Dentinal Hypersensitivity: A Review

Course Author(s): Patricia A. Walters, RDH, MSDH, MSOB
CE Credits: 1 Hour(s)
Intended Audience: Dentists, Dental Hygienists, Dental Assistants, Dental Students, Dental Hygiene Students, Dental Assistant Students
Date Course Online: 10/18/2005
Last Revision Date: 11/14/2024
Course Expiration Date: 11/13/2027
Cost: Free
Method: Self-instructional
AGD Subject Code(s): 161, 730, 739

Online Course: www.dentalcare.com/en-us/ce-courses/ce200

Conflict of Interest Disclosure Statement

- Ms. Walters is a retired employee of P&G and does consulting work for P&G. She has no relevant financial relationships to disclose.

Introduction

This free continuing education course will address the etiology, prevalence and diagnosis of dentinal hypersensitivity as well as review clinical evidence behind popular desensitizing dentifrices.

Course Contents

Overview

Dentinal hypersensitivity is a common clinical condition referred to as "dentinal hypersensitivity," "dentine sensitivity," "root sensitivity" or "tooth sensitivity" that is frequently encountered yet it is often under-reported by patients, or misdiagnosed by clinicians. This course will address the etiology, prevalence and diagnosis of dentinal hypersensitivity as well as review clinical evidence behind common treatments.

Learning Objectives

Upon completion of this course, the dental professional should be able to:

- Discuss the prevalence of dentinal hypersensitivity and common contributing factors.
- Explain the hydrodynamic theory, widely accepted as the cause for dentinal sensitivity.
- Discuss common diagnostic tools.
- List common ingredients used in at-home and in-office desensitizing products.
- Discuss the mode of action of common ingredients.
- Discuss the clinical evidence behind common treatment approaches including in-office treatments.

Prevalence

Dentinal hypersensitivity (DH) is a global oral health issue and a significant challenge for most dental professionals. Symptoms of dentinal hypersensitivity are generally reported by the patient and are difficult to describe and challenging to accurately diagnose because other dental diseases have to be ruled out first, such as dental caries, cracked-tooth syndrome, and defective restorations, among others. The condition has been defined by an international workshop on DH as follows: "DH is characterized by 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".[1,2] (Figure 1)).

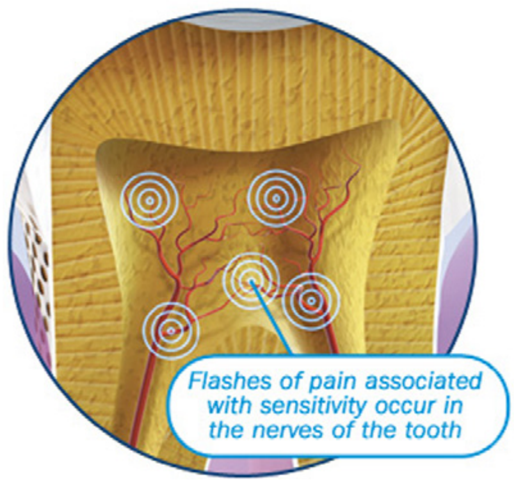

Figure 1. Figure 1. Pictorial display of origin of pain associated with sensitive teeth.

Dentinal hypersensitivity incidence ranges from 4-74%.[1] The variations in the reports may be because of difference in populations and different methods of investigations. The methods employed are usually patient questionnaires or clinical examinations. A slightly higher incidence of DH is reported in females than in males. While DH can affect the patient of any age, most affected patients are in the age group of 20–50 years, with a peak between 30 and 40 years of age.[3-9] The most common teeth affected by DH are the canines and premolars and the buccal aspect of cervical area is the commonly affected site.[4] Among periodontal patients, the frequency is much higher (60-90%).[5-7]

Pathogenesis:
Dentin is covered by enamel on the crown surface and by a thin layer of cementum on the root surface of the tooth and is sensitive to stimuli. DH develops in two phases: lesion localization and lesion initiation. Lesion localization occurs by loss of protective covering, enamel and cementum over the dentin, thereby exposing the dentinal tubules. Exposure is attributed to attrition, abrasion, erosion and abfraction. However, dentinal exposure mostly occurs due to gingival recession which can be due to toothbrush abrasion, pocket reduction surgery, tooth preparation for crown, excessive flossing or secondary to periodontal diseases.[18] In the second phase, the exposed dentin will only be sensitized if the tubular plugs and the smear layer are removed exposing them to the external environment. Both mechanical and chemical factors are effective in removing the smear layer from the dentinal tubules. A couple culprits involved in removing the smear layer are acidic foods and acidic drinks.

Theories
A variety of theories have been suggested to help explain the mechanism involved in the etiology of dentinal hypersensitivity.[11] The transducer theory, the modulation theory, the "gate" control and vibration theory, and the hydrodynamic theory have all been presented and discussed throughout the years. The latter, "hydrodynamic theory," developed in the 1960's and based upon two decades of research, is now widely accepted as the cause of tooth sensitivity.[12] Before explaining the "hydrodynamic theory" it is important to point out that none of these mechanisms full explain dentin hypersensitivity, indicating unexplained mechanisms are possibly responsible. The widely accepted hydrodynamic theory asserts that when the fluid within the dentinal tubules, absent of a smear layer, or enamel or cementum, is subjected to thermal, chemical, tactile or evaporative stimuli, the movement of the fluid stimulates the mechanical receptors which are sensitive to fluid pressure, resulting in the transmission of the stimuli to the pulpal nerves (Figure 2) ultimately causing the pain response.[11]

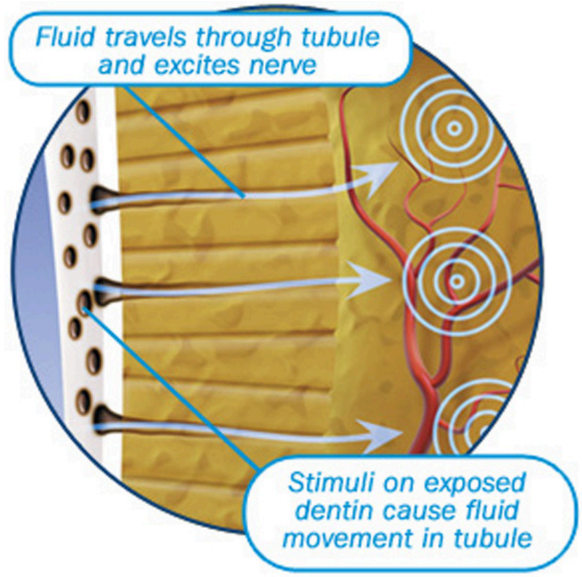

Figure 2. Depiction of Brannstrom's Hydrodynamic Theory.

Berman[11] describes this reaction as:

> "The coefficient of thermal expansion of the tubule fluid is about ten times that of the tubule wall. Therefore, heat applied to dentin will result in expansion of the dentinal fluid, and a cold stimulus will result in contraction of the fluid, both creating an excitation of the 'mechano-receptor'."

Based on the hydrodynamic theory, dentinal hypersensitivity is a transient tooth pain. In order to exhibit a response to the stimuli, the tubules would have to be open at the dentin surface as well as the pulpal surface of the tooth. Anatomically, the tubules in the area closest to the pulp chamber are wider, and the number of tubules per unit area increases almost two-fold from the outer surface to the pulp.[13]

The most important variable affecting the fluid flow in dentin is the radius of the dentinal tubules. If the radius is reduced by one-half, the fluid flow within the tubules falls to one-sixteenth of its original rate. Consequently, the creation of a smear layer or the occlusion of the tubules will significantly reduce sensitivity.[14,15]

Diagnosis

The reason(s) for tubules to be exposed or open should be assessed during a visual examination of the teeth. Additionally, a detailed dietary history should be taken. Useful diagnostic tools are the air/water syringe (thermal), dental explorer (touch), percussion testing, bite stress tests, and other thermal tests such as an ice cube, and assessment of occlusion. Since dentinal hypersensitivity is essentially diagnosed by exclusion, a comprehensive dental examination will ultimately rule out other underlying conditions for which sensitivity may be a symptom such as cracked tooth, fractured restoration, chipped teeth, dental caries, gingival inflammation, post-restorative sensitivity, marginal leakage and pulpitis. Excessive intake of dietary acids such as citrus juices and fruits, carbonated drinks, wines and ciders have been identified as potential risk factors for dental hypersensitivity.[4,12,14] The dietary history provided by the patient will assist in identifying the risk factors the patient may have for tooth sensitivity. Erosion is one of the most common causes of irreversible enamel loss.[15]

In addition, other risk factors should be ferreted out during an examination such as toothbrush abrasion (Figure 3), chemical erosion (Figure 4), thin enamel, gingival recession, exposed dentin, and eating disorders such as bulimia. The patient will be able to assist in diagnosis by identifying the pain-inciting stimuli, i.e., thermal, tactile, etc., as well as describing the pain. The response to stimuli varies from patient to patient. Factors such as individual pain tolerance, emotional state, and environment can contribute to the variety of responses between and among patients.[16]

The most commonly cited reason for exposed dentinal tubules is gingival recession (predisposing factor).[17] Gingival recession is the reduction of the height of the gingival margin to a location apical to the CEJ. Chronic exposure to bacterial plaque, toothbrush abrasion, abfraction, gingival laceration from oral habits such as toothpick use, excessive flossing, crown preparation, inadequate attached gingiva, inadequate labial plate of the alveolar bone and gingival loss secondary to disease or surgery are some but not all causes of gingival recession.[17]

Recessed areas may become sensitive due to the loss of cementum, ultimately exposing dentin. Probing depths, recessed areas (areas of gingival recession), and sensitivity reported by the patient must be accurately recorded and monitored to provide a reference for the patient's disease activity over time. By removing the etiology of DH (for example, over enthusiastic brushers, periodontal treatment patients, bulimics, people with xerostomia, high acid food/drink consumers, chewing smokeless or snuff tobacco) it can be prevented from occurring or reoccurring.

Figure 3. Tooth Abrasion.

Figure 4. Tooth Erosion.
Images courtesy, Dr. Beatrice Gandara,University of Washington, School of Dentistry

Treatments

Classifications of Desensitizing Agents based on:

I. Mode of administration

At home treatment

In-office treatment

II. Mechanism of action

Nerve desensitization
- Potassium nitrate

Protein precipitation
- Gluteraldehyde
- Silver nitrate
- Zinc chloride
- Strontium chloride hexahydrate

Plugging dentinal tubules
- Sodium fluoride
- Stannous fluoride
- Strontium chloride
- Potassium oxalate
- Calcium phosphate
- Calcium carbonate
- Bio active glasses (SiO2–P2O5–CaO–Na2O)

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

Lasers
- Neodymium:yttrium aluminum garnet (Nd-YAG) laser
- GaAlAs (galium-aluminium-arsenide laser)
- Erbium-YAG laser

Homeopathic medication
- Propolis

Treating dentinal hypersensitivity can be challenging for the dental professional because of the difficulty related to measuring the pain response as the response can often vary from patient to patient. In addition, if the dentin exposure is due to personal habits, it may be difficult for patients to change their behavior.

IIn 1935, Grossman addressed the requirements for an ideal desensitizing agent as: rapidly acting with long-term effects, non-irritating to the pulp, painless and easy to apply without staining the tooth surface. These requirements still exist today when considering an ideal solution to dentinal hypersensitivity.[18] There are two common approaches to treating dentinal hypersensitivity, nerve depolarization and tubule occlusion. Furthermore, treatment options can be classified as either invasive or non-invasive in nature. Examples of invasive procedures administered in-office include gingival surgery, application of resin adhesive materials such as dentin bonding agents, or a pulpectomy. Dentifrices and other products for home use are non-invasive. Finally, treatments can be categorized based on whether they can be applied by the patient (over-the-counter) or require professional application. For the purposes of this CE course, the focus is on those treatments that disturb the neural response to pain stimulus and those that block/occlude the dentinal tubules.

Over-the-Counter Products

OOver-the-counter products for the treatment of tooth sensitivity are considered to be a simple and cost-effective first line of treatment for most patients.[18] The primary at-home non-invasive treatment option has historically been anti-sensitivity dentifrices. The two most common ingredients are potassium nitrate, which interferes with the transmission of the nerve impulse, and stannous fluoride and arginine, which blocks dentinal tubules by forming a smear layer at the surface.

Potassium Nitrate Dentifrice

Potassium salts move along the dentinal tubules and through blocking the action of the intra-dental nerve fibers decrease the excitability of the tooth. Potassium salts such as potassium chloride, potassium citrate and potassium nitrate are known to interfere with the nerve impulse and is commonly found in desensitizing toothpaste.[19] Potassium nitrate products raise the extracellular potassium ion concentrations and affect polarization. When the concentration is sustained over time, the synapse between nerve cells is blocked, the nerve excitation is reduced and the tooth is less sensitive to the stimuli. A large number of studies, published since the early seventies, have investigated the use of potassium nitrate (KNO3) as an effective active ingredient in treating dentinal hypersensitivity.[20-34]

The use of toothpastes which contain potassium nitrate and fluoride has a positive effect on reducing DH. A four-week exposure time is widely used in these clinical trials because results have shown that this time is needed for 5% KNO3 to exert its desensitizing effect.23 The use of a broadly accepted positive or negative control toothpaste formulation or product has been increasingly used over the years in comparative trials because the condition itself can appear to be self-resolving within the time scale of the study. Over time, investigators have chosen various methods to capture subjective responses; controlled reproducible stimuli and objective measurements are preferred.

In 2006, the Cochrane Collaboration published a systematic review26 of potassium nitrate toothpastes for the treatment of dentinal hypersensitivity based on clinical trials conducted up to the year 2005 involving KNO3 toothpaste compared to non-KNO3 toothpaste. This review focused on studies that incorporated similar methods in order to determine if KNO3 is an effective agent in reducing dentinal hypersensitivity. The results were obtained by measuring tactile (Figure 5),

Figure 5. Illustration of the Yeaple Probe.

thermal, and air blast stimuli as well as patients' subjective assessment of pain during everyday life. The exposure periods ranged from six to eight weeks, reporting outcome measurements as a mean change from baseline.

The meta-analysis included six studies,27-32 and all showed a significant effect on sensitivity assessed by air blast and tactile methods at the 6 to 8 week follow-up. However, there was no significant effect observed at the 6 to 8 week follow-up for the subjective assessment. The authors concluded the support for the efficacy of potassium nitrate toothpaste for dentinal hypersensitivity was based on a very small sample size, thus evidence of the effectiveness of KNO3 is not clear, suggesting more clinical trials need to be conducted and published. There is no current research published to support a different conclusion than what is stated above even though new product lines are being marketed using this technology.

Products currently on the market that contain potassium nitrate include Sensodyne Repair & Protect, Sensodyne Fast Sensitive Teeth Relief, Crest® Sensitivity Whitening plus Scope, Crest Sensi-Relief Plus Scope Toothpaste, Colgate® Sensitive Prevent & Repair, Colgate® Enamel Health Sensitivity Relief, Arm & Hammer® Advanced Whitening Sensitive, Tom's of Maine™ Rapid Relief-Sensitive and Hello Sensitive Relief Fluoride Toothpastes.

Stannous Fluoride

Stannous fluoride has been incorporated into oral hygiene products to prevent dental caries,[35] reduction of plaque formation,[36] control of gingivitis[37] suppression of breath malodor,[38] and reduce dentinal hypersensitivity since the 1960s. The ADA has recognized the desensitizing properties of stannous fluoride by granting the ADA Seal of Acceptance to a non-aqueous stannous fluoride gel formulation (Gel-Kam) for the therapeutic prevention of sensitivity and caries as well as to Crest® PRO-HEALTH® toothpaste.[21] The effect of stannous fluoride is the result of the reaction between the stannous ion and the dental hard tissue which leads to a protective layer on the tooth surface that is resistant to an acid challenges.[40]

In situ research shows root dentin treated with stannous fluoride exhibits tubule occlusion at the surface by the formation of a smear layer (Figure 6).[39] When the tubules are blocked, fluid flow is limited and the stimulation of the mechanoreceptors does not occur, thus preventing the pain response.

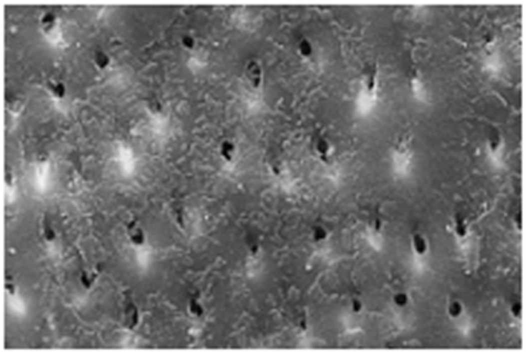

Figure 6a. Open tubules following treatment with non-sensitivity fluoride toothpaste.

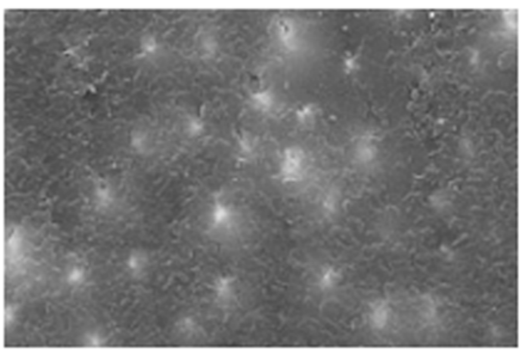

Figure 6b. Closed tubules following treatment with SnF_2 dentifrice.

Stannous fluoride has been delivered via a mouth rinse, dentifrice, and gel for some time. Research by Thrash et al.[43,44] in the 1990's suggested there is a gradual decrease in sensitivity starting at two weeks and continuing throughout the 16-week period from initiation of treatment. Thrash and colleagues conducted a two-phase experimental design study comparing a 0.4% stannous fluoride gel to an aqueous 0.717% fluoride solution and a placebo to evaluate the effect of the products on hypersensitivity tooth pain and to determine the precise time of onset of any effect on dentinal hypersensitivity. Sensitivity to thermal stimuli was assessed prior to the first application and then at 2, 4, 8, and 16 week intervals after the initial application. The results indicated subjects who applied the 0.4% stannous fluoride gel reported significantly less sensitivity during the four to eight week period. The stannous fluoride gel resulted in the lowest mean threshold temperature compared to the other products.

Historically, one limitation to the use of stabilized stannous fluoride has been the potential for temporary extrinsic tooth staining associated with the long-term use of these products. Due to advances in dentifrice technology, this occurrence has been mitigated by incorporating sodium hexametaphosphate, an advanced tartar control and whitening ingredient, in the formulation marketed as Crest® PRO-HEALTH® toothpaste. Also, a variety of stabilization chemistries have been employed in modern stannous formulations to mitigate stains.

A recent meta-analysis by West, et al, looked at randomized clinical trial conducted over the past three decades to assess their effects on DH and enamel erosion. Fourteen (14) randomized and controlled clinical trials (RCTs) were identified assessing DH relief specifically. The 14 studies were conducted involving 1287 participants across the US, Canada and China. Benefits for stannous fluoride over both negative and positive controls ranged from 22 to 142% for alleviation of DH. Six RCTs assesses SnF_2 versus a positive control (potassium nitrate, arginine) found a 22% benefit for SnF_2 using the Schiff assessment method. Final results in the meta-analysis supports better desensitizing efficacy of bioavailable gluconate chelated SnF_2 toothpaste. The scope of the research is robust involving 20 studies in different regions of the world and are consistent with the literature and provides support for the use of stannous fluoride for those experiencing dentinal hypersensitivity.[45]

In addition, there have been several studies demonstrating similar results during a 3 day study period in one study, as well as a 2 week study period in four studies.[46-49] In two randomized, double-blind clinical trials, this stabilized stannous fluoride toothpaste significantly reduced thermal and tactile sensitivity versus a negative control.[49,50] More recently, He et al, demonstrated in two different randomized controlled clinical trials that twice daily brushing with the stabilized stannous fluoride dentifrice provides superior dentinal hypersensitivity improvement versus a marketed sodium fluoride dentifrice, and a dentifrice containing 8.0% arginine, calcium carbonate and sodium monofluorophosphate.[51,52] The stannous fluoride dentifrice provided some relief after the first brushing relative to each control, with the benefit growing larger over the study period with twice daily use. When compared to sodium fluoride/triclosan dentifrice, there was a similar outcome, superior dentinal hypersensitivity improvement with significant greater relief after two weeks and a larger benefit at eight weeks with twice daily brushing.[53] In separate clinical research, this unique dentifrice provided significant extrinsic whitening relative to a positive control.[53,54] In addition, a systematic review was recently published by Konradsson et al included scientific evidence for the efficacy of stabilized stannous fluoride dentifrice in relations to DH when compared with standard fluoride dentifrices. Eleven (11) DH articles were published between 2005 and 2016 and represented 1361 patients with 618 in the test group and 617 in the control group. Toothpaste containing stabilized stannous fluoride with and without sodium hexametaphosphate was shown to have preventive and therapeutic effects on dentin hypersensitivity especially when used twice daily for 1 minute. The stannous fluoride product was shown to be efficacious after 3 days and lasting up to 8 weeks.[46] Crest Pro-Health products and Colgate Stannous Fluoride Daily Repair are examples of toothpastes with stannous fluoride ingredient.

Arginine

Arginine is an amino acid which occurs naturally in saliva. The mechanism of action for toothpastes containing arginine bicarbonate and calcium carbonate is plugging of open dentinal tubules. The plug is able to remain even after exposure to acids. In 2013 Sharif et al conducted a systematic review of Arginine toothpastes effectiveness in treating DH. The review highlighted that the studies conducted thus far involved small numbers of subjects and reduced DH in the short-term. The authors recommended that further well designed RCTs should be conducted looking at medium and long term effects of Arginine on DH.[47] There is a cross-over product (at home use and professionally applied) Colgate Anywhere Anytime Sensitivity Relief System that has the following ingredients: Arginine bicarbonate, benzyl alcohol, calcium carbonate, cellulose gum, flavor, glycerin, propylene glycol, sodium bicarbonate, sodium saccharin, sodium silicate, titanium dioxide and water. Very little research has been conducted to come to any conclusion regarding the effectiveness of Arginine products. In regards to clinical significance, the results of this clinical study, together with the results of published pivotal studies, demonstrate that a desensitizing toothpaste containing 8% arginine and calcium carbonate, with or without fluoride, provides statistically significant reduction in dentin hypersensitivity when applied by a dental professional prior to a professional dental prophylaxis. The results also demonstrate that this desensitizing toothpaste provides statistically significant reductions in dentin hypersensitivity when used subsequently as an adjunct to routine twice daily tooth brushing.[47]

Bioactive Glass

Dentifrices containing desensitizing agents have been the most popular first-line treatment for sensitive teeth, but there are some drawbacks. It typically takes time (approximately 4 weeks) to experience relief and on-going use is required to maintain the benefit.

One such product is NovaMin, a synthetic mineral composed of calcium, sodium, phosphorus and silica releases deposits of crystalline, hydroxyl-carbonate apatite which is structurally similar to tooth mineral composition. NovaMin is technically described as sodium calcium phosphosilicate.

Method of Action

The formation of bioglass reacts with the saliva in the mouth to form a protective layer of hydroxyapatite on the tooth, thereby, occluding dentin tubules. This layer prevents the discomfort that is tooth sensitivity.

A number of clinical studies (5) investigating the efficacy of NovaMin for four and six weeks have been conducted. A product name you may be familiar with is Sensodyne Repair and Protect. An overview of the clinical evidence for the use of NovaMin to treat dentinal hypersensitivity was addressed by Gendreau et al. Clinical evidence supports the effectiveness of the 5% and 7.5% product twice daily brushing for pain relief from this malady.[56] Burwell et al conducted four experiments to demonstrate the ability of NovaMin to rapidly occlude tubules, remain on the dentin surface in the face of acid challenges, and form a stable layer on the surface of the dentin. NovaMin was compared to other marketed calcium-based products that claim tubule occlusion after a single treatment, a 10 day acid challenge looking at tubule occlusion, changes in surface hardness and calcium release. The authors concluded from the 4 experiments that NovaMin adheres to an exposed tooth dentin surface and reacts to form a mineralized layer. The layer is resistant to acid challenges and is strong mechanically. In addition, there is a continuous release of calcium over time and after four hours a higher release of calcium was observed providing for continual occlusion of the tubules.[57]

In-office Treatments

Professional treatments are available for sensitivity cases that cannot be managed using over-the-counter products. Some in-office treatments include fluoride varnishes, prophylaxis pastes and laser treatments.

Fluoride Varnishes

The most popular in-office treatment is fluoride varnish, a resin-based fluoride. Various types are available. Fluoride varnish is primarily used to prevent tooth decay by entering the tooth enamel and making the tooth surface impenetrable. The mode of action involves calcium fluoride being deposited on the tooth surface with the formation of fluorapatite.[59-69] The varnish is applied after cleaning and drying the tooth surface. For caries protection, fluoride varnish is painted onto the tooth surfaces with a small brush. The varnish forms a sticky covering over the tooth and becomes hard as soon as saliva in the mouth touches it. Fluoride varnish prevents new cavities from forming and slows down or stops decay from progressing.

Many practitioners have begun using fluoride varnish as a desensitizing agent by applying the varnish to the exposed area to seal the dentin surface. Pashley et al., evaluated a series of commercial cavity varnishes and reported that all cavity varnishes tested decreased dentin permeability by 20 to 50%.[61] In 2012, Camilotti et al. conducted a randomized, split-mouth clinical trial in 42 patients (252 teeth) presenting with dentin hypersensitivity to thermal changes in the mouth. The treatment groups were 4 fluoride varnishes (Duraphat, Fluorniz, Duofluorid Xii, and Fluorphat), a neutral fluoride (Flutop), a potassium oxalate gel (Oxa-gel) and a placebo which were all applied 3 different times with a time interval of one week between applications. Sensitivity reduction using air blast and clinical probing was evaluated at the end of 1 week, 2 weeks, 3 weeks and 30 days after the last application. The 4 fluoride varnish groups and the oxalate gel group had significantly lower pain scores compared to placebo at the 30-day reassessment; there were no significant differences between the 5 groups. The neutral sodium fluoride group was not significantly different from placebo, nor was it significantly different from Fluoriniz, Duoluorid XII, or Oxa-gel.[60]

Fluoride varnish is easy to apply, low-cost and generally safe to use in the mouth, but should not be used if there is an allergy to one of the ingredients in the varnish.

Prophylaxis Pastes

Prophylaxis pastes with desensitizing agents are another professional treatment used for the relief of sensitivity. One example is paste containing 8% arginine (Pro-Argin), calcium carbonate, and 1450 ppm fluoride as sodium monofluorophosphate (Colgate Sensitive Pro-Relief). Arginine, an amino acid naturally present in saliva, is reported to work in conjunction with calcium carbonate and phosphate to occlude dentinal tubules.[70-76]

Results from a 12-week clinical trial showed that 8% arginine-containing prophylaxis paste was statistically significantly more effective in reducing dentinal hypersensitivity than a control pumice prophylaxis paste (NuPro) immediately following application and after 4 weeks. No statistically significant differences were noted between treatment groups post-scaling and after 12 weeks.70

In some markets outside the US, arginine is available in over-the-counter dentifrice and mouthrinse products.

Another prophylaxis paste for hypersensitivity relief contains sodium calcium phosphosilicate, marketed under the name of NovaMin®.[77,78] Sodium calcium phosphosilicate occludes the dentinal tubules by forming a protective hydroxyapatite-like layer on the dentin surface. A number of clinical studies investigating the efficacy of NovaMin® for the relief of dentinal hypersensitivity have been conducted.[79-82]

Neuhaus et al., conducted a randomized, controlled, double-blind, parallel study with three treatment groups - sodium calcium phosphosilicate prophylaxis paste, with and without fluoride, and a control group - in 151 subjects meeting dentinal hypersensitivity entrance criteria.[83] Tactile and air blast assessments were completed at baseline and day 28. The results indicate that after a single professional application of sodium calcium phosphosilicate prophylaxis paste, hypersensitivity was significantly reduced immediately and 28 days after scaling and root planing procedures. The effect was independent from the presence of fluoride in the paste.

Lasers

Four different kinds of light amplification by stimulated emission of radiation (lasers) have been used for the treatment of dentinal hypersensitivity with effectiveness ranging from 5.2 to 100%, depending on the laser type and parameters used.[84] The most common are: Nd-YAG (neodymium:yttrium-aluminum-garnet), GaAlas (gallium/aluminum/arsenide) and Erbium-YAG (yttrium-aluminum-garnet) lasers.[85-88] The mechanism of action of lasers in treating hypersensitivity is not very clear, but it has been proposed that the lasers coagulate the proteins inside the tubules and block the movement of fluid.

A 2011 systematic review of lasers for the treatment of sensitivity found only 3 randomized clinical trials for inclusion. The authors concluded that laser therapy can reduce dentinal hypersensitivity-related pain, but there is only weak evidence for its effectiveness and the placebo effect has to be taken into account.[89]

Conclusion

Dentinal hypersensitivity is a common problem that effects many dental patients. When a patient presents with dentinal hypersensitivity symptoms, they should be examined and informed of the treatment options available to alleviate the problem. The patient plays a role in this process since their daily habits may be contributing to the problem, and if not changed the condition may persist.

The initial cause of dentinal hypersensitivity, in the majority of cases, is recessed gingiva with the exposure of dentinal tubules. Once the tubules are exposed the patient is susceptible to pain in response to thermal, tactile, or osmotic stimuli. Desensitizing treatments should be delivered systematically.

Prevention and over-the-counter treatments, including desensitizing toothpastes, are a good place to start and can later be supplemented with in-office treatments if needed.

Course Test Preview

To receive Continuing Education credit for this course, you must complete the online test. Please go to: www.dentalcare.com/en-us/ce-courses/ce200/start-test

1. The prevalence of dentinal hypersensitivity has been reported over the years in a variety of ways; the variation in percentage is due to ____________.

A. Difference in populations
B. Methods of investigations
C. Not enough female participants
D. The wrong teeth being examined
E. A & B only

2. At what age does dentinal hypersensitivity typically peak?

A. 20-30s
B. 30-40s
C. 40-50s
D. Above 60

3. Dentinal hypersensitivity has been researched extensively through the years and many authors express an agreement that dentinal hypersensitivity is ____________.

A. under-reported by the dental patient population
B. under-diagnosed
C. A & B
D. neither A nor B

4. The ""hydrodynamic theory"" is widely accepted as the cause of tooth sensitivity.

A. TRUE
B. FALSE

5. Assumptions of the hydrodynamic theory conclude that when the fluids within the __________ are subjected to temperature changes or physical osmotic changes, the movement stimulates a nerve receptor sensitive to pressure, which leads to the transmission of the stimuli.

A. hydroxyapatite
B. cementum
C. dentinal tubules
D. periodontal ligaments

6. Various stimuli that are reported to cause the transmission of sensation are __________.

A. cold
B. hot
C. osmotic
D. A & B
E. A, B & C

7. The most important variable affecting the fluid flow in dentin is the pH of the stimulus.

A. TRUE
B. FALSE

8. A comprehensive dental examination will ultimately rule out potential underlying conditions where sensitivity is a symptom, EXCEPT one of the following. Which is the exception?
A. cracked tooth
B. fractured restoration
C. intrinsic tooth stain
D. dental caries

9. The response to stimuli ____________
A. is relatively constant among patients
B. varies from patient to patient
C. can be predicted based on gender
D. is directly correlated to the patient's age

10. One of the most common reasons a dental patient would have exposed dentinal tubules is:
A. fluorosis
B. interproximal plaque
C. class II malocclusion
D. gingival recession

11. Invasive treatments may include all the following EXCEPT one. Which one is the exception?
A. application of resins
B. use of home care OTC products
C. pulpectomy
D. gingival surgery

12. Over-the-counter desensitizing dentifrices are considered to be simple, cost-effective and an efficacious first line of treatment for most patients.
A. TRUE
B. FALSE

13. Stannous fluoride is a most widely available desensitizing toothpaste active ingredient. It works by ____________.
A. occluding dentinal tubules by forming a smear layer at the surface
B. numbing the surrounding tissue
C. blocking the synapse between nerve cells, reducing nerve excitation and associated pain
D. None of the above.

14. The following are examples of in-office treatments:
A. prophylaxis pastes with a desensitizing agent
B. fluoride varnishes
C. lasers
D. B & C
E. A, B & C

References

1. Addy, M. Dentin hypersensitivity: new perspectives on an old problem, Int. Dent J. 52(2002) 367-375 Accessed November 2, 2021.
2. Holland GR, Narhi MN, Addy M. Gangarosa, L, Orchardson, R, Guidelines for the design and conduct of clinical trials on dentine hypersensitivity, J. Clin Periodontol. 24 (1997) 808-813.
3. Rees JS, Addy M. A cross-sectional study of buccal cervical sensitivity in UK general dental practice and a summary review of prevalence studies. Int J Dent Hyg. 2004 May;2(2):64-9.
4. Addy M, Dowell P. Dentine Hypersensitivity – A review. Clinical and in vitro evaluation of treatment agents. J Clin Periodontol. 1983 Jul;10(4):341-50-63.
5. Irwin CR, McCusker P. Prevalence of dentine hypersensitivity in a general dental population. J Ir Dent Assoc. 1997;43(1):7-9.
6. Addy M. Etiology and clinical implications of dentine hypersensitivity. Dent Clin North Am. 1990 Jul;34(3):503-14.
7. Chabanski MB, Gillam DG, Bulman JS, Newman HN. Clinical evaluation of cervical dentine sensitivity in a population of patients referred to a specialist periodontology department: a pilot study. J Oral Rehabil. 1997 Sep;24(9):666-72.
8. Von Troil B, Needleman I, Sanz M. A systematic review of the prevalence of root sensitivity following periodontal therapy. J Clin Periodontol. 2002;29 Suppl 3:173-7.
9. Mantzourani M, Sharma D. Dentine Sensitivity: past, present and future. J Dent. 2013 Jul;41 Suppl 4:S3-S17.
10. Curro FA. Tooth hypersensitivity in the spectrum of pain. Dent Clin North Am. 1990 Jul;34(3);429-37.
11. Sood S, Nagpal M, Gupta S, et al. Evaluation of dentine hypersensitivity in adult population with chronic periodontitis visiting dental hospital in Chandigarh. Indian J Dent Res. 2016 May-Jun;27(3):249-55. doi: 10.4103/0970-9290.186239.
12. Uchida A, Wakano Y, Fukuyama O, et al. Controlled clinical evaluation of a 10% strontium chloride dentifrice in treatment of dentin hypersensitivity following periodontal surgery. J Periodontol. 1980 Oct;51(10):578-81.
13. Nishida M, Katamsi D, Uchida A, et al. Hypersensitivity of the exposed root surfaces after surgical periodontal treatment. J Osaka Univ Dent Soc. 1976;16:73-77. Accessed November 2, 2021.
14. Berman LH. Dentinal sensation and hypersensitivity. A review of mechanisms and treatment alternatives. J Periodontol. 1985 Apr;56(4);216-22.
15. Brännström M, Aström A. The hydrodynamics of the dentine; its possible relationship to dentinal pain. Int Dent J. 1972 Jun;22(2):219-27.
16. Michelich V, Pashley DH, Whitford GM. Dentin permeability. a comparison of functional versus anatomical tubular radii. J Dent Res. 1978 Nov-Dec;57(11-12):1019-24.
17. Pashley DH, Tao L, Boyd L, et al. Scanning electron microscopy of the substructure of smear layers in human dentine. Arch Oral Biol. 1988;33(4):265-70.
18. Dugmore CR, Rock WP. A multifactorial analysis of factors associated with dental erosion. Br Dent J. 2004 Mar 13;196(5):283-6.
19. Orchardson R, Colins WJ. Clinical features of hypersensitive teeth. Br Dent J. 1987 Apr 11;162(7):253-6.
20. Bal J, Kundalgurki S. Tooth sensitivity prevention and treatment. Oral Health. 1999 Feb;89(2):33-4, 37-8, 41.
21. Grossman L. A systematic method for the treatment of hypersensitive dentine. J Am Dent Assoc. 1935;22:592-8. Accessed November 2, 2021.
22. Jacobsen PL, Bruce G. Clinical dentin hypersensitivity: understanding the causes and prescribing a treatment. J Contemp Dent Pract. 2001 Feb 15;2(1):1-12.
23. Candadian Advisory Board on Dentin Hypersensitivity: Consensus-based recommendations for the diagnosis and management of dentin hypersensitivity. J Can Dent Assoc. 2003 Apr;69(4):221-6.

24. American Dental Association Council on Scientific Affairs: Acceptance Program Guidelines Products for the Treatment of Dentinal Hypersensitivity, 2004.
25. Clark DC, Hanley JA, Geoghegan S, et al. Effectiveness of a fluoride varnish and a desensitizing toothpaste in treating dentinal hypersensitivity. J Periodontol Res. 1985 Mar;20(2):212-9.
26. Addy M, Mostafa P, Newcombe R. Dentine hypersensitivity: A comparison of five toothpastes used during a 6-week treatment period. Br Dent J. 1987 Jul 25;163(2):45-51.
27. Tarbet WJ, Buckner A, Stark MM, et al. The pulpal effects of brushing with a 5 percent potassium nitrate paste used for desensitization. Oral Surg Oral Med Oral Path 1981 Jun;51(6):600-602.
28. Knight NN, Lie T, Clark SM, et al. Hypersensitive dentin: testing of procedures for mechanical and chemical obliteration of dentinal tubuli. J Periodontol. 1993 May;64(5):366-73.
29. Poulsen S, Errboe M, Lescay MY, et al. Potassium containing toothpastes for dentine hypersensitivity. Cochrane Database Syst Rev. 2006 Jul 19;(3):CD001476.
30. Nagata T, Ishida H, Shinohara H, et al. Clinical evaluation of a potassium nitrate dentifrice for the treatment of dentinal hypersensitivity. J Clin Periodontol. 1994 Mar;21(3):217-21.
31. Schiff T, Dotson M, Cohen S, et al. Efficacy of a dentifrice containing potassium nitrate, soluble pyrophosphate, PVM/MA copolymer, and sodium fluoride on dentinal hypersensitivity: a twelve-week clinical study. J Clin Dent. 1994;5 Spec No:87-92.
32. Silverman G. The sensitivity-reducing effect of brushing with a potassium nitrate-sodium monofluorophosphate dentifrice. Compend Contin Educ Dent. 1985 Feb;6(2):131-3, 136.
33. Schiff T, Dos Santos M, Laffi S, et al. Efficacy of a dentifrice containing 5% potassium nitrate and 1500 ppm sodium monofluorophosphate in a precipitated calcium carbonate base on dentinal hypersensitivity. J Clin Dent. 1998;9(1):22-5.
34. Orchardson R, Gillam DG. The efficacy of potassium salts as agents for treating dentin hypersensitivity. J Orofac Pain. 2000 Winter;14(1):9-19.
35. Sowinski J, Ayad F, Petrone M, et al. Comparative investigations of the desensitizing efficacy of a new dentifrice. J Clin Periodontol. 2001 Nov;28(11):1032-6.
36. Wara-aswapati N, Krongnawakul D, Jiraviboon D, et al. The effect of a new toothpaste containing potassium nitrate and triclosan on gingival health, plaque formation and dentine hypersensitivity. J Clin Periodontol. 2005 Jan;32(1): 53-58.
37. West NX, Addy M, Jackson RJ, et al. Dentine hypersensitivity and the placebo response. A comparison of the effect of strontium acetate, potassium nitrate and fluoride toothpastes. J Clin Periodontol. 1997 Apr;24(4):209-15.
38. Stookey GK, Mau MS, Isaacs RL, et al. The relative anticaries effectiveness of three fluoride containing dentifrices in Puerto Rico. Caries Res. 2004 Nov-Dec;38(6):542-50.
39. Sharma N, He T, Barker ML, et al. Plaque control evaluation of a stabilized stannous fluoride dentifrice compared to a triclosan dentifrice in a six-week trial. J Clin Dent. 2013;24(1):31-6.
40. Lussi A, Carvalho TS, The future of fluorides and other protective agents in erosion prevention, Caries Res. 49 (2015) 18-29.
41. Mallatt M, Mankodi S, Bauroth K, et al. A controlled 6-month clinical trial to study the effects of a stannous fluoride dentifrice on gingivitis. J Clin Periodontol. 2007 Sep;34(9):762-7.
42. Farrell S, Barker ML, Gerlach RW. Overnight malodor effect with a 0.454% stabilized stannous fluoride sodium hexametaphosphate dentifrice. Compend Contin Educ Dent. 2007 Dec;28(12):658-61.
43. Baig A, He T. A novel dentifrice technology for advanced oral health protection: A review of technical and clinical data. Comped Contin Educ Dent. 2005 Sep;26(9 Suppl 1):4-11.
44. Miller S, Truong T, Heu R, et al. Recent advances in stannous fluoride technology: antibacterial efficacy and mechanism of action toward hypersensitivity. Int Dent J. 1994 Feb;44(Suppl 1):83-98.
45. West N, He T, Zou Y, DiGennaro J, Biesbrock A, Davies M. Bioavailable gluconate chelated stannous fluoride toothpaste meta-analyses; effects on dentin hypersensitivity and enamel erosion. J of Dent 105 (2021) 1-11.

46. Konradsson K, Lingstrom P, Emilson C-G, Johannsen G, Ramberg P, Johannsen A. Stabilized stannous fluoride dentifrice in relation to dental caries, dental erosion and dentine hypersensitivity; a systematic review. Am J Dent 2020; 33:95-105.
47. Sharif M, Iram, S, Brunton P. Effectiveness of arginine-containing toothpastes in treating dentine hypersensitivity: a systematic review. J of Dent 41(6), 483-492, 2013.
48. Blong MA, Volding B, Thrash WJ, et al. Effects of a gel containing 0.4 percent stannous fluoride on dentinal hypersensitivity. Dent Hyg (Chic). 1985 Nov;59(11):489-92.
49. Snyder RA, Beck FM, Horton JE. The efficacy of a 0.4% stannous fluoride gel on root surface hypersensitivity. J Dent Res. 1985;62:201. Abstract.
50. Thrash WJ, Jones DL, Dodds WJ. Effect of a fluoride solution on dentinal hypersensitivity. Am J Dent. 1992 Dec;5(6):299-302.
51. Thrash WJ, Dodds MW, Jones DL. The effect of stannous fluoride on dentinal hypersensitivity. Int Dent J. 1994 Feb;44(1 Suppl 1):107-18.
52. Lanzalaco AC, Dykman AG, Shaffer JB, et al. In Situ Iodide permeability of root dentin following use of two SnF2 products. 1996 ADA/FDI World Dental Congress. Abstract. Accessed May 15, 2018. Accessed May 15, 2018.
53. Ellingsen JE, Rolla G. Treatment of dentin with stannous fluoride - SEM and electron microprobe study. Scand J Dent Res. 1987 Aug;95(4):281-286.
54. Addy M, Mostafa P. Dentine hypersensitivity. I. Effects produced by the uptake in vitro of metal ions, fluoride and formaldehyde onto dentine. J Oral Rehabil. 1988 Nov;15(6):575-85.
55. Miller JT, Shannon IL, Kilgore WG, et al. Use of water-free stannous fluoride-containing gel in the control of dental hypersensitivity. J Periodontol. 1969 Aug;40(8):490-1.
56. Schiff T, Saletta L, Baker RA, et al. Desensitizing effect of a stabilized stannous fluoride/Sodium hexametaphosphate dentifrice. Compend Contin Educ Dent. 2005 Sep;26(9 Suppl 1):35-40.
57. Schiff T, He T, Sagel L, et al. Efficacy and safety of a novel stabilized stannous fluoride and sodium hexametaphosphate dentifrice for dentinal hypersensitivity. J Contemp Dent Pract. 2006 May 1;7(2):1-8.
58. He T, Chang J, Cheng R, et al. Clinical evaluation of the fast onset and sustained sensitivity relief of a 0.045% stannous fluoride dentifrice compared to an 8.0% arginine-calcium carbonate sodium monofluorophosphate dentifrice. Am J Dent. 2011 Dec;24(6):336.40.
59. He T, Barker ML, Qaqish J, et al. Fast onset sensitivity relief of a 0.454% stannous fluoride dentifrice. J Clin Dent. 2011;22(2):46-50.
60. He T, Baker R, Bartizek RD, et al. Extrinsic stain removal efficacy of a stannous fluoride dentifrice with sodium hexametaphosphate. J Clin Dent. 2007;18(1):7-11.
61. Gerlach RW, Underwood J, Miner M. A Randomized Clinical Trial Evaluating a 2-step Stannous Fluoride Dentifrice and Whitening Gel System Versus a Potassium Nitrate Dentifrice for Sensitivity Relief. Data on file. 2016.
62. Gendreau L, Barlow AP, Mason SC. Overview of the clinical evidence for the use of NovaMin in providing relief from the pain of dentin hypersensitivity. J Clin Dent. 2011;22(3):90-5.
63. Terézhalmy G, Chaves E, Bsoul S, et al. Clinical evaluation of the stain removal efficacy of a novel stannous fluoride and sodium hexametaphosphate dentifrice. Am J Dent. 2007 Feb;20(1):53-8.
64. Cunha-Cruz J, Stout JR, Heaton LJ, et al. Dentin hypersensitivity and oxalates: a systematic review. J Dent Res. 2011 Mar;90(3):304-10.
65. Greenhill JD, Pashley DH. The effects of desensitizing agents on the hydraulic conductance of human dentin in vitro. J Dent Res. 1981;60:686-98.
66. Sharma D, McGuire JA, Amini P. Randomized trial of the clinical efficacy of a potassium oxalate-containing mouthrinse in rapid relief of dentin sensitivity. J Clin Dent. 2013;24(2):62-7.
67. Cunha-Cruz J, Wataha JC, Zhou L, et al. Treating dentin hypersensitivity: therapeutic choices made by dentists of the northwest PRECEDENT network. J Am Dent Assoc. 2010 Sep;141(9):1097-105.
68. Cummins D. Recent advances in dentin hypersensitivity: clinically proven treatments for instant and lasting sensitivity relief. Am J Dent. 2010 May;23 Spec No A:3A-13A.

69. Camilotti V, Zilly J, Busato Pdo M, et al. Desensitizing treatments for dentin hypersensitivity: a randomized, split-mouth clinical trial. Braz Oral Res. 2012 May-Jun; 26(3):263-8.
70. Pashley DH. Mechanism of dentin sensitivity. Dent Clin North Am. 1990 Jul;34(3):449-73.
71. Lan WH, Liu HC, Lin CP. The combined occluding effect of sodium fluoride varnish and Nd:YAG laser irradiation on human dentinal tubules. J Endod. 1999 Jun;25(6):424-6.
72. Hoang-Dao BT, Hoang-Tu T, Tran-Thi NN, et al. Clinical efficiency of a natural resin fluoride varnish (Shellac F) in reducing dentin hypersensitivity. J Oral Rehabil. 2009 Feb;36(2):124-31.
73. Ritter AV, del Dias W, Miguez P, et al. Treating cervical dentin hypersensitivity with fluoride varnish: a randomized clinical study. J Am Dent Assoc. 2006 Jul;137(7):1013-20.
74. Aranha AC, Pimenta LA, Marchi GM. Clinical evaluation of desensitizing treatment for cervical dentin hypersensitivity. Braz Oral Res. 2009 Jul-Sep;23(3):333-9.
75. Lambert RL. Topical fluoride treatment of cavity preparations. Chap. 36. In: Goldman HM (ed). Current Therapy in Dentistry, Vol 5, St. Louis, C.V. Mosby Company. 1974, pp. 280-282.
76. Krauser JT. Hypersensitive teeth. Part II: Treatment. J Prosthet Dent. 1986 Sep;56(3):307-311.
77. Malloy CM, Shannon IL. A single solution mixture of fluorides for treatment of cavity preparation. Gen Dent. 1982 May-Jun;30(3):225-27.
78. Clark MB, Slayton RL. Fluoride use in caries prevention in the primary care setting. Pediatrics. 2014 Sep;134(3):626-33.
79. Schiff T, Delgado E, Zhang YP, et al. Clinical evaluation of the efficacy of an in-office desensitizing paste containing 8% arginine and calcium carbonate in providing instant and lasting relief of dentin hypersensitivity. Am J Dent. 2009 Mar;22(Spec No A):8A-15A.
80. Carson SJ. Possible role for arginine-containing toothpastes in managing dentine hypersensitivity. Evid Based Dent. 2013;14(2):44-5.
81. Ayad F, Ayad N, Delgado E, et al. Comparing the efficacy in providing instant relief of dentin hypersensitivity of a new toothpaste containing 8.0% arginine, calcium carbonate, and 1450 ppm fluoride to a benchmark desensitizing toothpaste containing 2% potassium ion and 1450 ppm fluoride, and to a control toothpaste with 1450 ppm fluoride: a three-day clinical study in Mississauga, Canada. J Clin Dent. 2009;20(4):115-22.
82. Kakar A, Kakar K, Sreenivasan PK, et al. Comparison of the clinical efficacy of a new dentifrice containing 8.0% arginine, calcium carbonate, and 1000 ppm fluoride to a commercially available sensitive toothpaste containing 2% potassium ion on dentin hypersensitivity: a randomized clinical trial. J Clin Dent. 2012;23(2):40-7.
83. Chu CH, Lo EC. Immediate post-application effect of professional prophylaxis with 8% arginine-calcium carbonate desensitizing paste on hypersensitive teeth. A practioner-based clinical trial. Am J Dent. 2014 Feb;27(1):7-11.
84. Collins JR, Richardson D, Sotero K, et al. Beneficial effects of an arginine-calcium carbonate desensitizing paste for treatment of dentin hypersensitivity. Am J Dent. 2013 Apr;26(2):63-7.
85. Elias Boneta AR, Ramirez K, Naboa J, et al. Efficacy in reducing dentine hypersensitivity of a regimen using a toothpaste containing 8% arginine and calcium carbonate, a mouthwash containing 0.8% arginine, pyrophosphate and PVM/MA copolymer and a toothbrush compared to potassium and negative control regimens: An eight-week randomized clinical trial. J Dent. 2013 Mar;41 Suppl:S42-49.
86. Gillam DG, Tang JY, Mordan NJ, et al. The effects of a novel Bioglass dentifrice on dentine sensitivity: a scanning electron microscopy investigation. J Oral Rehabil. 2002 Apr;29(4):305-13.
87. Du Min O, Tai BJ, Jiang H, et al. Efficacy of dentifrice containing bioactive glass (NovaMin) on dentine hypersensitivity. J Dent Res. 2004;83(Special Issue A): Abstract 1546.
88. Burwell A, Jennings D, Muscle D, et al. NovaMin and dentin hypersensitivity--in vitro evidence of efficacy. J Clin Dent. 2010;21(3):66-71.
89. Salian S, Thakur S, Kulkarni S, et al. A randomized controlled clinical study evaluating the efficacy of two desensitizing dentifrices. J Clin Dent. 2010;21(3):82-7.
90. Sharma N, Roy S, Kakar A, et al. A clinical study comparing oral formulations containing 7.5% calcium sodium phosphosilicate (NovaMin), 5% potassium nitrate, and 0.4% stannous fluoride for the management of dentin hypersensitivity. J Clin Dent. 2010;21(3):88-92.

91. Gendreau L, Barlow AP, Mason SC. Overview of the clinical evidence for the use of NovaMin in providing relief from the pain of dentin hypersensitivity. J Clin Dent. 2011;22(3):90-5.
92. Neuhaus KW, Milleman JL, Milleman KR, et al. Effectiveness of a calcium sodium phosphosilicate containing prophylaxis paste in reducing dentine hypersensitivity immediately and 4 weeks after a single application: a double-blind randomized controlled trial. J Clin Periodontol. 2013 Apr;40(4):349-57.
93. Kimura Y, Wilder-Smith P, Matsumoto K. Lasers in endodontics: a review. Int Endodont J. 2000 May;33(3):173-85.
94. Pesevska S, Nakova M, Ivanovski K, et al. Dentinal hypersensitivity following scaling and root planing: comparison of low-level laser and topical fluoride treatment. Lasers Med Sci. 2010 Sep;25(5):647-50.
95. Kara C, Orbak R. Comparative evaluation of Nd:YAG laser and fluoride varnish for the treatment of dentinal hypersensitivity. J Endod. 2009 Jul;35(7):971-4.
96. Yilmaz HG, Kurtulmus-Yilmaz S, Cengiz E. Long-term effect of diode laser irradiation compared to sodium fluoride varnish in the treatment of dentine hypersensitivity in periodontal maintenance patients: a randomized controlled clinical study. Photomed Laser Surg. 2011 Nov;29(11):721-5.
97. Raichur PS, Setty SB, Thakur SL. Comparative evaluation of diode laser, stannous fluoride gel, and potassium nitrate gel in the treatment of dentinal hypersensitivity. Gen Dent. 2013 May-Jun;61(3):66-71.
98. Sgolastra F, Petrucci A, Gatto R, et al. Effectiveness of laser in dentinal hypersensitivity treatment: a systematic review. J Endod. 2011 Mar;37(3):297-303.

About the Author

Patricia A. Walters, RDH, MSDH, MSOB

Ms. Walters is an independent contractor. After earning an MS in Oral Biology and an MS in Dental Hygiene Education from the University of Missouri-Kansas City, she began her career in Dental Hygiene Education at the University of Texas at San Antonio followed by 17 years at P&G in clinical trials research and Professional Relations in the area of Oral Care and currently working with Cincinnati Consulting Inc.

Email: paw2161@yahoo.com