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REVIEW

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Dentinal hypersensitivity: a review

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Abstract

Tooth sensitivity is a very common clinical presentation which can cause considerable concern for patients. This condition is frequently encountered by periodontists, dentists, hygienists and dental therapists. The management of this condition requires a good understanding of the complexity of the problem, as well as the variety of treatments available. This review considers the aetiology, incidence and management of dentinal hypersensitivity.

Key words: Dentinal hypersensitivity, desensitizing agents, cervical sensitivity.

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INTRODUCTION

Dentinal hypersensitivity, or cervical dentinal sensitivity, is a significant clinical problem. It is defined as pain arising from exposed dentine typically in response to thermal, chemical, tactile or osmotic stimuli.[1]

Dentine may become exposed via several means. For example, the enamel or cementum which normally covers the dentine surface may be removed or denuded as a result of attrition, abrasion or erosion. Alternatively, in some individuals the cementum and enamel which normally cover the dentine do not meet and result in dentine exposure as a result of a developmental anomaly. In general, it appears that dentinal hypersensitivity is rarely a result of just one of the above factors, but rather a combination of more than one factor. Regardless of the aetiology of dentine exposure, one feature appears to be in common and that is open dentinal tubules which provide a direct link between the external environment and the internal pulp of the tooth. If the tubules are not exposed it seems unlikely that hypersensitivity will be found. In areas of sensitive dentine the apertures of the dentine tubules are patent and this results in more stimuli having closer contact with the dental pulp.

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Even after long periods of exposure to the oral environment, dentinal sensitivity may still be a significant problem despite the exposed tubules becoming occluded by the smear layer or pellicle. Thus, once sensitivity has become established the pulp may become irreversibly sensitive. Treatment is therefore aimed at not only restoring the original impermeability of the tubules by occluding them, but also controlling the neural elements within the pulp to dampen the external stimulatory effects. These two modes of control are either partial or total obliteration of the dentinal tubules or alteration of pulpal sensory activity, or both.

Theories for dentinal hypersensitivity

Odontoblastic transduction theory

According to this theory, odontoblastic processes are exposed on the dentine surface and can be excited by a variety of chemical and mechanical stimuli.[2,3] As a result of such stimulation neurotransmitters are released and impulses are transmitted towards the nerve endings. To date no neurotransmitters have been found to be produced or released by odontoblastic processes.

Neural theory

As an extension of the odontoblastic theory, this concept advocates that thermal, or mechanical stimuli, directly affect nerve endings within the dentinal tubules through direct communication with pulpal nerve fibres. While this theory has been supported by the observation of the presence of unmyelinated nerve fibres in the outer layer of root dentine[4] and the presence of putative neurogenic polypeptides,[5] this theory is still considered theoretical with little solid evidence to support it.

Hydrodynamic theory

By far the most widely accepted theory for dentinal hypersensitivity is the hydrodynamic theory proposed by Brannstrom and co-workers.[6,7] This theory postulates that fluids within the dentinal tubules are disturbed either by temperature, physical or osmotic changes and that these fluid changes or movements stimulate a baroreceptor which leads to neural discharge. The basis of this theory is that the fluid filled dentinal tubules are

Table 1. Dentinal hypersensitivity epidemiological studies

Authors	Country	Setting	Study type	n	Prevalence (%)
Jensen, 1964[67]	USA	University	Clinical	3000	30
Graf and Glase, 1977[68]	Switzerland	Practice	Clinical	351	15
Flynn *et al.*, 1992[10]	UK	University	Clinical	369	18
Orchardson and Collins, 1987[11]	UK	University	Clinical	109	74
Fisher *et al.*, 1992[8]	Brazil	University	Clinical	635	17
Murray and Roberts, 1994[69]	Indonesia	Not stated	Questionnaire	1000	27
Murray and Roberts, 1994[69]	USA	Not stated	Questionnaire	1000	18
Murray and Roberts, 1994[69]	Japan	Not stated	Questionnaire	1000	16
Murray and Roberts, 1994[69]	France	Not stated	Questionnaire	1000	14
Murray and Roberts, 1994[69]	Germany	Not stated	Questionnaire	1000	13
Murray and Roberts, 1994[69]	Australia	Not stated	Questionnaire	1000	13
Chabanski *et al.*, 1997[15]	UK	University	Clinical	51	73
Irwin and McCusker, 1997[16]	UK	Practice	Questionnaire	250	57
Liu *et al.*, 1998[70]	Taiwan	University	Clinical	780	32
Rees, 2000[9]	UK	Practice	Clinical	3593	4
Taani and Awartani, 2002[71]	Saudi Arabia	University	Clinical	295	42-60
Clayton *et al.*, 2002[72]	UK	Air force	Questionnaire	228	50
Rees and Addy 2002[9]	UK	Practice	Clinical	4841	4.1
Rees *et al.*, 2003[73]	Hong Kong	Hospital	Clinical	226	67.6

open to the oral cavity at the dentine surface as well as within the pulp.

In general, the excitement of nerve fibres by different kinds of stimuli can be explained by the hydrodynamic theory. For example, dehydration associated with desiccation following air movement over the exposed dentine surface results in outward movement of dentinal fluid towards the dehydrated surface, which triggers nerve fibres and results in a painful sensation. In a similar manner thermal changes can result in expansion or contraction of the dentinal tubules resulting in changes in dentinal fluid flow and associated excitation of nerve fibres causing pain. High osmotic stimuli such as sugar, acid and salt can also result in fluid flow within the dentinal tubules and induce nerve stimulation and painful sensations. Physical stimulation is more difficult to explain through this theory although it is possible that mechanical abrasion of the exposed dentine surface may be sufficient to induce unwanted fluid flow within the dentinal tubules with resulting pain from the stimulated nerve fibres.

Epidemiology of dentinal hypersensitivity

Dentinal hypersensitivity appears to be a common problem with various reports indicating an incidence of between 4 to 74 per cent of the population (Table 1). One reason for this large discrepancy relates to the variations in the methods of data collection. Several studies indicate that even though high percentages of a population may report to have sensitive teeth, a much smaller proportion are actually diagnosed as having cervical dentine hypersensitivity on the basis of defined clinical diagnostic criteria.[8] In general, it appears that the incidence of hypersensitivity in most populations ranges between 10 to 30 per cent of the general population. The incidence can vary considerably depending on the cohort being studied with periodontal patients, patients with gingival recession and smokers with periodontitis showing the highest incidence of diagnosed dentinal hypersensitivity.[9]

The teeth most commonly affected by dentinal hypersensitivity are the upper premolars followed by the upper first molars with the incisors being the least sensitive teeth.[8-15]

It has been reported that there is a slightly higher incidence of dentine hypersensitivity in females compared to males.[8,10-12] This difference is, however, not statistically significant.[10]

The relationship between dentine hypersensitivity and ageing is unclear. It has been suggested that with the lifespan of the general population increasing, and more people keeping their teeth longer, hypersensitivity will increase in prevalence. This seems to make sense on the basis that gingival recession and loss of enamel and cementum is more prevalent in older individuals. The above assumptions are somewhat confounded by reports in the literature which indicate that most sufferers of dentine hypersensitivity range in age from 20 to 40 years with the peak incidence occurring at the end of the third decade and decreases during the fourth and fifth decades of life.[10,16] This may be explained by the decrease in permeability of dentine and neural sensitivity with ageing. Such responses may arise from the natural desensitization of sclerosis and secondary dentine formation. In addition, long-term use of fluoride dentifrices can add to the occlusion of open dentine tubules resulting in a decrease in sensitivity.[17]

Clinical significance of hypersensitivity

Dentinal hypersensitivity, while neither life threatening nor a serious dental problem, can be a particularly uncomfortable and unpleasant sensation for patients and can dictate types of foods and drinks ingested. Patients may describe the condition as dull or sharp, vague or specific and intermittent or constant. Teeth

Table 2. Treatment strategies for dentinal hypersensitivity

1. Nerve desensitization
   Potassium nitrate
2. Anti-inflammatory agents
   Corticosteroids
3. Cover or plugging dentinal tubules
   a. Plugging (sclerosing) dentinal tubules
      Ions/salts
         Calcium hydroxide
         Ferrous oxide
         Potassium oxalate
         Sodium monofluorophosphate
         Sodium fluoride
         Sodium fluoride/stannous fluoride combination
         Stannous fluoride
         Strontium chloride
      Protein precipitants
         Formaldehyde
         Glutaraldehyde
         Silver nitrate
         Strontium chloride hexahydrate
      Casein phosphopeptides
      Burnishing
      Fluoride iontophoresis
   b. Dentine sealers
      Glass ionomer cements
      Composites
      Resins
      Varnishes
      Sealants
      Methyl methacrylate
   c. Periodontal soft tissue grafting
   d. Crown placement/restorative material
   e. Lasers

causing such symptoms are rarely considered to be as seriously affected as those affected by caries, endodontic problems or periodontal disease, however, the condition is nonetheless of sufficient concern to warrant appropriate and proper management. In most instances the condition can be managed by patients through appropriate home care using properly prescribed over-the-counter products. These features of the condition negate, in the majority of cases, the need for expensive and lengthy professional care. Notwithstanding the above, the condition still requires an appropriate differential diagnosis since carious exposure of dentine surfaces, inflamed pulps or cracked cusps can produce symptoms similar to cervical dentinal hypersensitivity.[18]

Treatment for hypersensitivity

There is a surprisingly large number of treatment options for managing dentinal hypersensitivity. Chemical or physical agents are used to either desensitize the nerve or cover the exposed dentinal tubules (Table 2). The most common form of management is the placement of a topically applied agent applied either by a dental professional or by the patient at home. All currently available treatments appear to work.[19]

Several criteria are recognized as constituting an ideal desensitizing agent. These include not irritating the pulp, being relatively painless to apply, easily applied, rapid action, permanently effective and should not discolour the teeth.[20] Overall, patient responses are very subjective and thus treatment results are largely dependent upon the individual's pain threshold.[21,22]

Role of dental plaque

While there are many factors which can contribute to dentine hypersensitivity, plaque accumulation has often been cited as an important factor.[23] Plaque accumulation on root surfaces may lead to demineralization of tooth structures which could be associated with patency of dentinal tubule orifices.[23] It has been reported that patients who maintain good levels of plaque control are less likely to report dentine hypersensitivity.[24] On the other hand, patients who have significant proportions of their root surfaces covered with dental plaque report more problems with dentine hypersensitivity.[25-27] Despite these findings, the influence of plaque on dentine hypersensitivity remains controversial. Interestingly, many patients with gingival recession have minimal plaque deposits yet still complain of sensitivity.

Nerve desensitization

Potassium nitrate

A number of studies have reported the efficacy of potassium nitrate for managing dentinal hypersensitivity.[28-31] While the Hodosh study was the first to report that potassium nitrate was a "superior desensitizer" this study was not well controlled and it was not until the studies of Tarbet *et al.*[29,30] that good evidence for the efficacy of potassium nitrate in managing dentinal hypersensitivity was demonstrated. These controlled studies demonstrated that potassium nitrate at a concentration of 5% in a low abrasive toothpaste was able to desensitize dentine for up to four weeks compared to a control paste. Potassium nitrate in bioadhesive gels at 5% and 10% has also been shown to be effective in reducing dentinal hypersensitivity.[32] Importantly, it has been shown that potassium nitrate does not induce any pulpal changes.[33] Despite these encouraging findings it is interesting to note that a recent Cochrane Database Systematic Review failed to find strong evidence supporting the efficacy of potassium nitrate toothpaste for dentine hypersensitivity.[34] Nonetheless, this review did report that the differences which were noted were statistically significant in favour of treatment with potassium nitrate toothpaste.

The mechanism of action of potassium nitrate is largely unknown, although an oxidizing effect or blocking of tubules by crystallization has been proposed[28] but not proven. The effect of potassium nitrate on dentinal fluid flow has been reported to be minimal even at a 30% concentration.[35] A more likely explanation is that the potassium ions are the active component and that potassium nitrate reduces dentinal sensory nerve activity due to the depolarizing activity of the K^+ ion,[36] although this proposal has never been confirmed in intact human teeth.[37]

Anti-inflammatory agents

Corticosteroids

Anti-inflammatory agents such as corticosteroids have been proposed for use to manage dentine hypersensitivity. However, trials have not found them to be particularly useful.[22] While it is presumed that these agents may induce mineralization leading to tubule occlusion, this view has yet to be validated[38] and the validity of using such agents has been questioned.[39]

Covering or plugging dentinal tubules

Calcium hydroxide

Several studies have reported on the effectiveness of calcium hydroxide in managing dentinal hypersensitivity.[40-42] Its mode of action has been proposed to be via occlusion of dentinal tubules through the binding of loose protein radicals by calcium ions[42] and increasing mineralization of the exposed dentine. Although immediately effective, the action of calcium hydroxide diminishes rapidly requiring multiple applications to maintain its effect.[19] A negative feature of calcium hydroxide is its reported irritation of gingival tissues.[22]

Casein phosphopeptides

A relatively new product on the market composed of casein phosphopeptides has been used for the management of dentinal hyperersensitivity. Despite a number of anecdotal case reports on various websites, to date there are no published studies reporting the efficacy of this material for dentinal hyperersensitivity.

Sodium fluoride

Many clinical studies have shown that treatment of exposed root surfaces with fluoride toothpaste and concentrated fluoride solutions is very efficient in managing dentinal hypersensitivity.[43-45] The improvement appears to be due to an increase in the resistance of dentine to acid decalcification as well as to precipitations in the exposed dentinal tubules. Tal *et al*.[46] suggested that the probable desensitizing effects of fluoride are related to precipitated fluoride compounds mechanically blocking exposed dentinal tubules or fluoride within the tubules blocking transmission of stimuli.

Sodium monofluorophosphate

Toothpastes containing sodium monofluorophosphate have been shown to be effective in managing dentinal hypersensitivity.[47,48] The mechanism of action of sodium monofluorophosphate is unclear.[21] It does not appear to act by occluding dentinal tubules since scanning electronmicroscopic studies have failed to demonstrate any visual changes to the dentine surface treated with sodium monofluorophosphate.[21] Any tubule occlusion which might occur does not appear to be permanent.[29]

Stannous fluoride

Stannous fluoride in either an aqueous solution or in glycerine gelled with carboxymethyl cellulose is effective in controlling dentinal hypersensitivity.[50] The mode of action appears to be through the induction of a high mineral content which creates a calcific barrier blocking the tubular openings on the dentine surface.[51] Alternatively, stannous fluoride may precipitate on the dentine surface leading to occlusion of the exposed dentinal tubules.[21]

Fluoride iontophoresis

Iontophoresis is the process of influencing ionic motion by an electric current and has been used as a desensitizing procedure in conjunction with sodium fluoride.[42] Studies report that there is an immediate reduction in sensitivity after treatment with iontophoresis, but the symptoms gradually return over the ensuing six months.[52] This method has enjoyed some popularity but more controlled studies are required.[53]

Formaldehyde or glutaraldehyde

Claims have been made that formaldehyde and glutaraldehyde, through their ability to precipitate salivary proteins in dentinal tubules, can be used to manage dentinal hypersensitivity. However, this effect has been questioned since various formulations have been found to have little or no effect on dentinal hypersensitivity.[49,54] Given that these agents are very strong tissue fixatives, they should be used with extreme caution to ensure they do not come in contact with the vital gingival tissues.

Dentine sealers

Resins and adhesives

Sealing of dentinal tubules with resins and adhesives has been advocated for many years as a means of managing dentinal hypersensitivity.[55] In general, results have been good but problems arise when the adhesive breaks away resulting in exposure of the tubules. This technique is generally reserved for cases of specific and localized dentinal hypersensitivity rather than generalized dentinal pain.[21]

Lasers

Both the Nd:YAG and CO_2 lasers have been studied for their use in managing dentinal hypersensitivity. Both applications rely on their ability to occlude the dentinal tubules. The Nd:YAG laser has been used in conjunction with sodium fluoride varnish with encouraging results showing up to 90 per cent of the dentinal tubules being occluded through use of this combined therapy.[56] CO_2 laser irradiation and stannous fluoride gel has also been shown to be effective for inducing tubule occlusion for up to six months after treatment.[57,58] While still largely experimental, this technique requires further scientific investigation before it becomes a clinically acceptable means of treatment.

Combination of desensitizing agents and fluoride

Because some effective desensitizing dentifrices (such as those containing potassium nitrate) were not

fluoridated, people requiring relief from dentinal sensitivity and protection against dental caries needed to use both desensitizing and fluoridated dentifrices. As a result a number of well-controlled clinical studies have been carried out to determine the efficacy of potassium nitrate as a desensitizing agent in dentifrices containing fluoride.[59-62] As early as 1992, the United States Food and Drug Administration granted Category 1 status to combination 5% potassium nitrate:fluoride dentifrices, indicating they were not only safe but also effective.[63]

Restorative materials

The use of restorative materials is generally an invasive solution to the problem of hypersensitivity. Commonly used materials include composite resins and glass ionomer restorations. Generally this approach is reserved for situations where there has been significant prior loss of cervical tooth structure or as a last resort for a tooth which does not respond to other less invasive desensitizing protocols.

Periodontal surgery

There are numerous soft tissue grafting procedures which can be carried out to cover exposed root surfaces including lateral sliding grafts, free gingival grafts, connective tissue grafts and coronally repositioned flaps. While these procedures may cover exposed dentinal tubules, some are not very predictable in terms of their efficacy in root surface coverage. Soft tissue grafting for localized recession defects requires careful planning and an understanding of the anatomical defect to be treated. In general, soft tissue grafting for the management of sensitivity is not regarded as a very predictable treatment strategy.

Safety of commonly used at home desensitizing agents

Most "at home" desensitizing agents are generally restricted to dentifrices and mouthrinses containing one or a combination of the agents discussed above. Of these, the most common "active ingredients" are potassium nitrate, stannous fluoride, sodium fluoride, sodium monofluorophosphate and strontium chloride.[64] For all products which are currently on the market as over-the-counter products extensive toxicity testing has already been carried out by the manufacturers and has been reviewed by various regulatory bodies to ensure both safety and efficacy.

Although there is only minimal evidence to demonstrate superiority of one desensitizing agent over another, there is ample evidence that desensitizing toothpastes do provide benefit to the patients suffering from dentinal hypersensitivity.[19,21,65,66]

As a general rule, all well performed clinical studies published in the literature report safety assessments of the products under study.[60] Moreover, in recent times, studies carried out within universities require the approval of a human ethics committee prior to commencement. All such committees require safety issues of products under investigation to be reported as part of the routine experimental protocol.

Safety measures for toothpastes generally include adverse event data to elicit complaints/symptoms of the subjects at each examination time point and by evaluating changes in medical history and concomitant medications. All adverse reactions are recorded, listing the date of onset, duration, frequency, maximum intensity, seriousness, action taken and outcome. In addition, the oral soft tissues and perioral area should always be visually examined to evaluate effects that could be manifested as a tissue response to an irritant, soft tissue pathology or other clinically meaningful deviations from the normal.

It is of particular note here that there are very few (if any) reports of adverse reactions to dentifrices containing the desensitizing agents listed above (Table 2). A Medline search (concluded December 2005) failed to find any citations regarding adverse reactions, toxicity or safety problems with strontium chloride or potassium nitrate. Only a few citations were found for fluoride formulations and these involved predominantly children.

Since dentinal hypersensitivity is considered to be a response to a localized condition within individual teeth and is not indicative of, or related to, any known systemic condition, management of dentinal hypersensitivity is not considered to mask or impact on any other systemic conditions which a patient might have. There is no literature to support the concept that dentinal hypersensitivity is anything other than a well-localized dental problem.

Finally, one of the most effective treatments for the majority of patients is simple daily plaque removal. Daily plaque removal over time allows demineralization of the dentinal tubules from salivary minerals and can alleviate much of the discomfort caused by exposed dentine. The use of an additional aid, such as a dentifrice containing additional agents which might expedite (or at least encourage) improved oral hygiene and daily plaque removal, is not considered harmful to either the tooth, the surrounding soft tissues, or the whole body.

CONCLUSION

Dentinal hypersensitivity is a relatively common and significant dental problem which can be successfully managed by a very wide variety of procedures, agents and formulations applied locally, either "in office" or "at home". It is clear that some products appear to be more effective than others. For those products developed for self application at home, potassium nitrate, stannous fluoride, sodium fluoride, sodium monofluorophosphate and strontium chloride have all been extensively studied and shown to be not only safe to use but of benefit to individuals suffering from dentinal hypersensitivity.

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