Community Dental Health (2014) **31,** 15–20
Received 6 September 2013; Accepted 2 October 2013

doi:10.1922/CDH_3287Cartwright06

Dentinal hypersensitivity: a narrative review

R.B. Cartwright

School of Biosciences, University of Birmingham, Edgbaston, Birmingham, UK

Introduction: Dentinal hypersensitivity is an exaggerated response to a sensory stimulus that usually causes no response in a normal healthy tooth. It is a source of chronic irritation that can severely affect an individual's eating and drinking habits. The management of tooth hypersensitivity by oral healthcare professionals requires an appreciation of the complexity of the problem together with knowledge of available treatments. ***Aim:*** To review the symptoms, contributing oral factors, prevalence, measurement and mechanisms of dentinal hypersensitivity, together with current and potential future therapies for the condition. ***Method:*** Narrative literature review. ***Principle findings:*** The permeability and fluid movement in open, exposed dentinal tubules has provided a favoured theory for stimulus transmission through dentine. Occlusion of dentinal tubules has been identified as a potential method of reducing pain associated with sensitive teeth. Current treatments work to occlude dentinal tubules. However these treatments can be expensive and their effects are often transient. In comparison, future therapies could be based upon either laser or iontophoresis techniques. ***Conclusion:*** Future therapies may provide a more permanent and cost effective way of treating dentinal hypersensitivity for health care professionals and their patients.

Key words: *dentinal hypersensitivity, dentinal tubules, desensitizing agents*

Symptoms of dentinal hypersensitivity

The symptoms of dentinal hypersensitivity are characterised by the rapid onset of pain that is sharp and short in duration; it has been described as a chronic condition with acute episodes (Addy *et al.,*1987; Orchardson and Collins, 1987). Dentinal hypersensitivity can occur due to the exposure of open dentinal tubules to various stimuli within the oral environment (Absi *et al.,* 1989; Brännström, 1963). These stimuli may be thermal, chemical, tactile or osmotic in nature. The pain may continue for a variable time as a dull vague sensation in the affected tooth even after the stimulus has been removed (Brissada, 1994). The use of the keywords, attributed to this paper, to conduct literature searches of journal citation databases such as Pubmed.gov, reveal a significant amount of research that has been conducted to understand the symptoms, contributing oral factors, prevalence, measurement and mechanisms of dentinal hypersensitivity (Montori *et al.,* 2005).

Before a diagnosis of dentinal hypersensitivity is made, other forms of dental defects must be ruled out since other conditions may elicit similar symptoms (Chu *et al.,* 2010). These include cracked or chipped teeth, fractured or improperly insulated metallic restorations, post restorative sensitivity, dentinal caries, hypoplastic enamel, or a congenitally open cementum–enamel junction (Wallace and Brissada, 1990). Alternatively, pain may arise in the pulp due to inflammation (Narhi *et al.,* 1994). A thorough diagnosis is required to rule out alternative causes of tooth pain. This should include a history and thorough clinical and radiographic examination of the tooth in question as well as all adjacent teeth (Brissada, 1994).

Oral factors that contribute to the development of dentinal hypersensitivity

Dentinal hypersensitivity may often occur due to the exposure of dentinal tubules after cervical abrasion or erosion, excessive intake of dietary acids or periodontal therapy (Pol *et al.,* 2011). Abrasive tooth brushing and the use of abrasive toothpastes may also initiate dentinal hypersensitivity (Addy, 2005). These actions can damage or remove tooth tissue and may also remove protective plaque layers, exposing open dentinal tubules to stimuli within the oral environment (West *et al.,* 1998). The ingestion of fruits, fruit juices and other acid beverages are able to produce oral conditions that can cause tooth erosion (Larsen, 1975). The critical pH below which enamel erodes, is pH 5.5 (±0.5), leading to dentinal tubule exposure and subsequent dentinal hypersensitivity. Many fruit juices have a pH below this (Touyz and Glassman, 1981). However, levels of calcium and phosphate, in the form of hydroxyapatite, are supersaturated in saliva at normal intra-oral pH. In acute episodes of hypersensitivity, exposed dentinal tubules may be plugged by calcium phosphate deposits from the saliva.

The equilibrium for the dissolution of hydroxyapatite is described by the formula:

$$Ca_{10}(PO_4)_6(OH)_2 \leftrightarrow 10Ca^{2+} + 6PO_4^{3-} + 2OH^-$$

If the pH of the oral environment falls, PO_4^{2-} is converted to HPO_4^{3-} or $H_2PO_4^{3-}$ and OH^- is neutralised to form water. Under these conditions, saliva will no longer be supersaturated with respect to free calcium and phosphate and will not easily plug exposed dentinal tubules; hypersensitivity will therefore, persist (Touyz,

Correspondence to: Richard Bryan Cartwright, School of Biosciences, University of Birmingham, Edgbaston, Birmingham, B15 2TT, UK.
Email: rcartwright81@outlook.com

1994). Dietary analysis and history taking are methods that can identify any excessive intake of dietary acids (Addy and Pearce, 1994). Common food acids that can lead to tooth erosion include malic, fumaric, citric and phosphoric acids. These acids usually have pH levels of 3 or below (Imfeld, 1983). This high acidity means that even brief periods of their exposure to dentinal tubules, (typically covered with cementum or smear layer material), would lead to loss of mineral and protective dental plugs (Absi *et al.,* 1989).

Finally, patients undergoing periodontal treatment may develop acute pain; however, they are also susceptible to the development of chronic and more severe hypersensitivity (Porto *et al.,* 2009). This is because sensitive sites frequently exist before periodontal treatment and may be aggravated by surgical interactions with exposed dentine. Patients who develop sensitivity due to treatment, generally improve with time. However, about 10-15% of patients may never achieve a spontaneous remission of symptoms (Pashley, 1990).

Prevalence of hypersensitive teeth

The prevalence of dental conditions that could be described as tooth hypersensitivity have been variably reported within the literature: between 8-30% of adults being affected (Irwin and McCuster, 1997); over 40 million people in the US annually (Kanapka, 1982); and, up to 14.3% of all dental patients (Dowell and Addy, 1983). To provide a comprehensive and comparable international set of data on the experiences of sufferers from hypersensitive teeth, a questionnaire survey was conducted during 1990 in six countries: Australia, France, Germany, US, Japan and Indonesia. About 1000 people were questioned and the overall mean prevalence of sensitive teeth was 14.8% (Murray, 1994). During this study, little difference was reported in prevalence between men and women, however, this was in contradiction to other reports which stated that proportionally more females were affected (Addy and Pearce, 1994).

Dentinal hypersensitivity is frequently encountered in individuals in their 20s to 40s (Graf and Galasse, 1977). Sensitivity is most likely to occur in younger individuals who experienced rapid exposure of dentinal surfaces and tubules. Older individuals generally show more exposed dentine and here it is often less sensitive than in the younger age group (Verzak *et al.,* 1998). This is due to dentinal tubules becoming filled with mineral deposits through dentinal sclerosis, which reduces the permeability and fluid movement through the tubules (Johnson *et al.,* 1973). Furthermore, there is a decrease in the number of viable sensory nerve fibres within the pulp chamber (Trowbridge, 1986). Finally, epidemiological studies have identified the sites of the tooth most prone to hypersensitivity. These include the exposed facial surfaces of the cervical root for canines and premolars (Flynn *et al.,* 1985; Graf and Galasse, 1977).

Measurement of dentinal hypersensitivity

The pain response associated with dentinal hypersensitivity may be elicited by stimuli that are thermal, tactile or osmotic in nature. These types of stimulation have been related to hydrodynamic fluid motion through the dentinal tubules that is detected by nerve cells within the pulp (Brännström, 1963). Electrical stimulation may also elicit a pain response, by the transmission of electrical energy through dentine in the form of either current or potential that also stimulates the nerve cells. Thus, these stimuli are the basis of various methods used to measure dentinal hypersensitivity within subjects (Gunjikar, 2012; Madhavan *et al.,* 2012).

The methods of assessing pain are based upon either a graded stimulus that elicits a pain response or a fixed level of stimulation that produces a pain response that can be graded. Many of the methods are semi-quantitative and stepped indices (such as 0, 1, 2 and 3) are used to indicate the different levels of pain intensity elicited when a stimulus is applied to the subject. Other methods that involve physical or chemical instruments usually use a continuous scale (Kleinberg, 1994).

The simplest method to elicit pain, based upon a thermal stimuli, is to blow room temperature air (approximately 20°C to 24°C) from a dental syringe for 1 second, briefly over the root surface of a sensitive tooth (approximately 32°C to 34°C). The air movement removes heat from the tooth, lowering its temperature and causes evaporation of fluid from open dentinal tubules, ultimately resulting in fluid motion that elicits pain (Brännström, 1960). Methods to elicit dental pain, involving those based upon tactile stimuli, are also straightforward. The simplest tactile device for testing hypersensitivity is a sharp dental explorer. The stimulus is passed along the cementum-enamel junction and the response of the subject is graded on a severity scale (McFall and Morgan, 1985). To produce pain with an osmotic stimulus, concentrated sugar or salt solutions are usually applied to the tooth at room temperature with a cotton applicator for a fixed period of time (McFall and Hamrick, 1987). The duration of application is controlled by rinsing or flushing with warm water to dilute and stop the stimulus and the response of the patient is graded on a severity scale.

Various devices have been used to stimulate teeth electrically. Most have been used to determine whether teeth are vital or not. However, problems with this method arise due to variation in tooth resistance. Different teeth have different thicknesses of dentine and enamel (Stark *et al.,* 1977). Due to the variation in electrical resistance, electrical current is considered the more suitable mode of electrical stimulation. As the electrical current is increased, a point is reached where the nerve fibres are excited at the threshold level of excitation (Kleinberg *et al.,* 1990).

Finally, when developing methods to assess pain an appreciation is needed of the morphological features and functions of the dentine-pulp complex, and their relationship to the proposed mechanisms for dentinal hypersensitivity (Kleinberg, 1994).

Mechanisms of dentinal hypersensitivity

Several theories have been developed to explain the mechanisms involved in dentinal hypersensitivity and all involve an understanding of the structure of the dentine and pulp (Bamise and Esan, 2011; Blaggana *et al.,* 2011). Firstly, it has been suggested that odontoblasts can act as sensory receptors. This model proposes that

sensitivity may be initiated by the direct excitation of the odontoblasts. The odontoblast is of a neural crest origin and has a high resting membrane potential. However, direct odontoblastic synaptic connections have never been established. Thus, in recent years, this theory has been given little support (Tsukada, 1987).

A second model suggests that odontoblast processes may influence the excitability of the pulpal nerves. Dentinal tubules contain odontoblast processes and in some cases, nerve axons. While no direct synaptic link has been established between these components, they are within close proximity of each other. Stimuli are able to cause fluid flow in the dentinal tubules where the odontoblast process will be one of the components of the dentinal tubules that may move during fluid flow (Avery *et al.,* 1980). This stimulation of the odontoblast process may result in changes in the ionic environment of the closely adjacent nerves. It has been proposed that fluid flow past the odontoblast may induce a shear stress, which if too great, might cause the release of some odontoblast intracellular K^+ which could alter the resting membrane potential of nearby nerves (Holland, 1994). Alternatively, hypersensitivity may involve pulpal inflammatory responses within the dentine-pulp complex, as a result of exposed dentine being penetrated by noxious stimuli and bacterial toxins. Dynamic and adaptive changes within these pulpal nerve receptor mechanisms may lead to persistent activation of neurones resulting in chronic sensitivity (Olgart and Keperzoudis, 1994).

Finally, the hydrodynamic theory, developed in the 1960s, is the most popular explanation of the mechanism responsible for coupling painful stimuli through fluid movement across dentine, to the stimulation of pulpal nerves and hence the initiation of dentinal hypersensitivity (Brännström, 1966; Walters, 2005). Pain is linked to the rate of fluid flow through the dentinal tubules. Stimuli which increase the rate of fluid flow across the dentine are proposed to increase nerve excitability leading to an increase in dentine sensitivity. The hydraulic conductance of a tissue, expresses the ease with which fluid can flow in unit of time across a unit surface area when under unit pressure. Hydraulic conductance is dependent upon a number of variables such as the patency, population and degree of tubular occlusion of the dentine (Brännström, 1963, 1965; Ishikawa, 1969). Various experiments have shown that tubules of hypersensitive teeth are typically characterised as being open and large, with tubules in some sensitive sites being twice as wide as tubules in non-sensitive sites (Absi *et al.,* 1987; Yoshiyama *et al.,* 1989). Some of these tubules, patent at the surface of the dentine, will also be patent all the way to the pulp. However, dye penetration studies have shown that some tubules are patent only part way through the tissue (Absi *et al.,* 1987) when the occlusion of these tubules may reduce or eliminate tooth sensitivity (Cuenin *et al.,* 1991).

Current methods of treating dentine hypersensitivity

Since the permeability and fluid movement in open, exposed, dentinal tubules, has provided a favoured theory for stimuli transmission through dentine, then the occlusion of those dentinal tubules has been identified as a potential method for reducing pain associated with sensitive teeth (Kim *et al.,* 2013). The sealing of exposed dentinal tubules with topical agents, varnishes and bonding resins has been suggested to create longer-lasting relief from dentine hypersensitivity (Holland, 1994). These treatments may be considered non-invasive, as in the cases of the topical agents and dentifrices, which contain the desensitising active ingredients. Alternatively, treatment may be invasive, as with the application of resins. However, according to the literature, the most widely available desensitising agent is the toothpaste ingredient, potassium nitrate (Walters, 2005).

The first attempts to treat dentine hypersensitivity, involved the use of caustic agents. Various substances were used in an attempt to precipitate proteins (to occlude the dentinal tubules) including, silver nitrate, zinc chloride, phenol, formaldehyde, concentrated alcohol and strong acids (Seltzer *et al.,* 1977). However, these substances are liable to irritate the pulp and may have a short-term efficacy, so they are not ideal desensitising agents. In contrast, the topical application of fluorides appears to be effective in treating hypersensitivity over periods from several days to several weeks (Thrash *et al.,* 1992). Fluoride applications are thought to create a barrier by precipitating calcium fluoride at the tooth surface. However these precipitates are relatively soluble in saliva which may account for the transient action of this chemical barrier (Porto *et al.,* 2009). The daily application of fluoride in a glycine vehicle has achieved popularity (Brännström and Nyborg, 1971). However, concentrated glycine may cause pain due to its high osmolarity causing water to be withdrawn from the tubules, leading to hypersensitivity in its own right. Finally, fluoride containing varnishes have been recommended for the treatment of sensitive teeth, but their action is more transient, usually lasting only several hours (Hansen, 1992).

If a patient is suffering from dentine hypersensitivity, due to severe abrasion or erosion, then restorative materials may be used to rebuild the functional and anatomical form of the tooth (Camilotti *et al.,* 2012). Restorative treatment of an eroded area can be accomplished with a variety of third or fourth generation dentine bonding agents and restorative materials. These bonding agents include Gluma, Scotchbond II, All-bond and C&B Meta Bond or glass-ionomer cement (Gangarosa, 1994). Restorative materials, such as light cured glass-ionomers are easy to handle and have been reported to be useful for the treatment of dentine hypersensitivity (Hinoura *et al.,* 1991). Alternatively, resins may be employed and good desensitising effects have been obtained with the application of cyanocrylate (Javid *et al.,* 1987). However, the use of restorative materials to treat dentine sensitivity is considered technique sensitive, expensive and complex in comparison to other techniques but offers the hope of longer lasting and more predictable results than topical agents (Hansen, 1992).

Furthermore, the development of a successful barrier to block open dentinal tubules has been reported, with the use of oxalates (Cunha-Cruz *et al.,* 2011). Examples of commercial oxalate systems include potassium oxalate (Protect, Butler Inc.) and ferric oxalate (Sensodyne, Block Drug). Experimental oxalate systems, whereby precipitates

of calcium oxalates have been formed, have also been reported (Vachiramon *et al.,* 2008). Although oxalates appear to be a successful treatment for blocking tubules, eventually saliva dissolves the surface precipitate that forms the barrier; thus frequent re-application is required. Alternatively, a number of studies have investigated the use of potassium nitrate as an effective ingredient in treating dentinal hypersensitivity; however it does not appear to work by tubule occlusion. (Hodosh, 1974). Instead, it is proposed that the potassium ions in the solution work by blocking the synaptic connections between nerve cells, reducing nerve excitation and associated pain (Markowitz and Kim, 1990). Potassium nitrate delivered in toothpaste is a widely available desensitising agent (Silverman *et al.,* 1996).

Potential future methods for treating dentine hypersensitivity

Due to the transient nature of current non-invasive techniques and greater financial costs associated with more permanent methods of treatment, potential future therapies for sensitive teeth should ideally be non-invasive and be able to provide long term pain relief, in a cost effective manner. One such method of treatment for dentinal hypersensitivity could be based upon iontophoresis (Gangarosa, 1994). Iontophoresis is the process of introducing ionic drugs to a body surface for therapeutic purposes. The technique allows high concentrations of drugs to be placed accurately, where they are needed, rather than depending upon diffusion or systemic administration. Iontophoresis requires that a charged drug be delivered, using an electrode with an appropriate means of application, at or near the surface of condition or disease (Gangarosa and Jeske, 1992). The use of iontophoresis with sodium fluoride based solutions has been proposed as a method of treating dentinal hypersensitivity and early studies by Gangarosa and Park (1978), initially demonstrated highly consistent desensitisation. The exact mechanism by which sodium fluoride based iontophoresis produces desensitisation of dentine is not known, but several hypotheses have been proposed. An initial hypothesis proposed that iontophoretic desensitisation is achieved by the applied electrical current altering sensory nerve conduction (Gangarosa *et al.,* 1977). Alternatively, fluoride ions introduced into dentinal tubules, may reduce dentine permeability (Pashley *et al.,* 1978). Finally, rapid formation of tertiary and/or peritubular dentine following the application of a current (with or without fluoride) to dentine may also reduce dentine permeability (Lefkowitz and Brown, 1966). However, it is conceivable that a combined mechanism of action by the iontophoresis based treatment, on both nerves and on the permeability of the dentinal tubules, may be occurring. Alternatively, another potential future therapy for dentine hypersensitivity may be based on the use of lasers. Studies by Ladalardo *et al.* (2004) compared the effectiveness of two types of lasers in individuals with sensitive teeth and their pain was evaluated before and after treatment. A 660nm red laser was reported to have greater desensitising effectiveness than the 830nm infrared laser. The types of tissue reaction responses occurring depended upon the active medium, wavelength and power density of the laser and the optical properties of the target tissue. In one such study, Villa *et al.,* (1988) observed the profiling of odontoblasts within irradiated teeth and noted a large quantity of tertiary dentine formation. It was proposed that in this manner, laser irradiation contributed to the repair of the dentine by an as yet, unknown mechanism.

In other studies, the use of carbon dioxide based lasers (with a wavelength of 10,600nm) has been employed with varying desensitising effectiveness. It has been postulated that this laser's mechanism of action, is to occlude dentinal tubules (Moritz *et al.,* 1995). This is in contrast to the mechanism of action of low power Gallium Aluminium-Asenium (GaAlAs) lasers (with a wavelength of wavelength 900nm). The use of this type of laser has also had some desensitising success. However, it is postulated that this laser type, mediates an analgesic effect that is related to the depression of nerve transmission by blocking the depolarisation of afferent nerves (Wakabayashi *et al.,* 1992). There have also been reports of successful, combinatorial use of laser irradiation together with chemical agents such as sodium fluoride. The combined use of these agents, together with GaAlAs based lasers enhanced treatment effectiveness by more than 20% above that of laser treatment alone (Kimura *et al.*, 2000).

Finally, a mechanism of action, common to both Iontophoresis and laser based treatments has been postulated to involve the up-regulation of either tertiary and/or peritubular dentinogenesis. Both types of dentinogenesis will affect the occlusion of dentinal tubules and will have consequences for their permeability and the transmission of stimuli through the tissue during episodes of dentine hypersensitivity (Baume, 1980). It is hypothesised that these dentinogenic responses may both involve a complex series of mineralisation events. Therefore, the induction of mineralisation events, by dental cells through their response to signalling molecules sequestered within the dentine matrix could also form the basis of a further therapy for dentine hypersensitivity (Holland, 1994; Mjor, 1985; Tziafas *et al.*, 2000).

Conclusion

Dentinal hypersensitivity is a common and significant dental problem with the symptoms, prevalence, measurement and oral factors that contribute to dentinal hypersensitivity having been well characterised. Several theories have been developed to explain the mechanisms involved in dentinal hypersensitivity with respect to the structure of the dentine and pulp. This has lead to the development of a variety of treatments that may be transient or permanent, non-invasive or invasive in nature. However a variety of future treatments for dentine hypersensitivity are currently under development that could combine the benefits of being both non-invasive and permanent yet cost effective for both oral health care professionals and their patients.

Acknowledgement

This review was supported by a grant from the Biotechnological and Biological Sciences Research Council.

References

Absi, E., Adams, D. and Addy, M. (1987): Dentine hypersensitivity - a study of the patency of dentinal tubules in sensitive and nonsensitive cervical dentine. *Journal of Clinical Periodontology* **14**, 280-284.

Absi, E., Adams, D. and Addy, M. (1989): Dentine hypersensitivity - the development and evaluation of a replica technique to study sensitive and non-sensitive cervical dentine. *Journal of Clinical Periodontology* **16**, 190-195.

Addy, M. (2005): Tooth brushing, tooth wear and dentine hypersensitivity - are they associated? *International Journal of Dentistry* **55**(Supp.1), 261-267.

Addy, M., Newcombe, R.P. and Mostafa, D. (1987): Dentine hypersensitivity: The distribution of recession, sensitivity and plaque. *Journal of Dentistry* **15**, 242-248.

Addy, M. and Pearce, N. (1994): Aetiological, predisposing and environmental factors in dentine hypersensitivity. *Archives of Oral Biology* **39**, 33-38.

Avery, J.K., Cox, C.F. and Chiergo, D.J. (1980): Presence and location of adrenergic nerve endings in the dental pulps of mouse molars. *Journal of Anatomical Records* **198**, 59-71.

Bamise, C.T. and Esan, T.A. (2011): Mechanisms and treatment approaches of dentine hypersensitivity: a literature review. *Oral Health & Preventative Dentistry* **9**, 353-367.

Baume, L. (1980): The biology of the pulp and dentine. *Monographs of Oral Science* **4**, 457-463.

Blaggana, A. Vohra, P. and Nagpal, A. (2011): Diagnosis and treatment of dentinal hypersensitivity. *Journal of Innovative Dentistry* **1**, 19-26.

Brännström, M. (1960): Dentinal and pulpal response III. Application of an air stream to exposed dentine. *Acta Odontologica of Scandinavia* **18**, 17-28.

Brännström, M. (1963): A hydrodynamic mechanism in the transmission of pain producing stimuli through dentine. *Sensory Mechanics in Dentistry* **71**, 73-79.

Brännström, M. (1965): The surface of sensitive dentine. *Odontics Review* **16**, 293-299.

Brännström, M. (1966): Sensitivity of dentine. *Oral Surgery* **21**, 517-529.

Brännström, M. and Nyborg, H. (1971): Pulp reaction to fluoride solution applied to deep cavities. An experimental histological study. *Journal of Dental Research* **50**, 1548-1552.

Brissada, N. (1994): Symptomatology and clinical features of hypersensitive teeth. *Archives of Oral Biology* **39**(Supp.), 31S-32S.

Camilotti, V. Zilly, J., Busato, P.do M.R., Nassar, C.A. and Nassar, P.O. (2012): Desensitizing treatments for dentin hypersensitivity: a randomized, split-mouth clinical trial. *Brazilian Oral Research* 26, 15-21.

Chu, C.H., Lui, K.S., Lau, K.P., Kwok, C.M. and Huang, T. (2010): Effects of 8% arginine desensitizing paste on teeth with hypersensitivity. *Journal of Dental Research* **89**(A): 21-28.

Cuenin, M., Scheidt, M., O'Neal, R., Strong, S., Pashley, D., Horner, J. and Van Dyke, T. (1991): An in vitro study of dentin sensitivity: The relation of dentin sensitivity and the patency of dentinal tubules. *Journal of Periodontology* **62**, 668-673.

Cunha-Cruz, J., Stout, J.R., Heaton, L.J. and Wataha, J.C. (2011): Dentin hypersensitivity and oxalates: a systematic review. *Journal of Dental Research* **90**, 304-310.

Dowell, P. and Addy, M. (1983): Dentine hypersensitivity - a review. *Journal of Clinical Periodontology* **10**, 341-363.

Flynn, J., Galloway, R. and Orchardson, R. (1985): The incidence of hypersensitive teeth in the west of Scotland. *Journal of Dentistry* **13**, 230-236.

Gangarosa, L. (1994): Current strategies for dentist-applied treatment in the management of hypersensitive dentine. *Archives of Oral Biology* **39**, 101S-106S.

Gangarosa, L. and Jeske. A. (1992): Iontophoresis: a system for solving some difficult dental problems. In: Hardin, J., Ed., *Clarke's Clinical Dentistry* Chap. 31B: 1-32. Philadelphia, US: J.B. Lippincott Co.

Gangarosa, L. and Park, N. (1978): Practical considerations in iontophoresis of flouride for desensitizing dentin. *Journal of Prosthetic Dentistry* **39**, 175-178.

Gangarosa, L. Park, N. and King, G., (1977): Iontophoresis of lidocaine into frog sciatic nerve fibres. *Life Science* **21**, 885-890.

Graf, H. and Galasse, R. (1977): Morbidity, prevalence and intraoral distribution of hypersensitive teeth. *Journal of Dental Research* **53**(Special issue A), 162.

Gunjikar, T. (2012): Methods for the evaluation of pain associated with dentinal hypersensitivity. *Journal of the Indian Dental Association* **6**, 57-59.

Hansen, E. (1992): Dentin hypersensitivity treated with a fluoride containing varnish or a light cured glass ionomer liner. *Scandinavian Journal of Dental Research* **100**, 305-309.

Hinoura, K., Miyazaki, M. and Onose, H. (1991): Dentin bond strength of light-cured glass-ionomer cements. *Journal of Dental Research* **70**, 1542-1544.

Hodosh, M. (1974): A superior desensitizer - potassium nitrate. *Journal of the American Dental Association* **88**, 831-832.

Holland, G.R. (1994): Morphological features of dentine and pulp related to dentine sensitivity. *Archives of Oral Biology* **39**(Supp.): 3S-11S.

Imfeld, T. (1983): Identification of low caries risk dietary components. *Monographs of Oral Science* **3**: 71-75.

Irwin, C. and McCusker. P. (1997): Prevalence of dentine hypersensitivity in a dental population. *Journal of the Irish Dental Association* **43**, 7-9.

Ishikawa, S. (1969): A clinical histological study on the hypersensitivity of dentine. *Journal of the Japanese Stomatological Society* **63**: 68-88.

Javid, B., Barkhordar, R. and Bhinda, S. (1987): Cyanoacrylate--a new treatment for hypersensitive dentin and cementum. *Journal of the American Dental Association* **114**, 486-488.

Johnson, G., Olgart, L. and Brännström, M. (1973): Outward fluid flow in dentin under a physiologic pressure gradient: experiments in vitro. *Oral Pathology* **35**, 238-248.

Kanapka, J. (1982): Current treatment for dentinal hypersensitivity. A new agent. *Compendium of Continuing Education in Dentistry* **3**(Supp.3): 188-220.

Kim, S., Kim, E., Kim, D. and Lee, I. (2013): the evaluation of dentinal tubule occlusion by desensitizing agents: a real-time measurement of dentinal fluid flow rate and scanning electron microscopy. *Operative Dentistry* **38**, 419-428.

Kimura, Y.P., Wilder-Smith, P. and Matsumoto,K. (2000): Lasers in endodontics: a review. *International Endodontics Journal* **33**, 173-185.

Kleinberg, I. (1994): Measurement of tooth hypersensitivity and oral factors involved in its development. *Archives of Oral Biology* **39**(Supp.), 63S-71S.

Kleinberg, I., Kaufman, H. and Confessore, F. (1990): Methods of measuring tooth hypersensitivity. *Dental Clinics of North America* **34**, 515-529.

Ladalardo, T. Pinheiro, A. and Campos, R. (2004): Laser therapy in the treatment of dentine hypersensitivity. *Brazilian Dental Journal* **15**, 144-150.

Larsen, M. (1975): Degrees of saturation with respect to apatites in fruit juices and acidic drinks. *Scandinavian Journal of Dental Research* **83**, 13-7.

Lefkowitz, W. and Brown, W.L. (1966): Influences of dentinal fluids on experimental caries. *Journal of Dental Research* **45**, 1493-1498.

Madhavan, S., Nayak, M., Shenoy, A., Shetty, R. and Prasad, K. (2012): Dentinal hypersensitivity: A comparative clinical evaluation of CPP-ACP F, sodium fluoride, propolis, and placebo. *Journal of Conservative Dentistry* **15**, 315-318.

Markowitz, K. and Kim, S. (1990): Hypersensitive teeth. Experimental studies of dentinal desensitizing agents. *Dental Clinics of North America* **34**, 491-501.

McFall, W. and Hamrick, S. (1987): Clinical effectiveness of a dentifrice containing fluoride and a citrate buffer system for treatment of dentinal sensitivity. *Journal of Periodontology* **58**, 701-705.

McFall, W. and Morgan, W. (1985): Effectiveness of a dentifrice containing formalin and sodium monofluorophosphate on dental hypersensitivity. *Journal of Periodontology* **56**, 288-292.

Mjor, I. (1985): Dentin-predentin complex and its permeability- physiologic overview. *Journal of Dental Research* **64**, 621-627.

Montori, V., Wilczynski, N. Morgan, D. and Haynes, R. (2005): Optimal search strategies for retrieving systematic reviews from Medline: analytical survey. *British Medical Journal* **8**, 68.

Moritz, A., Gutknecht, N. and Schoop, U. (1995): Effects of CO_2 laser irradiation on treatment of hypersensitive dentin: results of an in vitro study. *Journal of Clinical and Laser Medical Surgery* **13**, 397-400.

Murray, L. (1994): The prevalence of self-reported hypersensitive teeth. *Archives of Oral Biology* **39**(Supp.), 129S.

Narhi, M., Yamamoto, H., Ngassapa, H. and Hirvonen, T. (1994): The neurophysiological basis and the role of inflammatory reactions in dentine hypersensitivity. *Archives of Oral Biology* **39**(Supplemental), 23S-30S.

Olgart, L. and Keperzoudis, N. (1994): Nerve-pulp interactions. *Archives of Oral Biology* **39**, 47S-54S.

Orchardson, R. and Collins, W. (1987): Clinical features of hypersensitive teeth. *Brazilian Dental Journal* **162**, 253-256.

Pashley, D.H. (1990): Dentine permeability: theory and practice. In: Spangberg, L.S.W. (Ed.), *Experimental Endodontics* pp19-49. Boca Raton, FL: CRC Press.

Pashley, D.H., Livingston, M.J., Reeder, O.W. and Horner, J. (1978): Effects of the degree of tubule occlusion on the permeability of human dentine *in vitro*. *Archives of Oral Biology* **23**, 1127-1133.

Pol, S., Chute, M., Gunjikar, A., Jonnala, J. and Pol, D. (2011): Effects of in vitro acids and dietary acids on dentine hypersensitivity. *Journal of the Indonesian Dental Association* **5**, 1168-1170.

Porto, I., Andrade, A. and Montes, M. (2009): Diagnosis and treatment of dentinal hypersensitivity. *Journal of Oral Science* **51**, 323-332.

Seltzer, S., Rainey, E. and Gluskin, A. (1977): Correlation of scanning electron microscope and light microscope findings in uninflamed and pathologically involved human pulps. *Oral Surgery, Oral Medicine and Oral Pathology* **43**, 910-928.

Silverman, G., Berman, E. and Hanna, C. (1996): Assessing the efficacy of three dentrifices in the treatment of dentinal hypersensitivity. *Journal of the American Dental Association* **127**, 191-201.

Stark, M. Kempler, D. and Pelzner, R. (1977): Rationalization of electric pulp-testing methods. *Oral Surgery, Oral Medicine and Oral Pathology* **43**, 598-606.

Thrash, J., Jones, D. and Dodds, W. (1992): Effect of a fluoride solution on dentinal hypersensitivity. *American Journal of Dentistry* **5**, 299-302.

Touyz, L. (1994): The acidity (pH) and buffering capacity of Canadian fruit juice and dental implications. *Journal of the Canadian Dental Association* **60**, 454-458.

Touyz, L. and Glassman, R. (1981): Dentinal Hypersensitivity – a review. *Journal of the Dental Association of South Africa* **36**, 195-201.

Trowbridge, H. (1986): Review of dental pain - Histology and physiology. *Journal of Endodontics* **12**, 445-452.

Tsukada, K. (1987): Ultrastructure of the relationship between odontoblast processes and nerve fibres in dentinal tubules of rat molar teeth. *Archives of Oral Biology* **32**, 87-92.

Tziafas, D. Smith, A. J. and Lesot, H. (2000): Designing new treatment strategies in vital pulp therapy. *Journal of Dentistry* **28**, 77-92.

Vachiramon, V., Vargas, M., Pashley, D., Tay, F. Geraldeli, S., Qian, F. and Armstrong, S. (2008): Effects of oxalate on dentin bond after 3-month simulated pulpal pressure. *Journal of Dentistry* **36**, 178-185.

Verzak, Z., Buković Jr., D. and Bagić, I. (1998): Prevalence and intraoral distribution of dentin hypersensitivity among students. *Collegium Antropologicum* 22, 259-265.

Villa, R., Brugnera, A. and Aun, C. (1988): Dentine Hypersensitivity and Lasers. *European Journal of Dentistry* **1**, 101-105.

Wakabayashi, H., Tachibana, H. and Matsumoto, K. (1992): A clinical study on the hypersensitive dentine by 20 and 40 mW GaAlAs semiconductor laser. *Journal of Showa University Dental Society* **12**, 343-347.

Wallace, J. and Brissada, N. (1990): Pulpal and root sensitivity rated to periodontal therapy. *Oral Surgery, Oral Medicine and Oral Pathology* **69**, 743-747.

Walters, A. (2005): Dentinal Hypersensitivity: A review. *Journal of Contemporary Dental Practice* **6**, 107-117.

West, N., Addy, M. and Hughes, J. (1998): Dentine hypersensitivity: the effects of brushing desensitizing toothpastes, their solid and liquid phases, and detergents on dentine and acrylic: studies in vitro. *Journal of Oral Rehabilitation* **25**: 885-895.

Yoshiyama, M., Masada, J. and Uchida, A. (1989): Scanning electron microscopic characterization of sensitive and insensitive human radicular dentin. *Journal of Dental Research* **68**, 498-502.