate effect of physical stimuli applied to exposed dentin, results in the outward flow of the tubular contents (dental fluids) through capillary action (Figure 1). The changes to the dentinal surface lead to stimulation of the A-type nerve fibers surrounding the odontoblasts. For there to be a stimulus response, the tubules must be open at both the dentinal interface and within the pulp. Absi and coworkers reported that nonsensitive teeth were not responsive to any physical stimuli; sensitive teeth had up to eight times the number of open dentinal tubules per surface area compared to nonresponsive teeth. Another theory is an alteration in pulpal sensory nerve activity. The treatment of exposed, open dentinal tubules is based upon the physiology of the stimulus response.

**Location of Dentinal Hypersensitivity – Patients at Risk**

Why are some root surfaces hypersensitive and others are not?

Exposed root surfaces due to gingival recession are a major predisposing factor to dentinal root hypersensitivity (Figure 2). According to a recent report of adults over the age of 60, almost 32% had root caries or a restored root surface. Since root caries are an indication of periodontal attachment loss and subsequent recession, this defines the population of adults over 60 with an at-risk of recession in at least one or more teeth as at least 30%. Another study concluded that at least 22% of the adult population between 30 and 90 years of age will have evidence of recession in one or more teeth of 3 mm or more. Gingival recession is more common as patients age and in patients with better oral hygiene. Common causes include inadequate attached gingiva, prominent roots with a thin alveolar housing or bony dehiscence, toothbrush abrasion, periodontal surgery, factitial habits (e.g., picking at cervical of tooth with fingernail), excessive tooth cleaning, excessive flossing, loss of gingival attachment due to specific pathologies, and iatrogenic loss of attachment during restorative procedures.

**Figure 2. Gingival recession with exposed root surfaces**

Dentinal hypersensitivity can also occur as result of a routine dental cleaning, or be exacerbated during scaling and root planing or routine dental prophylaxis and polishing due to pre-existing dentin-root hypersensitivity. Patients who have had or are having periodontal therapy are at risk; the prevalence of root sensitivity has been reported as 9%–23% before and 54%–55% after periodontal therapy. An increase in the intensity of root sensitivity occurred one to three weeks following therapy, after which it slowly decreased. An assessment found that all patients experienced increased discomfort and dentinal hypersensitivity after periodontal treatment, including scaling and root planing. Fear of pain and discomfort during subgingival instrumentation has been reported to deter 10% of the population from seeking treatment. Once the root surfaces are exposed, the cementum/dentin is more susceptible to caries and loss of tooth substance due to erosion, abrasion and abfraction (Figure 3). Postprocedural sensitivity can also be a result of etching beyond restoration.
margins, leaving dentinal tubules open, or of finishing and polishing a restoration that extends to the root surfaces, which can also leave dentinal tubules open. Root surfaces on teeth adjacent to a tooth being extracted can be abraded and scarred with the use of dental elevators during the extraction procedure. Resective periodontal surgical procedures may also leave roots exposed. Enamel loss with exposed dentin due to attrition and tooth wear due to bruxism, occlusal habits and other forms of parafunctional activity can also contribute to the etiology of dentinal hypersensitivity (Figure 4).\(^1\)

**Figure 3.** Gingival recession with associated noncarious cervical lesions

In normal function, the tubules sclerose and become plugged, and when dentin is cut or abraded the mineralized matrix produces debris that spreads over the dentin surface to form a smear layer.\(^3,4\,44\) This occurs to both enamel and dentin,\(^4\) but the loss of this smear layer, the unplugging of the dentinal tubules, contributes to dentinal hypersensitivity (Figure 5).

**Figure 4.** Enamel loss with exposed dentin due to attrition

**Figure 5.** Scanning electron micrograph demonstrating open dentinal tubules

Biofilm deposits on root surfaces may also increase hypersensitivity. The opening of dentinal tubules can also occur due to poor oral hygiene techniques leaving bacterial plaque/biofilm on root surfaces, with the acidic by-products of the biofilm opening the dentinal tubules. Conversely, excellent oral hygiene techniques but with highly abrasive dentifrices can cause continued dentinal tubule exposure. Root surfaces exposed to the physical action of toothbrushing with and without toothpaste can be predisposing factors in removing the smear layer, leaving a tooth hypersensitive.\(^13,45\) Exposure of the oral cavity to acids, e.g., ingestion of acidic foods and beverages,\(^46-48\) or ingestion of chlorinated pool water,\(^49\) as well as bulimia and gastrointestinal reflux disease can also contribute to the opening of the end of the dentinal tubules (Figure 6).\(^50\) Brushing immediately after ingesting acidic foods or beverages should be avoided.\(^51\)

**Figure 6.** Erosion of the maxillary anterior teeth in a bulimic patient due to stomach acid

**Screening and Diagnosis of Dentinal Hypersensitivity**

Dentists and dental hygienists unfortunately do not all routinely include screening for dentinal hypersensitivity.\(^25\) In 1995, a random sample of Dutch dentists completed a survey on the prevalence, conditions and treatment of cervical hypersensitivity of their patients.\(^52\) A similar questionnaire was administered to U.K. dentists in 2002.\(^53\) For both groups, the results revealed discrepancies in screening, perceptions and knowledge of treatment. A separate study administered a questionnaire by mail to 5,000 dentists and 3,000 dental hygienists in Canada and revealed that fewer than half of the respondents considered a differential diagnosis for dentinal hypersensitivity, even though it is by definition a diagnosis of exclusion.\(^25\) Many misidentified the etiology: 64% of the dentists and 77% of the hygienists incorrectly cited bruxism and malocclusion as triggers for dentinal hypersensitivity, while only 7% of dentists and 5% of dental hygienists correctly identified erosion as a primary cause and 17% of dentists and 48% of hygienists were unable to identify the accepted theory of hypersensitivity. Only half of the respondents had the confidence to manage a patient’s pain and to consider the modification of predisposing factors to control a patient’s pain. This survey also demonstrated a lack of understanding of desensitizing toothpastes – most dentists (56%) and dental hygienists (68%) believed these helped prevent dentinal hypersensitivity, while 31% and 16%, respectively, did not
believe that desensitizing toothpastes provided relief from dentinal hypersensitivity.

Dental professionals need to fully understand the etiology and treatment of dentinal hypersensitivity, to screen for it and to diagnose it by exclusion. It is also worth noting that patients with unresolved hypersensitivity over many years provide the dental professional with varied behavioral and postural clues, some of which are easily recognized. These include avoidance of routine dental exams, necessary treatment and follow-up care, reluctance to schedule planned treatment or follow-up care, insistence on the use of local anesthesia for even the most minor of dental treatments, tense facial muscles, tooth clenching, a rigid torso, holding hands tightly on the arm rest, crossed arms, an awkward head position and an inability to follow routine instructions for head and body positioning. 19

As part of any screening for dentinal hypersensitivity, the clinician should assess whether there is a localized or generalized problem. In addition, for patients with identified isolated and generalized dentinal hypersensitivity, a routine dental cleaning can be anxiety provoking. 38 Consideration should be given to dentinal hypersensitivity associated with dental treatment – during treatment and postoperatively. While the focus of controlling pain for many dental professionals during periodontal scaling and root planing and routine dental cleanings has been the use of local and topical anesthetic agents, we should also give thought to providing our patients with treatments to relieve postprocedural dentinal hypersensitivity. 19,26,56,57

Treatment and Prevention of Dentinal Hypersensitivity

Once the diagnosis of dentinal hypersensitivity has been made and the etiologic factors identified, treatment and prevention should be primary goals, 19,38,59 and a treatment plan can be developed and implemented. Once a tooth or teeth are predisposed to dentinal hypersensitivity, they will need to be re-evaluated for continued treatment. The patient should be shown correct brushing techniques to prevent further loss of dentin that would contribute to dentinal hypersensitivity; improper toothbrushing has also been associated with dentinal hypersensitivity. 1 It has been shown that both a manual and a power brush used with a desensitizing toothpaste are almost equivalent in effectiveness. 60 If there are changes and behavior modifications or treatments that can be made, these should be discussed with the patient. Drisko summarized preventive recommendations (Table 1). 61

Treatment of Dentinal Hypersensitivity

Two major groups of products are used to treat dentinal hypersensitivity: those that block and occlude dentinal tubules, and those that interfere with the transmission of neural impulses. Localized dentinal hypersensitivity can usually be treated in-office. For generalized conditions where there is significant recession on multiple teeth, an at-home treatment regimen may be a better choice.

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Professional in-office treatments

In-office desensitizing agents work by occluding and sealing the dentin tubules. 62,63 When treating patients with an in-office treatment, American Dental Association treatment codes can be noted for insurance reimbursement (Table 2).

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<td>D9910 Application of desensitizing medicament</td>
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<td>Includes in-office treatment of root sensitivity. Typically reported on a “per visit” basis for application of topical fluoride or other desensitizing agents. This code is not used for bases, liners or adhesives used under restorations.</td>
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<tr>
<td>D9911 Application of desensitizing resin for cervical and/or root surface</td>
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<td>Typically reported on a “per tooth” basis for application of adhesive resins. This code is not used for bases, liners or adhesives used under restorations.</td>
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A recent novel approach is a technology based on arginine and calcium carbonate. This technology was introduced as a result of the need to provide patients with a treatment regimen to reduce and treat dentinal hypersensitivity after dental cleanings. In 2002, Kleinberg et al. reported on the development of this novel desensitizing technology based upon the role that saliva plays in naturally reducing dentinal hypersensitivity. Saliva provides calcium and phosphate, which over time will occlude and block...
open dentinal tubules from external stimuli associated with dentinal hypersensitivity.\textsuperscript{19,56} Reduced salivary flow, hyposalivation and xerostomia are risk factors for caries and tooth demineralization and may exacerbate dentinal hypersensitivity. While hyposalivation may be due to medical conditions and aging, it is also a side effect of more than 500 prescription and over-the-counter medications.\textsuperscript{64} The mechanism providing for the clinical effectiveness of this technology utilizes arginine, an amino acid; bicarbonate, a pH buffer; and calcium carbonate, a source of calcium. This technology, originally introduced as Sensistat\textsuperscript{®} (Ortek Therapeutics, Roslyn Heights, NY), effectively relieves dentinal hypersensitivity (Figure 7).\textsuperscript{56} The technology is proposed to block dentinal hypersensitivity pain by occluding dentinal tubules by using arginine, which is positively charged at physiologic pH of 6.5-7.5, to bind to the negatively charged dentin surface, and helps attract a calcium-rich layer from the saliva to infiltrate and block the dentinal tubules. An in-office product based upon this technology (ProClude) was used for the management of tooth sensitivity during professional dental cleanings. Early studies on this technology demonstrated instant relief from discomfort that lasted 28 days after a single application and reported a 71.7% reduction in sensitivity measured by air-blast and an 84.2% reduction by the “scratch” test immediately following application.\textsuperscript{56} The same technology was used in a toothpaste (DenClude).

In 2007, Colgate-Palmolive Company acquired the rights to the technology, now known as ProArgin\textsuperscript{™} technology, and has introduced Colgate® Sensitive Pro-Relief\textsuperscript{™} Desensitizing Paste (Figure 7). This is applied in-office using a prophylaxis cup on a prophy angle. The recommendation is that the paste be applied using a low speed handpiece with a moderate amount of pressure to burnish the paste into the exposed tubules, optimizing their occlusion. This product can be used before or after dental procedures.

**Figure 7. Colgate® Sensitive Pro-Relief™ Desensitizing Paste**

In clinical trials, this product has been found to provide immediate and lasting relief of hypersensitivity for four weeks when it is applied in patients who had scaling and root planing.\textsuperscript{77} A second study demonstrated its effectiveness in relieving dentinal hypersensitivity when applied prior to dental prophylaxis, with a significant reduction in dentinal hypersensitivity demonstrated postprocedurally.\textsuperscript{65} An evaluation of this desensitizing paste containing 8% arginine and calcium carbonate on dentin and enamel found no significant effect on surface roughness.\textsuperscript{56} In investigating the mechanism of action of arginine and calcium carbonate paste using scanning electron microscopy, confocal laser scanning microscopy and atomic force microscopy, Petrou et al. found that the technology totally occluded the dentinal tubules rapidly. This was the result of the formation of a deposit on the surface and plugs in the dentinal tubules that contained high amounts of phosphate, calcium and carbonate. In addition, it was determined through hydraulic conductance testing that these deposits significantly reduced the flow of dentinal fluid in the tubules.\textsuperscript{67} In-office paint-on surface treatments are a popular approach to treating root hypersensitivity, and are especially effective for localized dentinal hypersensitivity (single teeth). These products generally occlude and seal the dentin tubules. A variety of products has been reported to effectively reduce dentinal hypersensitivity, including resin-based materials.\textsuperscript{68-71} 5% sodium fluoride varnish (Duraphat, Colgate-Palmolive, New York, NY) painted over exposed root surfaces has been shown to be an effective treatment for dentinal hypersensitivity.\textsuperscript{62} An aqueous solution of glutaraldehyde and hydroxyethylmethacrylate (HEMA) (Gluma Desensitizer, Heraeus-Kulzer; Calm-It\textsuperscript{™}, Dentsply-Caulk) has been reported to be an effective desensitizing agent for up to nine months.\textsuperscript{71,72} The mechanism for tubule occlusion appears to be due to the glutaraldehyde.\textsuperscript{73} The use of oxalates has also been shown to be effective, with the oxalate precipitating and occluding the open dentinal tubules.\textsuperscript{74} In addition, while there have not been any controlled studies on its effectiveness, anecdotal evidence suggests that burnishing a 0.5% solution of prednisolone onto exposed sensitive root surfaces may mitigate intractable hypersensitivity.

Other treatment options include gingival grafts, adhesive resins, lasers and topically applied agents. Gingival grafts should be considered, in particular when the recession is progressive, there are aesthetic concerns or the sensitivity is unresponsive to more conservative treatment.\textsuperscript{75} When the exposed sensitive root surface has surface loss due to abrasion, erosion and/or abfraction leaving a notching of the root, consideration should be given to placing either an adhesive composite resin or glass ionomer restoration,\textsuperscript{76} which would both restore the tooth to full contour and seal the dentinal tubules. Lasers have been used successfully to seal open dentinal tubules either alone or with surface treatments.\textsuperscript{77,79} Iontophoresis can also be used, a technique that utilizes a low galvanic current...
to accelerate ionic exchanges and precipitation of insoluble calcium with fluoride gels to occlude the open tubules.

Figure 9. In-office paint-on surface treatments

Recommendations for use and technique are product specific. The clinician needs to understand the in-office desensitizing agents to select one that is appropriate for the patient.

Professionally dispensed self-applied treatments

A professionally prescribed at-home treatment for dentinal hypersensitivity that provides rapid and continued relief using a bioactive glass technology has been introduced (SootheRx™, 3M/ESPE Preventive Care). This contains a calcium sodium phosphosilicate bioactive glass (NovaMin®) that induces the formation of hydroxyapatite to seal and clog open dentinal tubules. The basis for this use comes from a clinical trial using the same active ingredient in toothpaste. Amorphous calcium phosphate and casein phosphopeptide-amorphous calcium phosphate products (Relief ACP, Discus Dental; MI Paste™, GC America) can also be used for desensitization by brushing them on the teeth, including before and after mouthguard or in-office bleaching. ACP has also been found to be effective for control of bleeding sensitivity when incorporated into bleaching gels. The use of ProClude, the precursor to Colgate® Sensitive Pro-Relief™ Desensitizing Paste, was also been reported to decrease sensitivity when used before bleaching.

Self-applied over-the-counter treatments

Over-the-counter (OTC) treatments for sensitive teeth can be the most cost-effective means to relieve sensitivity, and many people make the decision to self-medicate with desensitizing toothpastes. The claim of desensitizing teeth is a therapeutic claim and the toothpaste must contain an active ingredient that is recognized by the FDA as being an effective desensitizer at that concentration. For anything not recognized by the FDA as a desensitizing ingredient, a new drug application is required. The most popular desensitizing ingredient in toothpastes is potassium nitrate. According to the FDA monograph, for a potassium nitrate toothpaste to claim to be desensitizing, it must contain 5% potassium nitrate (Sensodyne®, GlaxoSmithKline; Colgate® Sensitive and Colgate® Sensitive Enamel Protect™, Colgate-Palmolive; Crest® Sensitivity, Procter & Gamble). The mode of action involves penetration of the potassium ions through the tubules to the A-fibers of the nerves, decreasing the excitability of these nerves. Studies have evaluated the effectiveness of 5% potassium nitrate toothpastes containing other additives, and there is no difference in the reduction of dentinal hypersensitivity. Many clinical trials have provided evidence of a reduction in tooth sensitivity with toothpastes containing potassium nitrate. These toothpastes may take up to two weeks to show any effectiveness. For best results, the toothpaste should be used twice a day as part of the patient’s oral care regimen.

In recent years, vital bleaching has become very popular, with transient tooth sensitivity as a primary reported side effect with an incidence of 7% to 75%. For many patients, this is a barrier to continuing treatment, and 5% potassium nitrate desensitizing toothpaste has been recommended for patients undergoing bleaching. Two effective strategies using a 5% potassium nitrate desensitizing toothpaste are brushing with it for two weeks prior to initiating bleaching and having the patient place it into his or her bleaching tray and wear the tray for 30 minutes a day one week prior to the initiation of bleaching.

Conclusion

As part of the routine dental examination and during every recall appointment, dental professionals should include in their patient questions queries about whether there are any sensitive teeth. Patients with dentinal hypersensitivity should be evaluated based upon risk factors and a proper diagnosis made, after which a treatment plan can be outlined for the patient. In most circumstances, the least invasive, most cost-effective treatment is the use of an effective desensitizing toothpaste. Depending on the severity of dentinal hypersensitivity, clinical management may include both in-office and self-applied at-home therapies, including recent and novel technologies that have been introduced.

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