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Research Paper

Dentin Hypersensitivity-Pathogenisis and Management

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ABSTRACT:- *Dentinal hypersensitivity is a common painful condition of the teeth, associated with the exposure of the dentin to external environment of the mouth. A thorough knowledge regarding etiology and predisposing factors is inevitable for the effective treatment approach. The aim of this paper is to summarize existing information regarding pathogenesis, etiology and prevalence, assessment strategies, self care strategies and treatment approach of dentin hypersensitivity.*

Keywords:- *Dentinal hypersensitivity, DH management, etiology*.

I. INTRODUCTION

Dentinal hypersensitivity (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 form of dental defect or pathology [1]. A modification of this definition was suggested by the Canadian Advisory Board on Dentine Hypersensitivity [2] in 2003, which suggested that 'disease' should be substituted for 'pathology'. The definition provides a clinical description of the condition and identifies DH as a distinct clinical entity. Other terms to describe DH have been created by substituting the word dentinal, adding site descriptors, such as cervical or root, and combining this with either hypersensitivity or sensitivity.

Despite the existence of these various terms, several authors prefer the term DH, commonly used and accepted for many decades to describe a specific painful condition of teeth, which is distinct from others types of dentinal pain having different etiologies. . The condition has been defined by an international workshop on DH as follows [3]. "Dentine hypersensitivity 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". DH is a painful clinical condition that affects 8 to 57% of the adult population and is associated with the dentin exposure to the oral environment [4], [5].

II. DISCUSSION

2.1. PATHOGENESIS

DH develops in two phases: lesion localization and lesion initiation [6]. Lesion localization occurs by loss of protective covering over the dentin, thereby exposing it to external environment. It includes loss of enamel via attrition, abrasion, erosion or abfraction. Another cause for lesion localization is gingival recession which can be due to toothbrush abrasion, periodontal surgery, tooth preparation for crown, excessive flossing or secondary to periodontal disease [7]. Not all exposed dentine is sensitive. For DH to occur, the lesion localization has to be initiated. It occurs after the protective covering of smear layer is removed, leading to exposure and opening of dentinal tubule

Several theories try to explain the dentinal hyper-sensitivity although none of them leads to a thorough understanding of how the stimuli applied in this surface can cause pain.

Transduction theory: According to this theory, the odontoblast has a special sensory function and the functional complex with the nerve ending in or near the odontoblastic layer acts as an excitatory synapse. The odontoblast and its process have been perceived as a transducer mechanism.

Modulation theory: According to this theory the nerve impulses in the pulp are modulated through the liberation of polypeptides from the odontoblasts, when injured These substances may selectively alter the

permeability of the odontoblastic cell membrane through hyperpolarization, so that pulp neurons are more prone to discharge upon receipt of subsequent stimuli.

Hydrodynamic theory: According to Brannstrom's hydrodynamic theory [8], when an appropriate stimulus is applied to the outer dentin surface, there is a displacement of the contents of the dentinal tubules that gives rise to a mechanical stimulation of the neurons at the pulpodentinal border. The stimulation of the nerve endings next to the odontoblastic layer is provoked by the variation of the intrapulpal pressure toward the pulp or in the opposite direction .The nerve fibers stimulation occurs because of the deformation of these fibers, caused by the fluid movement, leading to a widening of the nerve membrane ionic canals, allowing the entrance of $Na_{pain.}^{++}{}_3$in the cell, depolarizing the fibers and provoking it to cause pain

Histologically, sensitive dentin presents widened dentinal tubules, two times larger when compared with tubules of normal dentin and in a greater number per area, when compared with the dentin without sensitivity [9], (Figure 1). At a macroscopic level, dentine exhibiting hyper-sensitivity appears no different from non-sensitive dentine. The status of the pulp in DH is not known, although symptoms would suggest minor inflammation as a result of the length of time that symptoms persist without developing into true pulpits [10] .

Dentinal hypersensitivity is most likely to occur in younger people who experience rapid exposure of the root surface. Old people, in spite of presenting root exposure, usually do not show painful sensitivity; this can be explained by the following factors:

- Mineral deposition inside the tubules (dentinal sclerosis)
- Reduction in the number of tubules
- Pulp chamber reduction due to an increase of reparative dentin
- A reduction in cellularity, vascularity and nerve fibers in the pulp.

Pulp pressure is greater than the atmospheric pressure; this explains why the dentinal fluid is constantly flowing through the permeable exposed dentin, although in very low rates to activate the pulpal mechanoreceptors. Any event that increases the pressure gradient, increasing the fluid movement, may evoke pain [11]. The dentinal hypersensitivity can be triggered off by many factors including(Figure.2)

- Thermal: The pain is caused by the temperature variation which causes fluid contraction, when cold, or expansion, when hot, leading consequently to its movement;
- Evaporative: Through the application of a brief air blast on the tooth, there will be evaporation of the dentinal fluid.
- Tactile: The simplest device for testing hypersensitivity is scrubbing a sharp probe along the cemento-enamel junction. This induces the fluid movement probably due to a vibration mechanism
- Osmotic: The pain is provoked in the presence of sweet substances. Because they are hypotonic, they cause an osmotic pressure in dentin, which leads to a movement of the dentinal fluid toward the sweet substance

2.2. Etiology and Prevalence

DH can manifest when dentin is exposed by enamel loss (lesions of abrasion, erosion or corrosion) followed by the constant action of acids, which keep the tubules open on the dentin surface, or because the root surface has been denuded due to loss of structures such as cementum, which is easily removed by brushing or periodontal treatment (, or more commonly, by the association of two or more of these factors [12]. It may also be caused by gingival recession which occurs with aging, chronic periodontal disease and patient's deleterious habits [13].

Studies [14] indicate that dentin exposure can occur in the area of the enamel/ cementum junction because of enamel, or cementum loss due to one or more of the following processes

Lack or excessive tooth brushing. .

Low level of oral hygiene.

Periodontal therapy has been associated with DH due to the exposure of dentinal tubules.

Exposure to non bacterial acids in the diet, chemical products, medication, drugs or endogenous acids from reflux or regurgitation of stomach acid; that is, substances with low pH lead to the loss of dental structure by chemical dissolution without bacterial involvement. This process, called erosion, produces a more softened enamel zone.

Occlusal contact with excessive force and premature contact

Abfraction is not directly related to the diet, periodontal disease or abrasion. However, it may be a predisposing factor to DH.

Physiological causes. The increase in the number of teeth with root exposure is evident, as age advances. Dental extrusion, in the absence of an antagonist tooth, results in root exposure, which may lead to DH.

2.3. Assessment strategies

An appreciation for the impact of the pain on a patient's quality of life and a more thorough understanding of hypersensitivity will lead clinicians to make a systematic treatment approach. A detailed questionnaire helps to obtain further information regarding dentin hypersensitivity. The following questions are suggested to initiate the discussion of dentin hypersensitivity also aiding to evaluate the intensity of pain [15].

1. Which tooth or teeth is/are sensitive and on which aspect?
2. On a scale from one to 10, how much does it hurt, with 10 being the most painful?
3. How long does the pain last?
4. Can the pain be characterized as sharp, dull, shooting, throbbing, persistent, constant, pressure, burning, intermittent?
5. Does it hurt when you bite down (pressure)?
6. Does the discomfort linger or stop immediately after a stimulus such as cold water is removed?
7. On a scale from one to 10, how much does the pain impact your daily life?
8. Is the pain stimulated by certain foods: Sweet? Sour? Acidic?
9. Does sensitivity result from hot or cold food or beverages?
10. Does discomfort stop immediately upon removal of the painful stimuli, such as cold food or beverage?
11. How effectively are you managing the stress in your life?

There are a number of ways to evaluate the degree of hypersensitivity. Using a verbal rating scale and the use of a short, intermittent "air blast" to quantify the subjective level of pain as follows:

0—No discomfort/pain; no discomfort/pain but aware of stimulus
1—Mild discomfort/pain—described as mild discomfort during but not following the air blast
2—Marked discomfort/pain—described as definitive discomfort during the air blast
3—Marked discomfort/pain lasting more than 10 seconds following exposure to air.

There are a number of predisposing factors that individually or collectively place the patient at risk for dentin hypersensitivity. These include gingival recession, tooth wear, lifestyle, behaviors, oral self-care habits, and the pH of the oral environment which may be related to dietary as well as xerostomic conditions.

The assessment and evaluative phase includes the identification and elimination of any predisposing factors affecting the pH of the oral environment. The pH of the oral environment is the single most contributory factor to hypersensitive cervical and occlusal surfaces. This is due to both exogenous and endogenous contributing factors.

Any food substance with a critical pH value of less than 5.5 can become a corrosive and demineralize the tooth structure creating erosion. Enamel is comprised of 96% mineralized substrate with 4% being water and organic protein. Under normal conditions with an oral environment of pH greater than 5.5, the enamel is very resistant; however, once compromised by low pH, it becomes vulnerable to abrasion, attrition, and even abfraction.

Erosion of the dentin brings about a rapid loss of the smear layer and the opening of dentinal tubules. Those foods and beverages with a low pH readily remove the smear layer after a few minutes of exposure. Attention should be given to obtaining a dietary history in order to identify predisposing acidic factors in the patient's diet which causes surface softening .It can extend up to 3 to 5 μm and make the tissue is highly susceptible to physical wear [16].

Endogenous or internal contributory factors may include regurgitation associated with gastro esophageal reflux disease or eating disorders, both of which are capable of lowering the pH of the oral environment rapidly and significantly. Xerostomia due to intrinsic and extrinsic factors also can enhance sensitivity.

2.4. Self-care strategies and evidence-based recommendations

Oral self-care can influence the degree of dentin hypersensitivity. Patients who brush excessively or use undue pressure while brushing should be instructed on proper tooth brushing techniques to avoid gingival recession. Tooth brushing (with variations to be considered such as bristle stiffness; end rounding; and tooth-brushing force, duration, and frequency) has long since been associated with gingival recession.

Also, repeated brushing, immediately following the intake of a low pH food or beverage, can further exacerbate the dissolution of both the enamel and dentin. Wear of enamel and dentin can be dramatically increased if tooth brushing follows an erosive challenge. It is for this reason that one should avoid tooth brushing for a minimum of 30 to 60 minutes or longer after consuming acidic foods or drinks to reduce the co effects of acids and abrasion [13]

Currently, the paradigm has been focused on biofilm control instead of total elimination. The protective and re-mineralizing nature of the dental biofilm plays a significant role in dentin hypersensitivity .The opportunity for delivering and retaining other broad-spectrum antimicrobial or demineralization agents into dental plaque biofilms should be considered in developing novel innovative approaches to dentin hypersensitivity.

Erosive agents are also important agents in initiation and progression of DH. They tend to remove the enamel or open up the dentinal tubules [17]. 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 etc) [18]. 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 gastro esophageal 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 [19].Such a patient should be referred to the medical practitioner for expert treatment approach

III. CLASSIFICATION OF DESENSITIZING AGENTS

Currently, two main methods are used in the treatment of DH: tubular occlusion and blockage of nerve activity by means of direct ionic diffusion, increasing the concentration of potassium ions acting on the pulpal nerve sense activity [20], (Figure.3). The advantage of using products available for home use is that they are immediately available for treatment, when compared with those applied by professionals, disadvantage is that time is needed for remission of the symptoms (2-4 weeks), while theoretically, those applied in-office promote immediate relief.

3. I. Mode of administration

At home desensitizing agents
In-office treatment

3.2. On the basis of mechanism of action (Table 1)

Nerve desensitization
Protein precipitation
Plugging dentinal tubules
Dentine adhesive sealers
Lasers
Homeopathic medication

IV. AT HOME DESENSITIZING THERAPY

Grossman [21] 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. They present complex formulae with several ingredients, among them desensitizing agents such as strontium chloride, potassium nitrate; dibasic sodium citrate formaldehyde, sodium fluoride, sodium monofluorphosphate and stannous fluoride [22] are widely used. The mechanism of action is based on the obliteration of dentinal tubules, by the precipitation of calcium phosphate on the dentin surface and calcium is the most frequent component present in the dentifrices [23]. Many dentifrices contain abrasives (calcium carbonate, aluminum, calcium phosphate, silicate, etc) which may also cause obliteration of the tubules by the abrasive or indirectly by the formation of a smear layer during brushing [24].

Silver nitrate reduces DH by fast coagulation of the Tomes processes forming silver albuminate, which acquires a dark color when exposed to light, blackening the tooth surface. The subsequent use of sodium chloride reduces the pigmentation. Thus, due to tooth darkening, this technique is not well accepted among patients

Unlike other products, potassium nitrate does not diminish dentin hydraulic conductivity, or promote obstruction of dentinal tubules by the deposition of crystals.

According to Kim [25], Potassium salts act by diffusion along the dentinal tubules and decreasing the excitability of the intradental nerve fibers by blocking the axonic action [26].

Strontium chloride and zinc chloride are protein\ precipitants and their mechanism is through organic precipitation and odontoblast denaturation forming a sealing film that prevents fluid movement and has an occlusive action [27].

V. IN-OFFICE DESENSITIZING AGENTS

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).

5.1.Fluorides

Fluorides have been used as a caries preventive material which can help in remineralization of enamel [28].Clinical studies have shown that application of fluoride solution can decrease the DH. Fluorides decrease the dentinal permeability by precipitation of calcium fluoride crystals inside the dentinal tubules. These crystals are partially insoluble in saliva. SEM revealed granular precipitates in the peritubular dentin after application of fluorides [29].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.

Iontophoresis with sodium fluoride [30], is recommended, here electric current increase the ion diffusion. A clinical study has shown that 0.4% stannous fluoride along with 0.717% of fluoride can provide an immediate effect after a 5 minute professional application [31]. 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 [32]. 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 [33]. 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

5.2. Oxalates

Oxalate reacts with the dentin calcium and promotes deposition of calcium oxalate crystals on the dentin surface and/or inside its tubules, significantly reducing hydraulic conductivity inherent to this structure, sealing the tubules more effectively than the intact smear layer. If the hydrodynamic mechanism is responsible for pain, this effect observed after the application of potassium oxalate leads to the reduction of DH [34]. The calcium oxalate crystals formed on the dentin surface are easily removed by daily brushing. However when dentin is previously etched with 35% phosphoric acid the penetration depth of oxalate buffer into the dentinal tubules is about 6-7 μm [35], and thus, pain relief can be expected for a longer period. The application of potassium oxalate on the etched dentin can also be associated with l a covering of dentinal adhesives [36].

5.3. Varnish

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 [37] .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**.**

5.4. 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 [38]. 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 [39]. Newer bonding agents modify the smear layer and incorporate it in into the hybrid layer [40]. Recently, some dentin bonding agents have been introduced in the market with the sole purpose of treating DH.

5.5. Bioglass

It has been proved that a formulation of bioglass can promote infiltration and remineralization of dentinal tubules.[41,42] 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.

5.6. Portland cement

Some authors have shown that calcium silicate cement derived from Portland cement can help in the management of DH. [42] It helps to occlude the dentinal tubules by remineralization.

5.7. Casein phosphopeptide–amorphous calcium phosphate

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)[43]. The stabilized CPP–ACP prevents the dissolution of calcium and phosphate ions and maintains a supersaturated solution of bioavailable calcium and phosphates. By virtue of its remineralizing capacity, it has also been proposed by the manufacturers for treatment of sensitivity

5.8. Lasers

Laser can cause melting of dentine and closure of exposed dentinal surface without causing cracking [44] and results in the reduction of permeability and hydraulic conductance. Sealing of dentinal tubules can be up to 4mm in center and 3µm along the lateral margin

VI. CONCLUSION

The causative factors play key role in localizing and initiating dentin hypersensitivity. Active management involves a combination of chair side and oral self-care therapies. Multiple therapeutic modalities are designed to treat dentin hypersensitivity, including products to impede nerve conduction of the pain stimulus, products to mechanically occlude dentin tubules, and calcium-containing products designed to generate plugs in the tubules utilizing a remineralization mechanism.

Although dentin hypersensitivity is not the enigma it once was, there is still much to learn about the condition itself, the management, and prevention behaviors. Our profession is strategically positioned to not only evaluate but also to educate to improve the comfort and quality of life for our patients. It still remains as common cold in dentistry

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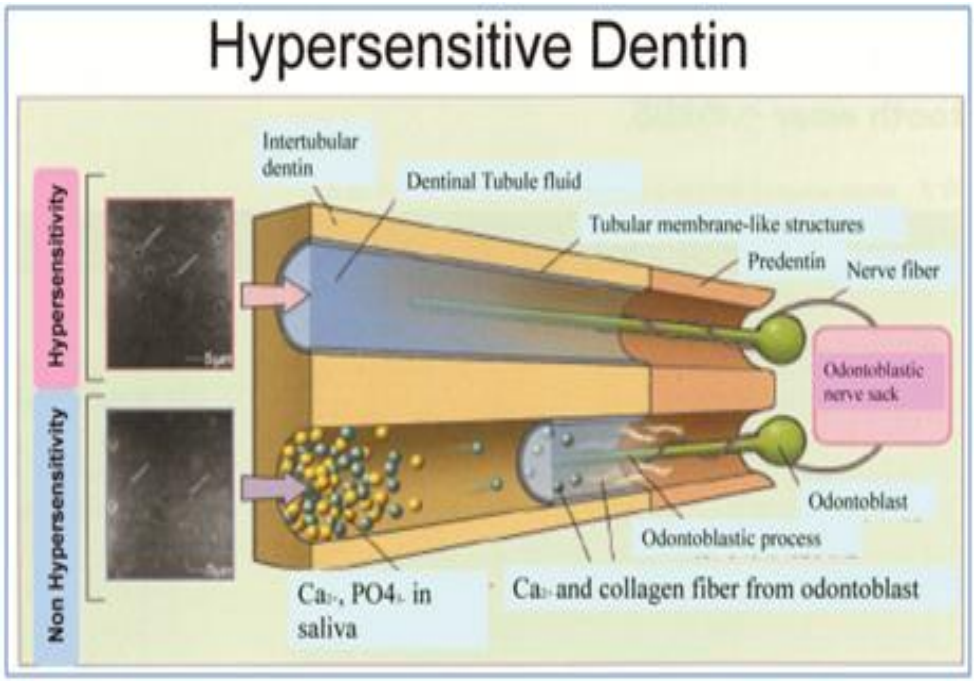

Figure 1

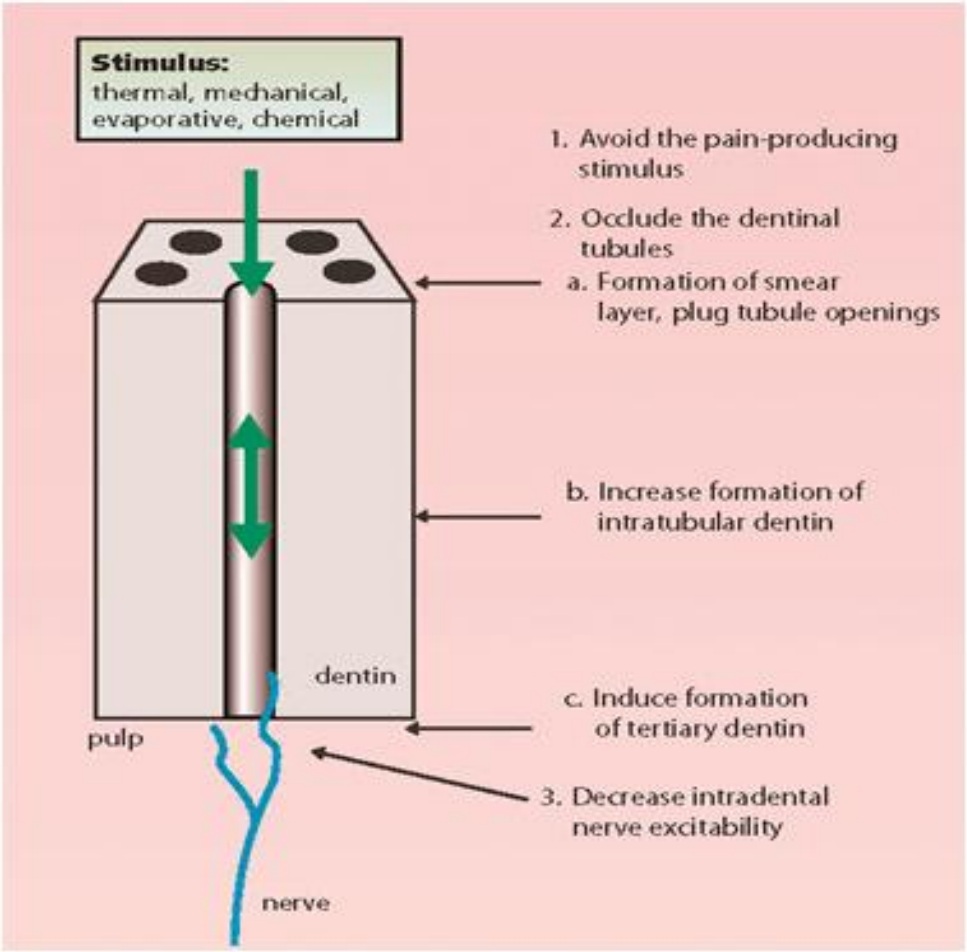

Figure .2.Stimulus activate intradental nerve to cause pain

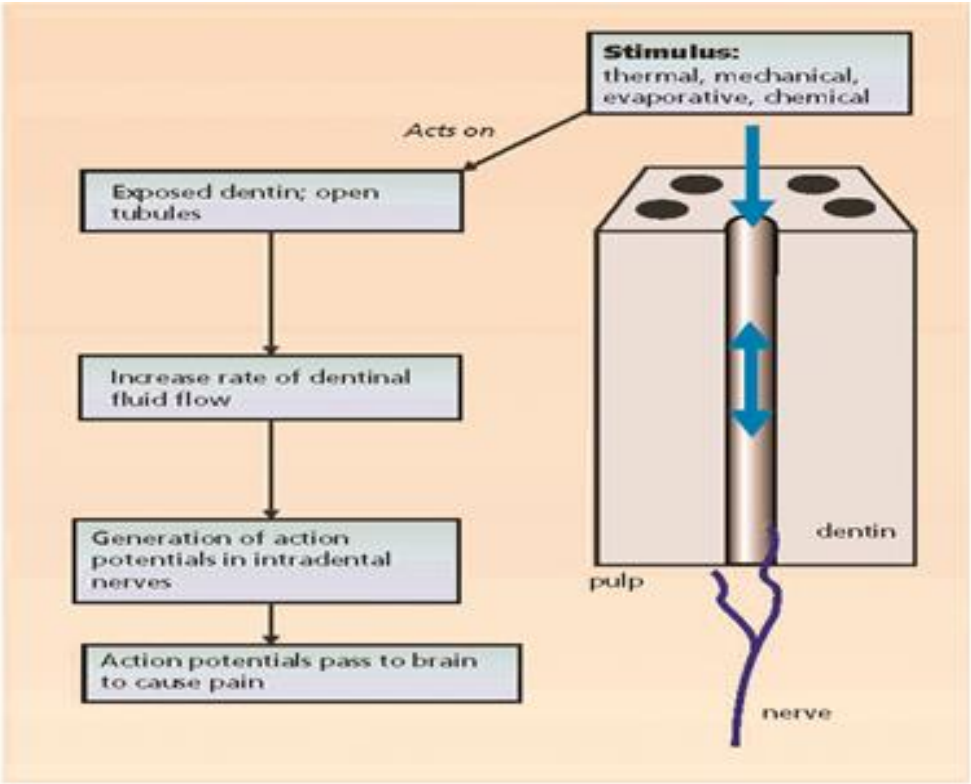

Figure.3. Areas that can be targeted by desensitizing agents

Table 1

Desensitizing Methods	Agents Used
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 sealers	Fluoride varnishes Oxalic acid and resin Glass ionomer cements Composites Dentin bonding agents
Lasers	Neodymium: yttrium aluminum garnet (Nd-YAG) laser GaAlAs (gallium-aluminum-arsenide laser) Erbium-YAG laser
Homeopathic medication	Propolis