Dentin sensitivity, or hypersensitivity, may be defined as a transient pain arising from exposed dentin, typically in response to chemical, thermal, tactile and/or osmotic stimuli. The enamel or cementum which normally covers the dentin surface may be removed by attrition from occlusal wear, abrasions, abrasive tooth brushing, erosion from acidic diet, gingival recession, or tooth preparation for restorative treatment. Alternatively, in some individuals the cementum and enamel which normally cover the dentin do not meet, and dentin is thus exposed as a result of a developmental anomaly. The pain response also varies substantially from one person to another. This review article describes the etiology, epidemiology, assessment, and conventional treatment of dentin hypersensitivity.

**Keywords:** assessment, conventional treatment, dentin hypersensitivity, dentin permeability, epidemiology, etiology, placebo effect.
The smaller nerve fibers which terminate in the dental pulp have been variously interpreted. Investigators differ in their opinions regarding the numbers of the unmyelinated and the smallest myelinated nerve fibers. The smaller myelinated fibers have been regarded as numerous and the sympathetic unmyelinated fibers as few in number.14,15 The diameter of most of the fibers is no larger than 4 μm. The total number of fibers varies considerably, with the extreme figures being 151 to 1296 fibers per single tooth. A relation between caliber ranges and nerve function seems to exist.16 Most unmyelinated fibers are found near or attached to the blood vessels, and it was hypothesized that they had an autonomic function.17 Experiments carried out in dogs have shown that there are two groups of intradental nerves which respond to thermal stimulation of dentin: one which is excited by cooling and another which is excited by heating.18,19

As already mentioned, the dental pulp is innervated by both myelinated and unmyelinated axons. The nerves in the apical region of normal human pulps were investigated with the transmission electron microscope.20 It was observed that there were more unmyelinated than myelinated axons within an apical pulpal cross section, 72% and 28%, respectively. Identification of myelinated and unmyelinated axons has often been based on conduction velocity measurements.19,21 Correspondingly, according to conduction velocities (cv), which is the rate at which these fibers conduct impulses, the nerve units can be classified into A (cv > 2 m/s) and C groups (cv ≤ 2 m/s). Most of the A-fibers have their conduction velocities within the Aδ range (< 30 m/s) and those with conduction velocities higher than 30 m/s can be classified as Aβ-fibers. The C-fibers of the pulp do not respond to the same type of dentinal stimulation. It seems that activation of the A-fibers produce a sharp, well-localized pain, whereas C-fibers may be related to the second dull, radiating pain sensation. Intradental C-fibers are activated by inflammatory mediators, prolonged application of heat, and direct mechanical irritation of the dental pulp.22-24

It is now generally accepted that the dentin is innervated. However, the question as to whether the nerve fibers end at the inner dentin or extend up to outer dentin is still unanswered. Physiological studies conducted in order to explain the etiology of dentin hypersensitivity failed to demonstrate that nerve fibers continue into the dentinal tubules.25 On the other hand, Fearnhead26 has shown that small nerve fibrils with an approximate diameter of 0.2 μm were in close relationship to the odontoblast process as far as 1.5 mm in the dentin. It was not possible, however, to determine whether these fine fibrils extend as far as the enamel-dentin or cementum-dentin junction. Moreover, the studies by Frank27 and Dahl and Mjör28 support the presence of stable connections between the odontoblast process and the nerve fibers.

THE STRUCTURE OF DENTIN AND ITS PERMEABILITY

Examination of human dentin by phase contrast microscopy has revealed that the odontoblast process continues with the form of a large number of lateral branches.29 Using scanning electron microscopy, the odontoblast processes in the tubules were seen in the predentin and dentin, and this up to a distance of 0.2 mm from the pulp. However, no nerve-like fiber was seen in the dentinal tubules.30

The number of tubules was found to be 45,000/mm² near the pulp with a diameter of 2.5 μm, whereas in the middle of the dentin there were 29,500/mm² tubules with a diameter of 1.2 μm. Odontoblast processes were observed only in tubules near the pulp and no peritubular dentin was apparent.31

Other investigators quantified the hydraulic conductance of dentin.32-34 This value, which by definition is a measure of the ease with which fluid under hydrostatic or osmotic pressure can pass through a permeable barrier (in this case, dentin), was found to increase as the surface area increases and/or as the dentin thickness decreases. Interestingly, the acid etching procedures led to a 32-fold increase due to removal of surface debris occluding the tubules.32 Temperature was found to have a great effect on the hydraulic conductance of dentin. Increasing the temperature by 40°C (ie, from 10 to 50°C) resulted in a 1.8-fold increase in fluid flow in unetched dentin and in a 4-fold increase in acid-etched dentin. This increment in hydraulic conductance was attributed to the thermal expansion-induced increases in tubular diameter.33 Finally, the radicular dentin hydraulic conductance decreased with distance from the pulp and with increasing dentin thickness. This value in radicular dentin was found to be much lower than that in coronal dentin, which suggests that the diffusive permeation across radicular dentin would be lower in an analogous manner.34

The peripheral dentin was found to be more permeable than the dentin in the central region, which may have a higher mineral content and more intertubular dentin matrix.35 According to Outhwaite et al36 and Pashley et al,37-40 once dentinal tubules are exposed,
the rate at which solutes permeate across dentin is determined by the following variables: molecular size, degree of occlusion of the tubule orifices by a “smear layer” of debris, area of dentin surface, thickness of the dentin, presence or absence of pulp tissue, and the proximity of the exposed dentin surface to the pulp.

A tendency towards natural desensitization with time is also evident, since not all patients with exposed root surfaces experience dentin hypersensitivity. Karlsson and Penney have shown that the formation of “acquired pellicle” in exposed root surfaces in dogs accounted for a desensitization with time. The surface smear layer was first described by Eick et al in 1970, as a film of microcrystalline debris created when dentin structure is cut. However, its occluding effect on the dentinal tubules seems to be relatively short-lived.

Pashley focused attention on the effects of plasma proteins from the underlying pulp vessels on dentin permeability. Increased amounts of plasma proteins may occur for various reasons, eg, inflammation due to trauma, and neurogenic inflammation. Fibrinogen was one of the most effective agents for producing acute reductions in dentin permeability. Tubular occlusion by peritubular dentin deposition and the formation of irregular secondary dentin is also expected to reduce permeability. As early as 1972, Mjör described different patterns of occlusion of the tubules which resulted in increased mineralization of the dentin tissue. When Vongsavan and Matthews examined the permeability of cat dentin, they observed that there was no diffusion of dye in the dentinal tubes when the experiments were performed in vivo. They considered that the outward rate of fluid was the most important factor that determined the diffusion of chemicals through the dentinal tubules.

Opinions differ as to the extent to which bacteria from saliva and plaque can penetrate dentinal tubules. The study by Olgart et al has shown that bacteria can grow into tubules in intact vital dentin, regardless of the outward fluid flow or the presence of smear layer, since this mechanical barrier is easily removed after some days. On the other hand, Michlich et al have suggested that bacteria do not enter etched dentinal tubules due to the presence of smear layer, but they easily penetrate acid-etched dentin. Moreover, in a study of the distribution of bacteria within the radicular dentin and cementum in periodontal patients, bacterial growth was detected in 87% of the periodontally diseased, caries-free teeth. Additionally, when cementum was absent, there was a faster rate and depth of acid demineralization than that observed in normal roots, suggesting the protective role of an intact cementum layer.

THE SENSITIVITY OF DENTIN

Experiments carried out by Anderson et al in 1958 indicated that the sensation of pain from dentin does not depend on direct stimulation of the endings of nerve fibers in the dentinal tubules. Rather, the stimuli seem to be transmitted through the dentinal tubules. The electrical excitability of odontoblasts was determined on both dog and human teeth. These findings indicated that electrical impulse conduction by odontoblast processes in the dentin was not possible, since the odontoblasts did not show electrical excitability. Additionally, recordings made from dentin at the tip of the canine teeth of cats suggested that the nerves were excited by a change in ionic composition of the extracellular fluid, rather than by displacement of the contents of the dentinal tubules.

In contrast, a series of experiments that were initiated in the early 1960s by Brännström et al provided evidence that the main cause of dentinal pain is a rapid outward flow of fluid in the dentinal tubules that is initiated by capillary forces. Based on the results of experiments, a hydromechanical theory was advanced to account for the sensitivity of the dentin (Fig 1). According to this theory, if movement occurs rapidly enough, the displacement of tubular contents may produce deformation of nerve fibers in the pulp or predentin, or damage the odontoblastic cells. Both effects can result in pain.

A feature of the response of the dentin and pulp to stimuli is the aspiration of odontoblastic nuclei into the dentinal tubules. In a study by Kramer, however, where the incidence of pain was compared with the incidence of aspiration of cells into the dentinal tubules, no correlation was found between the incidence of this disturbance and pain experience.

Nevertheless, further studies by Brännström et al supported the hydrodynamic theory of dentin sensitivity: the whole pulp seems to function as a kind of mechanoreceptor and painful stimuli applied to the exposed dentin stimulate this receptor structure mechanically. They have suggested that the role of the odontoblasts for the elicitation of pain must be questioned. However, Panopoulos et al demonstrated that there must be at least two different mechanisms of transmission of the stimuli through dentin. The results of their experiments supported an indirect mechanism based on the hydrodynamic theory when stimuli are...
applied on thick layers of dentin. In deep cavities, on the other hand, the stimuli seemed to directly excite the intradental nerves. More recently, a stimulus transmission mechanism has been hypothesized from the presence of indented structures 40 to 80 nm deep, known as coated pits, on the cell membrane of the odontoblastic processes in the proximity of the nerve fibers.65

Pashley et al also studied the effects of prolonged air blasts on human dentin permeability.66 The reduction in dentin sensitivity was attributed in part to the evaporation of water, which in turn raised the concentration of organic and inorganic components of dentinal fluid. Long-lasting irritation of the dentin surface of dog canine teeth has shown disruption of the odontoblast layer and its separation from the predentin. The condition of the dentin surface was supposed to be the decisive factor rather than the pulp tissue.67

Erosion, abrasion, attrition, and possibly abfraction lead to exposure of tubules. Exposure of the dentin by attrition will lead to secondary dentin formation.58,69 It seems that pain-producing stimuli are more readily transmitted from a dentin surface when the exposed tubule apertures are wide open.70 The sensitivity of dentin is directly related to the size and patency of the dentinal tubules.46,71,72 In a study to determine differences between sensitive and nonsensitive teeth, Absi et al73 reported that nonsensitive teeth were unresponsive to the tested stimuli and had very few exposed tubules. In contrast, sensitive teeth had 8 times as many visible tubules at the root surface than nonsensitive teeth. Similarly, the average diameter of tubules in sensitive teeth was almost 2 times greater. It is the width of the tubules which is particularly relevant, since fluid flow is proportional to the fourth power of the tubule radius: doubling the tubule’s diameter results in a 16-fold increase in flow rate.74

Scanning and transmission electron microscopic characterization clearly showed that the lumens of most of the tubules were occluded with mineral deposits in naturally desensitized areas, but they were empty in hypersensitive areas.75,76 A significantly high correlation between the dentinal tubule morphology and dentin hypersensitivity was also demonstrated using scanning electron microscopy on replica models.77 It was interesting to notice that hypersensitive points were more numerous in the distal and mesial parts than in the central, which suggested that there

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**Fig 1** The hydrodynamic mechanism by which stimuli activate intradental nerves to cause pain.
are many factors contributing to hypersensitivity. Additionally, no pain could be elicited in the presence of smear layer. The presence and morphology (amorphous or crystalline) of smear layers was studied with the use of replica models from teeth with exposed dentin. In the hypersensitive dentin, the smear layer was thinner and probably undercalcified compared to the nonsensitive dentin.

THE ROLE OF INFLAMMATION IN DENTIN HYPERSENSITIVITY

A two-way communication seems to exist between sensory nerves and pulpal cells. Such interactions could play an important part in the genesis and maintenance of inflammation in the dental pulp. The nerve fibers mainly responsible for dentin hypersensitivity in the initial stage are the A-fibers. The responsiveness of these fibers can be affected by obstructing and opening the dentinal tubules. Inflammation in the pulp can considerably alter dentin sensitivity. A patient that suffers from hypersensitive dentin may perceive an exaggerated, intense pain or a more continuous pain stimulus of longer duration which would not be expected to originate only from the hydrodynamic stimulus. The latter is likely to be associated with pulp inflammation. It has been shown that A-fibers are relatively insensitive to inflammatory mediators, whereas C-fibers may take an active part in the development of a pulp inflammation. Intradental C-fibers can release neuropeptides, including substance P and CGRP which function in the inflammatory reactions. It was concluded that although C-fibers do not respond to dentin stimulation, they may have an important role in dentin sensitivity.

A relationship of pulpite and hyperemia to thermal sensitivity was supported in the study by Dachi. Pulpal hyperemia can be significantly associated with increased sensitivity to both cold and heat. Stimulation of intradental afferent nerves can produce vasodilation in the pulp, and in turn an increase in the rate of outward flow of fluid through the dentin. Along with the increase in pulpal blood flow, there is also an increase in tissue pressure that in turn will cause excitation of both Aδ and C fibers. In contrast, reduction of fluid flow has an inhibitory effect on Aδ fibers, but no discernible effect on C fibers. Consequently, the vaso-dilation appears to be produced by an axon reflex mechanism that is part of a neurogenic inflammatory response which will decrease the rate of diffusion of toxic products into the pulp. The other components of neurogenic inflammation, including an increase in capillary permeability and in local blood flow, will also contribute to the defense of the pulp.

EPIDEMIOLOGY

According to a study of Orchardson and Collins, hypersensitive dentin was found in all types of teeth, but was commonest in canines (25%) and first premolars (24%), and especially (93%) on buccal surfaces. The age or the gender of the patients did not have a major influence on the prevalence of hypersensitive teeth, although Curro reported that the incidence of dentin hypersensitivity appears to peak around the third decade of life. Dentin hypersensitivity seems to be a common problem, with various reports indicating an incidence between 8% and 57% of the population. The wide range of results reported is possibly due to different methods used to diagnose the condition. Bartold mentioned that the most commonly affected teeth were the maxillary premolars, and the most frequent initiating factor was cold drinks.

According to a study by Chabanski et al, 72.5% to 98% of periodontal patients complained of hypersensitive dentin, with no significant gender difference. The condition was more prevalent in molars, followed by left canines and premolars. A tendency for a greater number of sensitive teeth was also found when these patients smoked or came from higher socioeconomic levels. The higher prevalence of dentin hypersensitivity in periodontal patients may reflect a different etiology, since in periodontal disease, bacteria are reported to penetrate into dentin to a considerable distance.
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THE PLACEBO EFFECT

In 1955, Henry K. Beecher 106 published the classic work entitled “The Powerful Placebo”. This publication is still the most frequently cited placebo reference. Placebo effects are commonly referred to in the dentin hypersensitivity clinical trial literature. A positive practitioner-patient relationship can motivate a patient to obtain relief. Furthermore, positive emotional and motivational behavioral responses can activate the central release of endorphins. 107 In the management of dentin hypersensitivity, faith in the practitioner and the desire to obtain relief contribute to the placebo effect. Yet another possible phenomenon which could occur is the “Hawthorne effect”. The “Hawthorne effect” is a response to non intervention procedures, such as frequent examinations and improved oral hygiene. Patients frequently appear to improve merely from the effects of being placed in a trial. 108,109

In placebo-controlled studies, 110,111 both the test and control groups often show significant improvement. It is concluded that either the therapeutic agents have no effect or the activity is masked by the magnitude of the placebo response.

False impressions of placebo effects can be produced in various ways: spontaneous improvement, assessment bias, answers based on politeness, misquotation, etc. A wide range of errors was found in the placebo literature, which produce false impressions of placebo effects. 112 Thus, the claimed extent of the placebo effect remains questionable.

CONVENTIONAL TREATMENT OF DENTIN HYPERSENSITIVITY

By definition, dentin hypersensitivity is associated with dentin exposed to the oral environment. Dentin may become exposed by two processes: either loss of covering periodontal structures, usually termed “gingival recession”, or loss of enamel.

Gingival recession has long been related to aging, but it seems that other factors can also contribute to this condition. Anatomical factors such as fenestration and dehiscence of the alveolar bone or malposition of the teeth and inflammation associated with various forms of injury have been implicated. 113,114 Periodontal therapy has also been considered a causative agent of gingival recession. Periodontal root planing removes the cementum and exposes the underlying dentin. However, a smear layer is produced that covers the dentin surface and initially reduces the dentin permeability. This protective layer can nevertheless be lost by dietary or plaque acids, and dentin hypersensitivity may occur. 115 In addition, periodontal surgery may expose the root surface of the teeth and this newly exposed dentin is frequently hypersensitive. 116

Loss of enamel can also occur due to attrition associated with parafunctional activity, abrasion from dietary components, zealously and vigorous toothbrushing, or due to erosion associated with environmental or dietary components, particularly acids. 117-120 According to a study by Smith and Knight, 121 dietary and regurgitation erosion were the most common causes of tooth wear. Regurgitation erosion caused the most severe damage; attrition was secondary in importance to erosion, and abrasion the least important. It has been advised that in cases of dentin sensitivity, toothbrushing should precede dietary acid application or at least be separated from mealtimes to protect the demineralized tissue from accelerated abrasion. 122,123

Dentin hypersensitivity and abfractive lesions seem to be associated with signs of occlusal trauma. 124,125 Additionally, the cementum-dentin junction is a wide zone of lower mechanical properties, compared with the adjacent hard tissues, cementum and dentin, probably because of lower mineral and higher organic content. 126 Since occlusal hyperfunction, eccentric loading, and parafunction are considered to be cofactors in the etiology of abrasion, a judicious occlusal analysis and
equilibration are indicated to eliminate dentin hypersensitivity.127

Dentin hypersensitivity treatment methods are numerous90,95,107,128-136 and can be classified according to their mode of delivery, while the causative factors must be appreciated so that prevention can be included in the treatment plan. Currently, there are at least two recognized mechanisms of action of desensitizing agents. One involves blocking fluid movement by occluding tubules (Fig 2). The other involves blocking pulpal nerve activity by altering the excitability of the sensory nerves. Some desensitizing agents are believed to act by reducing intradental nerve activity.137 To date, no single agent or form of treatment has been found effective for all patients. Grossman138 suggested that the ideal desensitizer should (1) not unduly irritate the pulp, be (2) painless when applied, (3) easy to apply, (4) consistently effective, (5) permanently effective, (6) quick acting, and finally (7) should not cause tooth discoloration. Most of the methods currently in use fall short of these criteria.

CLINICAL TRIALS AND IN VITRO STUDIES OF SOME DESENSITIZING AGENTS

Calcium Compounds

Calcium hydroxide
Calcium hydroxide has been a popular agent for the treatment of dentin hypersensitivity for many years, especially after periodontal treatment. Following periodontal surgery, Jorkjend and Tronstad139 applied a creamy paste of calcium hydroxide to the exposed root surfaces, and then covered the paste with a thin layer of methacrylate and a gingival pack. When the pack was removed 7 days later, the teeth were no longer sensitive to cold water, cold air, or carbohydrates.

The study by Levin et al140 evaluated the use of calcium hydroxide and magnesium hydroxide in the treatment of hypersensitive dentin. The calcium hydroxide was the most effective immediately and after 6 months. The authors advised that some caution be exercised in the application of calcium hydroxide, as irritation can result from the very alkaline pH. Magnesium hydroxide was also evaluated in the study because it is less alka-
line. According to the results, it proved less effective. A comparison between calcium hydroxide and potassium nitrate as a potential desensitizing agent was performed in order to eliminate hypersensitivity after periodontal surgery. In this study, controlled application of quantitative stimuli was used to evaluate the results of the treatment, in contrast with other studies that are used only the patient's subjective responses. Using a thermoelectric and a mechanical stimulating device, calcium hydroxide was found to be more effective than the potassium nitrate or the control.

Pashley et al\textsuperscript{142} studied the effects of calcium hydroxide on dentin permeability and concluded that topical treatment of dentin with calcium hydroxide paste decreases the hydraulic conductance of dentin by 52%. They proposed that the immediate desensitization reported by most of the clinical studies can be explained by a rapid physicochemical mechanism. The dentinal tubules in this in vitro study were filled with a phosphate-buffered saline. The combination of a high calcium concentration and high pH (pH: 13) is considered to produce aggregates of calcium phosphate from the phosphate-buffered saline in the dentinal tubules.

Dibasic calcium phosphate, calcium phosphate precipitation method (CPP)

Burnishing with a paste of dibasic calcium phosphate (CaHPO$_4$) for 1 min has demonstrated resolution of dentin hypersensitivity. However, dentin hypersensitivity associated with occlusal trauma did not always respond to treatment with CaHPO$_4$ crystals.\textsuperscript{143} The occlusion of dentinal tubules with calcium phosphate by a calcium phosphate precipitation (CPP) method was investigated.\textsuperscript{144} The addition of NaF to the post-treatment solution led to more apatitic precipitate formation.\textsuperscript{145} Studies after 1, 2, 5, and 7 days of immersion in synthetic saliva have shown a continuous effect in reducing the dental permeability.\textsuperscript{146} In another study,\textsuperscript{147} the addition of CaSiF$_6$ improved the CPP method, and several post-treatment solutions for the CPP method were evaluated.\textsuperscript{148}

Fluoride Compounds

Fluoride was first proposed as a desensitizing agent by Lukomsky in 1941.\textsuperscript{149} Further studies conducted by Hoyt and Bibby\textsuperscript{150} in 1943, in accordance with the suggestions of Lukomsky, demonstrated that the effects of the sodium fluoride treatment were very satisfactory and seemed to last for many months. Fluoride-treated surfaces showed increased resistance to acid decalcification, and fluoride compounds have long been considered effective cariostatic agents.\textsuperscript{151} Most compounds that are useful as caries prophylactics were also proposed for dentin desensitization. The fluoride penetrates the dentin along the dentinal tubules and forms calcified intratubular deposits.\textsuperscript{152} Along with its anticaries properties, a toothpaste containing 0.76% sodium monofluorophosphate was shown to perform significantly better in reducing dentin hypersensitivity than a 10% strontium chloride toothpaste.\textsuperscript{153} On the other hand, Pashley et al\textsuperscript{154} using an in vitro model, concluded that the efficacy of a NaF/kaolin/glycerin paste in reducing dentin hypersensitivity is due primarily to the burnishing action which created a smear layer in the dentin, rather than to the fluoride paste. The time required for total elimination of excess F was about 1 week for a topical treatment with an acidulated fluoride phosphate solution containing 1.2% F.\textsuperscript{155} However, in another study, scanning electron micrographs showed precipitations of fluoride on the peritubular dentin after 6 months.\textsuperscript{156} This indicates a long-lasting topical fluoride effect in spite of a relatively short-lived fluoride incorporation. This conclusion is supported by a study by Ritter et al,\textsuperscript{157} who found that two fluoride varnishes were effective in reducing dentin hypersensitivity for a period of up to 6 months.

Fluoride Iontophoresis

Iontophoresis with sodium fluoride solutions was also proposed as a method of treating hypersensitive dentin. This procedure uses electricity to enhance diffusion of ions into the tissues. The daily use of an iontophoretic toothbrush with fluoride toothpaste not only had an anticaries effect but also proved to be very effective in reducing dentin hypersensitivity.\textsuperscript{158,159} Murthy et al\textsuperscript{160} reported the effectiveness of fluoride iontophoresis in a doubled-blind, controlled clinical trial. They compared iontophoresis of sodium fluoride to a placebo and concluded that 1% fluoride iontophoresis provided a statistically more effective treatment than placebo or topical applications. Pashley et al\textsuperscript{161} indicated that iontophoresis may be useful for enhancing dentin permeation of ionized substances for therapeutic purposes. Several other reports have shown that iontophoresis of fluoride was more effective in reducing dentin hypersensitivity than was topical application alone. In a study by Wilson et al,\textsuperscript{162} the fluoride penetration in successive layers of dentin has been measured after topical and iontophoretic application to exposed dentin. The results demonstrated that iontophoresis significantly enhanced both the amount
and depth of penetration by fluoride ions in the outer 50 μm of dentin.

On the other hand, a clinical study that compared the effectiveness of sodium fluoride treatment with and without iontophoresis in the same group of patients indicated that both forms of treatment were effective in reducing dentin hypersensitivity, with no significant difference between them. Similarly, the study by Brough et al demonstrated that the use of sodium fluoride or even distilled water with and without iontophoresis was effective. They attributed the reduction in sensitivity when distilled water was used alone to the formation of reparative dentin or to a possible placebo effect.

**Strontium Compounds**

Strontium has a considerable affinity for dentin due to its high permeability and the possibility for absorption into or onto the organic connective tissues and the odontoblast process. The ability of strontium to exchange with calcium of root dentin and dentin in powder form was also studied. This study concluded that the exact mechanism of strontium interaction with dentin is not clear, since the displaced calcium was not proportional to the strontium uptake in root dentin, although in powdered dentin the ratio was 1:1. In an animal model, a possible desensitization after only 4 to 7 days of topical application of zinc fluoride or strontium chloride was studied. Both toothpastes penetrated deeply into the dentin, but for strontium chloride desensitization to be effective, it seemed that repetitive applications or longer application times may be needed.

Moreover, Gedalia et al reported that topical application of 10% strontium chloride prior to application of 2% sodium fluoride was more effective in decreasing sensitivity than sodium fluoride alone. They concluded that a mineralization process is the fundamental mechanism by which fluoride or strontium reduces hypersensitivity. It was also proposed that burnishing of strontium halide with a rubber prophylaxis cup will obtain maximum depth of penetration.

In another study, the effectiveness of two toothpastes containing sodium citrate in a pluronic gel with and without fluoride were compared with a toothpaste containing 10% strontium chloride. After 8 weeks of treatment, all three products significantly reduced the dentin hypersensitivity. Similarly, a comparable reduction of the symptoms was evident when two strontium and a conventional fluoride toothpaste were used.

**Oxalates**

In a series of studies, Pashley et al studied oxalate solutions and toothpastes, and found them superior to other popular toothpastes in occluding the dentinal tubules. Other investigators have indicated that products containing oxalate are capable of covering the dentin surface and occluding the tubules to varying degrees. Gillam et al studied the desensitizing effects of different agents using the dentin disk model and concluded that products containing oxalate were able to occlude the dentinal tubules to varying degrees. However, their long-term effectiveness has not been determined. Similarly, Santiago et al found that different oxalate compounds reduced the dentin permeability by an average of 75%. Another study showed that 6% ferric oxide (Sensodyne Sealant: GlaxoSmithKline; Brentford, UK) and a bis-GMA and HEMA resin combination (Scotchbond Multipurpose: 3M Dental; St Paul, MN, USA) were equally effective in sealing dentin.

Apart from these in vitro investigations, some clinical trials evaluated the effectiveness of oxalate compounds. The results showed a significant reduction of dentin hypersensitivity immediately after the application of the treatment solution and after 4 weeks.

**Potassium Nitrate**

The use of a 5% potassium nitrate toothpaste as a daily home treatment seems to be effective for patients with dentin hypersensitivity. The mode of action of potassium ions can be directly related to nerve excitability by raising the extracellular potassium ion concentration to prevent action potential generation. Peacock and Orchardson determined the concentration of potassium ions that is necessary to block nerve conduction in vitro, although they considered that it remains questionable whether this substance can diffuse through dentin in a clinical situation due to the outward flow of dentinal fluid.

**Dental Adhesives and Resins Composites**

New dentin adhesive systems seem to protect the exposed surfaces from further erosion and abrasion, although their efficacy seems to decrease with time, and their long-term effectiveness is questionable. A study by Belluz et al compared the quality of cervical restorations using a composite and a flow-
able resin. There was no statistically significant difference regarding the performance of the two materials tested, and a decrease of the quality of the restorations was recorded over time for all USPHS criteria. It seemed that the flowable resin tested did not perform better than the traditional composite regarding maintenance of the quality of restorations.

CONCLUSION

There are a number of alternative modalities that will desensitize hypersensitive dentin. The decisive factor is what works best for the individual dentist and/or patient. Apparently, the placebo effect is always present, and it appears that at least 30% of the time, anything that is done will achieve a result.

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Contact address: Professor Dr. Roeland De Moor, Department of Operative Dentistry and Endodontology, Ghent University, Ghent University Hospital, Dental School, De Pintelaan 185 - P8, B-9000 Gent, Belgium. Tel: +32-9-332-4000, Fax: +32-9-332-3851. e-mail: roeland.demoor@ugent.be