# **Dentin Hypersensitivity**

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#### Introduction

n one form or the other, pain is the major reason for patients to visit the dentist. Most often, this may be related to dental caries, traumatic injuries etc. which can be correctly diagnosed and treated successfully. However, there is a small percentage of cases where the exact reason for sensitivity cannot be easily identified or satisfactorily managed. These patients complain of a sharp pain in response to various stimuli like heat, cold, chemicals etc. This condition is called dentin or tooth hypersensitivity. It usually affects adults in the age group of 30 to 40 years most frequently. The response to a stimulus varies due to difference in pain tolerance, environmental factors and psychology of the patient.

Dentin hypersensitivity can fit the criteria of several pain terms described by Merskey (1979) for the International Association for the study of pain (IASP). Pain is described as an unpleasant sensory and emotional experience associated with actual or potential tissue damage. Careful clinical examination of the patients of dentin hypersensitivity may reveal no obvious pathology.

Though many theories have been proposed & several treatment options suggested, dentin hypersensitivity is still a vexing clinical problem to diagnose and manage.

#### **Definition**

# International Workshop on Dentin Hypersensitivity (1983)

Dentin hypersensitivity is characterised 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 form of dental defect or pathology.

### **Prevalence**

Dentin hypersensitivity is a fairly

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common condition with between 8 and 35% of the population being affected. Adults in the age group of 20 to 50 years are the most affected with a peak between 30 & 40 years. In general, a slight higher incidence of dentin hypersensitivity is reported in females than in males. The reduced incidence of dentin hypersensitivity in older individuals reflects age changes in dentin and the dental pulp.

#### **Intra Oral Distribution**

Hypersensitivity is most commonly noted on buccal cervical zones of permanent teeth. Although all tooth type may be affected, canines and premolars in either jaws are the most frequently involved. Regarding the side of mouth, in right handed toothbrushers, the dentin hypersensitivity is greater on the left sided teeth with equivalent contralateral teeth.

## **Etiology & Predisposing Factors**

The primary underlying cause for dentin hypersensitivity is **exposed dentin tubules**. Dentin may be exposed by two processes:-

- 1. By loss of covering periodontal structures (gingival recession)
- 2. By loss of enamel

The most common clinical cause for exposed dentinal tubules is gingival recession.

Various factors which can cause gingival recession are-

- 1. Inadequate attached gingiva
- 2. Prominent roots
- 3. Toothbrush abrasion
- 4. Oral habits resulting in gingival laceration i.e. traumatic tooth picking, eating hard foods.
- 5. Excessive tooth cleaning
- 6. Excessive flossing
- 7. Overzealous tooth cleansing habits
- 8. Gingival recession secondary to specific diseases i.e. NUG, periodontitis, herpetic gingivostomatitis
- 9. Crown preparation

The recession may or may not be associated with bone loss .If bone loss occurs, more dentinal tubules get exposed. When gingival recession occurs, the protective layer of root dentin i.e. cementum gets abraded or eroded away. This leaves the exposed underlying dentin which consists of protoplasmic projections of odontoblasts within the pulp chamber. These cells contain nerve endings and when distributed nerves depolarize and this is interpreted as PAIN.

Gingival recession removal of cemental layer exposure of dentin and thus dentinal tubules depolarization of nerve endings of odontoblasts PAIN

# Depolarization Of Nerves Will Be Presented As Pain

Reasons for continued dentinal tubular exposure are-

- Poor plaque control i.e. acidic bacterial byproducts
- Excess oral acids i.e. soda, lime juice, bulimia, swimming pool chlorine.
- · Cervical decay
- Toothbrush abrasion

The other reason for exposure of dentinal tubules is due to loss of enamel.

#### Causes of Loss of Enamel

- Attrition caused by exaggerated occlusal functions like bruxism.
- Abrasion from dietary components or improper brushing technique.
- Abfraction
- Erosion caused with environmental or dietary components particularly acids.
- Since dentinal tubules get sclerosed own their own and plug themselves up in the oral environment.
- Treatment should focus on eliminating factors associated with continued dentinal exposure.

## **Neurophsiology of Teeth**

• Myelinated a Delta Fibers Seems to be Responsible for dentin hypersensitivity.









- Mechanism of Pain Transmission
- Theories of Dentin Hypersensitivity
- 1. Direct innervation theory / neural theory
- 2. Odontoblast deformation theory transducer mechanism
- 3. Hydrodynamic theory

# **Direct Innervation Theory / Neural Theory**

This theory attributes to the direct activation of nerve endings within the dentinal tubules. These nerve signals are then conducted along the parent primary afferent nerve fibers in the pulp into the dental nerve branches & then into brain. This theory considered that entire length of tubules contains free nerve endings. However, histologic studies have shown that nerve fibers are present only in the predentin and inner dentinal tubules but do not extend all the way upto DEJ which is the most sensitive area of the dentin. These nerves are also absent in areas like the root dentin which is also very sensitive. Another finding that disputes this theory is that when pain inducing substances like potassium chloride, acetylcholine & histamine are applied to exposed dentin, they fail to elicit a painful response. Due to these shortcomings, this theory is no longer accepted.

# Odontoblast Deformation Theory / Transducer Mechanism

This theory assumed that odontoblasts extend to the periphery. The stimuli initially excite the process or body of the odontoblast. The membrane of odontoblast may come into close apposition with that of nerve endings in the pulp or in the dentinal tubule & the odontoblast transmit the excitation of these associated nerve endings. However in the most recent study, Thomas(1984) indicated that the odontoblastic process is restricted to the inner third of the dentinal tubules. So, this theory also fell into disfavour as research has shown that the odontoblastic processes extend only partly through the dentin & not upto the DEJ. Also several invitro studies have shown that the odontoblast membrane

potential is too low to permit transduction. Another valid finding was that there was no demonstrable neurotransmitters like acetylcholine in the neural transmission of pulp. Hence, this theory is also no longer valid.

## **Hydrodynamic Theory**

Given by Brannstrom M in 1962. This is the most accepted mechanism to explain dentin hypersensitivity. Structurally, the dentin has 3,00,000 dentinal tubules per square mm. They are filled with dentinal fluid which is the intercellular fluid of the pulpal connective tissue. In a vital tooth, there is a constant, slow outward movement of this fluid through the dentinal tubules. The hydrodynamic theory states that whenever exposed dentin is stimulated by tactile, chemical, thermal or osmotic stimuli, there is rapid movement of the dentinal fluid either towards the pulp or outwards. This can cause:-

- 1. Direct stimulation of low threshold A delta fibers in the pulp.
- Indirect stimulation of A delta fibers in the pulp by displacing the odontoblastic cell bodies. Such rapid displacement of dentinal fluid in thousands of dentinal tubules at the same time produces a cumulative effect & this cause hypersensitivity and presented as PAIN by the patient.

There is a lot of evidence to indicate that movement of fluid in the dentinal tubules is the basic event in the dentinal hypersensitivity. Studies have shown that stimuli like cold, sweets, air blasts etc. cause a rapid outflow of dentinal fluid causing pain. For this theory to be accepted as valid, teeth presenting with hypersensitivity must have dentinal tubules which are open at the dentin surface and patent till the pulp. Scanning electron microscope & dye penetration studies have demonstrated that there is a greater number and wider diameter tubules in hypersensitive dentin as compared to non sensitive dentin. Thus, presently the hydrodynamic theory of dentinal sensitivity is well accepted.

Mathews et al (1994) noted that stimuli such as cold causes fluid flow away from the pulp produces more rapid & greater pulp nerve responses that those such as heat which causes an inward flow. This certainly would explain the rapid & severe response to cold stimuli as compared to the slow dull response to heat.

\*\*The dehydration of dentin by airblasts or absorbent paper causes outward fluid movement and stimulates the mechanoreceptor of the odontoblast, causing pain. Prolonged air blast causes formation of protein plug into dentinal tubules, reducing the fluid movement and thus decreasing pain.

\*\*The pain produced when sugar or salt solution are placed in contact with exposed dentin can also be explained by dentinal fluid movement. Dentinal fluid is of relatively low osmolarity which have tendency to flow towards solution of higher osmolarity i.e. salt or sugar solution.

### **Clinical Features**

**PAIN** is the primary symptom of hypersensitive dentin. The patient usually experiences as short, sharp pain in response to heat, cold, tactile stimuli and sweet or sour foods. The pain is considered to be an exaggerated response to the normal pulp dentin complex & is only felt on application of the external stimulus. However, there is no lingering discomfort once the stimulus is removed. Thus, the clinical symptoms of dentin hypersensitivity are similar to those acute reversible pulpitis.

#### **Diagnosis**

When a patient presents with the symptoms of dentin hypersensitivity, the first step is to diagnose the condition accurately. This requires a careful history & clinical examination.

### **Case History**

Elicit the following information:-

- 1. The history & nature of pain (sharp, dull etc.)
- 2. The number & location of sensitive teeth and whether it is the same teeth that are always involved.



- 3. The intensity of pain (mild, moderate or severe)
- 4. The stimuli which initiate the sensitivity.
- 5. The frequency & duration of sensitivity.
- Other related events like history of recent restorative or periodontal treatment, dietary habits, any home bleaching or over the counter bleaching regimes etc.

### **Clinical Examination**

This should include the following tests & observations:-

- 1. Evidence of dentin exposure (gingival recession, loss of enamel)
- 2. Sensitivity or pain or tactile examination of the suspected teeth.
- 3. Percussion sensitivity
- 4. Pain lingering after the stimulus is removed
- 5. Vitality tests to rule out pulpal involvement.
- Radiographic examination to check for caries, pulpal or periodontal involvement
- 7. Signs of fractured, leaky or poor restorative margins.

# Difference between Dentin Hypersensitivity & Pulpal Hypersensitivity

In case of dentin hypersensitivity, patient's ability to locate the source of pain is very good where as in case of pulpal pain, it is very poor. The pain is intensified by thermal changes, sweet or sour foods. Intensity of pain is usually mild or moderate. The pain can be duplicated by hot or cold application or by scratching the dentin. The pulpal pain is explosive, intermittent & throbbing & can be affected by hot or cold.

## **Differential Diagnosis**

A number of dental conditions are associated with dentin exposure and may produce the same symptoms. Such conditions include:-

- 1. Chipped tooth i.e. fractured enamel exposing dentin
- 2. Fractured restorations
- 3. Post restorative sensitivity
- 4. Dental caries

### 5. Cracked tooth syndrome

6. Bleaching sensitivity

## **Treatment Strategies**

Hypersensitivity can resolve without the treatment or may require several weeks of desensitizing agents before improvement is seen. Treatment of dentin hypersensitivity is challenging for both patient & the clinician mainly for two reasons:-

- 1. It is difficult to measure or compare pain among different patients.
- 2. It is difficult for the patient to change the habits that initially caused the problem.

# **Management of Dentin Hypersensitivity**

it is well known fact that hypersensitivity often resolves without treatment. This is probably related to the fact that dentin permeability decrease spontaneously because of occurrence of natural processes in the oral cavity.

# Natural Processes Contributing To Desensitization

- 1. Formation of repairative dentin by the pulp.
- 2. Obturation of tubules by the formation of mineral deposits (dental sclerosis)
- 3. Calculus formation on the surface of the dentin.

# **Two Principal Treatment Options**

- 1. Plug the dentinal tubules preventing the fluid flow
- 2. Desensitize the nerve, making it less responsive to stimulation.

All the current treatment modalities address there two options.

Treatment of dentin hypersensitivity can be divided into:-

- 1. Home care with dentifrices
- 2. Inoffice treatment procedures
- 3. Patient education

#### **Home Care With Dentifrices**

Dentifrices has been defined as a substance used with a toothbrush to aid in cleaning the accessible surfaces of the teeth. **Strontium Chloride Dentifrices** 

It acts by penetrating the tubules and forming strontium apatite which occludes the exposed dentinal tubules.10% strontium chloride dentifrices have been found to be effective in relieving the pain of tooth

## hypersensitivity.

## **Potassium Nitrate Dentifrices**

- 1. They are used to block the pulpal sensory nerves from transmitting pain impulses.
- 2. Potassium ions from potassium nitrate toothpastes can easily pass through dentin to the pulp.
- 3. Here, they depolarize the sensory nerve endings present close to the odonto-blasts thus preventing the transmission of impulses to the brain.
- 4. The desensitizing effect of potassium nitrate toothpastes requires two applications a day for a minimum of two weeks.
- 5. 5% potassium nitrate dentifrices have been found to alleviate pain related to tooth hypersensitivity.

#### **Fluoride Dentifrices**

Sodium monofluorophosphate dentifrices are the effective of treating tooth hypersensitivity.

## **Commonly Used Dentifrices In India**

- Emoform dentifrice contains 5% potassium nitrate
- Sensodent-k contains 5% potassium nitrate.
- Colgate sensitive contains potassium nitrate & sodium monofluorophosphate.
- Ra thermoseal contains 5% potassium nitrate & 0.7% sodium monofluorophosphate.
- Close up active gel contains sodium fluoride. Approved by fdi.
- Hydent k contains 5% potassium nitrate, 0.7% sodium monofluorophosphate & 0.3% triclosan
- Caviseal- f contains 5% potassium nitrate, 0.7% sodium monofluorophosphate & 0.3% triclosan.

# **Inoffice Treatment Procedures**

- 1. Varnishes
- 2. Corticosteroids
- 3. Treatment which partially obturate dentinal tubules are:
- a. Burnishing of dentin
- b. Silver nitrate
- c. Zinc chloride potassium ferrocyanide
- d. Formalin



- e. Calcium compounds:-
  - dicalcium hydroxide
  - dibasic calcium phosphate
- f. Fluoride compounds
  - sodium fluoride
  - sodium silicofluoride
  - stannous fluoride
- g. Iontophoresis
- h. Strontium chloride
- i. Potassium oxalate
- j. Tubule sealants
  - restorative agents
- a. Gic
- b. Composite resin
  - dentin bonding agents
- k. Miscellaneous
  - Lasers (Co2, nd: YAG, er: YAG, He: Ne)

# **Inoffice Treatment Procedures Rationale of Therapy**

According to hydrodynamic theory of hypersensitivity, a rapid movement of fluid in the dentinal tubules is capable of activating intradental sensory nerves. Therefore, treatment of hypersensitive teeth should be directed towards:-

- 1. Reducing the anatomical diameter of the tubules.
- 2. Obliteration of the tubules
- 3. To surgically cover the exposed dentinal tubules so as to limit fluid movement.

Treatment options to reduce the diameter of dentin tubules can be-

- 1. Formation of a smear layer by burnishing the exposed root surface ( smear layer consists of small amorphous particles of dentin, minerals & organic matrix-denaturated collagen)
- 2. Applications of agents that form insoluble precipitates with in the tubules.
- 3. Impregnation of tubules with plastic resin
- 4. Application of dentin bonding agents to seal off the tubules.
- 5. Covering the exposed dentinal tubules by surgical means.

It must be recognized that simple procedure may not be consistently effective in the treatment of hypersensitivity, the dentist must be familiar with alternative methods of treatment. Prior to treating sensitive root surfaces, hard/ soft deposits should be removed from the teeth. Root planing on sensitive dentin may cause considerable discomfort, in this case, teeth should be anaesthetized prior to treatment & teeth should be isolated & dried with warm air.

#### **Varnishes**

They act by forming a barrier over the exposed dentin. This lowers hypersensitivity as it reduces dentin hypersensitivity to a considerable extent. Cavity varnishes containing copal, rosin etc. provide only a temporary relief as they can readily dissolve in the oral environment but fluoride varnishes like DURAPHAT provide a more sustained relief.

### Corticosteroids

Corticosteroids containing 1% prednisolone in combination with 25% parachlorophenol, 25% methacrylacetate and 50% gum camphor was found to be effective in preventing postoperative thermal sensitivity. The use of corticosteroids is based on the assumption that hypersensitivity is linked to pulpal inflammation. Hence, more information is needed regarding the relationship between these two conditions. The use of coricosteroids is controversial.

## **Partial Obliteration of Dentinal Tubules**

**Burnishing of Dentin**: is done with a toothpick or orangewood stick results in the formation of a smear layer which partially occludes the dentinal tubules & thus resulting in decreased hypersensitivity.

Formation of Insoluble Precipitates to Block Tubules: certain soluble salts react with ions in the tooth structure to form crystals on the surface of dentin. To be effective, crystals should occur in 1-2 minutes & the crystals should be small enough to enter the tubules & must also be large enough to partially obliterate the tubules

### **Calcium Oxalate Dihydrate Crystals**

These crystals are formed when potassium oxalate is applied to dentin.

These crystals are very effective in reducing permeability.

#### Silver Nitrate

It has the ability to precipitate protein constituents of odontoblast processes, thereby partially blocking the tubules.

## Zinc Chloride Potassium Ferrocyanide

When applied forms precipitate, which is highly crystalline & covers the dentin surface.

## Formalin 40%

It is topically applied by means of cotton pellets or orangewood sticks on teeth. It has been proposed by Grossman in 1935 as desensitizing agent of choice in treating anterior teeth because unlike silver nitrate, it does not produce stain.

## **Calcium Compounds**

Have been popular for many years for the treatment of hypersensitivity. The exact mechanism of action is unknown but evidence suggests that:-

- 1. It may block dentinal tubules
- 2. May promote peritubular dentin formation
- 3. On increasing the concentration of calcium ions around nerve fibers, may results in decreased nerve excitability. So, calcium hydroxide might be capable of suppressing nerve activity.

A paste of calcium hydroxide and sterile distilled water applied on exposed root surface & allowed to remain for 3-5 minutes, can give immediate relief in 75% of the cases .Dibasic calcium phosphate when burnished with round toothpick forms mineral deposits near the surface of the tubules & found to be effective in 93% of patients.

## Fluoride Compounds

Lukomsky (1941) was first to propose sodium fluoride as desensitizing agent, because dentinal fluid is saturated with respect to calcium & phosphate ions . Application of NaF leads to precipitation of CaF crystals, thus reducing the functional radius of the dentinal tubules.

### **Acidulated Sodium Fluoride**

Concentration of fluoride in dentin treated with acidulated sodium fluoride is



found to be significantly higher than dentin treated with sodium fluoride.

#### Sodium Silicofluoride

Silicic acid forms a gel with the calcium of the tooth & produces an insulating barrier. Thus, application of 0.6% sodium silicofluoride is much more potent than 2% solution of sodium fluoride as desensitizing agent.

#### **Stannous Fluoride**

10% solution of stannous fluoride forms dense layer of tin & fluoride containing globular particles blocking the dentinal tubules .0.4% stannous fluoride is also an effective agent, however, requires prolonged use (upto 4 weeks) to achieve satisfactory results.

## Fluoride Iontophoresis

Iontophoresis is a term applied to the use of an electric potential to transfer ions into the body for therapeutic purposes. The objective of fluoride iontophoresis is to drive fluoride ions more deeply into the dentinal tubules that cannot be achieved with topical application of fluoride alone. The iontophoresis unit consists of an adapted plastic tip placed around the tooth. This serves as a negative electrode while the positive electrode is placed on the patients's face or arm. A 2% solution of sodium fluoride is applied on the exposed dentin and this is tranferred deep into dentin on activation of the unit. Fluoride iontophoresis has been reported to provide a more long term relief from hypersensitive dentin. However, this method is expensive as it needs special equipment and often requires more than one application.

## **Strontium Chloride**

Studies have shown that topical application of concentrated strontium chloride on an abraded dentin surface produces a deposit of strontium that penetrates dentin to a depth of approximately 10 to 20 microns & extend into dentinal tubules.

#### Oxalates

Oxalates are relatively inexpensive, easy to apply & well tolerated by the patients. Potassium oxalate & ferric oxalate solution make available oxalate ions that can react with calcium ions in the dentinal fluid to insoluble calcium oxalate crystals that are deposited in the aperture of the dentinal tubules.

## **Dentin Resin and Adhesives**

The objective in employing resins & adhesives is to seal the dentinal tubules to prevent pain producing stimuli from reaching the pulp. Several investigators have demonstrated immediate & enduring relief of pain for periods of upto 18 months

following treatment. Although not intended for treatment of generalised areas of root sensitivity, this can be effective method of treatment when other forms of therapy have failed. GLUMA is a dentin bonding system that includes glutaraldehyde primer & 35% HEMA (hydroxyethylmethacrylate) It provides an attachment to dentin that is immediate & strong. GLUMA has been found to be highly effective when other methods of treatment failed to provide relief.

#### Lasers

Kimura et al (2000) reviewed of dentin hypersensitivity by lasers. The lasers used for the treatment of dentin hypersensitivity are divided into two groups:

- Low output power (low level) lasers:
  He:Ne and Gallium/ Aluminium/
  Arsenide diode lasers.
- **2. Middle output power lasers :** Nd: YAG & CO2 lasers.

They occlude the dentinal tubules by producing local changes around the exposed dentin. They are also reported to produce changes in the central pulp neuron. However, lasers are expensive and not available for routine use

#### **Patient Education**

- 1. Dietary Conselling
- 2. Tooth Brushing Technique
- 3. Plaque Control

## **Dietary Counselling**

Dietary acids are capable of causing loss of tooth structure, thereby removing cementum and resulting in opening of the dentinal tubules. Consequently, dietary counselling should focus on the quality and frequency of acid intake and intake occurring in relation to tooth brushing. Any treatment may fail if these factors are not controlled. A written diet history should be obtained on patients with dentinal hypersensitivity in order to advise those concerning eating habits. Because of the presence of a smear layer on dentin, teeth are not usually sensitive immediately following scaling and root planning. However, removal of the smear layer may result from exposure to certain components of the diet. Studies have shown that citrus fruit juices, apple juice and yoghurt are capable of dissolving the smear layer. Because of the loss of dentin is greatly increased when brushing is performed immediately after exposure of the tooth surface to dietary acids. Patient should be cautioned against brushing their teeth soon after ingestion of citrus food.

# **Tooth Brushing Technique**

Because incorrect toothbrushing appears to be an etiologic factor in dentin

hypersensitivity, instruction about proper brushing techniques can prevent loss of dentin and the hypersensitivity

### **Plaque Control**

Saliva contains calcium and phosphate ions and is therefore able to contribute to the formation of mineral deposits within exposed dentinal tubules. The presence of plaque may interfere with this process, as plaque bacteria, by producing acid, are capable of dissolving any mineral precipitates that form, thus opening tubules.Professional interest in the causes and treatment of dentin hypersensitivity has been evident in the dental literature for approximately 150 years or more. In generalized hypersensitivity (involving many teeth), desensitizing toothpastes containing potassium nitrate, fluoride or strontium chloride must be employed for a few weeks. In cases of severe hypersensitivity not responding to any of these methods of treatment, endodontic therapy may be necessary as the final option.

#### **Conclusion**

Dentin hypersensitivity is a matter of growing concern in the present times due to the increased life expectancy and consequent longer retention of natural teeth by patients. Hypersensitivity arises following loss of enamel or root denudation which exposes the underlying dentin. The hydrodynamic theory is the most accepted mechanism to explain this phenomenon. The ultimate goal in treating this condition is to provide immediate and longlasting relief of the associated painful symptoms. For this, the clinician must pay attention to diagnosis, prevention and selection of the appropriate treatment modality.

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