ECRONICON OPEN ACCESS

EC DENTAL SCIENCE
Review Article

Dentinal Hypersensitivity

Mohammed Hany Ahmad Fouad[1]*, Burair Muhammed Alameer[2], Fawaz Mohammed Sait[2], Wed Ali Albogami[2], Abdulelah Abdullah alharbi[2], Bashaer Abdualziz Alharbi[2], Afnan Hashim Balkhy[2], Mohammed Mansour Alghamdi[3], Israa Jawad Alali[3], Ahmed Ali AlGhamdi[4], Hasan Shafiq Barri[4], Abdulhadi Abdulrahman Alzughaibi[4], Emad Ali Alhrbei[5], Mohammed Saeed M Alqahtani[6] and Raghad Abdulhamid AlMunif[7]

[1]*Cairo University, Saudi Arabia*

[2]*Ministry of Health, Saudi Arabia*

[3]*Al-Farabi Private College, Saudi Arabia*

[4]*King Abdulaziz University, Saudi Arabia*

[5]*Tabih university, Saudi Arabia*

[6]*Armed Forces Hospital Southern Region, Saudi Arabia*

[7]*Imam Abdulrahman Bin Faisal University, Saudi Arabia*

***Corresponding Author**: Mohammed Hany Ahmad Fouad, Cairo University, Saudi Arabia. Number: 05JD0323.

E-mail: abdelghanymohamedhany@gmail.com

Received: December 06, 2021; **Published:** December 31, 2021

Abstract

Introduction: The complex sensory innervation of dental pulp gives rise to a complicated mechanism of pain transmission. Various stimuli, including thermal, chemical, or tactile, on the exposed dentine may lead to increased pain which is referred to as dentinal hypersensitivity. The unique arrangement of nerve fibers accounts for the increased sensitivity of teeth. The management of hypersensitivity aims at making dentinal tubules impermeable and controlling the nerves inside the pulp, which will bedew the stimulus acting on the pulp.

Aim of Work: This review aims at discussing the various theories of dentinal hypersensitivity and the different management techniques.

Methodology: This review is comprehensive research of PUBMED and Google Scholar from the year 1986 to 2021.

Conclusion: The complex nature of the nervous system in the dental pulp makes the mechanism of tooth pain an enigma to the dentists. In recent times, a better understanding of the nervous system has enabled practitioners to explore various theories behind dentinal sensitivity. Other mechanisms like the disruption of TRP and activation of ATP help in the management of dentine sensitivity. Pulpal inflammation also alters the sensitivity mechanism and causes peripheral sensitization-increased knowledge of the theories behind hypersensitivity help to develop better treatment options.

Keywords: *Dentine; Dentinal Hypersensitivity; Odontoblasts; Algoneurons; Sensitization*

Introduction

The complex sensory innervation of dental pulp gives rise to a complicated mechanism of pain transmission. Various stimuli, including thermal, chemical, or tactile, on the exposed dentine may lead to increased pain which is referred to as dentinal hypersensitivity [1]. Exposure to dentine is a fairly common occurrence and can happen in patients with attrition, erosion, or abrasion. During dentinal exposure, the outer covering of enamel or cementum is lost, which gets dentine in direct contact with the oral cavity and its stimulus. Dentine expo-

sure can also be a result of developmental defects where the enamel and cementum covering the dentine fail to meet each other resulting in the exposure of dentine. The etiology of dentinal hypersensitivity seems to be multifactorial, which arises as a result of open dentinal tubules causing a direct linkage between the oral cavity and dental pulp. The openings of the dentinal tubules seem to be more patent in areas of sensitive dentin, which causes a closer contact between the stimuli and dental pulp [2].

When the dentinal tubules are exposed for a long duration, there seems to be some occlusion by smear layer or pellicle on the aperture, but in severe cases of hypersensitivity, this occlusion might still not be helpful. The management of hypersensitivity aims at making dentinal tubules impermeable and controlling the nerves inside the pulp, which will bedew the stimulus acting on the pulp. This can be done by complete or partial annihilation of dentinal tubules or changes in the sensory activity of the pulp, or, in some cases, both [2].

The unique arrangement of nerve fibers accounts for the increased sensitivity of teeth. Nociceptive neurons make a larger volume in the dental pulp, which leads to increased sensation of pain. The pulpal stroma is innervated with Unmyelinated C fibers, and the myelinated fibers innervate the dentinal tubules. As a result, any stimulus applied on the tooth surface results in a pre-sensation of pain [3].

Mechanism of tooth pain and dentinal sensitivity

As discussed above, the complex arrangement of neurons in the pulp leads to a complex mechanism of tooth pain. A dentinal Hypersensitivity is a form of odontalgia. Tooth pain can be further explained in detail by the following six mechanisms (Table 1) [4].

Tooth Pain (Odontalgia)	Dentinal Hypersensitivity [5]
	Inflammation of the Pulp [6]
	Trauma from occlusion causing apical sensitization [7]
	Infection or inflammation leading to peripheral sensitization [7]
	Central sensitization which includes referred pain [8]
	Psychological Disorder [9]

Table 1: *Mechanism of Pain [4].*

Dentinal Sensitivity is one of the most commonly encountered dental pain. A better understanding of the mechanism of dentinal sensitivity leads to a better diagnosis and management of the condition. This review aims at highlighting the different theories of dentinal sensitivity and the published literature. The different theories of dentinal sensitivity are described in figure 1.

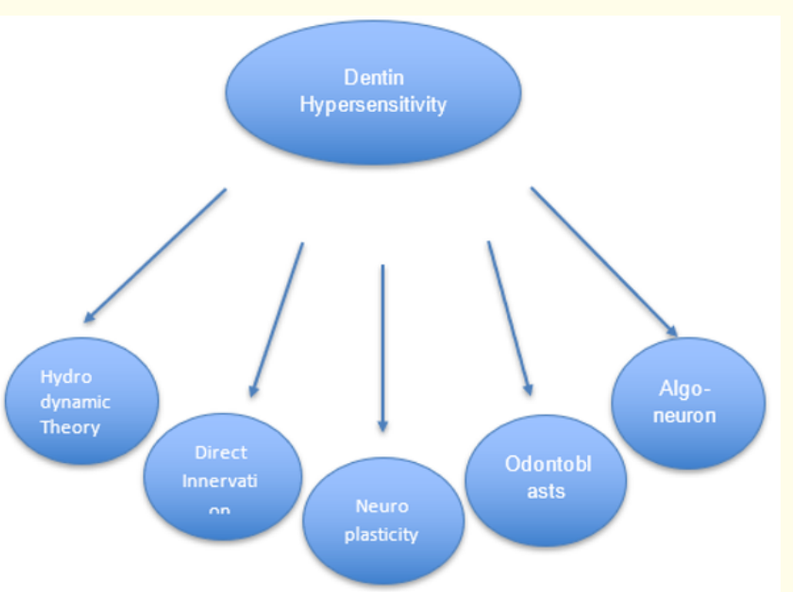

Figure 1: *Theories of Dentinal Hypersensitivity [4].*

Hydrodynamic theory

Brannstorm., *et al.* Put forward the hydrodynamic theory, which stated that neural discharge happens due to movement in the fluid present between the dentinal tubules on stimulation with changes in temperature, osmotic pressure, and physical changes; these changes stimulate a baroreceptor which leads to neural discharge [10]. In 1982, Lija., *et al.* Conducted a study where they studied the odontoblasts and nerves under infected cavities and their relation to dentin sensitivity. They reported that although infected caries does not have any nerves, the increased sensitivity to stimulation was a result of outward movement of fluid in the dentinal tubules, which activated nociceptive nerve terminals in the adjacent inflamed pulp [11]. They also concluded that the mechanism behind the hydrodynamic theory is the close proximity between the odontoblasts and nerves. In a similar manner, when the teeth experience a change in thermal conditions, there is an expansion of the dentinal fluid, which causes neural excitement resulting in pain. Osmotic stimulus refers to an excess of sugar, salts, or acid, which results in flow within the dential fluid. Physical stimulation, as described by Brannstorm, would be a result of air blasting, probing, and absorbent material, which causes an outward flow of dentinal fluid. Temporary restorative material Cavit (ESPE, GmbH., Seefeld, Germany) has also shown a large amount of fluid displacement in the dentine [12].

Theory of direct innervation or neural theory

The neural theory claims that instead of fluid movement, which activates the neurons, as stated in the hydrodynamic theory, the stimulus acting on the tooth surface directly activates the nerve fibers in the dentine [13]. The dental pulp is very densely innervated with afferent trigeminal axons, including myelinated A mostly present in the crown portion of the tooth, including and unmyelinated C fibers which are present in the pulp. The extent of the nerve fibers in the tooth has been examined by Tidmarsh under electron microscopy, where he studied and extracted the tooth by freeze-fracture method and concluded that nerve fibers extend till the dento-enamel junction. C fibers are polymodal in nature and do not respond to hydrodynamic stimulation. Their ability to release neuropeptides to mark inflammation contributes to its role in dentinal sensitivity. The polymodal nature of C fibers tends to increase their activation threshold and respond to various inflammatory markers like bradykinin and histamine. Osmotic pressure applied by hypertonic solutions tends to have an impact on A-fibers in the interdental nerves [15].

Neuroplasticity and sensitivity

Inflammation of the pulp alters the neurophysiology of the nerve, which in turn changes the sensitivity of A-delta nerve fibers. Pulpal inflammation also reduces the pain occurring because of sensitivity as the threshold of nociceptors is reduced due to the production of neuropeptides and neuroplasticity [16]. Changes in the pulpal pathology are seen following a prolonged pain stimulus; this inflammation ends up sensitizing the nerves to such an extent that even the smaller shifts in fluid activated the nerve. In a study performed by Park., *et al.* They studied the contribution of thermo-transient receptor potential (TRP) in tooth pain and concluded that once the TRP is activated by dental neurons, it causes toothache related to thermal stimuli [17]. In cases of dental caries involving or approaching the pulp, the nociceptive nerve endings produced neuroplasticity which led to the induction of neuropeptides. Following a dental injury, there is increased production of sensory nerve fibers as a result of the production of Nerve Growth factors (NGF). NGF also causes regulation of tetrodotoxin-resistant sodium current, and signal-regulated kinase plays a prime role in the induction of pain [18]. Multiple sodium channel isoforms in the peripheral afferents cause hyperexcitability, which might explain why some patients complain of pain in teeth that have been endodontically treated. This expression of inflammatory markers and plasticization of the central and peripheral nervous system make the tooth susceptible to hypersensitivity [19].

Odontoblastic theory

In 2011, Karim., *et al.* Came up with the concept that odontoblasts act as sensory cells [20]. cold response in human teeth is mediated by odontoblasts and fibroblasts. The arrangement of odontoblasts in dentin is in such a manner that it allows the transfer of sensory stimulation to an underlying pulp [21]. Odontoblasts have the ability to express and TRP channels. TRP channels present on the odontoblasts get excited by osmotic changes, which cause movement of the dentinal fluid. Odontoblasts are often associated with the expression of sodium, potassium, and calcium channels that play a major role in transduction [21]. The sodium channels associated with odontoblasts have been associated with the transduction of pain [5]. ATP release from odontoblasts has also been accounted for in dentinal pain [22].

Algoneurons

The term "Algoneurons" was coined by Fried., *et al.* Which basically described the low Threshold mechanoreceptors [LTM]. He said that the afferent nerves in the dentine were Low threshold mechanoreceptors rather than nociceptors. These highly receptive nociceptors turn into LTM because of sensitization of the central and peripheral nervous systems [24].

Management of dentinal hypersensitivity

The treatment options available for the management of Hypersensitivity are widespread. Treatment options can be divided into various categories, as mentioned in table 2.

Management of Dentine Hypersensitivity	
Technique	**Procedure**
Nerve Desensitization	Potassium Nitrate
Anti-Inflammatory	Corticosteroids
Plugging of dentinal tubules	Ions/Salts Sodium Fluoride Sodium monoflurophosphate Calcium Hydroxide Stannous Flouride Protein precipitants Dentine sealers Soft tissue grafting in Periodontium Placement of Crown or restoration Lasers

Table 2: *Management techniques for Dentinal Hypersensitivity [2].*

Plaque control is the mainstay of treatment options. Plaque accumulation leads to demineralization of the tooth structure that causes increased sensitivity of the tooth. Other management techniques include the application of desensitizing agents on the tooth surface. The desensitizing agents are often expected to be painless, should not cause discoloration, and should have a long-lasting effect post-application. Other management techniques are discussed in brief in this review article [24].

Desensitization of nerve by potassium nitrate

Tarbet., *et al.* In the 1980s gave solid evidence in favor of potassium nitrate being an effective desensitizing agent. Over time, Potassium nitrate has been used in various concentrations and consistencies as a desensitizing agent. At a concentration of 5%, it has been used in abrasive toothpaste, which showed evident improvement than the control group in 4 weeks. In-gel consistency 5-10% concentration is used [24].

Plugging of dentinal tubules

Sodium fluoride

Fluoride toothpaste and other concentrated fluoride solutions have shown to be very effective against hypersensitivity. There are two mechanisms for the management, firstly, increase in the resistance of dentin against decalcification because of fluoride, and secondly, precipitation of fluoride components on the dentinal tubules [2].

Stannous fluoride

Stannous fluoride forms a mineral content that makes a calcific barrier that occludes the dentinal tubules, thereby reducing sensitivity.

Calcium hydroxide

The calcium ions bind with the radical protein present in the dentine, thereby occluding the dentinal tubule [25].

Iontophoresis

Fluoride or oxalate ions are attracted to the tooth surface through the application of current through a negative electrode [26].

Lasers

Carbon Dioxide and Nd: YAG lasers have been used to reduce hypersensitivity with a huge success rate. In a study conducted in 2019, Nd: Yag laser occluded the dentinal tubules as effectively as sodium phosphosilicate paste.[28]. Nd: YAG laser ends up occluding almost 90% of the dentinal tubules. CO2 lasers have also shown a good occluding efficiency which lasts for around six months [27].

Periodontal surgery

Soft tissue grafting like lateral sliding grafts, coronally repositioned grafts, tend to cover the exposed area of the tooth surface, thereby reducing hypersensitivity [27].

Novel management techniques for hypersensitivity

- Casein-phosphopeptide-amorphous calcium phosphate (CPP) -(ACP) – It increases the availability of calcium and phosphate by ceasing their dissociation

- Pro-Arginine Technology- The positive charge on arginine helps it to bind with the negatively charged dentinal tubule and occludes it.

- Nanotechnology. A nanoparticle is defined as a particle with a size less than 100nm. These particles join together to form a nanocluster. Bioactive glass and nanohydroxyapatite nanoparticles are generally used in the case of hypersensitivity [28].

Conclusion

The complex nature of the nervous system in the dental pulp makes the mechanism of tooth pain an enigma to the dentists. In recent times, a better understanding of the nervous system has enabled the practioners to explore various theories behind dentinal sensitivity. Other mechanisms like disruption of TRP and activation of ATP help in the management of dentine sensitivity. Pulpal inflammation also alters the sensitivity mechanism and cause peripheral sensitization. Increased knowledge of the theories behind hypersensitivity help to develop better treatment options.

Bibliography

1. Kawashima N and Okiji T. "Odontoblasts: Specialized hard-tissue-forming cells in the dentin-pulp complex". *Congenital Anomalies* 56.4 (2016): 144-153.

2. Bartold P M. "Dentinal hypersensitivity: a review". *Australian Dental Journal* 51.3 (2006) 212-218.

3. Story GM., *et al.* "ANKTM1, a TRP-like channel expressed in nociceptive neurons, is activated by cold temperatures". *Cell* 112 (6 (2003): 819-829.

4. Aminoshariae A and Kulild JC. "Current Concepts of Dentinal Hypersensitivity". *Journal of Endodontics* 47.11 (2021): 1696-1702.

5. Hossain MZ., *et al.* "The role of transient receptor potential (TRP) channels in the transduction of dental pain". *International Journal of Molecular Sciences* 20.3 (2019): 526.

6. Lepinski AM., *et al.* "Bradykinin levels in dental pulp by microdialysis". *Journal of Endodontics 26*.12 (2000) 744-747.

7. Duffin PS., *et al.* "Nonodontogenic Odontalgia Referred from the Temporal Tendon: A Case Report". *Journal of Endodontics* 46.10 (2020)1530-1534.

8. Khan AA., *et al.* "Measurement of mechanical allodynia and local anesthetic efficacy in patients with irreversible pulpitis and acute periradicular periodontitis". *Journal of Endodontics* 33.7 (2007): 796-799.

9. Scully C., *et al.* "Munchausen's syndrome: oral presentations". *British Dental Journal* 178.2 (1995): 65-67.

10. Brännström M. "The Hydrodynamics of the Dentine; Its Possible Relationship to Dentinal-Pain". *International Dental Journal* 22.2 (1972): 219-227.

11. Lilja J., *et al.* "Dentin sensitivity, odontoblasts and nerves under desiccated or infected experimental cavities. A clinical, light microscopic and ultrastructural investigation". *Swedish Dental Journal* 6.3 (1982) 93-103.

12. Widerman FH., *et al.* "The physical and biologic properties of Cavit". *The Journal of the American Dental Association* 82.2 (1971): 378-382.

13. Närhi M., *et al.* "Neurophysiological mechanisms of dentin hypersensitivity". *Proceedings of the Finnish Dental Society. Suomen Hammaslaakariseuran Toimituksia* 88 (1992): 15-22.

14. Tidmarsh BG. "Contents of human dentinal tubules". *International Endodontic Journal* 14 (1981): 191-196.

15. Vongsavan N and Matthews B. "The relationship between the discharge of intradental nerves and the rate of fluid flow through dentine in the cat". *Archives of Oral Biology* 52.7 (2007): 640-647.

16. Närhi M., *et al.* "The neurophysiological basis and the role of inflammatory reactions in dentine hypersensitivity". *Archives of Oral Biology 39* (1994): S23-S30.

17. Park CK., *et al.* "Functional expression of thermo-transient receptor potential channels in dental primary afferent neurons: implication for tooth pain". *Journal of Biological Chemistry* 281.25 (2006): 17304-17311.

18. Sun S., *et al.* "Nav1. 7 via promotion of ERK in the trigeminal ganglion plays an important role in the induction of pulpitis inflammatory pain". *BioMed Research International* (2019).

19. Arslan H., *et al.* "Regenerative endodontic procedures in necrotic mature teeth with periapical radiolucencies: a preliminary randomized clinical study". *Journal of Endodontics* 45.7 (2019): 863-872.

20. El Karim IA., *et al.* "Human odontoblasts express functional thermo-sensitive TRP channels: implications for dentin sensitivity". *Pain*® 152.10 (2011): 2211-2223.

21. Magloire H., *et al.* "Topical review. Dental pain and odontoblasts: facts and hypotheses". *Journal of Orofacial Pain* 24.4 (2010): 335.

22. Hossain MZ., *et al.* "The role of transient receptor potential (TRP) channels in the transduction of dental pain". *International Journal of Molecular Sciences* 20.3 (2019): 526.

23. Shibukawa Y., *et al.* "Odontoblasts as sensory receptors: transient receptor potential channels, pannexin-1, and ionotropic ATP receptors mediate intercellular odontoblast-neuron signal transduction". *Pflügers Archiv-European Journal of Physiology* 467.4 (2015): 843-863.

24. Fried K., *et al.* "The paradox of pain from the tooth-pulp: Low-threshold "algoneurons?" *Pain* 152.12 (2011): 2685.

25. Frechoso SC., *et al.* "Evaluation of the efficacy of two potassium nitrate bioadhesive gels (5% and 10%) in the treatment of dentine hypersensitivity. A randomised clinical trial". *Journal of Clinical Periodontology* 30.4 (2003): 315-320.

26. McFall Jr WT. "A review of the active agents available for treatment of dentinal hypersensitivity". *Dental Traumatology* 2.4 (1986): 141-149.

27. Yadav BK., *et al.* "Dentine hypersensitivity: a review of its management strategies". *Journal of International Oral Health* 7.10 (2015): 137.

28. Maximiano V., *et al.* "Nd: YAG laser and calcium sodium phosphosilicate prophylaxis paste in the treatment of dentin hypersensitivity: a double-blind randomized clinical study". *Clinical oral Investigations* 23.8 (2019): 3331-3338.

29. Mrinalini UB., *et al.* "An Update on Dentinal Hypersensitivity-Aetiology to Management-A Review (2021).

Volume 21 Issue 1 January 2022