**Type of Article-** Review article

**Title :** Lasers in the treatment of Dentinal Hypersensitivity: A Brief review

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**Abstract:**

The purpose of this brief review was to evaluate the effectiveness of lasers in the treatment of dentinal hypersensitivity (DH). DH is a frequent condition that may be traced back to everyday dental treatment and is most commonly seen in people in their second to sixth decades of life. DH can be caused by faulty bushing, receding gums, an unsuitable diet, and a variety of other causes. Lasers are a significant treatment tool to treat dentinal hypersensitivity compared to conventional treatment modalities. But still further studies and follow-up is necessary for better results.

**Keywords:** Dentinal Hypersensitivity, Diagnosis, Laser treatment

## Introduction

DH is described as "pain produced from exposed dentin in reaction to chemical, thermal, tactile, or osmotic stimuli that cannot be explained as resulting from any other dental defect or illness," according to the Canadian consensus statement [1]. Dentinal hypersensitivity affects from 8% to 30% of the population, with the most afflicted age group being 20-40 years old. The first premolars are the most impacted teeth, accounting for more than half of all teeth, and the cervical area of the buccal surface is the most affected region [2]. The purpose of this review is to keep clinicians up to speed on the concerns and challenges involved with the clinical management of dentin hypersensitivity, as well as to give basic guidance on how to properly manage the disease within the clinical setting.

Lasers have recently been found to have an important role in treating dentinal hypersensitivity since they produce a long-term reduction in sensitivity while also providing patient satisfaction because they are painless and quick. According to previous studies, the effect of laser on the treatment of DH varies from 5-100 percent depending on the kind of laser and therapeutic factors such as the laser's length of beam, the amount of time spent using the laser, and the laser's strength [3-5]. Different modes of action for laser, its influence on the dentine, and its effect on DH reduction have been postulated. They are as follows: [5]

1. Coagulation of the fluid proteins inside the dentinal tubules causes occlusion.

2. Tubule occlusion caused by partial sub-melting

3. Internal tubular nerve discharge

## Predisposing Factors and Etiology

Abrasion, abfraction, erosion, gingival recession, buccal bone quality, periodontal disease and its treatment, surgical and restorative procedures, and patient destructive habits have all been identified as etiological and predisposing factors in the initiation of DH, according to Gillam and Orchardson [6].

According to the literature, dentinal hypersensitivity manifests itself in two stages [7]:

1. Lesion localization (Table 1)

2. Lesion initiation (Table 1)

| Lesion localization | Lesion initiation |
|---|---|
| It is caused by the removal of the protective coating over the dentin, exposing it to the external environment. Attrition, abrasion, and erosion all contribute to enamel loss. Gingival recession, which can be caused by tooth brush abrasion, pocket reduction surgery, crown preparation, excessive flossing, or owing to periodontal disorders, is another source of lesion localisation. | It happens when the protective covering of the smear layer is lost, exposing and opening the dentinal tubule. |

**Table 1:** Stages of Dentinal hypersensitivity (DH).

**Mechanism of Dentinal hypersensitivity:**

Three processes have been proposed [8]

Regarding the first idea, Direct Innervation theory (DI), it has been observed that nerve endings reach the dentin through the pulp and extend to the DEJ, where mechanical stimuli directly transfer pain. However, there is minimal evidence to support this notion; first, since there is little evidence to support the existence of nerve in the surface dentin, which has the highest sensitivity, and second, because the Rashkov plexus does not grow until the tooth is fully erupted. Newly formed teeth, on the other hand, can be sensitive [9].

According to the Odontoblast Receptor (OR) hypothesis, odontoblasts operate as pain receptors that send signals to the pulpal nerves. However, because the cellular matrix of odontoblasts is incapable of stimulating and creating neuronal impulses, this notion has also been dismissed. Furthermore, no connection between odontoblasts and pulpal nerves has been discovered [10].

Brannstorm [11] was the first to suggest a hydrodynamic theory for sensitive dentine. The most commonly recognised explanation for DH is this one. The notion is based on the passage of fluid within the tubules of the dentition. Tubules, according to this notion, are open between the visible dentine surface and the pulp.

**Diagnosis**

A detailed clinical history and examination are required to diagnose dentinal hypersensitivity. The use of a jet of air or an exploratory probe on the exposed dentin in a mesiodistal orientation while evaluating all the teeth is a straightforward clinical approach of identifying dentinal hypersensitivity. The severity or degree of pain can be measured using either a category scale or a visual analogue scale. A detailed clinical history and examination are required to diagnose dentinal hypersensitivity. The use of a jet of air or an exploratory probe on the exposed dentin in a mesiodistal orientation while evaluating all the teeth is a straightforward clinical approach of identifying dentinal hypersensitivity. The severity or degree of pain can be measured using either a category scale or a visual analogue scale [12-16].

**Causes** [17]:

Dentin hypersensitivity develops in stages, beginning as a local lesion with enamel erosion and proceeding to the exposing of dentin tubules. The following are some of the major reasons that have contributed to this (Figure 1).

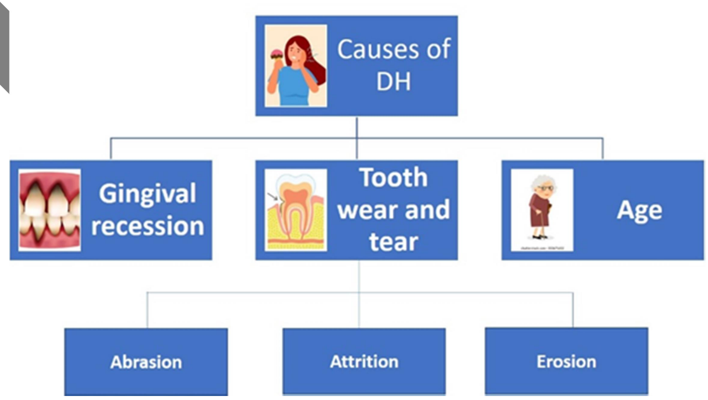


**Figure 1:** Causes of dentine Hypersensitivity (DH)

1**. Gingival recession**: Gingival recession is the exposure of dentin in the teeth's roots as a result of gum tissue loss over the teeth's roots. Periodontal disease, often known as receding gums, is a kind of gum disease. It is more common in persons who have poor oral hygiene and arises as a result of inappropriate or excessive tooth cleaning. Due to a lack of tooth brushing, dental plaque builds up at the base of the teeth, causing gingival recession and exposing the cementum root of the teeth. The tooth structures are also demineralized as a result of this.

2. **Wear and tear on the teeth**:

a) **Tooth abrasion** - Tooth enamel is lost when subjected to vigorous brushing, hard fibrous foods, or low pH oral fluids that cause mineral content of the enamel to dissolve.

b) **Tooth attrition** - Tooth to tooth contact caused by severe teeth grinding or jaw clenching causes tooth enamel deterioration. These practises are referred to as para-functional habits, or bruxism.

c) **Tooth erosion** - Tooth erosion occurs when teeth are repeatedly exposed to anaerobic chemical reactions or acids, whether via the intake of acidic meals or the regurgitation of intrinsic acids.

3**. Age:** Primary dentin begins to wear away with age, whereas secondary dentin is deposited and repaired throughout life.

**Treatment administration (Table 2):**

There are two main approaches in the treatment of DH:

A. Modification or blocking of pulpal nerve response.
B. Alteration of fluid flow in dentinal tubules

   These two approaches can be fulfilled by using following two techniques of treatment:

1. At home Treatment
2. In-office treatment

| At home Treatment | In-office treatment |
|---|---|
| Tooth dentifrice and tooth pastes | Potassium nitrate |
| Mouthwashes and chewing gums | Fluorides |

| | |
|---|---|
| | Oxalates |
| | Varnishes |
| | Adhesive resins |
| | Bio glass |
| | Portland cement |
| | Casein-phosphopeptide-amorphous calcium phosphate (CPP)-(ACP) |
| | Laser |

**Table 2:** Some examples of treatment administration

**Laser treatment in dentinal hypersensitivity**

In 1985, laser therapy was originally proposed as a possible treatment for dentinal hypersensitivity [18]. Since then, several studies on laser applications for dentine hypersensitivity therapy have been conducted, yielding a wealth of knowledge.

In-office DH laser therapy has some drawbacks compared to traditional treatments (e.g., high cost, complexity of usage, declining efficacy with time, etc.) that limit its therapeutic relevance [19]. Furthermore, the effectiveness and mechanism of action of laser therapy for DH treatments are hotly debated [5]. It's important to evaluate the likelihood of a placebo effect, especially because patient reports were good just after laser therapy [20].



**Mechanism of Laser:**

Laser light is monochromatic and has a one wavelength of light. It is made up of three major components: an energy source, an active lasing material, and two or more mirrors that create an optical cavity or resonator. The laser light is supplied from the laser to the target tissue through a fibreoptic cable, hollow waveguide, or articulated arm in dental lasers. The system is completed with focusing lenses, a cooling system, and other controls (Figure 2). The composition of an active medium, which can be a gas, a crystal, or a solid-state semiconductor, determines the wavelength and other parameters of the laser. A laser's light energy can interact with a target tissue in four different ways [21-23]: Absorption, Reflection, Scattering, and Transmission. When a laser is absorbed, the temperature rises and photochemical processes occur, depending on the water content of the tissues. When a temperature of 100°C is achieved, the water within the tissue vaporises, a process known as ablation. Proteins begin to denature at temperatures below 100°C but beyond roughly 60°C, without vaporisation of the underlying tissue. At temperatures exceeding 200°C, however, the tissue is dehydrated and eventually burnt, resulting in an undesirable consequence known as carbonization [23].

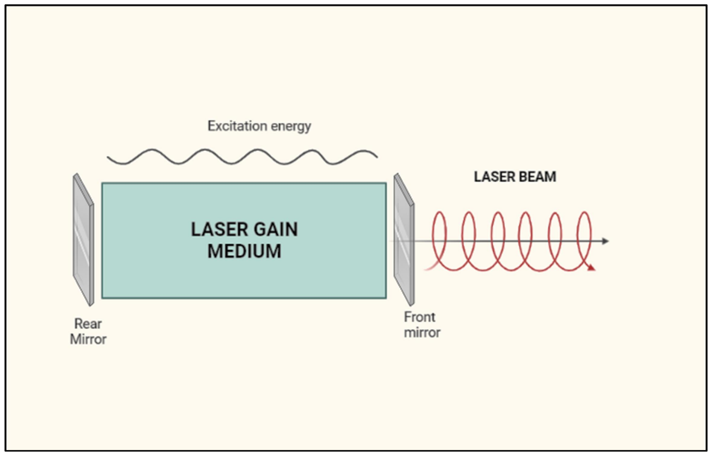


**Figure 2:** Mechanism of laser

In dentinal hypersensitivity the laser beam blocks dentinal tubules. It increases the excitability threshold of the free nervous terminations causing an analgesic effect. It blocks the open tubules by coagulation of proteins in the dentinal fluid and hence reduce permeability. It creates an amorphous sealed layer on the dentine surface which appears due to partial melt down of the surface.

**Types of lasers used in Dentistry (Figure 3):**

1. **Carbon dioxide Laser**: As the CO2 laser wavelength has a very strong affinity for water, it can achieve fast soft tissue excision and hemostasis with a relatively small depth of penetration. Although it has the highest [24] absorbance of any laser, the CO2 laser's downsides are its relatively big size, expensive cost, and harsh tissue damaging interactions.
2. **Neodymium Yttrium Aluminum Garnet Laser**: Because the Nd: YAG wavelength is significantly absorbed by pigmented tissue, it is a particularly efficient surgical laser for cutting and coagulating oral soft tissues while maintaining good hemostasis. In addition to surgical applications,[25] research on employing the Nd: YAG laser for

nonsurgical sulcular debridement in periodontal disease control [26] and the Laser Assisted New Attachment Procedure has been conducted (LANAP). [27]

3. Erbium Laser: Erbium lasers are classified into two types based on their wavelength: Er, Cr: YSGG (yttrium scandium gallium garnet) lasers and Er: YAG (yttrium aluminium garnet) lasers. Erbium wavelengths have the strongest affinity for hydroxyapatite and the most water absorption of any dental laser wavelength. As a result, it is the laser of choice for treating dental hard tissues. [28] Erbium lasers can be utilised for soft tissue ablation in addition to hard tissue operations since dental soft tissue includes a high amount of water. [29]
4. Diode Laser: The diode laser's active medium is a solid-state semiconductor composed of aluminium, gallium, arsenide, and, on occasion, indium, which generates laser wavelengths ranging from around 810 nm to 980 nm. Tissue pigment (melanin) and haemoglobin absorb the majority of diode wavelengths. They, on the other hand, are poorly absorbed by the hydroxyapatite and water found in the enamel [30].

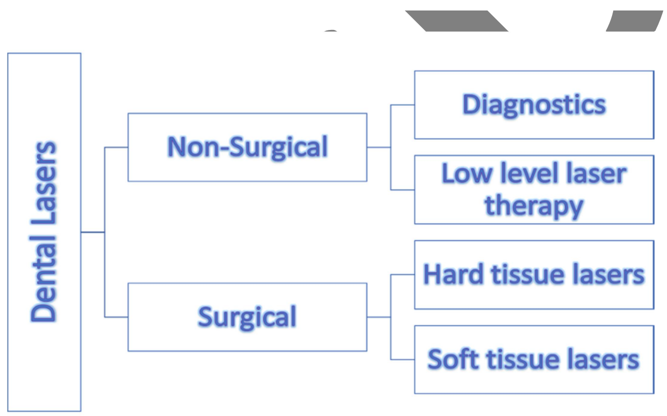


**Figure 3:** Types of Dental lasers

**Discussion:**

Dentinal hypersensitivity (DH) is a severe toothache that can cause dental discomfort during eating and drinking. This discomfort is thought to be caused by heat, tactile, chemical, and/or osmotic stimuli being applied to exposed dentinal tubules [31].There are several treatments that have been proposed by various dentists to treat DH, out of which lasers have shown the potential to provide a long-term effect in treating DH. Following are some studies that prove the role of lasers in DH.

Marsilio et al, 2003, [32] evaluated the effective ness of Gallium-aluminium-arsenium (GaAIAs) laser with maximum and minimum energies to treat DH. They included 106 cases of DH and irradiated teeth with 3 and 5 J/cm2 for up to six sessions, with an interval of 72 h between each application. The result showed a significant decrease in DH of selected teeth and concluded the low-level lasers are effective in treating DH. A similar study, performed by Gojkov-Vukelic et al, 2016 [33], used low level laser therapy to treat DH. Their study included 18 patients with 82 sensitive teeth. The degree of sensitivity was calculated using VAS score. They used diode laser over span of 3 visits per patient and calculated the effectivity of laser. The result showed a significant difference in VAS score of before and after first session of laser, confirming the effectiveness of laser.

Another study that compared the dentin desensitizing effect of 3 type of lasers (diode, Nd:YAG and Er:YAG) with placebo on teeth with gingival recessions concluded that lasers can be used for DH reduction [34]. Although lasers alone area significant treatment for DH but there are many studies that have shown some consequential results when lasers were used along with the desensitizing agents compared to alone. A study performed by Yadav et al, 2019,[35] in which they compared and evaluated the effect of Nd: YAG laser and desensitizing agents and found out that lasers along with Nanocrystalline hydroxyapatite (desensitizing agent) showed the better result compared to treatment with only Nd:YAG laser.

A study performed by Romeo et al, 2012[36] compared the efficacy of Laser (G1 group), sodium fluoride (G2 group), and in combination of both (G3 group), among 10 patients with DH and fount out that the G3 group showed maximum reduction in DH compared to the other two groups. Similarly, Lopes et al, 2013 [37] used Gluma desensitizer and Nd: YAG laser to treat DH. They divided patients in 3 groups out of which the combination group of laser and desensitizer showed the best long-lasting results for DH.A contradictive study reported by Pandey et al, 2017, [38], showed no extra benefits of using desensitizer along with lasers.

**Conclusion:**

Dental hypersensitivity should be treated on a regular basis, first with at-home therapy and then progressing to complementary therapies. After undergoing periodic treatments, it is advised that all patients have follow-up appointments scheduled.

Desensitizing dentifrices and other self-applied treatments are still widely utilised as home-made desensitising agents. One of the most successful therapeutic therapies is the use of lasers and adhesive systems and its effect can be enhanced when used with desensitizing agents.

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