DENTINE HYPERSENSITIVITY AND ITS MANAGEMENT

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ABSTRACT

The objective of this review is to inform practitioners about dentin hypersensitivity (DHS); to provide a brief overview of the prevalence, diagnosis, etiopathogenesis,

and clinical management of dentin hypersensitivity and to discuss technical approaches to relieve sensitivity. This clinical information is described in the context of the underlying biology.

The authors used PUBMED, Google Scholar, Research gate to find relevant English-language literature published in the period 1999 to 2021. The authors used combinations of the search terms “dentin”, “tooth”, “teeth”, “hypersensitivity”, “desensitizing”. Abstracts and also full text articles to identify studies describing, prevalence,distribution,etiology, clinical features, mechanisms of action of various desensitizing agents.

Key Words; Dentine Hypersensitivity, Etiology, Desensitizing agent, Diagnosis

INTRODUCTION

The term dentine hypersensitivity has been used for many decades to describe a common painful condition of the teeth. Despite this, there are many gaps in our knowledge concerning dentin hypersensitivity [1]. It is perhaps not surprising therefore that one can still have sympathy with statement made in 1987 by Johnson and Co-workers that dentine hypersensitivity is an Enigma, being frequently encountered yet ill understood [2].

DEFINITION

Dentine hypersensitivity is defined as short, sharp pain arising from exposed dentine in response to stimuli, typically thermal, evaporative, tactile, osmotic or chemical and which cannot be ascribed to any other dental defect or pathology [3].

PREVALENCE

- 15-18% of the general populations [4]
- 72-98% - in periodontal patients {2]
- Age incidence: - 20-40 years peak [3]
- Gender: females > males
- Differences in diet - favoring healthy but often acidic foods and drinks
- Either periodontal disease and / or periodontal treatment predisposed to dentine hypersensitivity, presumably through both having effects on dentine and gingival recession [3,5].

DISTRIBUTION

- Buccal cervical area of teeth
- Most commonly affected are canines and Ist premolars, then incisor and 2nd premolars, least often molars.

- Significantly greater proportions of left side tooth sensitivity compared with their right contralateral tooth types [5,7]

ETIOPATHOGENESIS

Two processes need to occur in other to give rise to dentine hypersensitivity.

- Lesion localization
- Lesion initiation

LESION LOCALIZATION

This involves the exposure of the dentin as a result of various mechanical or chemical processes such as, Attrition, Erosion, Abfraction, Abrasion, Pocket reduction surgery, Tooth preparation for crown, Excessive flossing and Secondary to periodontal diseases.[6,8]

LESION INITIATION

This involves the actual mechanism that results in the sensitivity felt by the patient. It is on this ground that different theories have been postulated as to how the response is brought about.[2,9]

THEORIES OF HYPERSENSITIVITY

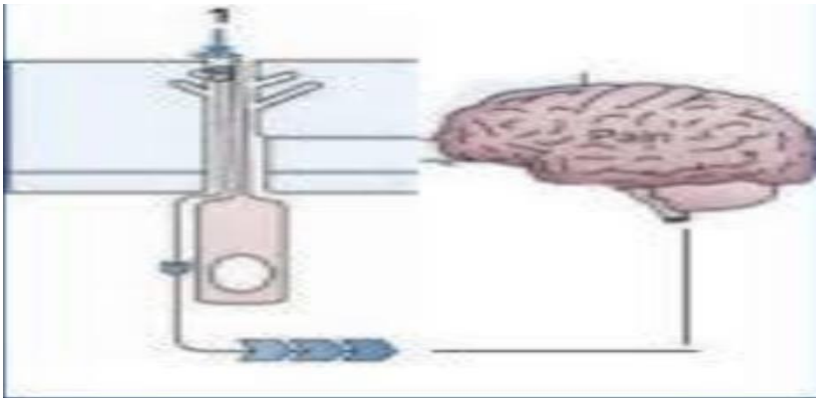
- Direct neural theory

- Odontoblasts receptor theory
- Transducer theory
- Gate control theory
- Fluid or Hydrodynamic theory

DIRECT NEURAL THEORY

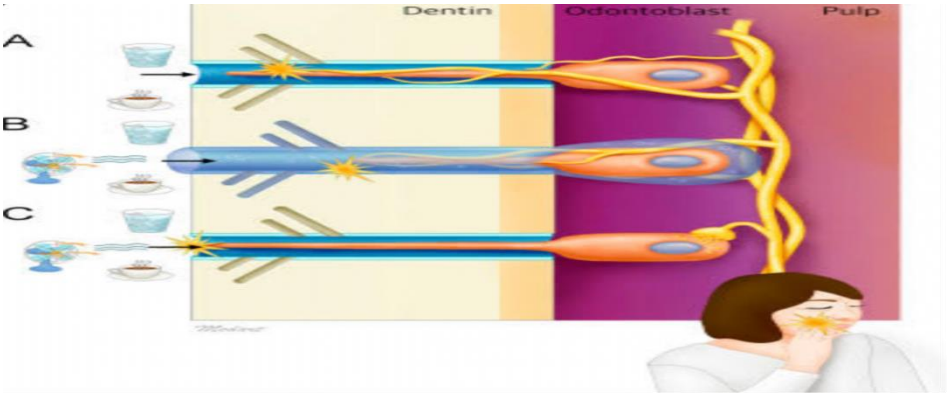
The dentin contains nerve endings that respond when it is stimulated.

The pulp is well innervated, especially below the odontoblasts (the plexus of rack show) and that some nerves penetrate a short distance in to some tubules. Whether these intratubular nerves are involved in dentin sensitivity is not known. No evidence has been found for nerves in the outer dentin, which is most sensitive [3,5].



ODONTOBLAST RECEPTOR THEORY

This mechanism explains dentin sensitivity considers the odontoblasts to be a receptor cell. This attractive concept has been considered, abandoned and reconsidered for many reasons. It was once argued because the odontoblasts is of neural crest origin and it retains an ability to transduce and propagate an impulse was not accepted, since there is no neurotransmitter vesicles in the odontoblast process to facilitate the synapse or synaptic specialization [5,10].



TRANSDUCER THEORY

This theory of dentinal sensation takes into consideration the synaptic -like relationship between the terminal sensory nerve endings and odontoblastic process. If a true synapse were present between these two elements to facilitate the transmission of dentinal sensations, then a neural transmitting substance such as acetylcholine could be expected, but there is no direct evidence of its presence [11,12].

GATE CONTROL THEORY AND VIBRATION

When the dentin is irritated, for example, by cavity preparation, all of the pulpal nerves become activated from the vibrations.

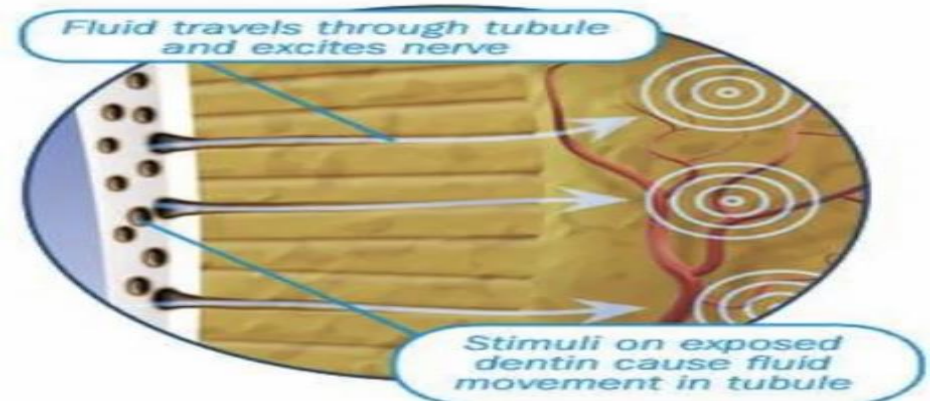
The larger myelinated fibers may accommodate to the sensations. The smaller C-fibers may tend to be maintained and not adjust to the stimulus.

Thus, as the low-intensity pain gates from the larger fibers are closed, the high-intensity "pain gates" from the smaller fibers are enhanced.

However, the gate theory does little to explain how pain responses from the dentin are transmitted and perceived by the nerve endings of the pulp-only how they may be centrally interpreted [9,13]

HYDRODYNAMIC THEORY

Rapid shifts of the fluids within the dentinal tubules, following stimulus application, result in activation of sensory nerves in the inner dentin region of the tooth [4].



MANAGEMENT OF DHS

- History
- Examination
- Investigation
- Diagnosis
- Treatment

IMPORTANT STEPS TO FOLLOW

• Correct diagnosis of dentin hypersensitivity including a patient's history and a brief clinical examination

- Identification of etiologic and predisposing factors
- Differential diagnosis, to exclude all other dental conditions
- If present, treatment of all conditions with symptoms similar to dentin hypersensitivity

• Removal or minimization of etiologic and predisposing factors [6,8,13]

HISTORY

- Sex: Females> Males

• Age: Middle age group of 30-40 years affected mostly Presenting complain: Pain, shocking tooth/teeth

• Character: Sharp

• Duration: Chronic, short lasting

• History of excessive tooth brushing, flossing and oral habit should be checked.

• Past dental Hx: like vital tooth bleaching, periodontal procedures 54%- 55%

• Medical condition that results in toothwear lesion, bulemics and gastrointestinal reflux disease

• Social practices: Intake of acidic foods and drinks. [1-3,13,14]

EXAMINATION

• Evidence of tooth wear lesion (attrition, abrasion, erosion)

• Gingival recession

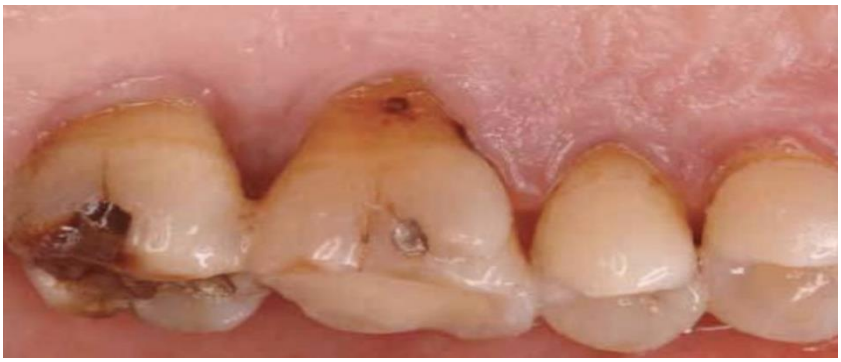
• Radiographs

• Caries diagnostic devices

• Percussion testing

• Assessment of occlusion

- Bite stress tests. [12,14]



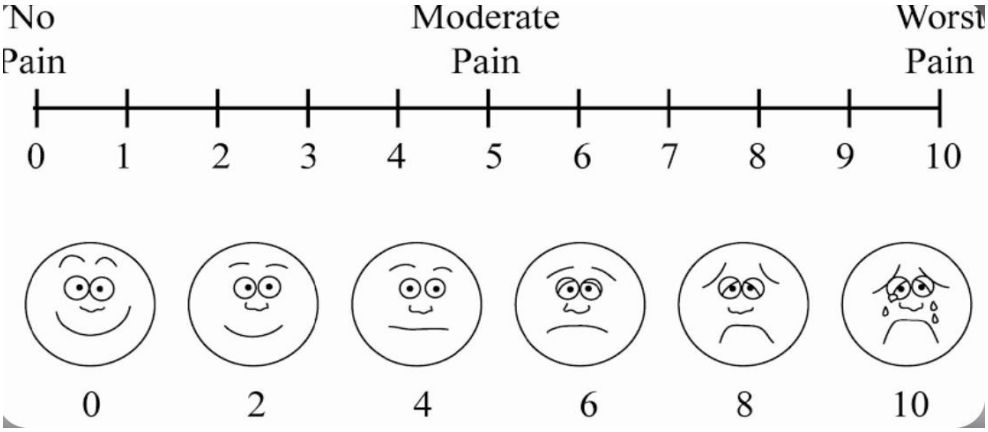
SUBJECTIVE EVALUATION:

VERBAL RATING SCALES:

Keele 1948 described four point scale grading pain as slight, moderate, severe and agonizing. Verbal rating scales (VRS) offer a choice of words that may not represent pain experience with significant precession for all patients [15]

VISUAL ANALOGUE SCALES:

A visual analogue scale (V AS) is a line 10 cm in length, the extreme of the line representing the limits of pain a patient might experience from external stimulus. No pain at one end and most severe pain at the other end. Patients are asked to place a mark on the 10 cm line which indicates the intensity of their current level of sensitivity or discomfort following application of stimuli. VAS can give only a one -dimensional assessment of pain and as such cannot distinguish between the sensory, intensity and affective aspects of pain [15].



OBJECTIVE EVALUATION:

• Mechanical (tactile) stimuli - explorer, constant pressure

probe, Mechanical pressure stimulators,

• Chemical (osmotic) stimuli - hypertonic solutions. e.g.

sodium Chloride, glucose, sucrose, and calcium Chloride.

• Electrical stimulation - electrical pulp testers

• Evaporative stimuli - cold air blast, air thermal system, air Jet stimulator,

• Thermal stimuli- electronic threshold measurement device, cold water testing, heat, Ethyl chloride, ice stick, thermo -electric Devices (e.g.: bio mat thermal probe) [13,15].

DIFFERENTIAL DIAGNOSIS

• Cracked tooth syndrome

- Fractured restorations
- Restorations left in traumatic occlusion
- Chipped teeth
- Dental caries, root caries
- Postoperative sensitivity
- Pulpal response to restorative treatment or certain materials
- Marginal leakage of restorations
- Pulpitis, pulpal status
- Vital bleaching procedures. [11]

DEFINITIVE DIAGNOSIS

Dentin hypersensitivity is always a diagnosis of exclusion, it is confirmed only after all possible other conditions have been diagnostically eliminated. A simple clinical method of diagnosing DHS includes a jet of air or using an exploratory probe on the exposed dentin, in a mesio-distal direction. [12]

TREATMENT OF DHS

- Classification of treatment options

- Ideal properties of a desensitizing agent
- Methods of treatment

CLASSIFICATION OF DESENSITIZING AGENTS

- Base on mode of administration:
  - i. at-home
  - ii. in-office
- Base on mode of action
  - i. Nerve desensitization
  - ii. Protein precipitation
  - iii. Plugging dentinal tubules
  - iv. Dentine adhesive sealers
  - v. Lasers
  - vi. Homeopathic medication
- Others"
  - i. gingival graft. [12-16]

On the basis of mechanism of action

A. Nerve desensitization - Potassium nitrate

B. Protein precipitation - Gluteraldehyde, Silver nitrate, Zine chloride, Strontium chloride hexahydrate

C. Plugging dentinal tubules - Strotium Acetate, Sodium fluoride, Stannous fluoride, Potassium oxalate, Calcium phosphate, Calcium carbonate, Bio active glasses (SiO-PO-CaO-_Na2O)

D. Dentine adhesive sealers- Fluoride varnishes, Oxalic acid and resin, Glass ionomer cements, Composites, Dentin bonding agents

E. Periodontal soft tissue grafting

F. Anti-inflammatory- corticosteroids

G. Crown placement and restorative materials

H. Lasers - GAlAs (galium-aluminium-arsenide laser), Erbium-YAG laser, He:Ne laser

I. Homeopathic medication - Propolis

Ideal properties of a desensitizing agent

- Rapidly acting with long-term effects,

- Non-irritant to pulp,
- Painless
- Easy to apply, and
- Should not stain the tooth. [1-3,12,16]

TREATMENT METHODS

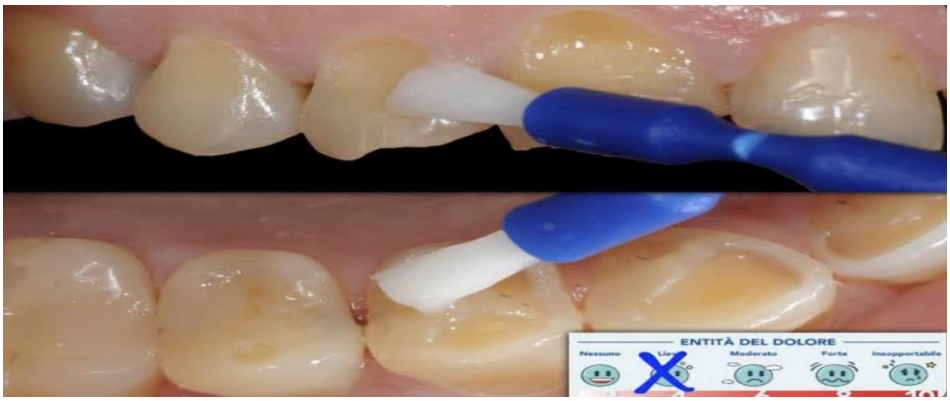
- ***Adhesive composite resin, GIC and dentin bonding agents***

Indicated when the exposed sensitive root surface has surface loss due to abrasion, erosion and/or abfraction leaving a notching of the root.

The adhesive resins can seal the dentinal tubules effectively by forming a hybrid layer. Newer bonding agents modify the smear layer and incorporate it into the hybrid layer.[3,12,16]

B. Fluoride varnish: (e.g. sodium fluoride, stannous fluoride)

Fluorides decrease the dentinal permeability by precipitation of calcium fluoride crystals inside the dentinal tubules. 5% sodium fluoride varnish is painted over exposed root surfaces. [1,12,16]



C. Oxalate.

Precipitates and occlude the open dentinal tubules.

Oxalate reacts with the calcium ions of dentine and forms calcium oxalate crystals inside the dentinal tubules as well as on the dentinal surface.

Topical application of 3% potassium oxalate can reduce DHS post periodontal surgery. Avoid using with tray for a long time as it can cause gastric irritation. [12,16]

D. Desensitizing dentifrices.

Desensitizing ingredient in toothpastes is potassium nitrate.

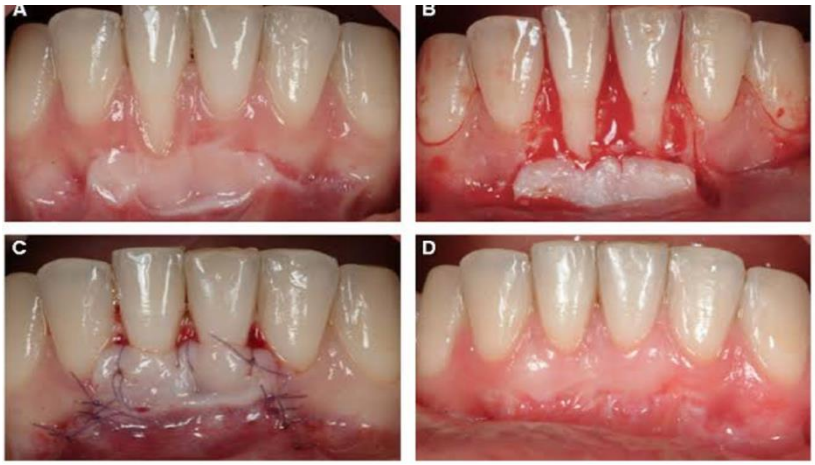
It acts by penetrating the A-fibres of the nerves reducing its excitabilty

For a potassium nitrate toothpaste it must contain 5% potassium nitrate.

This takes up to two weeks to show any effectiveness. [12,16]

E. Gingival graft

This is indicated when recession is progressive, aesthetics is a major concern and when the treatment is not responding to convention treatment, including coronally reposition flaps, lateral sliding graft, free gingival and connective tissue graft. [4,12,16]



F. Anti-inflammatory

Topical application 0.5% solution of prednisolone on exposed root surface will induce remineralisation leading to tubular occlusion.[13,16]

Others.

Fluoride 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. [10]

PREVENTION

• Ensure proper toothbrush consistency, ensure proper brushing technique, highly abrasive tooth powder or pastes should be avoided.

• Avoid over-brushing with excessive pressure or for an extended period of time or excessive flossing

• Avoid brushing immediately after taking acidic drinks

• Avoid over-polishing exposed dentin during

stain removal.

• Avoid over-instrumenting the root surfaces during scaling and root planing, particularly in the cervical area of the tooth

• Avoid violating the biologic width during restoration placement, as this may cause recession.

• Patient with gastrointestinal reflux disease should be properly managed by the physician and fabrication of occlusal splint to cover the affected areas, to prevent their contact with the acids.[1-6,11,14]

CONCLUSION

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. The least invasive, most cost-effective treatment is the use of an effective desensitizing toothpaste.

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