**Review Article**

# Dentine Hypersensitivity: A Review of its Management Strategies

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**Abstract:**
Dentine hypersensitivity or more colloquially "sensitive teeth" is a common problem encountered in routine clinical practice. The diagnosis of it, at times, is intriguing for even the most experienced clinician. The condition, though not alarming, is important diagnostically and therapeutically because it tends to disturb the routine day to day life of the patient, often becoming troublesome during eating, drinking, brushing, and in some cases mere breathing through mouth. This article attempts to discuss this topic in regards to its epidemiology, hypothesis regarding pain production, various etiological factors with special emphasis on the management strategies in a routine clinical scenario.

**Key Words**: Dentine bonding agent, dentine hypersensitivity, hydrodynamic theory, treatment

## Introduction

Dentine hypersensitivity is simply stated is the sensation produced by the stimulation of dentinal nerve endings. Dowell and Addy have defined dentinal hypersensitivity as a transient pain arising from exposed dentine, typically in response to chemical, thermal, tactile or osmotic stimuli, which cannot be explained as arising from any other form of dental defect or pathology.[1] It may affect as many as one in seven of adult patients attending for dental treatment[2] with the buccal surface of canines and premolars being affected the most. Hypersensitivity has its peak prevalence in young adult and then decreases with age.[3] It is reported to exhibits a slight female predilection.[4]

Hypersensitivity is caused by exposure of dentine due to loss of enamel or denudation of root[4] owing to varied reasons predominantly being gingival recession with advancing age, chronic periodontal disease, certain form of periodontal surgery, tooth brushing and chronic trauma from habits.[5]

The mechanism of pain production because of dentine hypersensitivity is still not clearly understood. Many hypotheses have been propounded of which the Hydrodynamic theory is the most accepted.[6,7]

The pain arising from exposed dentine varies in both frequency and severity. While, it may be sudden, sharp and of short duration in some in others it might be a persistent, dull or vague sensation in one or more tooth.[8]

Management of a patients suffering from dentine hypersensitivity begins with correct diagnosis. The diagnosis of this condition is usually arrived at, by ruling out other conditions that may present with similar feature in the orofacial region.

Various treatment agents have been used in treatment of dentinal hypersensitivity, namely, Silver nitrate, Zinc chloride, Formalin, Calcium hydroxide, Sodium fluoride with and without iontophoresis, Strontium chloride (SrCl2), Potassium nitrate, Dentin bonding agents Lasers, Periodontal surgery etc.

## Clinical Signs and Symptoms

The term "dentine hypersensitivity" has been variedly used in literature to describe a common painful dental condition. Several other terms, such as – dentine sensitivity, pulpal sensitivity, tooth sensitivity, cervical sensitivity, tooth hypersensitivity has been used synonymously.[9] The pain arising from exposed dentine, typically occurs in response to chemical, thermal, tactile and/or osmotic stimuli, and may be rapid in onset, sharp in character and of short duration or in some, while in others it may present as a dull or vague sensation.[8] Dentine hypersensitivity besides discomfort may indirectly pose other problems, especially, those associated with reduced oral hygiene in respect to gingival health both short and long term.[10]

## Pulpal Consideration of Dentinal Hypersensitivity

Nerve fibers originating from subodontoblastic plexus extend through odontoblastic layer, predentine, dentine to terminate as free nerve endings. These innervations are predominantly composed of A-delta- ($A\delta$) and C-fibers. It is postulated that $A\delta$-fibers are responsible for dentinal pain, and C- are predominantly nociceptors and are responsible for the pain

from external irritants that reach the pulp. Morphologically, nerve fibers may penetrate into the dentin as far as 150-200 μm only. A -fibers are said to be responsible for the sensitivity of dentin. They respond to stimuli that induce sharp pain in human teeth, for example, drilling of dentin and drying of dentin with air blasts. C-fibers are activated only when the stimuli reaches the pulp proper and are thought to be responsible for the pain which is chronic, dull and aching in nature.[11]

**Prevalence and Incidence**

Dentine hypersensitivity may affect as many as one in seven (approx. 14%) of adult patients attending for dental treatment.[2] The distribution of hypersensitive dentine shows that the buccal surface of canines and premolars are affected most.[3] Dababneh *et al*.[4] reports that the prevalence of dentine hypersensitivity ranges from 8-57%. Moreover, it has been reported to be considerably higher in periodontal patients, ranging between 72.5% and 98%. In general, a slightly higher incidence of dentine hypersensitivity is reported in females than in males, which may be a reflection of better reporting as females have a documented history of being more watchful about their oral health than their male counterparts. Von Trall *et al*.[9] concluded that root sensitivity occurs in approximately half of patients following scaling and root planning. The intensity of root sensitivity increases during a few weeks following non-surgical periodontal therapy, after which it decreases. Rees and Addy[12] in a cross-sectional study of dentine hypersensitivity demonstrated that the overall male:female ratio of 1:2.5, the highest numbers of patients with dentine hypersensitivity belonged to the 30-50 years age group.

**Etiology**

The etiology of dentinal hypersensitivity is multifactorial and as stated previously is attributed mainly to denudation of root surface or wear of the enamel causing exposure of the dentinal tubules and consequential stimulation of the nerve endings due to irritants- local or environmental.

Taani and Awartani[13] demonstrated the prevalence of dentine hypersensitivity related to the plaque. They examined dental hospital population and reported that 53% of the patients reported hypersensitive teeth. Women presented with a higher prevalence of hypersensitive teeth than men, with a significant statistical difference. Causes of gingival recession played a major role in dentine hypersensitivity in approximately 20% of the patients. Another subset of 29% patients had periodontal disease and 3.9% patients reported of having received periodontal treatment. Only 1.3% patients gave a history of previous orthodontic treatment or rehabilitation for trauma from occlusion.

Over-enthusiastic, tooth brushing has long been associated with gingival recession and sensitivity. Patients with dentine hypersensitivity exhibit a high, even excessive, standard of buccal tooth cleaning, and tooth sites affected show the lowest

plaque score. Clinical experience also indicates that areas of toothbrush abrasion/erosion appear free of plaque.[14] The difficulty may arise in the interpretation of available clinical evidence when individuals with poor oral hygiene and large amounts of buccal cervical plaque appear to exhibit dentine hypersensitivity. The probability exists that tubules have been opened, presumably due to plaque metabolism, and the production of organic acids leading to sensitivity.

Tooth surface lesions as attrition, abrasion, erosion, corrosion, and abfraction[15] by similar mechanism may also cause hypersensitivity.

**Mechanism**

Four theories[16,17] have been proposed to explain the mechanism of action of dentin hypersensitivity; the transducer theory, the modulation theory, the gate control theory and the hydrodynamic theory. Of these theories, the hydrodynamic theory is currently believed to be most responsible for the transmission of dentinal sensation

*Transducer theory*

This theory of dentinal sensation takes into consideration the "synaptic like" relationship between the odontoblastic processes. If the true synapse were present between these two elements to facilitate the transmission of dentinal sensation, then a neural transmitting substance such as acetylcholine would be expected in this area of the odontoblastic process and the predentin. There is no direct evidence for the presence of acetylcholine activity in the neural transmission in the pulp.[18]

*Modulation theory*

Modulation theory stated that, upon irritation, the odontoblast may become injured and release a variety of neurotransmitting agents, vasoactive and pain producing amines and protein which may modulate the nerve fiber action potentials by raising neuronal cyclic AMP levels through cell membrane adenylate cyclase receptors.[19]

*Gate control theory and vibration*

Irritation of dentine leads to neural stimulation. While, the larger myelinated fibers may adapt to the sensations over time, the smaller C-fibers may be non-accommodating to the stimulus. Thus, as the low intensity "pain gates" from the larger fibers are closed, the high intensity "pain gates" from the smaller fibers may remain open.[17] The gate control theory, however, does not adequately explain how pain responses from the dentin are transmitted and perceived by the nerve endings of the pulp.[18]

*Hydrodynamic theory*

Fish, referring to the interstitial fluid of the dentin as dental lymph, stated that pressure variation in the surrounding can bring about change in the fluid flow.[20] This change in fluid flow can cause dentinalgia according to Brannstrom and

Astrom, which subsequently brings about deformation in the odontoblast, or its process and elicits a response perceived as pain by the stimulation of associated "mechanoreceptor like" nerve ending.[21]

This is most aptly exhibited by a blast of air which causes the dentin to dehydrate leading to sensitivity. Moreover, when dry absorbent paper is applied to exposed dentin it caused pain, but no pain was elicited using wet paper.[21-23] This occurs due to the outward movement of the dentinal fluid into the dehydrating source (i.e., air blasts or absorbent paper) which stimulate the "mechanoreceptor" of the odontoblast causing pain.

The perception of acute thermal stimulation can also be explained by the hydrodynamic theory. The coefficient of thermal expansion of dentinal tubule fluid is about 10 times that of the tubule wall. Therefore, heat applied to dentin will result in an expansion of the fluid and conversely cold will result in a contraction of the fluid, with both creating an excitation of the mechanoreceptor[24,25] and eliciting pain.

Bender[26] found that pain was produced when sugar or salted solutions were placed in contact with exposed dentin. When the irritant was rinsed or washed away, the discomfort subsided. This again can be explained by dentin tubule fluid movements along the osmotic gradient towards that of higher concentration eliciting a pain response. Furthermore, support to this hypothesis was provided by the fact that when iso-osmotic solutions were applied, no pain was perceived.

**Differential Diagnosis**[27]

It is worthy of note that although many individuals are seen to have exposed dentine, not all experience symptoms. For those who do, it is important to eliminate other possible causes of patient's pain by considering a differential diagnosis such as:
1. Chipped teeth
2. Fractured restoration
3. Pupil response to caries
4. Pulpal response to restorative treatment
5. Cracked tooth syndrome
6. Palatogingival groove

**Management**

Dowell *et al.*[27] explained the management of the patient suffering from dentine hypersensitivity into three different categories:

*Diagnosis*

a. The identification of a zone or zones of exposed dentin which when suitably stimulated produces pain.
b. Identification of those factors which exposed the dentine and could be related to the opening of the contained dentinal tubules.
c. Elimination of other causes of pain, either as separate entities or coexisting with dentine hypersensitivity.

*Prevention*

Removal of etiological factors importantly:
a. Advice on correct non-traumatic tooth brushing techniques.
b. Dietary counseling with respect to the intake and frequency of acidic fruits and beverages, particularly as they relate to the times of tooth brushing.
c. Elimination of other habits or disease which cause dentine exposure.

*Therapy*

Therapy can be conveniently considered on the basis of extent and severity. For isolated problems therapy is largely professionally delivered and should be directed toward varnishes, adhesives, adhesive filling materials, and cervical restorations, for general hypersensitivity suitably formulated toothpaste including those containing fluoride or strontium and fluoride mouth rinses. In more severe and intractable cases consideration should be given to root canal therapy.

Treatment of hypersensitivity essentially involves the reduction of the functional diameter of the tubule to limit its fluid movement. This can be variously achieved by:
1. Formation of a smear layer by burnishing the exposed root surface.
2. Topical application of agents that form insoluble precipitates within the tubules.
3. Impregnation of tubules with plastic resins.
4. Applications of dentin bonding agents/adhesives the sealing the dentinal tubules.

Grossman[28] suggested the following requirements for a satisfactory desensitizing material:
1. Non-irritant to the pulp
2. Relatively painless upon application
3. Easily applied
4. Rapid in action
5. Effective for a long time
6. Without staining effects
7. Consistently effective.

Trowbridge and Silver[29] lists of various treatment agents that have been utilized for professional applications in desensitization of hypersensitive dentin:
- Cavity varnishes
- Anti-inflammatory agents
- Treatment that partially obturate dentinal tubules
  - Burnishing of dentin
  - Silver nitrate
  - Zinc chloride potassium ferrocyanide
  - Formalin
- Calcium compounds
  - Calcium hydroxide
  - Dibasic calcium phosphate

- Fluoride compounds
  - Sodium fluoride
  - Sodium silicofluoride
  - Stannous fluoride (SnF2)
- Iontophoresis
- Strontium chloride (SrCl2)
- Potassium oxalate
- Restorative resins
- Dentine bonding agents.

## Cavity Varnishes

It has been observed that dentin becomes insensitive if a thin film of a varnish is applied topically over the exposed dentin surface, but this is reported to provide only temporary relief. The varnish application is painless and adheres to the teeth for hours or days. The coating effect induces immediate relief of pain.

## Corticosteroids (Anti-inflammatory Agents)

Corticosteroids are also reported to be effective in relieving tooth hypersensitivity. It has been reported that when a cavity liner consisting of 1% prednisolone with 2.5% para-chlorophenol, 2.5% m cresyl acetate, and 50% gum camphor was extremely effective in preventing post-restoration thermal sensitivity.

## Treatment that Partially Obturate Dentinal Tubules

### Burnishing of dentine

Burnishing of dentin with a toothpick or orangewood stick results the formation of a smear layer that partially occludes the dentinal tubules. According to Pashley et al.[30] burnishing reduces the fluid movement across dentin by a factor of 50-80%. It has also been shown that dry orangewood stick is superior to glycerin alone or glycerin in combination with NaF in reducing dentin permeability.

### Silver nitrate

Burchard[31] described silver nitrate as a desensitizing agent having its action by coagulation or destroying the dentinal protoplasm. This has been told to effect mainly on the protein constituent of odontoblastic processes. Greenhill and Pashley[32] found that as compared with controls, $AgNO_3$ followed by 10% formalin decreased the hydraulic conductance by 59.2%, whereas $AgNO_3$ alone reduced it by 47.4%. Other protein precipitants such as zinc chloride and phenol had a similar effect on hydraulic conductance. Markowitz and Kim[33] demonstrated that silver nitrate is not used frequently because it stains the teeth permanently.

### Potassium nitrate

In 1974, a potassium nitrate-containing dentifrice was shown to be relieving to 35 dental patients with hypersensitivity.[34] Kim[35] suggested that the active moiety may be potassium ions which reached the dentinal nerve endings diffusing through the tubules and modified the exchange of sodium and potassium

in nerves. In a controlled study, Tarbet et al.[36] reported that 5% potassium nitrate paste reduced sensitivity to cold by 65% as compared with 20% for a placebo.

### Zinc chloride-potassium ferrocyanide

Gottlieb[37] developed the zinc chloride-potassium ferrocyanide impregnation method for controlling sensitivity due to exposed root surfaces and post-cavity preparation. In this procedure, a 40% solution of aqueous zinc chloride was rubbed onto the surfaces of sensitive teeth and allowed to remain for 1 min, followed by vigorous rubbing with a 20% aqueous potassium ferrocyanide until an orange, curdy crystalline precipitate is formed covering the dentine (Greenhill and Pashley).[32]

### Dibasic sodium citrate

Dibasic sodium citrate, formulated into a pluronic F-124 containing dentifrice (Protect), is a product currently recognized by ADA as being safe and effective for the treatment of dental sensitivity.[38]

### Formalin

Grossman[28] based on 108 cases treated by him and followed up for 7 years recommended formalin as the desensitizing agent of choice in treating anterior teeth due to its non-staining nature, unlike sodium nitrate. Formalin in the concentration of 40% (full strength) has been used for topical application by means of cotton pellets or orangewood stick.

## Calcium Compounds

### Calcium hydroxide

Calcium hydroxide $Ca(OH)_2$ has been popularly used for the management of hypersensitivity particularly after root planning. It functions by probably blocking dentinal tubules or promoting peritubular dentin formation.[39] Levin et al.[40] found the efficacy of calcium hydroxide to be 98% in immediately relieving cervical hypersensitivity. Furthermore, Green et al.[41] reported that calcium hydroxide application was consistently effective in relieving cervical hypersensitivity for a period of 3 months studied.

### Dibasic calcium phosphate

Hiatt and Johansen compared the effectiveness of burnishing CaHPO4 into sensitive areas of roots with only burnishing and reported a significant relief in 93% of patients studied as compared to the controls who received burnishing only.[42]

## Fluoride Compounds

### Sodium fluoride

Lukomsky[43] was probably one of the first to suggest the use of topical fluoride for the treatment of hypersensitive dentin. Clement,[44] Hoyt and Bibby[45] using 33.3% NaF and found that NaF was very effective in reducing dentinal hypersensitivity in subjective, non-controlled studies. Tal et al.[46] demonstrated that because dentinal fluid has a high concentration of calcium and phosphate ions, application of NaF to dentin leads to

precipitation of CaF2 crystals which functions to decrease the radius of the dentinal tubules. However, fluoride is lost fairly rapidly which explains the limited effectiveness of topical fluoride application in managing sensitivity on a long-term basis.[47]

### Sodium silicofluoride
Bhatia[48] proposed that the application of a saturated solution (0.6%) of sodium silicofluoride for 5 min was superior to a 2% NaF solution in managing cervical sensitivity.

### Stannous fluoride
Blong et al.[49] found that a 0.4% SnF2 gel used for the protracted duration of 4 weeks was effective in controlling sensitivity. Furthermore, Blank and Charbeneau[50] advocated burnishing a 10% solution of SnF2 into sensitive root areas. Thrash et al.[51] reported that topical application of 0.717% aqueous SnF2 provided immediate relief from sensitivity.

### Iontophoresis
Manning[52] described an iontophoretic device which would work electrophoretically to desensitize dentin using 2% NaF with iontophoresis. The exact mechanism, by which fluoride iontophoresis produce desensitization is not known, but the following hypothesis has been proposed. According to Lefkowitz et al.[53] application of electric current to the dentin leads to the formation of reparative dentine. Another possible explanation is that the electrical current produces paresthesia by altering the sensory mechanism of pain conduction. A third alternative explanation of iontophoretic desensitization is the rise in fluoride concentration in the dentinal tubules following fluoride iontophoresis leading to microprecipitation of calcium fluoride which may function to block hydrodynamically mediated pain-inducing stimuli.[54] Iontophoresis, by itself, produced no favorable effects. They speculated that the effects were from fluoride ion deposition and secondary dentin formation. Zodak et al.[55] reported that fluoride concentrations in surface and subsurface layers of iontophoretically-treated teeth were significantly greater than those treated by topical application of NaF only.

### Strontium Chloride
About, 10% SrCl2 used as the desensitizing agent which have been widely available. Hodge et al.[56] showed that strontium strongly absorbs to calcified tissues. A report[57] stated that SrCl2 combined with the biocolloids of teeth and thus exerted a favorable effect, the product Sensodyne toothpaste was formulated with SrCl2 hexahydrate, and since strontium ions are reactive with many commonly used dentifrice ingredients, it was necessary to formulate all unreactive dentifrice base subsequently, a patent was issued to Rosenthal and assigned to Block Drug Co. Inc. Sensodyne with 10% SrCl2, which was introduced nationally in 1961. Ross[58] reported the result of a study conducted among 78 office patients, 73% of subject reported complete relief of the condition after using 10% SrCl2

dentifrice. Alleviation of symptoms usually occurred within 1 month of use. Uchida et al.[59] reported that after 7 weeks of treatment, a 10% SrCl2 6H$_2$O dentifrice group demonstrated a 75.5% reduction in hypersensitivity in post-periodontal surgery patients, whereas a control group had only a 34.2% reduction. Dowell et al.[27] suggested that the formation of strontium apatite complex involves the exchange of strontium with calcium in the dentin, subsequently resulting in re-crystallization.

### Oxalates
The oxalates (as 6% ferric oxalate, 30% dipotassium oxalate, and 3% monohydrogen-monopotassium oxalate) are increasing in popularity off-lately particularly among periodontists as an inexpensive, easy and well-tolerated way of managing sensitivity. Pashley et al.[30] reported that topical application of 3% W/V half-neutralized monohydrogen-monopotassium oxalate (KHOx) is a highly effective means of reducing dentin permeability.

### Dentine Bonding Agent
Considering the hydrodynamic theory, Brannstrom and Nordenvall[60] felt that if dentinal fluid flow could be reduced by impregnating it with resin (the unfilled "bonding agent") dentinal pain could be controlled. Unfilled resins produce little adverse pulpal inflammation. After applying these to dentin, Brannstrom et al.[61] obtained "immediate and lasting blockage of sensibility" in 20 patients studied from 2 to 12 months. This is in agreement with Dayton et al.[62] who tested various unfilled resins in 44 teeth.

The dentin surface is first cleaned with EDTA and an antibacterial detergent. The area is dried with a continuous air spray for 20 s. Then the resin is applied, which enters into the tubules through the capillary and reactive forces. The superficial surface of the resin is then removed with a cotton pellet before it hardens. Results administrated an immediate and protracted blockage of sensitivity on most surfaces for between 1 month and 1 year.[61] Inoue et al.[38] states dentine bonding agents are available as three step, two step, one step system depending on how the three-step etching, priming and bonding to the tooth substrates are accomplished or simplified. They act by occluding the dentinal tubules, thus reducing the dentinal hypersensitivity. Among the first bonding agent to be used dall'Orologio et al.[63] found that single application of Gluma 3 primer and Gluma 2000 successfully eliminated or reduced dentinal sensitivity over a 6 months period.

### Laser
Laser therapy has been recommended by Kimura et al.[64] to treat dentine hypersensitivity with effectiveness ranging between 5.2% and 100%. The mechanism of laser treatment for dentine hypersensitivity is still not quiet understood.[64] According to Pashley[65] laser, may cause coagulation and protein precipitation of the plasma in the dentinal fluid or by alter the nerve fiber activity. Another opinion held by McCarthy et al.[66] is that the reduction

in dentine hypersensitivity could be the result of alteration of the root dentinal surface by physically occluding the dentinal tubules.

**Periodontal Surgery**

The periodontal procedures which can be carried out to cover exposed root surfaces like lateral pedicle grafts, free gingival grafts, connective tissue grafts, etc. However, recession coverage requires careful treatment planning and understanding of the defect to be treated. Though these procedures cover the exposed dentine, soft tissue grafting for management of sensitivity is not considered as a very predictable treatment strategy.

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