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Desensitizing agents in dentistry

A project

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بِسْمِ ٱللَّهِ ٱلرَّحْمَٰنِ ٱلرَّحِيمِ

۞ وَعِندَهُۥ مَفَاتِحُ ٱلْغَيْبِ لَا يَعْلَمُهَآ إِلَّا هُوَ ۚ وَيَعْلَمُ مَا فِى ٱلْبَرِّ وَٱلْبَحْرِ ۚ وَمَا تَسْقُطُ مِن وَرَقَةٍ إِلَّا يَعْلَمُهَا وَلَا حَبَّةٍ فِى ظُلُمَٰتِ ٱلْأَرْضِ وَلَا رَطْبٍ وَلَا يَابِسٍ إِلَّا فِى كِتَٰبٍ مُّبِينٍ ﴿٥٩﴾

صَدَقَ اللهُ العَظِيمُ

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Dedication

To my parents who were their for me in every step of the way with their have love and support…

To my supervisor for his guidance, help and endless support throughout this project…

Dania Mohanned Dawood

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Introduction

In dentistry, there are various reasons that are capable of causing unbearable and agonizing pain and one such major cause of pain and discomfort is dentin hypersensitivity (DH). DH has been defined as a sharp, sudden, painful reaction when the teeth are exposed to hot, cold, chemical, mechanical, touch, or osmotic (sweet or salt) stimuli and cannot be attributed to any other form of dental pathology or defect ***(Holland GR, Narhi MN., et al. 1997).***

The hydrodynamic theory of dentin sensitivity is now widely accepted . dentin hypersensitivity is thought to be caused by displacement of dentine fluid within the dentin tubules . the dentin tubules of hypersensitive teeth are open, more numerous and larger than in normal teeth (***Yoshiyamam M, Noiri Y., et al 1990***).

Dentine sensitivity presents a challenge to the dentist. Modern treatments for hypersensitivity teeth are intended either to reduce tubular fluid movement by reducing dentin permeability or to reduce the excitability of interdental nerve with neutrally active agents (***Pashley DH., 1994***) A variety of materials, such as calcium hydroxide, cavity varnishes, topical fluorides, fluoride iontophoresis, laser irradiation, strontium chloride and potassium nitrate dentifrices have been used in an attempt to alleviate dentin sensitivity. Oxalates, glutaraldehyde, benzalkonium chloride and dentin bonding agents with and without resin-based composite are some of the materials currently being used for the treatment of this condition. ***(Jain P, Reinhardt JW., et al. 2000).***

There are five different types of stimuli that can trigger pain when dentine is exposed:

A. **Tactile (mechanical) stimulation** can be attributed to toothbrush bristles or filaments, friction from dental clasp or prosthesis and metal object such as eating utensils or dental instruments.

B. **Chemical stimuli** are possibly the most overlooked triggers of dentin hypersensitivity. Acids presents in many foods and beverages: such as citrus fruit, vitamins, condiments, spices, wine, sauces and carbonated drinks should be suspected more than any other stimuli of dentin pain .

C. **Acid foods and drinks** have been shown to soften dentin and may remove deposits on the dentin surface. Ascorbic acid, from chewable vitamin C tablets, can even be stimulus.

D. Up to 9 percent of individuals suffering from dentin hypersensitivity report that the effect of a **thermal** a stimulus, particularly a cold stimulus such as breathing through the mouth on a cold day or consuming a cold drink, causing the painful sensation associated with sensitive teeth.

E. **Osmotic flow** within the dentinal tubules is important, there may be variations in the way in which different stimulus affect fluid flow.

F. **Bacteria produce acid** when fermentable carbohydrates are available, it is this acid when fermentable carbohydrates are available, it is this acid by-product, as it relates to demineralization or root caries, which can also cause sensitivity.

Since then, several authors have used other terms to describe (DH) by replacing the word dentine, adding site descriptors, such as cervical or root and joining this with either hypersensitivity or sensitivity. This practice resulting in a significant number of variations to describe the same condition. ***(Halland GR, Narhi MN, et al., 1997)***.

1.1. Prevalence and epidemiology

Various studies showed that the incidence of DH in most population ranges between 10-30% of the general population and the age range varies from 20-50 years with the peak incidence occurring at the end of the third decade and decreases during the fourth and fifth decades of life ***(Flynn J, Galloway R., et al 1985)*.**

In general, this incidence can vary considerably depending on the cohort being studied with periodontal patients, patients with gingival recession and the smokers with periodontitis showing the highest incidence of diagnosed dentinal hypersensitivity.

The teeth most commonly affected by dentinal hypersensitivity are the canines and premolars of both arches and buccal aspect of cervical area is the commonly affected site. It has been reported that there is a slightly higher incidence of dentine hypersensitivity in females compared to males. In contrary, one study showed no difference on prevalence of dentinal hypersensitivity in either gender, suggesting overall that as many males as females are susceptible. ***(Bartold PM., 2006)***

1.2 Etiopathogenesis

Dentine is covered and protected by hard tissues such as enamel or cementum. Dentin itself is a vital tissue, consisting of dentinal tubules, and is naturally sensitive because of extensions of odontoblasts and formation of dentine– pulp complex. Although dentin and pulp are histologically different, their origin is embryologically from the same precursor, i.e., the ectomesenchyme. Pulp is integrally connected to dentine, i.e., physiologic and/or pathologic reactions in one of the tissues will also affect the other. Dentin consists of small canal like spaces, dentinal tubules. These tubules occupied by odontoblastic processes.

The odontoblastic processes may extend through the entire thickness of dentin from pulp to dentino-enamel junction. The odontoblastic processes are actually the extensions of odontoblasts, which are the major cells of pulp–dentin complex.

The odontoblastic processes are surrounded by dentinal fluid inside the tubules.

The dentinal fluid forms around 22% of total volume of dentin. It is an ultrafiltrate of blood from the pulp via dentinal tubules and forms a communication medium between the pulp (via the odontoblastic layer) and outer regions of the dentin. ***(West NX, Sanz M., et al 2013).***

It has been stated in the literature that DH develops in two phases:
lesion localization and lesion initiation.

1. Lesion localization

In the first phase, dentinal tubules, due to loss of enamels, are exposed by attrition, abrasion, erosion, and abfraction.

However, dentinal exposure mostly occurs due to gingival recession along with the loss of cementum on the root surface of canines and premolars in the buccal surface. It is worth noticing that not all the exposed dentins are sensitive.

Their calcified smear layer, as compared to non sensitive dentin, is thin and this leads to an increase in the fluid movement and consequently the pain response. ***(Orchardson R, Cadden SW., 2001)***.

2. Lesion initiation

In the second phase, for the exposed dentin to be sensitized, tubular plugs and the smear layer are removed and consequently, dentinal tubular and pulp are exposed to the external environment. Plug and smear layer on the surface of exposed dentine are composed of elements of protein and sediments which are derived from salivary calcium phosphates and seal the dentinal tubules inconsistently and transiently.

The findings of laboratory research indicate that both mechanical and chemical factors are effective in removing the smear layer from the dentinal tubules.

However, the results of clinical investigations, the mechanical factors are not the only key factors in removal of the smear layer and when they are accompanied with acidic foods or drinks they lead to the removal of smear layer.

Lesion initiation requires removal of cementum or smear layers. This is achieved by abrasive or erosive agents.

The evidence available indicates erosion is the more dominant factor but can be potentiated by abrasion. (***Lussi A, .2006***).

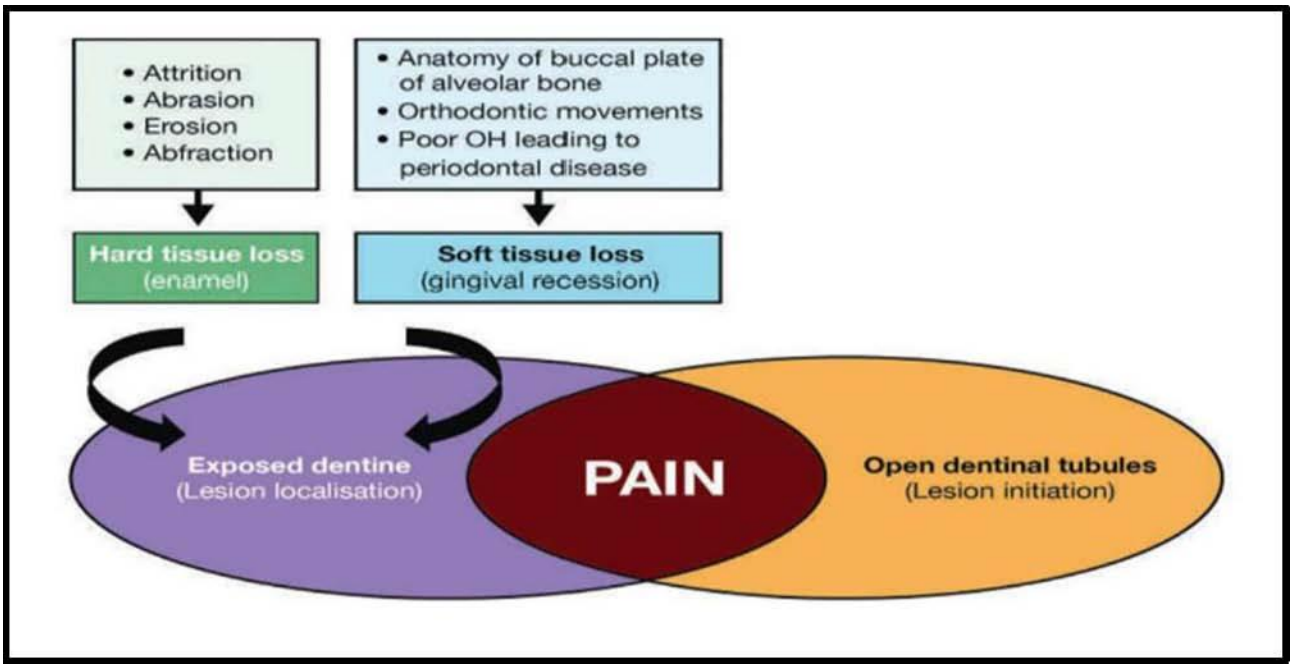


Figure (1): lesion localization and lesion initiation

Froma clinical point of view, dentinal hypersensitivity is caused by:

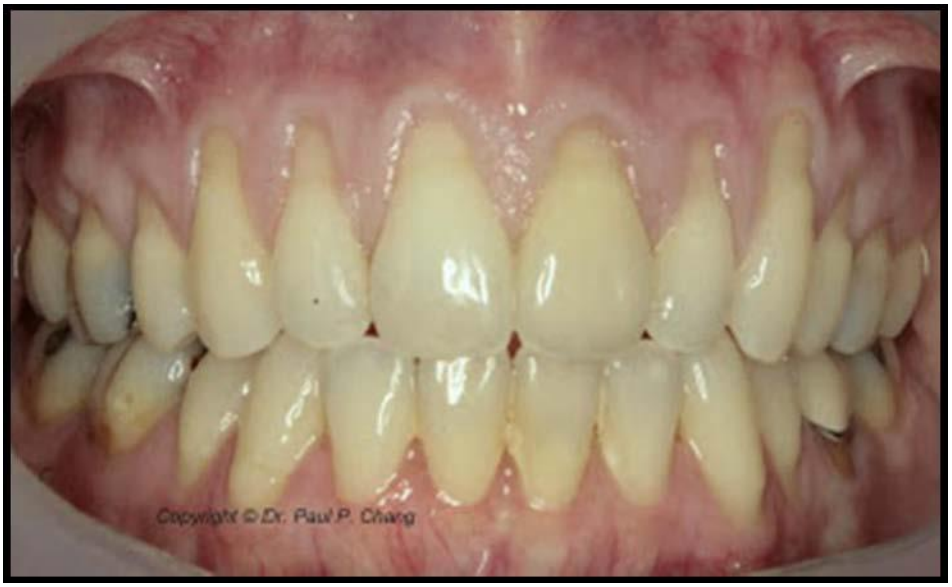
· gingival recession · tooth wear (abrasion, erosion, abfraction) · other factors.

1.2.2 Gingival recession

Gingival recession is usually recognized in patients with a high level of oral hygiene. The causes of gingival recession in population having good oral hygiene are due to overzealous tooth brushing, improper brushing technique or using of an excessive brushing forces and it is frequently seen on the buccal surfaces of the teeth.

On other hand lack of tooth brushing, with consequent accumulation of dental plaque on root surfaces in patient with bad oral hygiene may lead to periodontal complication and migration of gingiva in apical direction, exposing the cementum and then demineralization of tooth structure which could be associated with patency of dentine tubules orifices causing DH. (GillamDG,Orchardson R., 2006).

Figure (2)gingival recession



1.2.2 Tooth wear

It is considered an alternative pathway of cervical dentine exposure in the coronal aspect of the tooth due to the loss of dental hard tissue, especially enamel. It involves loss of enamel surface by attrition, abrasion, erosion or abfraction. Attrition is the loss of tooth substance or a restoration caused by tooth to tooth contact between opposing occluding or adjacent proximal surfaces as a result of parafunctional habits such as bruxism.

Abrasion is the loss of tooth structure caused by factores other than tooth contact as in case of over vigorous use of a tooth brush, or the consumption of abrasive and fibrous diets. Erosion is the progressive loss of hard dental tissues by chemical processes or acids not produced by cariogenic bacteria as in case of acidic agents associated with regurgitation or extrinsic acids associated with dietary and medication source. Abfraction lesions are wedge shape defects developed at the cervical region of the teeth and are not directly related to the diet, periodontal disease or abrasion. They occur as a result of mechanical overloading of cervical enamel regions initiated by cuspal flexure and occlusal over loading, resulting in fracture of the enamel crystals in this area with subsequent exposure of the underlying dentin (***Suge T, Kawasaki A., et al 2008***).

Figure (3) difference between dentalattrition,abfraction erosion and abrasion

1.2.3 Other factors

Moreover, dentinal hypersensitivity may also have an iatrogenic origin: in conservative treatments, when dentin is exposed to an etching acid for too long or as a result of periodontal surgery (mucogingival, resective or regenerative). ***(Orchardson R,Cadden SW.,2001).***

1.3 Theories and Mechanisms of sensitivity:

- Direct innervation theory (Neural theory).
- Odontoblast receptor (Odontoblastic transduction).
- Fluid movement/hydrodynamic theory. hree major mechanisms of dentinal sensitivity have been proposed in the literature:

1.3.1 Direct innervation theory (Neural theory)

According to direct this theory, nerve endings penetrate dentine and extend to the dentino-enamel junction. Direct mechanical stimulation of these nerves will initiate an action potential. There are many shortcomings of this theory. There is lack of evidence that outer dentin, which is usually the most sensitive part, is innervated. Developmental studies have shown that the plexus of Rashkow and intratubular nerves do not establish themselves until the tooth has erupted; yet, newly erupted tooth is sensitive. Moreover, pain inducers such as bradykinin fail to induce pain when applied to dentine, and bathing dentine with local anesthetic solutions does not prevent pain, which does so when applied to skin (***Canadian Advisory.,2003***).

1.3.2 Odontoblastreceptor (Odontoblastic transduction theory):

Odontoblast receptor theory states that odontoblasts acts as receptors by themselves and relay the signal to a nerve terminal. But majority of studies have shown that odontoblasts are matrix forming cells and hence they are not considered to be excitable cells, and no synapses have been demonstrated between odontoblasts and nerve terminals.(***Canadian Advisory.,2003***).

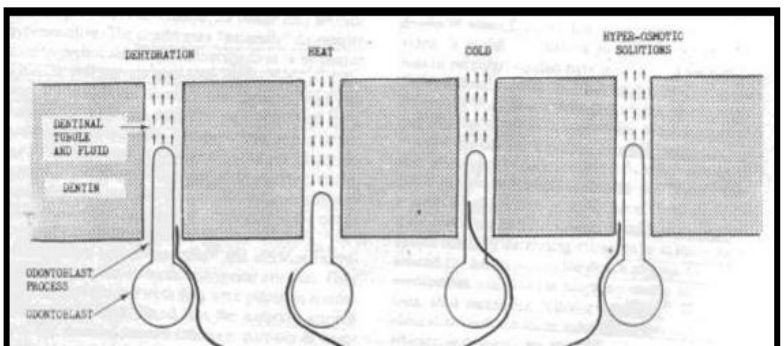


Figure (4) Transduction theory

1.3.3 Fluid movement theory (hydrodynamic theory):

Brannstrom (1964) has proposed that dentinal pain is due to hydrodynamic mechanism, i.e., fluid force. Scanning electron microscopic (SEM) analysis of "hypersensitive" dentin shows the presence of widely open dentinal tubules.

The presence of wide tubules in hypersensitive dentin is consistent with the hydrodynamic theory. This theory is based on the presence and movement of fluid inside the dentinal tubules. This centrifugal fluid movement, in turn, activates the nerve endings at the end of dentinal tubules or at the pulp–dentine complex. This is similar to the activation of nerve fibers surrounding the hair by touching or applying pressure to the hair.

The response of pulpal nerves, mainly A intradentinal afferent fibers, depends upon the pressure applied, i.e., intensity of stimuli. It has been noted that stimuli which tend to move the fluid away from the pulp–dentine complex produce more pain.

These stimuli include cooling, drying, evaporation and application of hypertonic chemical substances.

Approximately, 75% of patients with DH complain of pain with application of cold stimuli. In spite of the fact that fluid movement inside the dentinal tubules

produces pain, it should be noted that not all exposed dentine is sensitive.

As stated before, the “hypersensitive” dentin has more widely open tubules and thin/under calcified smear layer as compared with “non-sensitive” dentine. The wider tubules increase the fluid movement and thus the pain response. (***Orchardson R,Cadden SW., 2001***).

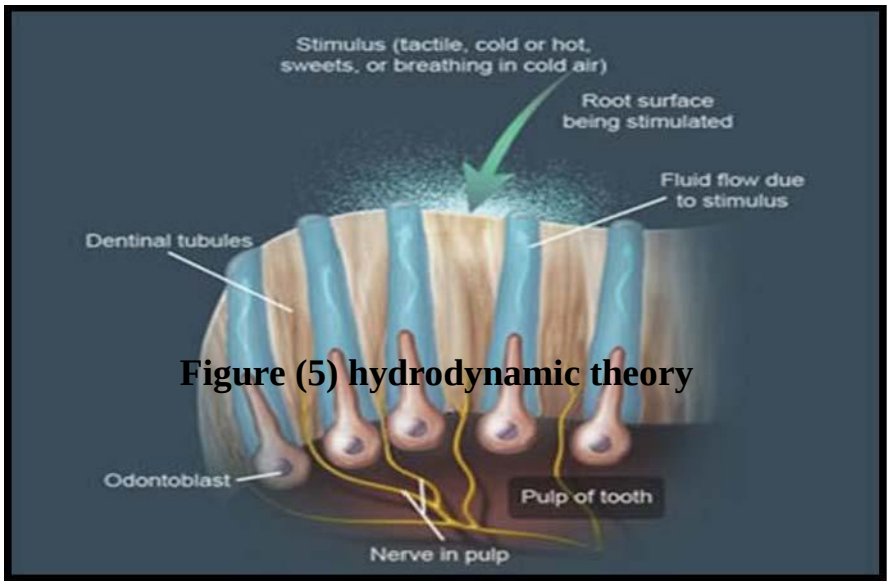


Figure (5) hydrodynamic theory

1.4 Clinical Management of DH

1.4.1 Diagnosis

As like any other clinical condition, an accurate diagnosis is important before starting the management of DH. DH has features which are similar to other conditions like caries, fractured or chipped enamel/dentine, pain due to reversible

pulpitis, and post dental bleaching sensitivity (***Chidchuangchai W,Vongsavan., et al*.,*2007***).

Diagnosis of DH starts with a thorough clinical history and examination. The other causes of dental pain should be excluded before a definite diagnosis of DH is made. Some of these techniques include pain response upon the pressure of tapping teeth (to indicate pulpitis/periodontal involvement), pain on biting a stick (suggests fracture), use of transilluminating light or dyes (to diagnose fractures), and pain associated with recent restorations. A simple clinical method of diagnosing DH includes a jet of air or using an exploratory probe on the exposed dentin, in a mesio-distal direction, examining all the teeth in the area in which the patient complains of pain. The severity or degree of pain can be quantified either according to categorical scale (i.e., slight, moderate or severe pain) or using a visual analogue scale. (***Minoux M,Serfaty R., et al. 2008***).

1.4.2Prevention of DH/removal of etiological factors

An often, neglected phase of clinical management of DH is the identification and treatment of the causative factors of DH. By removing the etiological factors, the condition can be even prevented from occurring or recurring. The etiological factors include faulty tooth brushing, poor oral hygiene, premature contacts, gingival recession because of periodontal therapy or physiological reasons, and exogenous/endogenous non-bacterial acids. Faulty tooth brushing includes hard brushes, excessive forces, excessive scrubbing at the cervical areas or even lack of brushing which causes plaque accumulation and gingival recession. (***Suge T,Kawasaki A,Ishikawa., et al. 2007***).

The patient should be taught the correct method of tooth brushing with the help of a model. Highly abrasive tooth powder or pastes should be avoided. Also, the patients should be instructed to avoid brushing for at least 2 hours after acidic drinks to prevent agonist effect of acidic erosion on tooth brush abrasion.

Erosive agents are also important agents in initiation and progression of DH. They tend to remove the enamel or open up the dentinal tubules The erosive agents can be either exogenous dietary acids or endogenous acids. The exogenous dietary acids include carbonated drinks, citrus fruits, wines, yogurt, and professional hazards (workers in battery manufacturing, wine tasters). A detailed dietary history should be taken. The quantity and frequency of the foods containing acids should be reduced. Patient should be advised to take something alkaline (milk) or at least neutral (water) after acidic drinks and to use a straw to sip the drink and avoid swishing it around the teeth. The endogenous acid comes from gastroesophageal reflux or regurgitation. It is also common in patients with eating disorders. The condition is characterized by generalized erosion of the palatal surfaces of maxillary anterior teeth. Such a patient should be referred to the medical practitioner for expert management of the underlying disease. An occlusal splint can be fabricated to cover the affected areas, to prevent their contact with the acids. (***Suge T,Kawasaki A,Ishikawa., et al. 2007***).

1.5 Classification of Desensitizing Agents

1.5.1 Mode of administration

At home desensitizing agents

In-office treatment

1.5.2 on the basis of 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 (SiO_2–P_2O_5–CaO–Na_2O)

- **Dentine adhesive sealer**

  Fluoride varnishes
- Oxalic acid and resin
- Glass ionomer cements
- Composites

- **Lasers**

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

Homeopathic medication

· Propolis

1.5.1.1 At home desensitizing therapy

Grossman listed the requirements for an ideal dentine desensitizing agent as: rapidly acting with long-term effects, non-irritant to pulp, painless and easy to apply, and should not stain the tooth. Traditionally, the therapy for management of DH is primarily aimed at occluding the dentinal tubules or making coagulates inside the tubules. Patients are often prescribed over-the-counter desensitizing agents. These "at home" desensitizing agents include toothpastes, mouthwashes and chewing gums. (***Eisenburger M,Addy M., et al. 2002***).

Majority of the toothpastes contain potassium salts (potassium nitrate, potassium chloride or potassium citrate), sodium fluoride, strontium chloride, dibasic sodium citrate, formaldehyde, sodium monofluorphosphate and stannous fluoride. Potassium salts act by diffusion along the dentinal tubules and decreasing the excitability of the intradental nerve fibers by blocking the axonic action. (***Markowitz K, Bilotto G, et al. 1991***).

It has been shown that toothpastes containing 5% potassium nitrate and 0.454% stannous significantly reduced the DH. Also, toothpastes containing

potassium nitrate and fluorides have been shown to reduce post-bleaching sensitivity. The desensitizing toothpastes should be used with the help of a toothbrush with soft bristles. Patients should be advised to use minimal amount of water to prevent the dilution of the active agent. Along with the desensitizing toothpastes, mouthwashes and chewing gums containing potassium nitrate, sodium fluoride or potassium citrate are also recommended. The results of "at-home" desensitizing therapy should be reviewed after every 3–4 weeks. If there is no relief in DH, "in-office" therapy should be initiated. (***Orchardson R, Gilliam D., 2006***).

Mechanism of Action

Tarbet et la. Studied the affect of a 5% KNO3 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 result demonstrate the 5% KNO3 dentifrice did not cause any observable tooth surface changes.

1.5.1.2 In-office desensitizing agents

Theoretically, the in-office desensitizing therapy should provide an immediate relief from the symptoms of DH. The in-office desensitizing agents can be classified as the materials which undergo a setting reaction (glass ionomer cement, composites) and which do not undergo a setting reaction (varnishes, oxalates).

Fluorides

Traditionally, fluorides have been used as a caries preventive material which can help in remineralization of enamel/dentin. Fluorides decrease the dentinal permeability by precipitation of calcium fluoride crystals inside the dentinal tubules (***Markowitz K, Bilotto G., et al 1991***).

These crystals are partially insoluble in saliva. SEM revealed granular precipitates in the peritubular dentin after application of fluorides. Various fluoride formulations are used to treat DH. These include sodium fluoride, stannous fluoride, sodium monofluorophosphate, fluorosilicates and fluoride combined with iontophoresis. Sodium fluoride has been used in dentifrices or may be professional applied in a concentration of 2%. The precipitates formed by sodium fluoride can be mechanically removed by the action of saliva or mechanical action. Therefore, an addition of acid formulation is recommended. The acidulated sodium fluoride can form precipitates deep inside the tubules. Stannous fluoride acts in a similar fashion as that of sodium fluoride, i.e., formation of calcium fluoride precipitates inside tubules. Also, SEM studies have shown that stannous fluoride itself can form insoluble precipitates over the exposed dentine. Fluorosilicates act by formation of precipitates of calcium phosphates from saliva. Ammonium hexafluorosilicate has been used as a desensitizing agent. It can present a continuous effect of dentinal tubule occlusion via precipitation of a mixture of calcium fluoride and fluoridated apatite.If the precipitate is predominantly composed of fluoridated apatite, it can form stable crystals deposited deep inside the dentinal tubules. These crystals are resistant to removal from the action of saliva, brushing or action of dietary substances. (***Kern DA, McQuade MJ., et al 1989***).

Method of action

In situ research shows root dentin treated with stannous fluoride exhibits tubules occlusion. Several other studies using analysis by scanning electron microscopy showed that partial or complete occlusion of dentin tubules occurred after treatment with SNF2. In addition Miller et al.36 reported a tin-rich surface deposit forms in vitro and in situ with two weeks use of 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.

Figure (6) stannous fluoride toothpaste

Oxalates

Oxalates can reduce dentinal permeability and occlude dentinal tubules. Thirty percent potassium oxalate had shown a 98% reduction in dentinal permeability. Also, topical application of 3% potassium oxalate reduced DH after periodontal therapy. The oxalate reacts with the calcium ions of dentine and forms calcium oxalate crystals inside the dentinal tubules as well as on the dentinal surface. This results in a better sealing as compared with an intact smear layer. It has been shown that the effect of oxalates on DH diminishes over a period of time. This can be attributed to the removal of the calcium oxalate crystals by brushing or dietary acids. The condition can be improved by acid etching of the dentinal surface, thus increasing the penetration of calcium oxalate crystals deep into the dentinal tubules. Many vegetables like rhubarb, spinach and mint contain oxalates. It has been shown that

phytocomplexes obtained from these natural products can reduce the dentinal permeability. This can also be followed by covering the exposed surface with a dental adhesive. (***litonjua LA, Andreana S, et al., 2003***) Potassium oxalate can lead to gastric irritation. Therefore, it should not be used with a tray with prolonged placement.

Varnishes are commonly used useful in-office measures to treat DH. Copal varnish can be applied to cover the exposed dentinal surface. But its effect is for short term and is not recommended for long term management of DH.To improve its efficacy, removal of smear layer is advocated. Also, the varnishes can act as a vehicle for fluoride. The fluoride varnishes can be acidulated to increase the penetration of ions (***Pillon FL, Romani IG, et al., 2004***).

Figure (7) D/Sense Crystal Desensitizer from Centrix

Adhesive materials

Resin-based dental adhesive systems can provide a more durable and long lasting dentine desensitizing effect. The adhesive resins can seal the dentinal tubules effectively by forming a hybrid layer. Traditionally, resin composites or dentin

bonding agents are used as desensitizing agents. The conventional dentin bonding agents (DBA) removes the smear layer, etches the dentinal surface and forms deep dentinal resin tags inside the dentinal tubules. The combined dentin–resin layer (consisting of penetrating resinous tags) has been termed as hybrid layer. It effectively seals the dentinal tubules and prevents DH. (***Hack GD, Thompson VP., 1994***).

Newer bonding agents modify the smear layer and incorporate it in into the hybrid layer. Recently, some dentin bonding agents have been introduced in the market with the sole purpose of treating DH. Gluma Desensitizer (Heraeus Kulzer, Hanau, Germany) contains hydroxyethyl methacrylate (HEMA), benzalkonium chloride, gluteraldehyde and fluoride. Gluteraldehyde causes coagulation of the proteins inside the dentinal tubules. (***Baysan A, Lynch E., 2003***).

It reacts with the serum albumin in the dentinal fluid, causing its precipitation. HEMA forms deep resinous tags and occludes the dentinal tubules.Gluma has shown promising results in the clinical trials (***Baysan A, Lynch E., 2003***).

Figure (8) Gluma Desensitizer from Kulzer

Potassium Nitrate use in Bleaching

Potassium nitrate has completely different mechanism of action than fluoride. Potassium nitrate penetrates the enamel and dentin to travel to the pulp and creates a calming effect on the nerve by affecting the transmission of nerve impulses. After the nerve depolarizes in the pain stimulus-response, it cannot re-polarize, so the excitability of the nerve is reduced. Potassium nitrate almost has an “anesthetic like effect” on the nerve.

One study demonstrate that applying potassium nitrate for 10 to 30 minute in the bleaching tray could be successful in reducing sensitivity in more than 90% of the patient, and allow them to complete the bleaching procedure successfully. Tray application could be used either before or after the bleaching treatment. Because the pain can occur remotely from the bleaching treatment, the potassium nitrate could be used as needed during the day or night. In several situation, the potassium nitrate could be substituted for the bleaching material on alternating nights of wear. the more readily source of 5% potassium nitrate in the United States is desensitizing toothpastes that contain 5% potassium nitrate. Five percent is the maximum amount of potassium nitrate approved by the US food and drug administration, and is the primary ingredient for sensitivity treatment allowed in OTC tooth paste. Based on the tray application study, desensitizing toothpaste can be placed in the tray for 10 to 30 minutes whenever sensitivity occurs. The only caution with tooth paste application is that some patients may experience a gingival reaction to the foaming ingredient sodium lauryl sulfate. This reaction is not caused by the potassium nitrate. The reaction generally produces a tissue burn or reddening of the gingiva. If this irritation occurs with one brand or flavor of tooth paste, the clinician may have to experiment with various OTC formulation for certain patients. Initially there was only one toothpaste available which had potassium nitrate, but not sodium laurel sulfate, and that was the original “pink packaged” Sensodyne. More recently, the advent of “pronamel Sensodyne” has provide a new option for a non-sodium laural sulfate,

potassium-nitrate containing toothpaste to be used in brushing or in the tray for treatment of sensitivity. If suitable toothpaste cannot be found for the patient, then the clinician should use the professionally available products containing 3% to 5% potassium nitrate and fluoride.

Several companies provide 3%to 5% potassium nitrate in a syringe for application in the bleaching tray as needed. The syringe material, which must be purchased from the companies, may be more appropriate for episodic sensitivity associated with the bleaching itself where the toothpaste was not acceptable because of the gingival respone. There are also disposable trays containing potassium nitrate which may be helpful, especially if there is no bleaching tray available for in-office techniques being used alone. (Dondi dall'Orologio G, et al.2002).

Figure (9) UltraEZ sustained-release potassium nitrate gel from Ultradent products

Bioglass

Bioglass was developed to stimulate the formation of new bone. It is used in orthopedics to cover the implants to promote union between implant and bone. It has

been used in dentistry to fill up the osseous defects during periodontal surgery. It has been reported that a formulation of bioglass can promote infiltration and remineralization of dentinal tubules. The basic component is silica, which acts as a nucleation site for precipitation of calcium and phosphate. SEM analysis has shown that bioglass application forms an apatite layer, which occludes the dentinal tubules. The use of bioglass in management of DH has been shown by some products such as NovaMin (NovaMin Technology Inc., FL, USA). (***Haywood VB, Caughman WF, et al., 2001***).

Figure (10) Dental self curing class ionomer cement Bioglass C

Portland cement

Some authors have shown that calcium silicate cement derived from Portland cement can help in the management of DH. It helps to occlude the dentinal tubules by remineralization. (***Markowitz K, Bilotto G. et al., 1991***).

Figure (11) Medizinischer Portland cement

Laser

Laser is an acronym for light amplification by stimulated emission of radiations. The mechanism of action of lasers in treating DH is not very clear. Some studies have shown that Nd–YAG laser application occluded the dentinal tubules. GaAlA laser is thought to act by affecting the neural transmission in the dentinal tubules. It has also been proposed that lasers coagulate the proteins inside the dentinal tubules and block the movement of fluid.(***Forsback AP, et al., 2004***).

Figure (12) laser application in dentin hypersensitivity

Casein phosphopeptide – amorphous calciumphosphate

Recently, milk protein casein has been used to develop a remineralizing agent (GC Tooth Mousse). The casein phosphopeptide (CPP) contains phosphoseryl sequences which get attached and stabilized with amorphous calcium phosphate (ACP). The stabilized CPP–ACP prevents the dissolution of calcium and phosphate ions and maintains a supersaturated solution of bioavailable calcium and phosphates. Various studies have shown that CPP–ACP can effectively remineralize the enamel subsurface lesions. By virtue of its remineralizing capacity, it has also been proposed by the manufacturers that it can also help in prevention and treatment of DH (***Corona SA, et al., 2003***).

Figure (13) GC tooth mousse

1.6 Management Strategy

- Take a detailed clinical and dietary history.
- Differentially diagnose the condition from other dental pain conditions.
- Identify and manage etiological and predisposing factors.
- In case of mild-to-moderate sensitivity, advice at-home desensitizing therapy.
- If there is no relief or in case of severe sensitivity, initiate in-office treatment.
- In extreme cases, if patient does not respond to the therapy and there are individual teeth exhibiting the symptoms, then endodontic therapy can be initiated.
- A regular review should be made with an emphasis on prevention of the condition.

1.7 Some studies about desensitizing agents:

1.7.1 desensitizing effect of Gluma desensitizer on prepared teeth

felton et la in 1991 assessed the ability og gluma dentin bone*(=Gluma Desensitizer) to inhibit dentinal sensitivity in teeth prepared to receive complete cast restoration. He concluded gluma dentin bond provided a significant reduction in dentin sensitivity when placed on exposed dentin of complete veneer crown preparation. The presence of a dentinal smear layer had no appreciable effect on dentin sensitivity responses for either Gluma treatment group.

1.7.2 Desensitizing effect of Gluma desensitizer on hypersensitive dentin

Orologio et al in 1993 investigated the effects of topical applications of Gluma 3 primer*(=Gluma desensitizer) or Gluma 2000 conditioning solution on hypersensitive erosion/abrasion lesions, they concluded that eliminated or at least significantly reduced dentin sensitivity throughout the 6 months observation time .

1.7.3 Efficiency of desensitizing treatment with Gluma Desensitizer

Blunck et la in 2000 evaluated the effectiveness of four different treatments for teeth with severe sensitivity, they concluded that even a single topical application of Gluma primer without prior cleaning significantly reduced the severe hypersensitivity of exposed cervical dentin and was as effective as the more time consuming application of a total dentin adhesive system.

1.7.4 Effectiveness of Glouma Desensitizer after periodontal therapy

Tenorio et la in 2002 were analyzed the effectiveness of two desensitizing agents on 48 teeth of male and female patients who had presented with hypersensitive dentin after periodontal therapy the data indicate that desensitizing agents were efficient to decease hypersensitive dentin after periodontal therapy.

1.7.5 Effects of desensitizer agents on dentinal tubules occlusion

Arrais it al in 2004 evaluated the features of dentinal tubules occlusion following application of three commercially available desensitizing agents: potassium-based / Oxa-Gel (OX), HEMA and glutaraldehyde-based /Gluma Desensitizer (GD) and acidulated phosphate fluoride-based / Nupro Gel (AF) by SEM analysis, OX promoted tubule occlusion by crystal-like deposits in the lumen of the tubules. While GL created a thin layer over the dentin surface, AF application produced precipitates that occluded the tubules.

According to the SEM analysis, all desensitizing agents were able to occlude the

dentinal tubules.

1.7.6 Clinical effectiveness of Gluma desensitizer on tooth cervical hypersensitivity.

Kakaboura et al in 2005 investigated the desensitizing ability of a one-bottle agent and a glutraaldehyde-based HEMA formulation on sensitive tooth cervical areas for a period up to 9 months, they concluded even through the one-bottle agent tested may offer a short term, adequate reduction of hypersensitivity, a significant reversal of the sensitivity may occur long-term, particularly for air-blast stimulation. The glutaraldehyde-based agent was proven more efficient in treating cervical sensitivity up to the 9-month follow-up.

1.7.7 Clinical evaluation of three desensitizing agents in relieving dentin hypersensitivity

Pamir et al in 2007 this in vivo study determined whether the application of three different desensitizing agents (seal & protect, Vivasens and BisBlock) on exposed dentin surfaces was effective in reducing dentin hypersensitivity in subjects with slight-to-moderate sensitivity. They were concluded that the desensitizing agents used in this clinical study were effective in alleviating dentin hypersensitivity.

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