Cervical Dentin Hypersensitivity: Etiology, Diagnosis, and Management

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Cervical Dentin Hypersensitivity: Etiology, Diagnosis, and Management

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LEARNING OBJECTIVES
After reading this article, the individual will learn:

- The accepted mechanism of dentinal sensation.
- The etiology, differential diagnosis, and management of cervical dentin hypersensitivity (CDH).

ABOUT THE AUTHORS

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INTRODUCTION
Cervical dentin hypersensitivity (CDH) is a common clinical condition that has been reported to annoy and affect 15% to 20% of the adult population, and most clinical practitioners. Approximately 40 million adults in North America may have some degree of dentin hypersensitivity at some time in their life, whereas in other areas of the world the prevalence may approach 50% of the population. Further, this incidence should continue to rise with the increase in life expectancy and patients retaining their natural dentitions longer. Thus, this clinical manifestation presents a significant clinical challenge in dentistry now and in the future.

CDH is a condition characterized by transient sharp tooth pain arising from exposure of dentin and opening of dentinal tubules as well as to inflammatory processes in the underlying pulp tissue. The condition is associated with a variety of exogenous stimuli that elicit pain, including thermal (cold), tactile (touch), evaporative, chemical (acid exposure), or osmotic changes (sweets or drying the surface). A considerable variation exists in the degree and extent of dentin hypersensitivity that can vary from individual to individual due to differences in patency of exposed dentinal tubules, state of the pulp, and the differences in pain tolerance, emotional state, and environmental factors for each individual. The condition may affect any tooth, but it most often affects canines and premolars.

This scientific review describes the accepted mechanism of dentinal sensation and discusses the etiology, differential diagnosis, and management of CDH.

MECHANISMS FOR CERVICAL DENTIN HYPERSENSITIVITY
Although the morphological characteristics of the dentinal tubule have been described through autoradiographic, histochemical, and electron microscopic studies, the precise mechanism of pain transmission from the exposed dentin surface to the terminal nerve ending is only
Many theories have been proposed to explain the mechanism of CDH, including the modulation, transducer, gate control, and hydrodynamic theories. One of these, the odontoblastic transducer mechanism theory proposed by Rapp, et al suggested that odontoblasts act as receptor cells, mediating changes in the membrane potential of the odontoblasts via synaptic junctions with nerves. This theory is inconclusive and not well accepted.

A hydrodynamic hypothesis for dentin sensitivity that was proposed 100 years ago provided the catalyst for the confirmatory evidence produced in the 1960s by Brännström and Aström. This “Hydrodynamic theory” has become the most widely accepted theory for explaining the mechanism of dentinal sensation. This theory postulates that the dentin tubules, which are open and wide, contain a fluid. Various stimuli (ie, thermal, tactile, chemical, or osmotic changes) displace the fluid in the dentinal tubules inwardly or outwardly. This liquid movement stimulates the odontoblast process, and the subsequent mechanical disturbance stimulates a baroreceptor (a nerve receptor sensitive to pressure) that leads to neural discharge (depolarization); this neural pulp activation is perceived as pain.

Pain seems to be produced by a rapid outward displacement of the fluid in the dentinal tubules at the pulpo-dentinal border that is initiated by strong capillary forces, and if sufficiently rapid, it may activate nerves located some distance from the tubules corresponding to the exposed dentin. Furthermore, studies of surface morphologies of hypersensitive and nonsensitive dentin report that dentin which is exposed and sensitive exhibits more numerous, patent, and wider dentinal tubules than in nonsensitive areas.

Another scanning electron microscope study showed that in hypersensitive dentin, the smear layer was thinner, different in structure, and was more likely to be undercalcified than nonsensitive dentin. These findings appear consistent with the hydrodynamic theory. According to Poiseuille’s law, the volume rate of flow within a tube is directly proportional to the fourth power of the tube’s internal radius. Therefore, an increase in the tubule diameter results in an increase of fluid flow by a power of 4, thus pain is amplified when the tubules are open to the oral cavity. Therefore, a greater number of open and wider tubules at the dentin surface enhances fluid permeability through dentin and thus increases the potential for stimulus transmission and subsequent pain response. These findings support the theory of increased hydrodynamic permeability of hypersensitive dentin and have provided strategies for the treatment of hypersensitive dentin. According, dentin hypersensitivity can be treated by minimizing movement of intratubular fluid (Figure 1).

ETIOLOGY AND PREDISPOSING INFLUENCES

There are numerous and varied etiological factors and predisposing influences involved with CDH. More than 90% of hypersensitive surfaces occur at the cervical region on the buccal and labial aspects of the involved teeth. In the ideal anatomical position, most teeth have only the enamel exposed to the oral environment, and dentin that is protected by enamel or cementum is not sensitive. Cervical tooth sensitivity occurs when this enamel or cementum layer is removed and the underlying dentinal tubules are open and exposed to the oral environment.

There are 2 phases in the development of CDH. First, “lesion localization” occurs by exposure of the dentinal tubules from either gingival recession and loss of cementum or through loss of enamel from wear. Since all
exposed dentin is not sensitive, this localized lesion requires the second phase. The second phase, “lesion initiation,” occurs when the smear layer or tubular plugs are removed, which opens the outer ends of the dentinal tubules.\textsuperscript{10,12}

This dentin exposure can be the result of numerous etiologic factors which include abrasion, corrosion, attrition, abfraction, and gingival recession.\textsuperscript{22} These etiologic cofactors can be attributed to a multitude of conditions and phenomena which include aging,\textsuperscript{23} improper oral habits, lack of or excessive tooth brushing,\textsuperscript{24} incorrect tooth brushing,\textsuperscript{25} improper use of bleaching agents,\textsuperscript{12,26} acidic dietary habits,\textsuperscript{13,27} low pH mouth rinses,\textsuperscript{12,13,28} bulimia nervosa,\textsuperscript{12,13,29-32} pyrophosphates, poor oral hygiene,\textsuperscript{13} airborne acids,\textsuperscript{32,33} clenching,\textsuperscript{32,34-37} bruxism,\textsuperscript{13,35} overeruption,\textsuperscript{23} a developmental anomaly, occlusal disharmony,\textsuperscript{13} periodontal disease,\textsuperscript{23} and periodontal therapy.\textsuperscript{38}

The following discussion of several of these phenomena may provide the dental professional with insight and information for determining etiology through differential diagnosis and methods for prevention, management, and treatment:

- With the increasing life span and the increased retention of teeth by the older patient, dental professionals must be aware of the physiological changes associated with the aging process. As age advances, the number of teeth with root exposure increases.\textsuperscript{13} One recent survey revealed that 88% of people age 65 years and older have one or more sites with recession.\textsuperscript{39} The presence and extent of gingival recession also increases with age. Despite these dramatic improvements in tooth retention for the older patient, there still remains a substantial portion of the aged population with missing natural teeth. With the absence of opposing teeth, dental extrusion can occur, resulting in root exposure and the increased potential for dentin hypersensitivity.\textsuperscript{23}

- Improper or inadequate oral hygiene maintenance can result in accumulation of plaque, causing gingival inflammation that can result in gingival recession. This root exposure can be related to CDH and intensified by acids excreted by bacteria, which can result in further opening of the dentinal tubules. Malpositioned teeth and toothbrush trauma have been reported to be the most frequent etiologic factors associated with gingival recession.\textsuperscript{39-41} The incidence of gingival recession increases with age and is greater in males than females of the same age.\textsuperscript{39-41}

- Dentin hypersensitivity can be a consequence of periodontal disease and surgical and nonsurgical periodontal therapy.\textsuperscript{38,42-51} After periodontal treatment, there can be a reduction of the gingival protective barrier as a result of excision of tissue which exposes the root surface. In addition, removal of cementum during root planing may expose the dentin to external stimuli.\textsuperscript{13,42,47,52} Reports by Tammaro, et al\textsuperscript{43} indicate a significant change in dentin hypersensitivity after scaling and root planing, while von Troil, et al\textsuperscript{50} found that dentin hypersensitivity occurred in approximately 50% of patients following scaling and root planing. Tamminen, et al\textsuperscript{48} reported that dentin hypersensitivity occurred after periodontal surgeries using the modified Widman flap procedure. This postoperative dentin hypersensitivity may be due to increased surface exposure of root dentin to external stimuli and gingival recession following periodontal surgery.\textsuperscript{53,54} In addition, age, sex, and type of therapy are factors that can influence postoperative pain and postoperative dentin hypersensitivity during periodontal therapy.\textsuperscript{45}

- Normal function generates considerable stresses within teeth and supporting tissues.\textsuperscript{55} Since teeth are not rigid structures, they undergo deformation (strain) when a functional load is applied,\textsuperscript{2,56} and this strain is proportional to the amount of stress. The functional load is influenced by the number of teeth, the type of occlusion, and the occlusal behavior of the patient (ie, premature contacts, parafunctional habits).\textsuperscript{2} During occlusal loading, the tooth undergoes a lateral or an axial bending called tooth flexure.\textsuperscript{55,57-64} The tooth flexure theory posits that occlusal forces are transmitted through the cusp and can become concentrated in the cervical region of the tooth.\textsuperscript{57,65} This theory has been demonstrated and supported by engineering studies\textsuperscript{50-64,66} reporting that these horizontal loading forces cause a microscopic flexing of the anatomical crown of the tooth. Subsequently, this physiologic bending generates a maximal strain in the cervical region of the tooth with resulting tensile stress concentrations in the cervical region on the side of the tooth from which the force is directed. At the same time, the opposite region of the tooth is under compressive stresses. When the direction of the force
changes (ie, bruxism), the tooth flexes in the opposite direction and the stresses correspondingly reverse at the cervical region. These cyclic tensile and compressive stresses which occur in the mouth during chewing or parafunctional habits can reach a fatigue limit and can result in tooth structure loss and dentin hypersensitivity.

Corrosion is tooth surface loss caused by chemical or electrochemical action; there are 2 sources of origin: endogenous and exogenous. Endogenous sources of corrosion involve unique patterns of enamel loss that are associated with endogenous acids from gastroesophageal reflux disease, bulimia, anorexia nervosa, hiatal hernia, and pregnancy morning sickness. Another endogenous source is gingival crevicular fluid which can be acidic and corrosive when in contact with the cervical region of the tooth. Exogenous sources of corrosion involve a chemically-induced loss of tooth substance from extrinsic origin and are associated with diet (ie, carbonated soft drinks, candies that contain phosphoric or citric acid, citrus fruits or juices, and baby bottle syndrome), airborne acids such as industrial chemicals, and chlorinated swimming pool water. Corrosion produces a more softened enamel zone, and the dentin becomes exposed to the oral environment. An acid environment can further open

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<td><strong>COVER THE DENTINAL TUBULES—PREVENTING FLUID FLOW</strong></td>
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<td>A. Plug the dentinal tubules</td>
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<td>1. Ions/salts</td>
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<td>Strontium chloride hexahydrate</td>
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<td>Zinc chloride</td>
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<td>CPP-ACPF</td>
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the dentinal tubules, resulting in dentin hypersensitivity.

- Postdental bleaching sensitivity and gingival irritation are the 2 main adverse effects of vital tooth bleaching. Gingival irritation can be caused from prolonged contact of the peroxide gel with the gingival tissues. Tooth sensitivity can be attributed to the penetration of the bleaching agent through enamel and dentin and into the pulp chamber during the whitening procedure. The hypersensitivity that occurs in association with bleaching has been attributed to patient factors, the concentration of the bleaching agent, length of exposure to the bleaching agent, pH of the bleaching solution, and factors related to tray fabrication.

**ESTABLISHING A DIAGNOSIS**

The most important part of any clinical treatment is to establish a good diagnosis and to find and eliminate the predisposing factors of the condition. After considering all factors related to cervical tooth sensitivity from corrosion, attrition, abrasion, abfraction, gingival recession, or a combination of these processes, a differential diagnosis should be developed. Dentinal hypersensitivity must be differentiated from other clinical conditions that may cause teeth to be sensitive, and an appropriate diagnosis must be made before any treatment is initiated. The characteristic response to a specific stimulus is that the pain should be sharp, localized, brief, and usually diminishes after removal of the stimulus, but may remain as a dull ache.

Clinical conditions that should be considered include postrestorative sensitivity, postoperative sensitivity from bleaching, fractured teeth or restorations, dental caries, and irreversible pulpitis. This differential diagnosis provides information for determining etiology and can require additional information such as age, diet, oral hygiene routine, occupation, medical and dental factors, abnormal oral habits, and occlusal disharmonies. The information acquired during the differential diagnosis will provide a systematic approach for the management of hypersensitive surfaces through preventive and restorative therapy.

Management of CDH begins with prevention and elimination of the predisposing factors associated with continued dentinal tubule exposure. Preoperative treatment considerations and procedures may include preventive measures such as fluoride therapy, iontophoresis, brushing with desensitizing dentifrices, professional application of potassium oxalate or other tubule occluding agents, application of dentin adhesives, occlusal adjustments, dietary instruction, toothbrushing and oral hygiene instruction, discontinuation of poor oral habits, and occlusal guard fabrication.

**TREATMENT STRATEGIES**

Three fundamental treatment strategies should be considered for the management of CDH (Table). The first treatment strategy is to desensitize the nerve tissue by modifying neural response within the dentin tubule. Potassium nitrate is an effective therapeutic agent that is currently used in all over-the-counter (OTC) desensitization dentifrices with ADA and US Food and Drug Administration approval. Tarbet, et al demonstrated in well-conducted clinical trials that a toothpaste containing 5% potassium nitrate was effective, with daily use, to desensitize for up to 4 weeks, and that potassium nitrate does not induce changes in the pulp. Potassium nitrate is believed to increase the extracellular potassium ion concentration and thus may depolarize the nerve and prevent it from repolarizing. This change disrupts the ionic tubular membrane transmission and prevents sending pain signals to the brain until ionic concentrations restabilize.

Potassium nitrate desensitizing gels (ie, in-office or home application) can be used for preoperative and postoperative bleaching sensitivity. Haywood, et al concluded that 10 to 30 minutes of wearing time of the gel in the tray before or after whitening may reduce sensitivity in more than 90% of patients and make the bleaching procedure tolerable. In addition, additives such as 3% potassium nitrate and 0.11 fluoride ion weight/volume have been reported to reduce but not eliminate sensitivity when added to a 10% carbamide peroxide bleaching gel (Figures 2a and 2b).

A second treatment strategy is to occlude the distal terminal ends of the exposed dentinal tubules. The tubules can be sealed through natural desensitization from secondary dentin formation or mineralization or by utilization of compounds that can precipitate to form an accumulation of denatured protein or a calcified plugging layer. These OTC and professionally applied desensitizing
dentifrice treatments for cervical sensitivity contain various chemicals that include denaturing salts (ie, strontium chloride, formaldehyde) and precipitating agents (ie, sodium fluoride, stannous fluoride, monofluorophosphate, casein phosphopeptide-amorphous calcium fluoride phos-phate, and oxalates) (Figure 3). This treatment is cost-effective, noninvasive, and can be applied at home or in the dental office.

Other effective clinical techniques include iontophoresis and adhesive resin application. Iontophoresis utilizes a charged electrical current to accelerate and precipitate insoluble calcium with fluoride gels. Adhesive resin impregnation is another clinical technique that has increased in popularity in recent years and is currently considered one of the most definitive and rapidly acting methods of desensitization. This procedure reduces sensitivity with the application of a dentin adhesive to form a hybrid layer, and this resin barrier prevents continued diffusion of toxins and bacterial invasion toward the pulp while producing minimal adverse pulpal inflammation (Figure 4). A combination of iontophoresis followed by resin impregnation is one of the most effective clinical techniques for eliminating cervical tooth sensitivity. In addition, one clinical technique that utilizes an application of calcium hydroxide paste has been effective in relieving cervical sensitivity by increasing peritubular dentin mineralization.

A third treatment strategy when there is gingival recession and/or a cervical lesion is to cover the exposed surface of the dentinal tubules by utilizing a periodontal surgical procedure and/or dental restoration. The periodontal procedures include free autogenous mucosal grafts, subepithelial connective tissue grafts, coronally advanced flap technique, guided periodontal tissue regeneration, and acellular dermal matrix grafts. Restorative methods can involve the use of conventional glass ionomers, resin-modified glass ionomers, compomers, flowable composites, hybrid composites, microfill composites, laboratory-processed composite and porcelain veneers, laboratory-processed composite Class V inlays, all-ceramic Class V inlays and crowns, and PFM crowns and bridges.
Since gingival recession is a primary cause of dentin exposure and a major predisposing factor for dentin hypersensitivity, a combined treatment modality should be considered for more advanced cervical lesions with root caries. This treatment strategy combines the use of a glass ionomer to seal the exposed surfaces of the dentinal tubules and to define the root emergence contour while repositioning the gingival contour using a connective tissue graft procedure\textsuperscript{99,101,102} (Figures 5a to 5c). Postoperative sensitivity can be prevalent following periodontal procedures and usually decreases considerably after one to 4 weeks.\textsuperscript{50} Addressing the occlusal status of a patient should also be examined as has been shown in the retrospective study done by Coleman, et al.\textsuperscript{91} Judicious occlusal analysis and equilibration can frequently eliminate CDH. It is suggested that a thorough oral hygiene protocol be integrated with the aforementioned strategies.

**CONCLUSION**

Management of any clinical situation begins with prevention, knowledge of the various etiological factors and predisposing influences, and an understanding of the various therapeutic methods available for treatment. Professionals should recognize the role causative factors play in initiating dentin hypersensitivity and inform and communicate those various clinical considerations and solutions effectively with their patients. This article has attempted to provide the dental professional a general overview for understanding the biology, etiology, and different mechanisms and clinical manifestations of CDH. By utilizing this knowledge with clinical experience and sound judgment, the dental restorative team can identify and determine etiology through differential diagnosis and thus provide methods for prevention, management, and treatment of dentin hypersensitivity.
REFERENCES

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POST EXAMINATION QUESTIONS

1. CDH most often affects:
   a. Maxillary molar teeth.
   b. Mandibular molar teeth.
   c. Incisor teeth.
   d. Canine and premolar teeth.

2. The most widely accepted theory for explaining the mechanism of dentinal sensation is the:
   a. Hydrodynamic theory.
   b. Odontoblastic transducer mechanism theory.
   c. Gate control theory.
   d. Modulation theory.

3. The volume rate of flow within a tube is determined by:
   a. Brännström's law.
   b. Rapp's law.
   c. Poiseuille's law.
   d. Aston's law.

4. Approximately ____% of hypersensitive surfaces occur at the cervical region on the buccal and labial aspects of teeth.
   a. 30.
   b. 50.
   c. 75.
   d. 90.

5. Dentin exposure can result from:
   a. Abrasion.
   b. Corrosion.
   c. Abfraction.
   d. All of the above.

6. One recent survey revealed that ____% of people age 65 years or older have one or more sites with gingival recession.
   a. 65.
   b. 77.
   c. 88.
   d. 93.

7. The incidence of gingival recession increases with age. The incidence of gingival recession is greater in females than males of the same age.
   a. The first statement is true, the second is false.
   b. The first statement is false, the second true.
   c. Both statements are true.
   d. Both statements are false.
8. Tooth surface loss caused by chemical or electrochemical action is called:
   a. Abrasion.
   b. Wear.
   c. Corrosion.
   d. Abfraction.

9. Hypersensitivity associated with tooth bleaching has been attributed to:
   a. The pH of the bleaching agent.
   b. Concentration of bleaching agent.
   c. Length of exposure to the bleaching agent.
   d. All of the above.

10. Pain from CDH usually diminishes after removal of the stimulus. The pain may remain as a dull ache.
    a. The first statement is true, the second is false.
    b. The first statement is false, the second true.
    c. Both statements are true.
    d. Both statements are false.

11. The following is currently used in over-the-counter (OTC) desensitization dentifrices:
    a. Stannous fluoride.
    b. Potassium nitrate.
    c. Sodium fluoride.
    d. Monofluorophosphate.

12. The following precipitating agent is used in OTC and professionally applied desensitizing dentifrices:
    a. Strontium chloride.
    b. Formaldehyde.
    c. Sodium fluoride.
    d. Both a and b.

13. Haywood, et al concluded that wearing a tray containing potassium nitrate desensitizing gel for _______ minutes may reduce dentin sensitivity in 90% or higher of patients:
    a. 5 to 10.
    b. 10 to 30.
    c. 30 to 60.
    d. 60 to 90.

14. A toothpaste containing 5% potassium nitrate used daily can desensitize for up to 4 weeks. However, such use may induce changes in the pulp.
    a. The first statement is true, the second is false.
    b. The first statement is false, the second true.
    c. Both statements are true.
    d. Both statements are false.

15. A combination of iontophoresis followed by resin impregnation:
    a. Is contraindicated due to risk of pulpal changes.
    b. Is rarely helpful in reducing dentin hypersensitivity.
    c. Is one of the most definitive and rapidly acting methods of desensitization.
    d. Has not been studied for clinical effectiveness in reducing dentin hypersensitivity.

16. One clinical technique that relieves cervical sensitivity by increasing peritubular dentin mineralization is:
    a. Iontophoresis.
    b. Calcium hydroxide paste.
    c. Adhesive resin impregnation.
    d. Glass ionomer.
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