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Dentinal Hypersensitivity: A Review

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Abstract

Dentinal hypersensitivity is generally reported by the patient after experiencing a sharp pain caused by one of several different stimuli. The pain response varies substantially from one person to another. The condition generally involves the facial surfaces of teeth near the cervical aspect and is very common in premolars and canines. The most widely accepted theory of how the pain occurs is Brannstrom's hydrodynamic theory, fluid movement within the dentinal tubules. The dental professional, using a variety of diagnostic techniques, will discern the condition from other conditions that may cause sensitive teeth. Treatment of the condition can be invasive or non-invasive in nature. The most inexpensive and efficacious first line of treatment for most patients is a dentifrice containing a desensitizing active ingredient such as potassium nitrate and/or stannous fluoride. This review will address the prevalence, diagnosis, and treatment of dentinal hypersensitivity. In addition the home care recommendations will focus on desensitizing dentifrices.

Keywords: Dentinal hypersensitivity, hydrodynamic theory, stannous fluoride, potassium nitrate

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Introduction

Dentifrices and mouth rinses are routinely used as a delivery system for therapeutic agents such as antimicrobials and anti-sensitivity agents. Therapeutic oral care products are available to assist the patient in the control of dental caries, calculus formation, and dentinal hypersensitivity to name a few. The dental practitioner makes recommendations regarding selection of the appropriate therapeutic dentifrice based on diagnosis of the disease or condition. These recommendations are based on extensive knowledge of the etiology of the disease/condition, the mechanism of action of the various active agents in the dentifrice and mouth rinse, and the host's needs and response to treatment.

This review will address the etiology of the condition commonly referred to as "dentinal hypersensitivity" or "tooth sensitivity." More specifically, this paper will review the prevalence and diagnosis of the condition as well as reviewing clinical evidence behind popular home care recommendations.

Prevalence

Dentinal hypersensitivity is generally reported by the patient after experiencing a sharp pain caused by one of several different stimuli (Figure 1).

The prevalence of dentinal hypersensitivity has been reported over the years in a variety of ways: as greater than 40 million people in the U.S. annually[1], 14.3% of all dental patients[2], between 8% and 57% of adult dentate population[3], and up to 30% of adults at some time during their lifetime.[4]

Dentinal hypersensitivity has been shown to peak in 20 to 30 year olds and then rise again when in their 50's.[4, 5] The condition generally involves the facial surfaces of teeth near the cervical aspect and is very common in premolars and canines.4 Patients undergoing periodontal treatment are particularly susceptible to this condition because of the recession following periodontal surgery or loss of cementum following non-surgical periodontal therapy.[6, 7] In addition periodontal disease and improper brushing habits can also result in gingival recession accompanied by sensitive teeth. Dentinal hypersensitivity has been researched extensively through the years and many authors express an agreement that dentinal hypersensitivity is either under-reported by the dental patient population or misdiagnosed.

Theories

Several theories have been cited to explain the mechanism involved in dentinal hypersensitivity.[8] The transducer theory, the modulation theory, the "gate" control and vibration theory, and the hydrodynamic theory have all been presented and discussed throughout

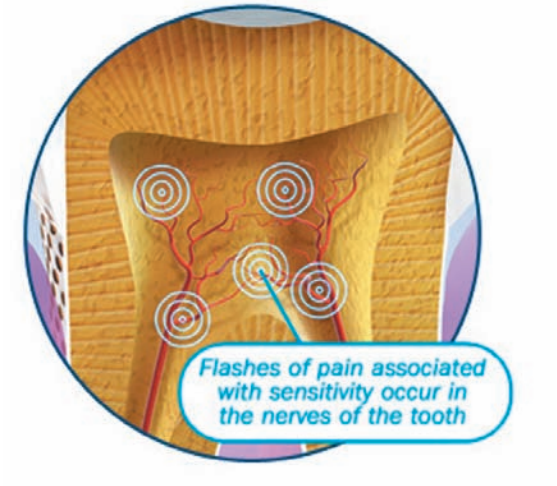

Figure 1. Pictorial display of origin of pain associated with sensitive teeth.

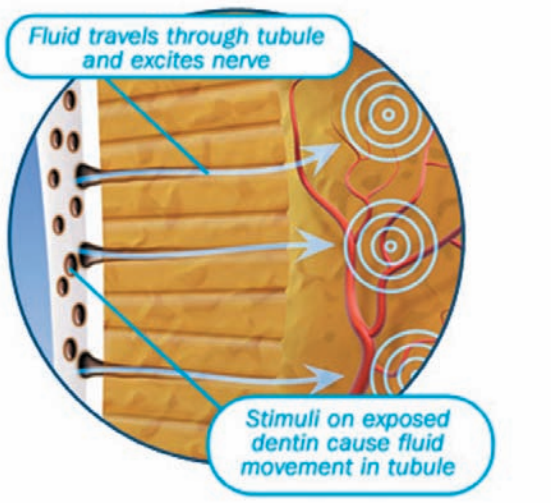

Figure 2. Depiction of Brannstrom's Theory.

the years. The latter, "hydrodynamic theory", developed in the 1960's and based upon two decades of research, is widely accepted as the cause of tooth sensitivity.[9] Assumptions of the hydrodynamic theory conclude that when the fluids within the dentinal tubules 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 (Figure 2).

The various stimuli that are reported to cause this transmission of sensation are cold, hot, osmotic, electrical, dehydration, and chemical.[10] Berman[8] 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 fluid and cold 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. The disease is characterized by a short, sharp pain arising from exposed dentin in response to a stimulus that cannot be ascribed to any other form of dental defect or pathology.[11, 12] Therefore, 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.

The most important variable affecting the fluid flow in dentin is the radius of the tubuli. If the radius is reduced by one-half, the fluid flow within the tubuli falls to one-sixteenth of its original rate. Consequently, the creation of a smear layer or obliteration of the tubule can greatly increase the effectiveness of the treatment of this malady.[13, 14]

Diagnosis

The reason(s) for tubules to be exposed or open should be assessed during a visual examination of the teeth as well as a detailed dietary history. 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. A comprehensive dental examination will ultimately rule out other underlying conditions for which sensitivity is a symptom such as cracked tooth, fractured restoration, chipped teeth, dental caries, gingival inflammation, post-restorative sensitivity, marginal leakage, and pulpitis. Excessive dietary acids such as citrus juices and fruits, carbonated drinks, wines, and ciders have been identified as potential risk factors for dental hypersensitivity.[4, 9, 11] The dietary history provided by the patient will assist in identifying the risk factors the patient may have for tooth sensitivity.

In addition other risk factors will 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. 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.[15]

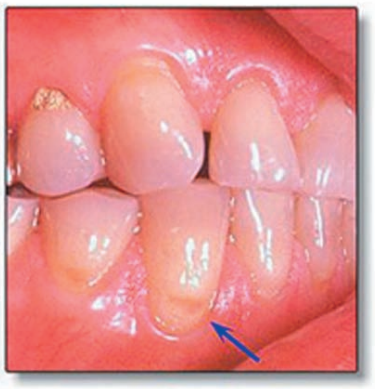

Figure 3. Tooth abrasion. (Courtesy, Dr. Beatrice Gandara, University of Washington, School of Dentistry)

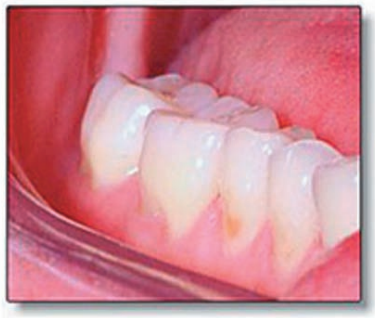

Figure 4. Tooth erosion. (Courtesy, Dr. Beatrice Gandara, University of Washington, School of Dentistry)

The most common cited reason for exposed dentinal tubules is gingival recession (predisposing factor).[16] Chronic exposure to bacterial plaque, toothbrush abrasion, gingival laceration from oral habits such as toothpick use, excessive flossing, crown preparation, inadequate attached gingiva, and gingival loss secondary to disease or surgery are some but not all causes of gingival recession.[16] Gingival recession is the reduction of the height of the marginal gingiva to a location apical to the CEJ. Recessed areas may become sensitive due to the loss of cementum, ultimately exposing dentin. Probing depths, recessed areas, and sensitivity reported by the patient must be accurately recorded and monitored to provide a reference for the patient's disease activity over time.

Treatments

Treating dentinal hypersensitivity can be challenging for the dental professional because of the difficulty related to measuring the pain response since the response varies 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(s). If the diagnosis confirms dentinal hypersensitivity in the absence of underlying diseases or structural problems, then the following steps can be initiated: (1) remove the risk factors by educating the patient about dietary acids and other oral care habits; (2) recommend different toothbrushing methods, if appropriate; (3) initiate treatment by recommending a desensitizing agent for home use; or (4) applying topical desensitizing agents professionally.

Treatment can be invasive in nature or non-invasive. Invasive procedures may include gingival surgery, application of resins, or a pulpectomy. In addition it has been reported that four kinds of lasers have been used for the treatment of dentinal hypersensitivity and the effectiveness ranged from 5.2 to 100%, which was dependent on the laser type and parameters used.[17] The mechanism involved in laser treatment for this condition is unknown at this time.

Non-invasive treatment options are topical agents and dentifrices that contain a desensitizing active ingredient. These are considered to be the simplest, cost-effective, and efficacious first line of treatment for most patients.[18] According to the literature, the most widely available desensitizing toothpaste ingredient is potassium nitrate.[19] The potassium ions are thought to block the synapse between nerve cells, reducing nerve excitation and the associated pain. Another active ingredient that exhibits a similar mechanism is potassium chloride.

Other treatments are designed to reduce flow into the dentin tubules by occluding or sclerosing the tubules. Active ingredients include stannous fluoride, strontium chloride hexahydrate, and aluminum, potassium or ferric oxalates and fluorides (Figure 5).

Other active agents that have been proven to be effective as a desensitizing agent are dentin sealers (resins), sodium citrate, and sodium monofluorophosphate.

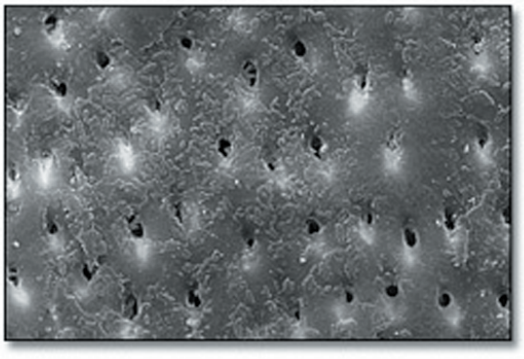

Figure 5a. Open tubules following treatment with non-sensitivity fluoride toothpaste.

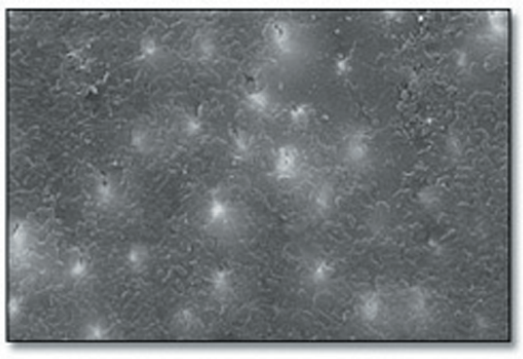

Figure 5b. Closed tubules following treatment with SnF_2 dentifrice.

More recently, dentifrices have been introduced that contain a combination of a desensitizing agent, fluoride source, anticalculus, and/or whitening ingredients to provide multiple therapeutic and cosmetic benefits. There are also new sensitivity dentifrices with improved flavors and esthetics, following trends in the general dentifrice category (Figure 6). These products may be attractive for patients with dentinal hypersensitivity because of their multiple benefits and/or esthetic attributes.

Figure 6. New Crest Sensitivity Dentifrices.

Currently, desensitizing dentifrices that carry the American Dental Association (ADA) Seal of Acceptance[20] for prevention of tooth sensitivity are Crest Sensitivity Protection Soothing Whitening Mint Toothpaste and Orajel Sensitive Pain Relieving Toothpaste for Adults. A few fluoride desensitizing products (Sultan 33 1/3% NaF and Gel-Kam 0.4% SnF_2) sold through a dental professional also carry the seal. (www.ada.org/ada/seal/index.asp)

There are several other dentifrices available over-the-counter for consumers to purchase that also claim they reduce tooth sensitivity that do not carry the ADA seal. In addition the literature mentions fluoride varnishes and sealants as a possible treatment for dentinal hypersensitivity.[21, 22]

In making treatment recommendations it is important to have an understanding of the clinical data that support the product's efficacy and safety. Reviewing the literature is a good approach to better evaluate the product benefits and make appropriate treatment planning. A review of published data on two ingredients most frequently used to treat hypersensitivity, potassium nitrate and stannous fluoride, follows:

Potassium Nitrate (KNO_3)

A number of studies, published since the early seventies, have investigated the use of potassium nitrate (KNO_3) as an effective active ingredient in treating dentinal hypersensitivity.[22, 23, 24, 25, 26] In order to address the effectiveness of potassium nitrate (5%) in a dentifrice to reduce dentinal hypersensitivity it will be necessary to compare studies using similar, controlled reproducible stimuli and objective measurements. It is often desirable to evaluate product efficacy relative to other dentifrices marketed for that purpose. Over time, investigators have chosen various methods to capture subjective responses, but it is not known which methods used are more valid than others.

A four-week exposure time is widely used in clinical trials because results have shown that this time is needed for 5% KNO_3 to exert its desensitizing effect.[22] Cited studies in this review range in exposure time from 2 weeks to 12 weeks with little rationale given for why the time was selected. In clinical trials of desensitizing agents, the use of a broadly accepted positive or negative control toothpaste formulation or product is recommended because the condition itself can appear to be self-resolving within the time scale of the study. A benchmark control, a branded fluoride toothpaste[23], has been increasingly used over the years in comparison studies.

Mechanism of Action

Tarbet et al.[24] studied the affect of a 5% KNO_3 dentifrice to the tooth surface to determine safety of the product. The dentifrice was used two times daily for four weeks. The teeth, scheduled for extraction, were examined microscopically at the end of product use for any histological discernible alterations. The results demonstrated the 5% KNO_3 dentifrice did not cause any observable tooth surface changes.

The potassium ions in potassium nitrate desensitizing products are reported to work by blocking the synapse between nerve cells, reducing nerve excitation, and the associated pain.[24, 25, 26, 27, 28]

Other research has shown 5% KNO_3 can obliterate the tubules. Knight et al.[24] reported

results of various mechanical and chemical procedures in obliterating dentinal tubuli by utilizing the scanning electron microscope. The toothpaste (5% KNO_3) and a professionally applied sealant product (6% ferric oxalate) both produced some obliteration of the tubuli. The findings indicate, if the radius of the tubuli is reduced via obliteration, the fluid flow within the tubuli falls, ultimately reducing dentin hypersensitivity.[25]

Efficacy Data

In 2004 the Cochrane Collaboration published a systematic review[26] of potassium nitrate toothpastes for the treatment of dentinal hypersensitivity based on clinical trials conducted up to the year 2000 involving KNO_3 toothpaste compared to non-KNO_3 toothpaste. This review focused on studies that incorporated similar methods in order to determine if KNO_3 is an effective agent in reducing dentinal hypersensitivity. The results were obtained by measuring tactile, thermal, and air blast stimuli as well as patients' subjective assessment of pain during every day life. The exposure periods ranged from six to eight weeks, reporting outcome measurements as mean change from baseline.

The final comparison in the Cochrane review included four studies[27, 28, 29, 30], and all showed significant differences in mean sensitivity scores at all time points assessed by tactile, air blast, and thermal stimulation. The authors concluded the support for the efficacy of potassium nitrate toothpaste for dentinal hypersensitivity was based on a sample size of 245 subjects.

In 2000 Orchardson et al.[31] evaluated the clinical evidence that potassium salts are effective desensitizing agents. Thirteen studies are cited as using a 5% KNO_3 toothpaste in double-blind, randomized designs. Again, the authors note the variability in exposure time, methods of measurements, and other design elements making it difficult to generalize results. For example, the median duration of the trials was eight weeks. Some of the studies utilized the Yeaple probe (Figure 7) for the objective measurement and an air sensitivity scale described by Schiff et al.[27] and Sowinski et al.[31] The most commonly used sensitivity measures were tactile stimulation, air blast stimulation, and subjective questionnaires.

The conclusions for all studies assessed in the review indicate toothpaste containing 5% KNO_3 significantly reduced dentinal hypersensitivity to both tactile and air blast stimuli as well as subjective response. Some of the studies assessed were similar to those used in the Cochrane review. In 2004 Wara-aswapati et al.[33] studied an experimental toothpaste containing 5% KNO_3 and other active ingredients aimed at reducing plaque formation and inflammation, in addition to reducing sensitivity. The duration of the study was 12 weeks of home use and the design was double-blind, randomized, parallel group comparison of three toothpaste groups. This study supports previously reported outcomes that 5% KNO_3 toothpaste effectively reduces dentinal hypersensitivity.

In comparison, results of some studies showed little or no effectiveness of KNO_3. For example, West et al.[33] compared three commercially available dentifrices, including a potassium nitrate dentifrice for the alleviation of dentinal hypersensitivity using a conventional fluoride dentifrice as the control. The design included a four week lead phase utilizing the control product in an attempt to reduce the variability in pain reduction produced by previous dentifrice use and to provide information on placebo effects. Instead

Figure 7. Illustration of the Yeaple Probe.

of a four week exposure period, West et al. used a six week usage period with brushing two times daily. Subjective pain scales were used in response to a cold air stimulus.[34]

The subjective pain questionnaire used a visual analog scale of 0-10 for rating the severity of tooth pain, 0 = no pain and 10 = excruciating pain. This scale is based on the subjects' perception of the pain experienced during everyday routine from hot/cold food or drink, cold air, tooth brushing, or sweet/sour foods. The tactile sensitivity was determined using a straight probe drawn across the cervical area of each tooth, and the cold air blast responses were again rated on subjects' perception of pain. The results of this study demonstrated a trend towards reduction of dentinal hypersensitivity over time for all variables (overall sensitivity score, tactile stimulus, and cold air stimulus) independent of treatment group. Although not statistically significant, overall results indicated a trend towards reduction in sensitivity measured by all variables (tactile, cold air, and general sensitivity) for all three treatment groups.

Over the years, the effectiveness of KNO_3 dentifrice in reducing dentinal hypersensitivity has been extensively researched. More research can be done concerning the mode of action, the potential synergistic action with other toothpaste ingredients, as well as the various design methods used by investigators. When comparing studies using similar methods and subjective measurements, there is clear evidence KNO_3 is effective in reducing pain due to tooth sensitivity.

Stannous Fluoride

Stannous fluoride (SNF_2) has been shown to be effective in the prevention of dental caries[35], reduction of plaque formation[36], control of gingivitis[37, 38], and as suppression of breath malodor.[39] Research shows stannous fluoride is effective against dentinal hypersensitivity as well.[40, 41, 42, 43] The ADA has recognized the desensitizing properties of stannous fluoride gel 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.[18]

Method of Action

In situ research shows root dentin treated with stannous fluoride exhibits tubule occlusion.[44] Several other studies using analysis by scanning electron microscopy showed that partial or complete occlusion of dentin tubules occurred after treatment with SNF_2.[45, 46] In addition Miller et al.[36] reported a tin-rich surface deposit forms in vitro and in situ with two weeks use of an anhydrous 0.4% stannous fluoride gel, providing nearly complete surface coverage and occlusion of the tubules. When the tubules are blocked, the stimulation of the mechanoreceptors does not occur, thus, preventing the pain response.

Efficacy Data

Stannous fluoride has been delivered via a mouth rinse, dentifrice, and gel for some time. In 1985 Blong et al.[40], using a precise thermo-electric stimulator, demonstrated 0.4% SNF_2 gel as an effective agent in reducing dentinal hypersensitivity when used twice a day over a two week period. Conclusions supported prolonged use (up to four weeks) and consistent use in order to achieve this affect. This investigation supports the previous work of Miller et al.[47] further establishing the effectiveness of the 0.4% stannous fluoride gel on reducing pain associated with dentinal hypersensitivity.

More recent research, Thrash et al.[42, 43] supports the theory the time required for a decrease in sensitivity is between two and four weeks from initiation of treatment. Thrash and colleagues compared a 0.4% stannous fluoride gel to an aqueous 0.717% fluoride solution and a placebo at 2, 4, 8, and 16 week intervals following a twice daily application. The results indicated subjects who applied the 0.4% SNF_2 reported significantly less sensitivity during the four to eight week period. The effect continued throughout the 16 week assessment period.

Another demonstration of stannous fluorides effect on sensitivity has been seen with the use of fluoride cavity washes. Topical stannous fluoride has been shown to reduce sensitivity on exposed cervical root surfaces.[47, 48, 49] The findings suggest topical SNF^2 cavity washes reduce thermal sensitivity following amalgam

restoration placement.[50, 51] Peterzen et al.[20] tested the theory in a clinical trial, and results indicated the treated tooth was less sensitive at most of the post-operative examinations as determined by patient report and thermal testing, therefore, recommending the use of SNF^2 as a cavity wash.

Historically, one limitation to its use 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 can be mitigated by incorporating certain tartar control and/or whitening ingredients in the formulation provided they do not suppress the desensitizing effects.

Conclusions

Dentinal hypersensitivity is a problem that plagues many dental patients. When a patient presents with dentinal hypersensitivity symptoms, they should be examined and informed of the multiple treatment options that may be necessary to eliminate the problem. The patient should be responsible for the decision making process since some of their daily habits may be contributing to the problem and if not changed the condition will persist.

The initial cause, in the majority of cases, is recessed gingiva. Once the tubules are exposed the patient will experience pain and the initial treatment choice is to cover up the tubules to desensitize the nerves (e.g., stannous fluoride) or interfere with the transmission of pain signal at the synapse (e.g., potassium nitrate). The product of choice is based on the scientific evidence that supports each active ingredient and the patient's preference for products that will fit most easily into his or her oral hygiene regimen. Based on the volume of scientific evidence, the most effective active ingredients available in toothpaste today to treat dentinal hypersensitivity may be potassium nitrate and stannous fluoride.

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Pat Walters is a Senior Scientist/Engineer at the Procter & Gamble Health Care Research Center in Cincinnati, OH, USA. 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. Her current position is at P&G in clinical trials research in the area of Oral Care.