# Dentin Hypersensitivity: Part I

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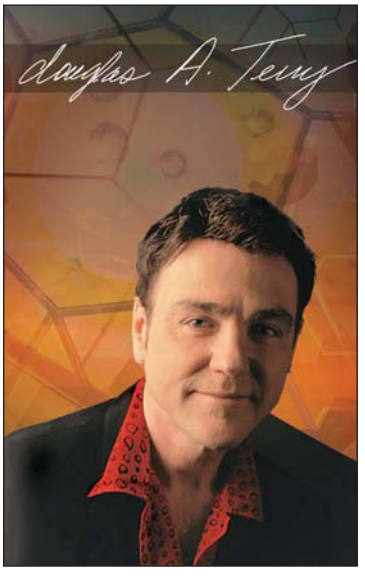
The condition, etiology, and treatment of dentin hypersensitivity, or hyperalgesia, has been reported in the literature for over 100 years. As early as 1884, Calvo stated that there is a great need of a medicament that, while lessening the sensibility of dentine, will not impair the vitality of the pulp.[1] Approximately 40 million adults in North America may have some degree of dentin hypersensitivity at some point in their lives, whereas in other areas of the world, the prevalence may even reach 50% of the population.[2,3] This incidence should continue to rise with the increase in life expectancy.

Dentin hypersensitivity is a transient tooth pain—associated with a variety of exogenous stimuli—that is characterized by exposure of dentin and the opening of dentinal tubules as well as to inflammatory processes in the underlying pulp tissue.[4] The degree and extent of dentin hypersensitivity can vary from individual to individual due to differences in patency of exposed dentinal tubules, state of the pulp, and differences in pain tolerance, emotional state, and environmental factors for each. This two-part discussion will describe the differential diagnosis and management of two types of dentin hypersensitivity: cervical and restorative sensitivity. Part I reviews the mechanism of dentinal pain, etiology of dentin hypersensitivity, and the differential diagnosis and treatment modalities for cervical sensitivity. Part II will review a pretreatment differential diagnosis and describe considerations and clinical techniques for the management of restorative postoperative sensitivity.




## Mechanism for Dentinal Sensation

Although the morphological characteristics of the dentinal tubule have been described through numerous studies, the precise mechanism of pain transmission from the exposed dentin surface to the terminal nerve ending is only theorized. The theories that have been proposed include the transducer, modulation, gate control, and hydrodynamic theory. Brännström's hydrodynamic theory is the currently accepted mechanism of dentinal sensation. This theory postulates that the dentin tubules are open, wide, and contain fluid. Various stimuli (eg, thermal, tactile, chemical, 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, which leads to neural discharge (ie, depolarization). This neural pulpal activation is perceived as pain.[5,6] Pain seems to be produced by a rapid outward displacement of the fluid in the tubules at the pulpodentinal border, which is initiated by strong capillary forces that, if sufficiently rapid, may even activate nerves located some distance from the tubules corresponding to the exposed dentin.[6,7] Furthermore, studies of surface morphologies of hypersensitive and nonsensitive dentin report that dentin that is exposed and sensitive exhibits more numerous, patent, and wider dentinal tubules than in nonsensitive areas. According to Poiseuille's law, the volume flow rate within a tube is directly proportional to the fourth power of the tube's internal radius.[8] An increase in the tubule diameter thus results in an increase of fluid flow by a power of four; hence, pain is amplified when the tubules are open to the oral cavity.[2] These findings support the theory of increased hydrodynamic permeability of hypersensitive dentin[9] and have provided strategies for the treatment of hyperalgesia.

## Etiology of Cervical Tooth Sensitivity

While the aforementioned scientific hypothesis of dentin hypersensitivity assists in understanding the condition and treatment of cervical tooth sensitivity, the clinical cause is exposed and open dentinal tubules. In the ideal anatomical position, only the enamel of most teeth is exposed to the oral environment, and dentin that is protected by

enamel or cementum is not sensitive. Cervical tooth sensitivity occurs when this protective layer is removed and the underlying dentinal tubules are exposed. This exposure can be the result of numerous etiologic factors that include abrasion, erosion, attrition, abfraction, and gingival recession. These etiologic factors can be attributed to a multitude of conditions (eg, aging, improper oral hygiene habits, dietary habits, low pH mouth rinses).[10] Preoperative considerations and procedures may include preventive measures such as fluoride therapy, iontophoresis, brushing with desensitizing dentrifices, 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.

## Developing a Differential Diagnosis

After considering all factors related to cervical tooth sensitivity from erosion, attrition, abrasion, abfraction, gingival recession, or a combination of these processes, a differential diagnosis should be developed. Dentinal hypersensitivity must be differentiated from other conditions that may cause teeth to be sensitive; an appropriate diagnosis must be made before any treatment is initiated. The characteristic response to a specific stimuli is that the pain should be sharp, localized, and brief, and that it usually diminishes following removal of the stimulus.[11] 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, etc.[12] The information acquired during the differential diagnosis will provide a methodical approach for the management of hypersensitive surfaces through preventive and restorative therapy.

**Figure 1. Cervical hypersensitivity is eliminated with an application of an adhesive bonding agent.**

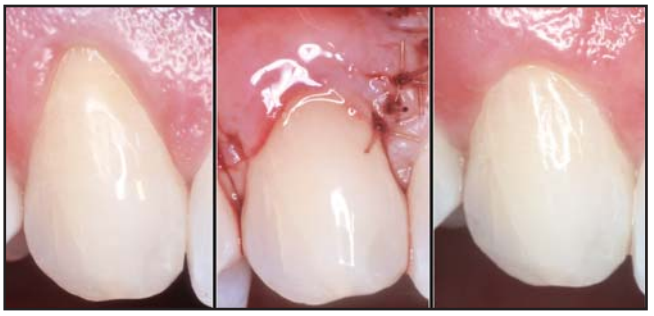


**Figure 2. A free, autogenous connective tissue graft is placed over the exposed dentinal tubules of the Class I gingival recession-type defect.**

## Treatment Strategies for Cervical Sensitivity

An understanding of the previously mentioned mechanism of dentinal sensation provides the following three fundamental treatment strategies for hypersensitive dentin.[13] The first treatment strategy is to desensitize the nerve tissue by modifying neural response within the dentin tubule. Potassium nitrate ($KNO_3$) is an effective therapeutic agent that is currently used in virtually all over-the-counter desensitization dentirices with ADA and FDA approval. Potassium nitrate is believed to increase the extracellular K ion concentration and thus may depolarize the nerve and prevent repolarization. This change disrupts the ionic tubular membrane transmission and prevents sending pain signals to the brain until ionic concentrations restabilize.

The 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 over-the-counter and professionally applied desensitizing dentirice treatments for cervical sensitivity contain various chemicals that include denaturing salts (eg, strontium chloride, formaldehyde) and calcium-precipitating agents (eg, sodium fluoride, stannous fluoride, monofluorophosphate, oxalates) that are cost-effective, noninvasive, and applied at home or in office. Iontophoresis is another effective clinical technique that 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

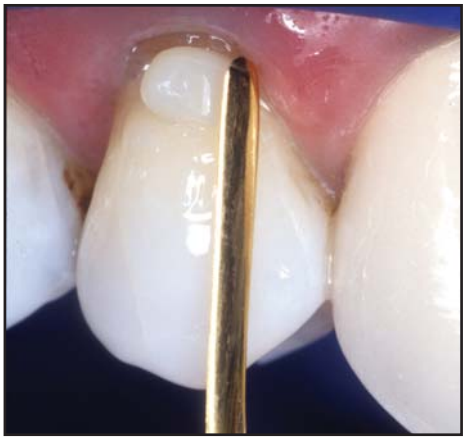


**Figure 3. A hybrid composite resin is used to restore the anatomic contour of the cervical lesion and eliminate sensitivity.**

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 1). A combination of iontophoresis followed by resin impregnation is one of the most effective clinical techniques for eliminating cervical tooth sensitivity.[14] Another clinical technique that utilizes an application of calcium hydroxide paste has been effective in relieving cervical sensitivity by increasing peritubular dentin mineralization.[15]

The final treatment strategy is to cover the exposed surface of the dentinal tubules by utilizing connective tissue graft procedures and/or dental restorations. The periodontal procedures include free, autogenous-mucosal grafts, subepithelial connective tissue grafts, a coronally advanced flap technique, guided periodontal tissue regeneration, and acellular dermal matrix grafts (Figure 2). Restorative methods can involve the use of:

- Conventional glass-ionomer cements;
- 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
- Porcelain-fused-to-metal crowns and bridges (Figure 3).[16]

## Conclusion

Management of dentin hypersensitivity begins with prevention and elimination of the predisposing factors associated with continued dentinal tubule exposure. Knowledge of the mechanism of dentinal sensation and etiology of cervical tooth sensitivity, and an understanding of the various treatment strategies may provide the clinician with alternative solutions for this challenging clinical dilemma. Part II of this series will discuss postoperative restorative sensitivity.

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