

Dentine hypersensitivity: new perspectives on an old problem

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Dentine hypersensitivity is a prevalent, painful condition of the teeth. Despite the fact that the accuracy of the terminology is questionable and other terms have been proposed, 'dentine hypersensitivity' has been in long-term use and is, therefore, the preferred term. In dentine hypersensitivity, lesions exhibit patent tubules at the exposed dentine surface and appropriate stimuli trigger pulpal nerves via a hydrodynamic mechanoreceptor mechanism to produce a typically short, sharp, painful response. This accepted definition of the condition indicates the need to consider a differential diagnosis. This review will consider evidence that dentine hypersensitivity is a tooth-wear phenomenon characterised predominantly by erosion, which both exposes dentine and, more importantly, initiates the lesions. Abrasion caused by brushing teeth with toothpaste appears to be a secondary aggravating factor, which may even act in synergy with erosion. Gingival recession probably accounts for most dentine exposure at the gingival margin, but the process is poorly understood. Management strategies, which take into account aetiological and predisposing factors, rather than treatment alone, should be considered. There is little clinical research on many aspects of the aetiology and particularly on the management of the condition.

Key words: Dentine hypersensitivity, mechanisms, erosion, abrasion, prevalence

In 1982, dentine hypersensitivity was described as an enigma, because it was frequently encountered yet poorly understood¹. Some 20 years later, it is worthwhile reconsidering the statement, as the title of a recent article seems to suggest². This review will discuss the terminology, definition, epidemiology, mechanisms, anatomy, aetiology and management of the condition. It is hoped that it will demonstrate that, although dentine hypersensitivity is not the enigma it once was, there is still much to be discovered about the condition, its prevention and its management.

Terminology

Dentine hypersensitivity is a relatively common, painful dental condition. Typically, the pain is short and sharp and occurs in response to certain stimuli applied to exposed dentine. At a macroscopic level, dentine exhibiting hypersensitivity appears no different from non-sensitive dentine. The histopathological state of the pulp in teeth exhibiting dentine hypersensitivity has not been clearly established either, despite extensive early work on the correlation of pulpal histopathology to clinical signs and symptoms associated with other dental disease, notably caries and its sequelae³. Taking all these factors into consideration, the term dentine hypersensitivity would appear to be at the least inaccurate and, at the most, totally inappropriate to describe the condition. Dentine

sensitivity would seem to be a more correct term⁴⁻⁶, accepting, of course, that dentine, in itself, cannot be sensitive, but that the stimuli, when applied to the dentine surface, evoke a response from the pulp nerves. Other terms to describe dentine hypersensitivity have been originated by substituting the word dentinal, adding site descriptors, such as cervical or root, and combining these with either hypersensitivity or sensitivity. This practice has resulted in a significant number of permutations to describe the apparently same condition.

The literature also refers to cemental hypersensitivity or sensitivity; however, evidence indicates that exposed cervical dentine rapidly loses the cementum layer⁷. Site descriptors are almost certainly inappropriate, as dentine hypersensitivity can occur at any area on a tooth, even though the cervical or root surface is by far the most commonly affected part. Interestingly, root sensitivity has been adopted at the 2002 workshop of the European Federation of Periodontology to describe the sensitivity of teeth, with periodontal disease before and after non-surgical or surgical therapies. The term was adopted because of the uncertainty as to whether sensitivity associated with periodontal disease and its treatment was truly dentine hypersensitivity⁸, particularly since, in periodontal disease, bacteria were shown to invade dentine tubules to depths close to the pulp⁹. Appreciating the fact that the term, dentine hypersensitivity, may be inaccurate and even inappropriate, alternative descriptors would be difficult to introduce. The term has been commonly used and accepted for many decades to describe a specific painful condition of teeth, which is distinct from other types of dentinal pain having differing aetiologies. Perhaps it would be more appropriate, at this juncture, to encourage universal adoption of the terminology and discourage the use of new terminology (*Table 1*).

Table 1 Terms in common usage for dentine hypersensitivity

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|---|
| <ul style="list-style-type: none"> • Dentine Sensitivity • Dentine Hypersensitivity • Dentinal Hypersensitivity • Cervical Hypersensitivity / Sensitivity • Root Hypersensitivity / Sensitivity • Cemental Hypersensitivity / Sensitivity |
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Definition and differential diagnosis

A definition for dentine hypersensitivity was suggested in 1983¹⁰ and, with minor amendment, was adopted by an international workshop on the design and conduct of clinical trials for treatments of the condition¹¹. The definition states: "Dentine hypersensitivity is characterised by short, sharp pain arising from exposed dentine in response to stimuli typically thermal, evaporative, tactile, osmotic or chemical and which cannot be ascribed to any other form of dental defect or pathology". The Canadian Advisory Board on Dentine Hypersensitivity in 2002 suggested that it would be more correct to substitute 'disease' for 'pathology'. The definition provides a clinical descriptor of the condition and identifies dentine hypersensitivity as a distinct clinical entity, thereby encouraging the clinician to consider a differential diagnosis. A number of other conditions are known to present with similar symptoms of 'dentinal pain'. It would not be unreasonable, therefore, to exclude other possible causes of pain before proceeding with any management strategy for dentine hypersensitivity. Other causes of the typically short, sharp, dentinal pain include caries, chipped teeth, fractured restorations, marginal leakage around restorations, some restorative materials, cracked tooth syndrome and palato-gingival grooves¹². Such conditions clearly require treatment options that are usually quite different from those used for dentine hypersensitivity.

Mechanisms of dentine sensitivity (review¹³)

Several hypotheses have been put forward over more than a century to explain the sensitivity of dentine. Circumstantial and direct evidence disproved the theory of 'innervation of dentine' and 'odontoblast transducer' mechanisms¹³. This left the hydrodynamic hypothesis first proposed in 1900¹⁴, and for which significant evidence accrued in the 1950s and 1960s¹⁵, as the most widely accepted theory to date. The hydrodynamic theory postulates that most pain-evoking stimuli increase the outward flow of fluid in the tubules (*Figure 1*). This increased flow, in turn, causes a pressure change across the dentine, which activates A- δ intradental nerves at the pulp-dentine border or within the dentinal tubules. The stimulation is thought to occur via a mechanoreceptor response that distorts the pulp nerves¹⁶, perhaps not unlike the 'touch' response which occurs when gentle pressure is applied to skin hair. In addition, when fluid moves in tubules, an electrical discharge known as streaming potential occurs, which is directly proportional to pressure^{17,18}. Whether this discharge reaches levels sufficient to stimulate nerves has not been established, although it is theoretically possible^{19,20}. In dentine hypersensitivity, the definition highlights different stimuli inducing pain. Of these, cold or evaporative stimuli are usually identified as the most problematic for sufferers²¹. Heat is not commonly reported perhaps because it is the exception to stimuli evoking pain, causing relatively slow inward movement of dentinal



Figure 1. Diagrammatic representation of hydrodynamic theory showing fluid moving away from the pulp in response to a cold stimulus

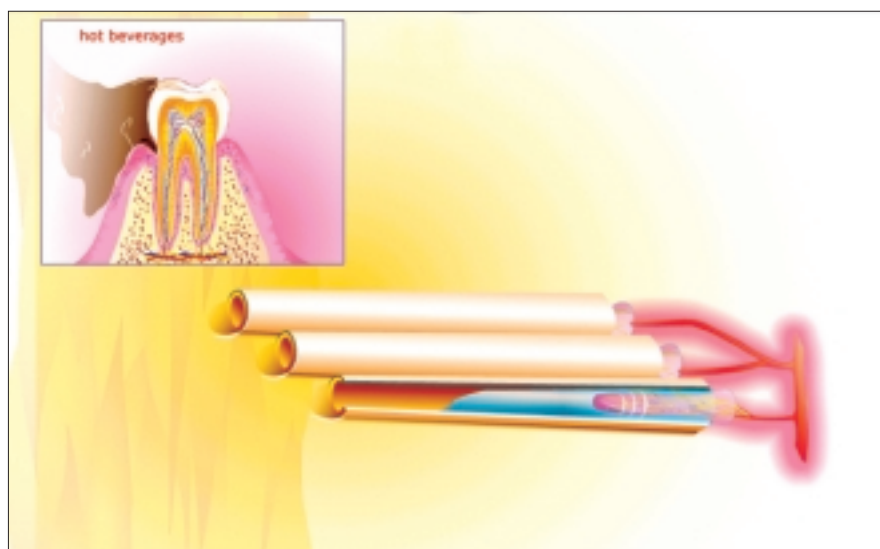


Figure 2. Diagrammatic representation of hydrodynamic theory showing fluid moving towards the pulp in response to a hot stimulus

Table 2 Differences in the number and diameter of tubules between hypersensitive and non-sensitive dentine (after Absi^{24,25})

Tubules	Hypersensitive dentine	Non-sensitive dentine
Number – ratio	8	1
Diameter (microns)	0.83	0.4

fluid²² (Figure 2).

If the hydrodynamic theory for dentine sensitivity is to be accepted as the mechanism involved in dentine hypersensitivity, then lesions must have dentinal tubules open at the dentine surface and patent to

the pulp. Scanning electron microscopic and dye penetration studies provided such evidence, demonstrating the presence of a greater number (8 times) and wider tubules (2 times diameter) on 'hypersensitive' dentine compared

to 'non-sensitive' dentine^{23–25} (Table 2). Figure 3 provides an illustration of dye penetrating a cervical lesion in an extracted tooth with a history of hypersensitivity, and a subsequent SEM of the lesion demonstrating large numbers of open tubules. Standard texts on dentinal tubules indicate that tubule numbers and diameters increase from the outer dentine towards the pulp. This raises the possibility that fluid flow, and therefore hypersensitivity, may increase as dentine is lost through tooth wear processes – assuming such wear does not induce reparative processes in dentine. The difference in tubule diameter may be the more important variable since fluid flow is proportional to the fourth power of the radius (i.e., doubling the tubule diameter results in a 16-fold increase in fluid flow). This information has important implications for treatment strategies. Although, by definition, dentine hypersensitivity is characterised by short, sharp pain, some sufferers report rapid, severe pain on initial stimulation followed by a persistent dull ache. Evidence suggests that, in these uncommon cases, there is pulp inflammation, which anecdotally indicates the need for a different treatment strategy – namely, endodontics or exodontia.

Prevalence (reviews^{2,8})

Whether classical epidemiological surveys have been used to determine the prevalence and incidence of dentine hypersensitivity is subject to question. Published studies show extreme variations and prevalence figures from cross-sectional surveys range from 3–57 per cent^{6,27–34}. Studies from periodontal patients suggest figures in the order of 72–98 per cent^{35,36}; however, as discussed above, such sensitivity may have a different aetiology, does not fit with the definition of dentine hypersensitivity and, for the present time, is better termed root sensitivity. A number of studies where patients were examined and

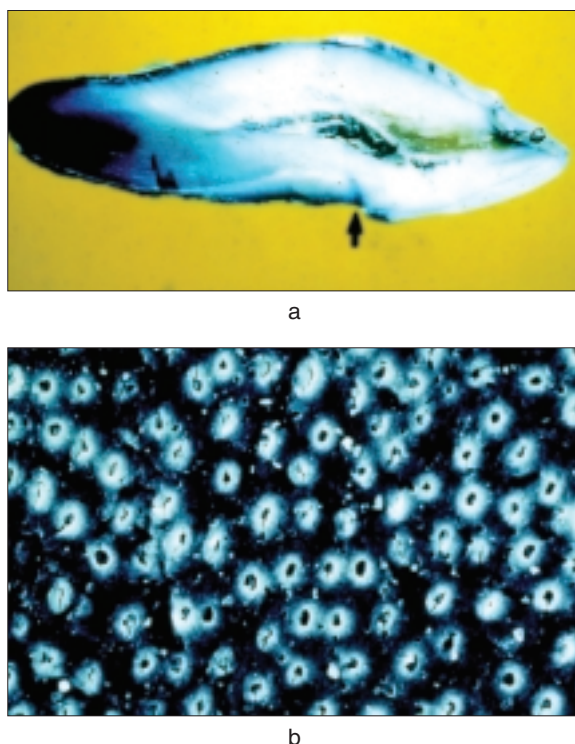


Figure 3. a) Lateral view of hypersensitive tooth longitudinally sectioned through cervical abrasion cavity. Methylene blue dye penetration can be seen (arrow). b) SEM of the same abrasion cavity showing large numbers of open tubules (magnification x1000) (Courtesy of Absi *et al.*²⁴)

Table 3 Frequency of dentate adults who responded positively to having or ever having sensitive teeth (pain or discomfort caused by cold, hot, sweet, sour, foods/drinks or toothbrushing)

Region/Country	% sensitive teeth sufferers (current or previous)		
	Male	Female	Total
North America	31	42	37
Europe	39	50	45
Others	50	54	52
Overall estimate	35	41	36

Table 4 Frequency of respondents who have ever consulted a dentist due to sensitive teeth

Country/Region	% ever consulted a dentist
North America	60
Europe	47
Others	34
Overall	48

evaluated for dentine hypersensitivity consistently report prevalence figures of around 15 per cent^{6,27,28}; an apparently similarly conducted study recently reported much lower figures³⁴.

Of greater interest, perhaps, are the data from such studies that

relate to the demographics of sufferers and the intra-oral distribution of the condition. Although the age range is extremely wide, the majority of sufferers are from 20–50 years old, with a peak between 30–40 years. Females tend to be affected more often and at a

younger age than males. A global survey of 11,000 adults conducted by the Research Quorum, Basingstoke, Hampshire, UK, in 2002 (unpublished) produced interesting findings about sensitive teeth based on patient perception. In the countries surveyed, prevalence figures differed from 37–52 per cent, suggesting that data included sensitivity for reasons other than just dentine hypersensitivity; again, however, females invariably showed a higher prevalence (*Table 3*). Only about half of those reporting sensitivity complained to the dentist (*Table 4*), and only half of these had treatment recommended. Interestingly, whereas dentists perceived a prevalence of dentine hypersensitivity in their practices similar to the 15 per cent figure found in studies, hygienists perceived the condition to be present in almost twice as many patients as reported by the Canadian Advisory Board on Dentine Hypersensitivity, 2002.

The majority of studies report a tooth site predilection order of canines and first premolars, followed by incisors and second premolars and, finally, molars, with the vast majority of sites being buccal cervical. Some studies show a similar predilection distribution for gingival recession, and both conditions have been shown to be more common on the left than on the right sides of the arches and to have an inverse relationship with plaque scores³⁷. Taken together, these findings suggest that tooth brushing is associated with dentine hypersensitivity. Thus, the oral hygiene of females is known to be better than males from an early age³⁸, and right-handed tooth brushers brush the left buccal surfaces more effectively than surfaces on the right side^{38,39}; interestingly, the converse does not appear to hold for left-handed tooth brushers³⁹. Unfortunately to date, there is a lack of randomised, controlled clinical trial data to demonstrate a causal relationship

between tooth brushing and gingival recession or dentine hypersensitivity.

Aetiology

Two processes need to occur for dentine hypersensitivity to arise: dentine has to become exposed (lesion localisation), and the dentine tubule system has to be opened and be patent to the pulp (lesion initiation). Lesion localisation and lesion initiation require both differing and similar aetiological agents in order to occur.

Lesion localisation (reviews^{2,8,40-44})

Exposure of dentine may occur by loss of either enamel or periodontal tissues, the latter of which is often termed gingival recession. Loss of enamel is generally considered under the heading of tooth wear, which encompasses attrition, abrasion and erosion. None of these physical and chemical processes probably ever acts alone to produce tooth wear; depending on the tooth surface concerned, all three could interact. For example, at contacting enamel surfaces or non-contacting surfaces, abrasion and erosion are likely to collaborate in enamel loss. Indeed, given the site of predilection for dentine hypersensitivity, namely buccal cervical areas, exposure of dentine through enamel loss is almost certainly due to an interaction of erosion with abrasion. In certain teeth, abfraction may act as a predisposing or co-destructive factor⁴⁵. This theoretical process, modelled in finite element analysis studies, suggests that eccentric occlusal loading leads to cusp flexure setting up cervical stress lesions, which, in turn, increase the susceptibility of enamel to abrasion and/or erosion. Attrition occurs due to tooth-to-tooth contact. Tooth wear due to attrition can reach pathological levels with parafunctional habits such

as bruxism^{46,47} as a result, occlusal dentine hypersensitivity may ensue. The interaction of abrasion and erosion with attrition has not been researched to any great degree. Recent studies *in vitro* demonstrated that enamel attrition was markedly reduced in an acid environment⁴⁸. An explanation for this somewhat surprising finding was the maintenance of very smooth contacting enamel surfaces due to the acid erosion, which reduces friction.

Interaction between abrasion and attrition, such as from the chewing of coarse diets or abrasive materials, has been the subject of only anecdote or case reports^{49,50}. Such cases suggest that some abrasive materials regularly introduced into the mouth and chewed, either as a habit or from an occupational environment, can cause marked enamel loss on contacting surfaces. Moreover, if combined in an acid medium, such as chewing fibrous acidic fruits like apples, tooth wear escalates dramatically. A model *in vitro* simulating the chewing of abrasive acid foods confirmed the potential for rapid enamel loss⁵¹.

Most interest in abrasion has centred on the effects of tooth brushing with toothpaste, with the majority of studies conducted *in vitro* and on dentine. As such, they are more relevant to the initiation of dentine hypersensitivity. A toothbrush alone has no measurable effects on enamel. Indeed, most toothpastes have very low relative enamel abrasivity (REA) values, as determined using the International Standards Organisation's Standard for toothpastes methodology⁵². Most toothpastes alone contribute little to enamel loss even over a lifetime of use. Erosion causes significant tooth wear and thereby dentine exposure at all sites on the anatomical crowns of teeth and, particularly, in the cervical area, where the enamel is very thin. Acids are usually classified as intrinsic or extrinsic: the former is hydrochloric acid from the stomach;

the latter originates from the diet or the environment, particularly in certain occupations⁴¹. Dentine hypersensitivity has been reported in association with erosion caused by acids from both intrinsic and extrinsic sources.

However, with respect to the buccal cervical site of predilection for dentine hypersensitivity, lesion localisation due to enamel loss is almost certainly the result of extrinsic acid erosion alone or, more likely, combined with tooth brushing with toothpaste. Thus, when acids come into contact with enamel, not only is there bulk loss of tissue but surface softening as well^{53,54}. Studies *in vitro* suggest that the surface softening can extend to 3–5 microns and that the tissue is highly susceptible to physical insults: a few strokes with a toothbrush and toothpaste, even a toothbrush alone, can remove this fragile layer⁵¹. Re-hardening can occur; however, evidence *in vitro* suggests that this may take hours⁵⁵, thus emphasising the need to avoid brushing teeth after food and/or drink. Indeed, the preventive potential of most toothpastes supports recommending brushing teeth before meals rather than the often cited advice to brush after meals.

The potentially serious nature of erosion was highlighted by a review of prevalence figures⁵⁶. In the 1993 UK Child Dental Health Survey⁵⁷, dentine exposure on deciduous teeth was found in a quarter of 5–6-year-olds and was even present on permanent teeth in 2 per cent of 11-year-olds. A review of the literature suggests the relevance of soft drink consumption from a very early age as important to tooth wear⁴¹. Studies *in situ* confirm the role of such drinks in enamel erosion and highlight a tenfold difference of individual susceptibility to erosion by acidic drinks⁵⁸⁻⁶⁰. The data from such studies indicated that, depending on susceptibility, and without the synergistic effects of other tooth wear factors, such

as abrasions, individuals consuming one litre of soft drinks per day could lose one millimetre of enamel in 2 to 20 years. Recently, some drinks have been modified successfully to minimise erosion and surface softening of enamel^{59,60}. Such modifications have thus far centred on adding calcium to drinks and making changes to titratable acidity and pH. Interest has also focused on polyphosphates; however, unpublished data from our laboratory studies indicate that, while these compounds may minimise surface loss of enamel, they may cause quite deep subsurface demineralised lesions.

Gingival recession and its aetiology have been reviewed^{61,62}. Recently, one author has described the condition as an enigma⁶², a description that now seems more aptly attributable to gingival recession than to dentine hypersensitivity. The aetiology of gingival recession appears to be multi-factorial and is made more complex by suggested predisposing factors. With few exceptions, aetiological and predisposing factors are implicated on the basis of circumstantial evidence and/or epidemiological association data. This applies, in particular, to tooth brushing, which has long been associated with gingival recession. Numerous factors ranging from filament stiffness and end rounding, to tooth brushing force, duration and frequency, have been considered relevant. Interestingly, toothpaste, and not the brush, is felt to produce abrasion to hard tissues, yet its role in soft tissue damage and gingival recession has never been considered. Other aetiological agents in gingival recession include acute ulcerative gingivitis (periodontitis), self-inflicted injury, periodontal disease, periodontal non-surgical and surgical procedures, with buccal or lingual alveolar bone dehiscence or fenestration acting as predisposing factors^{61,62}.

In conclusion, it is perhaps not surprising that the buccal cervical

area is predisposed to dentine hypersensitivity since erosive and abrasive factors alone or in combination are most likely to impact at this site to expose dentine. Although not studied, clinical experience suggests that gingival recession rather than loss of cervical enamel would account for the majority of exposed dentine. However, erosion alone or combined with abrasion and/or attrition may expose dentine through enamel loss at other sites on the anatomical crown.

Lesion initiation (reviews^{2,8})

Evidence already presented indicated that the lesions of dentine hypersensitivity have many more and wider open tubules than do non-sensitive dentine²³⁻²⁵ (Table 2). Replica studies demonstrated that cementum at the cervical area of teeth is rapidly lost and is never seen to cover the dentine once recession has occurred⁷. This observation suggests that the layer is easily removed by physical and/or chemical influences. Dentine is thought to be covered by a smear layer or the tubules occluded by calcium phosphate deposits derived from saliva. Removal of these occluding materials could also occur as a result of physical or chemical agents that open the dentinal tubules. Most research on and, therefore, conclusions about lesion initiation are based on studies *in vitro*.

In view of the manufacturers' and standards organisations' interest in the abrasivity of toothpaste to dentine, the influence of tooth brushing with toothpaste has attracted some interest by researchers. The toothbrush alone has little effect on dentine: it takes several hours of constant brushing *in vitro* to either remove the smear layer or re-create a smear layer⁶³ (these experiments represent years of normal tooth brushing). Toothpastes, their abrasives and, to some degree, the common toothpaste detergent, sodium lauryl sulphate, all cause

wear to dentine^{64,65}. Based on laboratory data, an associated review concluded that, under normal circumstances, tooth brushing with most toothpaste has little or no effect on enamel and clinically insignificant effects on dentine⁶⁶. Studies *in situ*, however, suggest that excessive or abusive tooth brushing habits could cause pathological dentine loss⁶⁷.

In dentine hypersensitivity, however, the following question begs to be asked: what effects does brushing teeth with toothpaste have on the dentine surface and, in particular, the smear layer and the tubules? Several scenarios can be envisaged, including: abrasive removal of the smear layer, abrasive creation of a smear layer, detergent removal of the smear layer, occlusion of tubules by abrasive particles, or occlusion of tubules by active desensitising ingredients. Again, studies *in vitro* indicate that most toothpastes readily remove the dentine smear layer to expose tubules⁶⁸.

Erosion of dentine appears to bring about rapid loss of the smear layer and the opening of dentinal tubules. Most soft drinks, some alcoholic beverages and yoghurt all readily remove the dentine smear layer after a few minutes exposure⁶⁹. Moreover, these sources of extrinsic acid dramatically reduce the resistance of the smear layer to gentle forces such as a nylon toothbrush used without toothpaste⁶³. Interestingly, some mouthrinses with pH values below 5 also readily dissolved the smear layer⁷⁰, and were even shown to erode enamel both *in vitro* and *in situ*⁷¹. Like enamel, erosion causes bulk loss of dentine and surface softening, the softened dentine being similarly very susceptible to physical insults⁷². Moreover, what little evidence is available throws into question the ability of softened dentine to rehardens⁷³.

In conclusion, available evidence suggests that lesion initiation in dentine hypersensitivity can be

induced by abrasive and erosive agents, Whereas erosion alone is probably the more dominant factor, in synergy with abrasion, it may bring about dentine wear and tubule opening.

Management strategies (reviews^{2,73})

In clinical experience, the professional approach to dentine hypersensitivity has been heavily treatment-based with little regard for the control of the aetiological and predisposing factors, which created the problem. This is perhaps not surprising since the practitioner and the sufferer are virtually bombarded with a vast array of products formulated to treat dentine hypersensitivity. The Canadian Consensus board, previously referred to, conducted surveys of dental professionals and indicated that there was confidence about diagnosing but not about managing dentine hypersensitivity. The principles of treatment will be discussed here, but this is not the place to describe or debate the therapeutic options, and the reader is directed to reviews on home use and in-office treatments⁷⁴. Nevertheless, it should be noted that the literature contains evidence, albeit often equivocal, for the apparent efficacy of a wide range of quite different, if not downright bizarre, agents for the treatment of dentine hypersensitivity. Few other human conditions or diseases, with the possible exception of haemorrhoids, appear treatable by such a diverse range of compounds.

As with all conditions or diseases, management strategies, which include treatment, are usually more successful than treatment alone. Failure to consider causation in the management of dentine hypersensitivity, as with caries and periodontal disease, may result at least in recurrence or, at worst, failure of treatment. Unfortunately, unlike caries and periodontal disease management, strategies for dentine hypersensi-

tivity are not data-driven but rather are based on logic derived from an understanding of the nature of the aetiology of and the predisposition to the condition.

Accepting that logic and biologic are often not the same, the following management strategy is proposed:

1. Ensure the correct diagnosis of dentine hypersensitivity is based on a history and examination, and is compatible with the definition's clinical descriptor.
2. Consider a differential diagnosis, as suggested by the definition of dentine hypersensitivity, which alone may explain the symptoms or identify the presence of other conditions contributing to the pain of dentine hypersensitivity.
3. Treat any and all secondary conditions that induce symptoms similar to dentine hypersensitivity (see above: Definition and differential diagnosis¹²).
4. Identify aetiological and predisposing factors, particularly with respect to erosion and abrasion. Consider detailed, written dietary histories and oral hygiene habits (frequency, duration and timing of brushing, brushing technique, estimation of brushing force, frequency of brush change, and appearance of brush at change). Some of these aspects of tooth brushing behaviour are best appraised by observing the patient brushing in the dental practice.
5. Remove or modify identified aetiological or predisposing factors. Offer dietary advice to minimise erosion and oral hygiene instruction to minimise abrasion and to divorce abrasion from erosion.
6. Recommend or provide treatments appropriate to the individual needs of the sufferer. The number of teeth involved and the severity of the pain are important variables, and should influence the treatment options.

Overall, there are two treatment approaches: to occlude dentinal

tubules, thereby blocking the hydrodynamic mechanism; to block neural transmission at the pulp. The majority of treatments, whether home-use or applied in-office, are formulated to occlude tubules. Blockage of neural transmission at the pulp theoretically can be achieved using topically applied potassium salts and completely by endodontics or tooth extraction. It is worth remembering that clinical trials on professionally-applied and, more particularly, home-use treatments show a significant improvement in symptoms due to either, or both, a placebo response or/and regression to the mode (natural improvement). Such studies even suggest that the mere recommendation of a home-use desensitising product, or the professional application of anything to exposed dentine, results in, on average, a 40 per cent and even greater improvement, irrespective of the specific treatment.

Conclusions

The evidence reviewed indicates that much has been learnt about dentine hypersensitivity since it was described as an enigma some 20 years ago. A great deal still remains to be discovered, however, and there are only very limited data derived from controlled clinical trials. Management strategies in particular have little scientific support and are based on what is known about the lesions of dentine hypersensitivity, the mechanism of stimulus transmission and the condition's aetiology. Little is known of the actual effects achieved by home-use desensitising dentifrice products *in vivo*. Dentine hypersensitivity, both in the localisation of lesions and certainly in the initiation of symptoms, has all the hallmarks of a tooth-wear phenomenon. Gingival recession probably accounts for most cervical dentine exposure but remains an under-researched and poorly understood process.

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