Dentine hypersensitivity — an enigma? a review of terminology, epidemiology, mechanisms, aetiology and management

R. H. Dababneh,[1] A. T. Khouri,[2] and M. Addy,[3]

Abstract Dentine hypersensitivity is a relatively common problem experienced in clinical dental practice. This condition may disturb the patient during eating, drinking, brushing and sometimes even breathing. Therapeutic intervention by desensitising agents may provide only partial pain relief and recurrence is common. Much remains unknown about dentine hypersensitivity, even the terminology can be questioned. Most of the literature over decades has been concerned with reporting clinical trials proving the efficacy of numerous treatments for dentine hypersensitivity. Indeed, besides haemorrhoids, there can be few other diseases or conditions known to man that can apparently be successfully treated by so many and extremely varied agents and formulations applied topically. This paper will discuss the epidemiology, mechanisms of pain production and aetiological factors for the condition in the hope of developing ideas for more realistic prevention and management strategies.

Tooth hypersensitivity, or more precisely dentine sensitivity or hypersensitivity, is described clinically as an exaggerated response to non-noxious stimuli and satisfies all the criteria to be classified as a true pain syndrome.[1] The suitability of the terms dentine sensitivity and dentine hypersensitivity have been questioned since both terms are often used to describe the same clinical condition. Although it has been suggested that true hypersensitivity can develop as a result of pulp inflammation,[2,3] the symptoms are thought to be more severe and persistent than the typical short sharp pain of dentine hypersensitivity. In such cases the management is likely to be completely different where it is directed at the pulp pathology. To date there is no information concerning the state of the pulp in dentine hypersensitivity.

Reviews on the subject have suggested that the term dentine sensitivity may be considered more appropriate since there is no evidence to indicate that 'hypersensitive' dentine differs in any way from normal dentine or that the pulp response is anything but a normal response to stimulation of exposed dentine.[4] However, it should be noted that not all exposed dentine is sensitive,[5,6] thus both terms could be considered suitable. Nevertheless, the term dentine hypersensitivity has been used for decades and is appreciated as a distinct entity by clinicians. Furthermore, the condition has been defined[7] and with a minor amendment, the definition was adopted by an international workshop on dentine hypersensitivity.[8] Thus, 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 dental defect or pathology. The definition clearly has two aspects. The first is a clinical descriptor of the condition. The second, perhaps more importantly identifies dentine hypersensitivity as a distinct clinical entity and invites the clinician to consider a differential diagnosis, since other conditions may have identical symptoms but require different management strategies.[7] Based upon these points it would not seem unreasonable to keep the term; accepting that it may not be totally accurate.

In general, conventional therapy for dentine hypersensitivity is based on using topical applied desensitising agents which can be applied either professionally or can be prescribed to the patient for home use. The ideal desensitising agent should not irritate or endanger the integrity of the pulp, should be relatively painless on application or shortly afterward, should be easily applied, rapid in action, permanently effective and finally should not discolour tooth structure.[9] To date no such treatment has been discovered and there is no 'gold standard' by which to assess new treatments. Topical desensitising agents had been classified[10] on the basis of their chemical and physical properties as follows:

Chemical agents	Physical agents
Corticosteroids	Composites
Silver nitrate	Resins
Strontium chloride	Varnishes
Formaldehyde	Sealants
Calcium hydroxide	Soft tissue grafts
Potassium nitrate	Glass-ionomer cements
Fluorides	Lasers
Sodium citrate	
Iontophoresis with 2% Sodium fluoride	
Potassium oxalate.	

In studying the above lists, it is somewhat surprising that such a varied array of surface treatments could all possibly be effective treatments for this single condition.

Epidemiological aspects of dentine hypersensitivity

Discomfort from dentine hypersensitivity is a common finding in adult populations, with the available prevalence data ranging from 8–57%.[11–15] The diversity of reports may be in part caused by different methods used to diagnose the condition and it is generally considered that surveys which rely on patient questionnaires alone greatly exaggerate the prevalence figures and thereby yield misleading data. Indeed, those studies which employed careful patient

[1]*Specialist in Periodontology,* [2]*Specialist in Conservative Dentistry, Jordanian Royal Medical Services, Queen Alia Military Hospital, Jordan;* [3]*Professor Honorary Consultant in Periodontology, Division of Restorative Dentistry, Dental School, Bristol BS1 2LY, UK*

REFEREED PAPER
Received 27.05.99; accepted 24.09.99
 187: 606–611

examinations produced surprisingly similar prevalence figures of around 15%.[11–13] Interestingly, the prevalence of cervical dentine sensitivity, another term used to describe dentine hypersensitivity, was found to be much higher in periodontal patients, ranging between 72.5–98%.[16] This may reflect a different aetiology, particularly since in periodontal disease bacteria are reported to penetrate dentine to a considerable distance.[17] Such sensitivity of dentine therefore may not be consistent with the agreed definition of dentine hypersensitivity[8] and may be a separate clinical entity possibly requiring different preventive and management strategies.

In general, a slightly higher incidence of dentine hypersensitivity is reported in females than in males,[12, 13, 18–20] which may reflect their overall healthcare and better oral hygiene awareness.[4] Most sufferers from dentine hypersensitivity range in age from 20 to 40 years but the peak occurrence is found at the end of third decade.[11, 12,14]Regarding the intra-oral distribution, dentine hypersensitivity is most commonly reported from the buccal cervical zones of permanent teeth. Sites of predilection in descending order are canines and first premolars, incisors and second premolars and molars.[11–13, 18–21]

In conclusion, although dentine hypersensitivity is already perceived as a common painful condition, it is possible that the prevalence will increase. Thus, as a result of the continuing emphasis on preventive dentistry, more adults will retain their teeth into later life, this in turn could lead to increased numbers of exposed dentine surfaces through periodontal therapy and home care procedures.

Mechanisms of dentine sensitivity

Several theories have been proposed to explain the mechanism of dentine sensitivity and therefore of dentine hypersensitivity.[22] Of these the most widely accepted theory is the so-called hydrodynamic theory of sensitivity. This theory postulates that rapid shifts, in either direction, of the fluids within the dentinal tubules, following stimulus application, result in activation of sensory nerves in the pulp/inner dentine region of the tooth.[23,24] The hydrodynamic hypothesis for dentine sensitivity was proposed a hundred years ago[23] with confirmatory evidence produced in the 1950s and 1960s.[24] Essentially, certain stimuli create a pressure change across the dentine which can excite individual intradental nerves.[25] Studies performed *in vivo* revealed that the response of the pulpal nerves was proportional to the pressure and therefore the rate of fluid flow.[26] Interestingly stimuli, such as cold, which cause fluid flow away from the pulp produce more rapid and greater pulp nerve responses than those, such as heat, which cause an inward flow.[26] This certainly would explain the rapid and severe response to cold stimuli compared to the slow dull response to heat. The exact mechanism by which the fluid flow stimulates pulpal nerves is not known with any certainty. However from animal experiments a mechano-receptor response is suggested.[26] Thus the pressure change across dentine distorts the pain receptors at the pulp dentine border. This would be similar to the activation of touch sensitive nerves around hair skin follicles by the application of light pressure to the protruding hair. The role of the odontoblast and the odontoblast process in dentine sensitivity has been reviewed.[27] Essentially, the odontoblast process is thought to extend only a short distance into dentinal tubules and thereby cannot be directly involved in stimulus transmission across dentine: a hypothesis previously described as the odontoblast transducer mechanism. Nevertheless, the large shear forces created by fluid flow in tubules could damage odontoblasts resulting in a local neurogenic inflammation of the pulp.

The pain producing stimuli can be thermal, tactile, osmotic, chemical or evaporative,[8] but the cold stimulus appears to be the strongest and causes the greatest problem to those troubled by dentine hypersensitivity.[14,18] It should be mentioned, however, that not all exposed dentine is sensitive. Evidence from a scanning electron microscopic investigation of extracted teeth would suggest that there are differences between 'hypersensitive' and 'non-sensitive' dentine in that there are more and wider open dentinal tubules in sensitive dentine.[5] Additionally, another scanning electron microscope study, based on replica models of hypersensitive and non-sensitive dentine, showed that, in hypersensitive dentine, the smear layer was thinner, different in structure and probably under-calcified than in non sensitive dentine.[6] These findings appear consistent with the hydrodynamic theory. The greater number of open and wider tubules at the dentine surface would enhance fluid permeability through dentine and as such increase the possibility for stimulus transmission and subsequent pain response. 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 diameter results in a 16-fold increase in flow rate. This is of relevance to treatment regimens which, as will be discussed later, aim to narrow or occlude the tubules.

Aetiological and predisposing influences

There are potentially numerous and varied aetiological and predisposing factors to dentine hypersensitivity. Certainly, no prime cause can be identified. By definition, dentine hypersensitivity may arise as a result of loss of enamel and or root surface denudation with exposure of underlying dentine. Enamel loss as a part of tooth wear may result from attrition, abrasion or erosion. Although tooth wear has usually been divided into attrition, erosion and abrasion, in reality it is a combination of these but often with differing proportional effects. Attrition describes the wear of teeth at sites of direct contact between teeth.[28] Attrition is associated with occlusal function and may be exaggerated by habits or parafunctional activity such as bruxism. Thus bruxism was found to be the sole cause of pathological tooth wear in 11% of referred tooth wear cases and was a contributory factor in two-thirds of the combined aetiology cases.[29] Abrasion describes the wear of teeth caused by objects other than another tooth,[28] examples include toothbrush/toothpaste abrasion and the variety of facets which can be caused by pipe smoking or other similar habits. Typical toothbrush abrasion lesions are side dependent, for example being greater on the left-side in right-handed people The buccal cervical area of the teeth are the sites of predilection. Furthermore, canines and premolars are most affected because of their position within the dental arch where they receive the most attention during toothcleaning.[30] The toothbrush itself has little or no effects on dental hard tissues.[31] Even toothpaste on a toothbrush alone causes almost no enamel abrasion and only clinically insignificant effects on dentine. However when combined with erosive agents tissue loss from toothbrushing with toothpaste is increased enormously.[32] Little is known about abrasion from chewing: the crushing of bones between the teeth[33] and chewing tobacco[34] were believed to lead to abrasion of teeth.

Erosion

Erosion is currently believed to be the major factor involved in tooth wear, and has been defined as the dissolution of teeth by acids which are not of bacterial origin.[35] Erosion may be by either extrinsic or intrinsic acids.[36,37] Extrinsic erosion can be subdivided into dietary and environmental, while intrinsic erosion is the result of exposure of teeth to gastric juice. Dietary erosion may result from foods or drinks containing acids such as citrus fruits, pickled food, fruit juices, carbonated drinks, wines and ciders. A recent publication showed that a raw food diet bears an increased risk of dental erosion compared to conventional nutrition.[38] Additionally, a study *in vitro* showed that red and white wine, citrus fruit juices, apple juice, and yoghurt were capable of rapidly dissolving the dentine smear layer within a few minutes.[39] Perhaps surprisingly, a carbonated cola drink was considerably less erosive.[39] Vitamin C (ascorbic acid), which is considered a healthy additive in many drinks, has been implicated in dental erosion.[40] In addition citric acid, found in

many drinks, has the potential to both demineralise dental hard tissues and chelate calcium.[41] Industrial erosion results from occupational exposure to acids or acidic vapour, such as, workers in battery manufacture[42] and wine tasters.[43] Other extrinsic sources of erosion have been reported, including swimmers who trained in poorly maintained pools with water at pH 2.7.[44] Improper use of bleaching agents particularly delivered in night guards is another risk factor for erosion of enamel and dentine and has been implicated in the development of dentine hypersensitivity.[45] Some mouthrinses have low pH values[46] and have the potential to dissolve the smear layer and thereby expose dentinal tubules; an effect enhanced by post treatment brushing.[47]

Intrinsic erosion may result from gastric reflux as in patients with hiatus hernia,[48] chronic alcoholism[49] and eating disorders.[50–52] When erosion is caused by gastric regurgitation, the palatal aspects of the upper incisors and the occlusal and buccal aspects of lower posterior teeth are primarily affected.[51,53]

Abfraction

Abfraction or cervical stress lesions has been hypothesised as an aetiological factor in tooth wear.[54,55] The process is thought to involve eccentric occlusal loading leading to cusp flexure. This in turn leads to compressive and tensile stresses at the cervical fulcrum area of the tooth with the resultant weakening of the cervical tooth structure. The process may be co-destructive rather than directly causal whereby abrasion and or erosive processes are potentiated. It is difficult to diagnose such lesions properly, but generally, in cases where a deep V-shaped cervical notch is present or when cervical restorations are repeatedly lost, the practitioner should look for wear facets or other signs of occlusal trauma.[56]

Gingival recession

Gingival recession and subsequent root surface exposure allow more rapid and extensive exposure of dentinal tubules because the cementum layer overlying the root surface is thin and easily removed.[57] Gingival recession, as with dentine hypersensitivity,[58] has been described as enigma,[59] having what appears to be a multifactorial aetiology.[59, 60]

Toothbrushing has long been implicated in gingival recession on buccal surfaces and is a frequent finding in subjects with a high standard of oral hygiene,[61, 62] or with a history of hard toothbrush use.[63] Also recession increases with increasing brushing frequency.[64] Gingival recession is frequently cited to result from periodontal treatment particularly surgery as is dentine hypersensitivity.[13,15,65]

Clinical studies,[16,66] that aimed to assess the prevalence of dentine hypersensitivity in a population of patients referred to a periodontal department, found a much higher percentage affected than cited in other epidemiological studies.[11–13] This could suggest periodontal disease and/or treatment may play a role in the aetiology of dentine hypersensitivity[16,66] or as alluded to previously, this type of sensitivity is not dentine hypersensitivity as defined. Relevant to this, the role of plaque in the aetiology of dentine hypersensitivity is an area of controversy. Some authors point out that most sensitive surfaces, particularly on the buccal aspect of the teeth, show very low plaque scores[19] and enthusiastic toothbrushing has long been associated with gingival recession and sensitivity.[67]

Clinical experience also indicates that areas of toothbrush abrasion/erosion appear free of plaque. Other authors have suggested a possible role of plaque and bacterial contamination of exposed dentine in dentine hypersensitivity,[68,69] particularly since gingival recession can be associated with both high inflammatory and plaque scores.[63] A possible interpretation of the above observations is that toothbrushing behaviour played a role in determining the distribution of gingival recession while plaque induced the dentine sensitivity possibly through dissolution of the smear layer and bacterial penetration of the tubules.[17] The management of such a condition therefore would be quite different from that of true dentine hypersensitivity.

Two phases in dentine hypersentivity development

In summary, there would appear two phases to the development of dentine hypersensitivity. Firstly, 'lesion localisation' by exposure of dentine and then 'lesion initiation' by opening of dentinal tubules. Given the buccal cervical site of predilection of the condition, lesion localisation will mainly result from abrasion and erosive influences to enamel and the gingivae. Clinical evidence indicates that gingival recession accounts for a much greater dentine area exposure than cervical enamel loss. Lesion initiation requires removal of cementum or smear layers. These could be achieved by abrasive or erosive agents. The evidence available and cited suggests erosion is the more dominant factor but can be potentiated by abrasion.

The clinical management of dentine hypersensitivity

The greatest clinical implication of dentine hypersensitivity is how the condition may be prevented either from occurring or recurring, and this can only be debated by considering the probable aetiologic factors. There is a need for greater professional and thereby public health awareness, through education, of the causes, effects and prevention of tooth wear and gingival recession. Management of a patient suffering from dentine hypersensitivity should be based on a correct diagnosis of the condition by the dentist, who should be aware of other clinical conditions which are similar in their presenting features. Conditions that can produce symptoms mimicking those of dentine hypersensitivity are cracked tooth syndrome, fractured restorations, chipped teeth, dental caries, post-restorative sensitivity, and teeth in acute hyperfunction.[7] Patients generally complain that pain arising from dentine hypersensitivity is usually rapid in onset, sharp in character, and short in duration.[22] More rapid response to stimuli or the persistence of pain after removal of the stimuli have been ascribed to inflammatory changes in the pulp.[70] In such cases conventional approaches to the treatment of dentine hypersensitivity are unlikely to be successful and recourse to endodontics even exodontia may be necessary.

Toothbrushing

Because incorrect toothbrushing appears to be an aetiological factor in dentine hypersensitivity, instruction in proper brushing technique can prevent further loss of dentine and the resulting hypersensitivity. Excessive force, hard toothbrushes, highly abrasive toothpastes should be avoided. The use of none or low abrasive dentifrices or brushing with water resulted in closure of dentinal tubules; while brushing with a dentifrice containing calcium hydrogen phosphate as an abrasive system led to opening of the dentinal tubules.[71] Nevertheless, a study *in vitro*[72] showed that some dentifrice abrasives in desensitising products, notably artificial silica, can occlude tubules and remain resistant to removal by both water and orange juice washing.

Clearly at this time, there is a dearth of information as to the effects of any particular toothpaste on an exposed dentine surface. Nevertheless, toothbrushing should be avoided after consuming acidic foods and drinks since tooth brushing, in combination with acid decalcification of superficial dentine, is capable of accelerating the loss of tooth structure[32] and opening dentinal tubules.[31] This finding raises the question of whether toothbrushing should be performed before meals rather than after meals if the diet contains acidic components. In order to clarify this point, an *in vitro* scanning electron microscope study was conducted to evaluate the effect of toothbrushing prior and/or subsequent to dietary acids application on smear layer formation and the patency of dentinal tubules.[73] The results of this study[73] suggested that in cases of den-

tine hypersensitivity, toothbrushing should not precede or follow dietary acid application but be separate from mealtimes. Certainly, the weight of available evidence strongly contradicts the message to brush after meals. Logically, this is not surprising since toothpastes are formulated to prevent disease and are not therapeutic agents.

Dietary acid

In view of a very clear role of dietary acid in the initiation of dentine hypersensitivity, dietary counselling is an important factor for the management of the condition. A written diet history should be obtained from patients with dentine hypersensitivity in order to identify aetiological agents and form a basis from which to provide advice.[7] As with the prevention of caries advice should be directed towards reducing the quantity and frequency of acid intake. Other suggestions have been to, drink something neutral or alkaline after consuming acids such as water or milk, avoid sipping, use a straw, chill the drink which can reduce its erosive potential and finally, avoid acids as a snack just before bed time or during the night.[74, 75] When mouthrinses or bleaching agents are to be prescribed, the dental practitioner should be aware of the potential disadvantages of these products as well as the possible therapeutic gain from their use in order to advise their patients with respect to usage of such products.

If intrinsic erosion is suspected as a factor in tooth wear and dentine hypersensitivity, then the cause should be investigated and treated by referring the patient to the medical practitioner. Rinsing the mouth with liquid antacid, especially after vomiting or regurgitation, is advised.[76] In severe or chronic cases, fabrication of an occlusal splint to be inserted at high risk times can reduce the insult to the teeth. If the splint is loaded with an alkali such as sodium bicarbonate, it may further neutralise the effect of gastric acids.[28] Non-acidulated fluoride gels may also be useful within the splint.[77]

Patient communication

Finally successful patient management relies heavily on good communication skills which are of vital importance in dentistry because it improves the quality and amount of information obtained from the patient, increases the likelihood of patient compliance, decreases the patient anxiety and improves the probable outcome of treatment.[78] As dentine sensitivity or hypersensitivity may result from dental treatments, every patient must be informed of the potential treatment risks. Informing the patient in advance regarding the possibility of potentially painful events following periodontal therapy and recently placed restorations can greatly strengthen the dentist-patient relationship and reduce pain and anxiety. In addition, explaining to the patient the possible causes of dentine hypersensitivity may help to modify or control these causative factors which in turn may improve the outcome of treatment.

Treatments

Treatments for dentine hypersensitivity are so numerous and diverse that a detailed review is outside the scope of this text. A classification according to chemical or physical characteristics is given at the beginning of this review. Treatments can be categorised and summarised according to delivery and therapeutic aims into professional (in office) and home use treatments. Professional treatments have been reviewed[79] and usually involve the application of a material to the tooth surface with the aim of occluding tubules. Similar literature reviews are available for the numerous over-the-counter proprietary products purchased or prescribed for home use: usually mouthrinses or toothpastes.[80–83] The therapeutic aims of both professional and home use treatments are to either interrupt the pulp neural response or block the sensitivity mechanism through tubule occlusion. Interruption of the neural response can be achieved through endodontia or exodontia, at the one extreme, to the application of potassium salts to dentine at the other. In the latter case delivery is usually through mouthrinses or toothpastes used at home. Direct evidence that potassium salts used in such vehicles achieves such an effect is lacking. Professional treatments used to produce tubule occlusion usually involve resins, varnishes, dentine bonding agents or restorative materials. In most cases a macroscopic coating is apparent providing evidence that tubule occlusion has been achieved. The same cannot be said for home use products. Thus, there is a dearth of information as to the changes produced at the dentine surface either by so called desensitising agents or by products containing these agents.

The choice of professional, home use treatments or indeed both combined is at present somewhat arbitrary and dependent on the practitioner. There are at present no tried and tested regimens proven superior to others. At best there are available in the literature the results of many studies supporting the efficacy of numerous agents and formulations. A few of the investigations have compared one professional treatment with another or one home use product with another, but mostly comparisons are with a control, minus active or placebo treatment. There appear to have been no comparisons of professional treatments with home use products. As already stated, studies reveal that an unusually large number of very diverse agents or formulations are apparently effective in reducing, even eliminating, the symptoms of dentine hypersensitivity. Two possible explanations for this most peculiar finding, other than all such agents are indeed effective, can be proposed. Firstly, dentine hypersensitivity is a painful condition and as such symptoms can improve apparently irrespective of treatment, a process described as regression to the mode. Secondly, painful conditions can show a placebo response to a treatment whereby the mere perception by the patient of treatment provision brings about some amelioration of symptoms. Recently, three clinical trials of treatment agents for dentine hypersensitivity, one by trial design[84] and two employing water as a treatment,[85,86] suggested placebo response improvements in symptoms of at least 40%.

Summary of management strategy

- Consider a differential diagnosis and deal with any dental pathology which could cause similar 'dentinal' symptoms.
- Identify aetiological and predisposing factors, these will mainly relate to abrasion and erosion. Written diet histories should be considered at an early stage.
- Remove, reduce or modify aetiological and predisposing factors. This will usually involve dietary advice to minimise erosion and oral hygiene advice to minimise abrasion.
- Treatment strategies will vary, being dependent on each individual case, and in particular on the number of teeth involved and the severity of symptoms. However some improvement for a majority of cases will be obtained by the recommendation of a desensitising toothpaste. Anecdotally, the prescription of a sodium fluoride mouthrinse, for use once daily for a month, appears to offer adjunctive benefits to the desensitising toothpaste. The topical application of the toothpaste directly onto sensitive cervical dentine just before retiring (without post application rinsing or expectoration) is also frequently cited as beneficial. Certainly active ingredients and abrasives such as the artificial silicas will have a much greater time to bind to the dentine and occlude tubules.[69]
- Patients or more usually individual teeth refractory to the above approach should firstly be re-evaluated for possible alternative dental pathology and appropriate treatment instituted. If a diagnosis of dentine hypersensitivity is reaffirmed, consideration should be given to the application of a professional product. Resins or other materials which bond to dentine and are resistant to wear are recommended; fluoride varnishes are not substantive and provide relief of limited duration.
- Individual teeth exhibiting severe symptoms, particularly when the pain persists after removal of the initiating stimulus should be

considered for endodontic treatment, but then only after obtaining informed consent from the patient.
- Regular review and reinforcement of the preventive aspects of management.

Conclusion

The increase of longevity of dentition through periodontal therapy and plaque control procedures may increase the incidence of dentine hypersensitivity. The ultimate goal in the treatment of dentine hypersensitivity is the immediate and permanent relief of pain. For many patients, conventional desensitising agents often produce unsatisfactory results, since the action is usually directed toward the occlusion of the opened dentinal tubules without considering the causative factors that created the problem. Once a definitive diagnosis of dentine hypersensitivity has been made, after considering a differential diagnosis, a careful assessment of the aetiologic factors must be considered, which in turn if identified and correctly managed may enhance the outcome of the currently used desensitising agents and ensure more successful management.

1 Curro F A. Tooth hypersensitivity in the spectrum of pain. *Dent Clin North Am* 1990; **34**: 429-437.
2 Trowbridge H O. Mechanism of pain induction in hypersensitive teeth. *In* Rowe N H (ed) *Hypersensitive dentine: origin and management* Ann Arbor: University of Michigan, pp1-10, 1985.
3 Narhi M, Kontturi-Narhi V, Hirvonen T, Ngassapa D. Neurophysiological mechanisms of dentine hypersensitivity [Review]. *Proc Finn Dent Soc* 1992; **88** suppl (1): 15-22.
4 Addy M. Etiology and clinical implications of dentine hypersensitivity. *Dent Clin North Amer* 1990; **34**: 503-514.
5 Absi E G, Addy M, Adams D. Dentine hypersensitivity: a study of the patency of dentinal tubules in sensitive and non sensitive cervical dentine. *J Clin Periodontol* 1987; **14**: 280-284.
6 Rimondini L, Baron C, Carrassi A. Ultrastructure of hypersensitive and non-sensitive dentine. *J Clin Periodontol* 1995; **22**: 899-902.
7 Dowell P, Addy M, Dummer P. Dentine hypersensitivity: Aetiology, differential diagnosis and management. *Br Dent J* 1985; **158**: 92-96.
8 Holland G R, Narhi M N, Addy M, Gangarosa L, Orchardson R. Guidelines for the design and conduct of clinical trials on dentine hypersensitivity. *J Clin Periodontol* 1997; **24**: 808-813.
9 Grossman L. A systematic method for the treatment of hypersensitive dentine. *J Am Dent Assoc* 1935; **22**: 592-598.
10 Scherman A, Jacobsen P L. Managing dentin hypersensitivity: What treatment to recommend to patients. *J Am Dent Assoc* 1992; **123**: 57-61.
11 Graf H, Galasse R. Morbidity, prevalence and intraoral distribution of hypersensitive teeth. *J Dent Res* 1977; **56** (Spec Issue A) 162, abstr 479.
12 Flynn J, Galloway R, Orchardson R. The incidence of hypersensitive teeth in the west of Scotland. *J Dent* 1985; **13**: 230-236.
13 Fischer C, Fischer R G, Wennberg A. Prevalence and distribution of cervical dentine hypersensitivity in a population in Rio de Janeiro, Brazil. *J Dent* 1992; **20**: 272-276.
14 Irwin C R, McCusker P. Prevalence of dentine hypersensitivity in general dental population. *J Irish Dent Assoc* 1997; **43**: 7-9.
15 Liu H C, Lan W H, Hsieh C C. Prevalence and distribuation of cervical dentine hypersensitivity in a population in Taipei, Taiwan. *J Endodont* 1998; **24**: 45-47.
16 Chabanski M B, Gillam D G, Bulman J S, Newman H N. Clinical evaluation of cervical dentine sensitivity in a population of patients referred to a specialist periodontology department: a pilot study. *J Oral Rehabil* 1997; **24**: 666-672.
17 Adriaens P A, DeBoever J A, Loesche W J. Bacterial invasion in root,cementum and radicular dentine of periodontally diseased teeth in humans — a reservoir of periodontopathic bacteria. *J Periodontol* 1988; **59**: 222-230.
18 Orchardson R, Collins W J N. Clinical features of hypersensitive teeth. *Br Dent J* 1987; **162**: 253-256.
19 Addy M, Mostafa P, Newcombe R G. Dentine hypersensitivity: the distribution of recession, sensitivity and plaque. *J Dent* 1987; **15**: 242-248.
20 Oyama T, Matsumoto K. A clinical and morphological study of cervical hypersensitivity. *J Endod* 1991; **17**: 500-502.
21 Lussi A R, Schaffner M, Hotz P, Suter P. Epidemiology and risk factors of wedge-shaped defects in Swiss population. *Schweiz Monstsschr Zahnmed* 1993; **103**: 276-280.
22 Dowell P, Addy M. Dentine Hypersensitivity: A review: Aetiology, symptoms and theories of pain production. *J Clin Periodontol* 1983; **10**: 341-350.
23 Gysi A. An attempt to explain the sensitiveness of dentin. *Br J Dent Sci* 1900; **43**: 865-868.
24 Brannstrom M. A hydrodynamic mechanism in the transmission of pain producing stimuli through the dentine. *In* Anderson D J ed *Sensory mechanism in dentine.* Oxford: Pergamon, pp 73-79, 1962.
25 Narhi M V O, Hirvonen T. The response of dog intradental nerves to hypertonic solutions of $CaCl_2$ and NaCl, and other stimuli applied to dentine. *Arch Oral Biol* 1987; **32**: 781-786.
26 Matthews B, Vongsavan N. Interactions between neural and hydrodynamic mechanisms in dentine and pulp. *Arch Oral Biol* 1994; **39**: (Suppl): 87-95.
27 Pashley D H. Dynamics of the pulpo-dentinal complex. *Crit Rev Oral Biol Med* 1996; **7**: 104-133.
28 Smith B G N. Toothwear: aetiology and diagnosis. *Dent Update* 1989; **16**: 204-212.
29 Smith B G N, Knight J K. A comparison of patterns of toothwear with the etiological factors. *Br Dent J* 1984; **157**: 16-19.
30 Addy M, Griffiths G, Dummer P, Kingdon A, Shaw W C. The distribution of plaque and gingivitis and the influence of brushing hand in a group of 11-12 year old school children. *J Clin Periodontol* 1987; **14**: 564-572.
31 Absi E G, Addy M, Adams D. Dentine hypersensitivity — the effect of toothbrushing and dietary compounds on dentine in vitro: an SEM study. *J Oral Rehabil* 1992; **19**: 101-110.
32 Davis W B, Winter P J. The effect of abrasion on enamel and dentine after exposure to dietary acids. *Br Dent J* 1980; **148**: 253-256.
33 Kerr N W. Diet and tooth wear. *Scot Med J* 1988; **33**: 313-315.
34 Milosevic A, Lo M S F. Tooth wear in three ethnic groups in Sabah (North Borneo). *Int Dent J* 1996; **46**: 572-578.
35 Pindborg J J. *Pathology of the hard dental tissues.* Munksgaard. Copenhagen: pp312-321, 1970.
36 Zero D T. Etiology of dental erosion-extrinsic factors. *Eur J Oral Sciences* 1996; **104**: 162-177.
37 Scheutzel P. Etiology of dental erosion-intrinsic factors. *Eur J Oral Sci* 1996; 104: 178-190.
38 Ganss C, Schlechtriemen M, Klimek J. Dental erosion in subjects living on a raw food diet. *Caries Res* 1999; **33**: 74-80.
39 Addy M, Absi E G, Adams D. Dentine hypersensitivity: The effects *in vitro* of acids and dietary substances on root planed and burred dentine. *J Clin Periodontol* 1987; **14**: 274-279.
40 Grenby T H, Phillips A, Desai T, Mistry M. Laboratory studies of the dental properties of soft drinks. *Br J Nutr* 1989; **62**: 451-464.
41 Jarvinen V K, Rytomaa I I, Heinonen O P. Risk factors in dental erosion. *J Dent Res* 1991; **70**: 942-947.
42 Petersen P E, Gormsen C. Oral conditions among German battery workers. *Comm Dent Oral Epidemiol* 1991; **19**: 104-106.
43 Gray A, Ferguson M M, Wall J C. Wine tasting and dental erosion. Case report. *Aust Dent J* 1998; **43**: 32-34.
44 Centerwal B S, Armstrong C W, Funkhouser L S, Elzay R P. Erosion of dental enamel among competitive swimmers at a gas-chlorinated swimming pool. *Am J Epidemiol* 1986; **123**: 641-647.
45 Leonard R H Jr, Haywood V B, Phillips C. Risk factors for developing tooth sensitivity and gingival irritation associated with nightguard vital bleaching. *Quint Int* 1997; **28**: 527-534.
46 Bhatti S A, Walsh T F, Douglas C I W. Ethanol and pH levels of proprietary mouthrinses. *Comm Dent Health* 1994; **11**: 71-74.
47 Addy M, Loyn T, Adams D. Dentine hypersensitivity — effects of some proprietary mouthwashes on the dentine smear layer: a SEM study. *J Dent* 1991; **19**: 148-152.
48 Howden G F. Erosion as the presenting symptom in hiatus hernia.
49 Robb N D, Smith B G N. Prevalence of pathological toothwear in patients with chronic alcoholism. *Br Dent J* 1990; **169**: 367-369.
50 Spigset O. Oral symptoms in bulimia nervosa. A survey of 34 cases. *Acta Odont Scand* 1991; **49**: 335-339.
51 Robb N D, Smith B G N, Geidrys-Leeper E. The distribution of erosion in the dentitions of patients with eating disorders. *Br Dent J* 1995; **178**: 171-175.
52 Rytomaa I, Jarvinen V, Kanerva R, Heinonen O P. Bulimia and tooth erosion. *Acta Odont Scand* 1998; **56**: 36-40.
53 Bartlett D W, Evans D F, Anggiansah A, Smith B G N. A study of association between gastro-oesophageal reflux and palatal dental erosion. *Br Dent J* 1996; **181**: 125-132.
54 Grippo J O. Abfraction: A new classification of hard tissue lesion. *J Esthet Dent* 1991; **3**: 14-19.
55 Braem M, Lambrechts P, Vanherle G. Stress-induced cervical lesions. *J Prosthet Dent* 1992; **67**: 718-722.
56 Milosevic A. Toothwear: Aetiology and presentation. *Dent Update* 1998; **25**: 6-11.
57 Bevenius J, Lindskog S, Hultenby K. The micromorphology *in vivo* of the buccocervical region of premolar teeth in young adults. A replica study by scanning electron microscopy. *Acta Odont Scand* 1995; **52**: 323-334.

58 Johnson R H, Zulgar-Nairn B J, Koval J J. The effectiveness of an electro-ionising toothbrush in the control of dentinal hypersensitivity. *J Periodontol* 1982; **53**: 353-359.
59 Smith R G. Gingival recession. Reappraisal of an enigmatic condition and a new index for monitoring. *J Clin Periodontol* 1997; **24**: 201-205.
60 Watson P J C. Gingival recession. *J Dent* 1984; **12**: 29-35
61 Serino G, Wennstrom J L, Lindhe J, Eneroth L. The prevalence and distribution of gingival recession in subjects with a high standard of oral hygiene. *J Clin Periodontol* 1994; **21**: 57-63.
62 Carlos M C, Muyco M M, Caliwag M L, Fajardo J A, Uy H G. The prevalence and distribution of gingival recession among U.E. dental students with a high standard of oral hygiene. *J Philipp Dent Assoc* 1995; **47**: 27-48.
63 Goutoudi P, Koidis P T, Konstantinidis A. Gingival recession: a cross-sectional clinical investigation. *Eur J Prosthodont Rest Dent* 1997; **5**: 57-61.
64 Khocht A, Simon G, Person P, Denepitiya J L. Gingival recession in relation to history of toothbrush use. *J Periodontol* 1993; **64**: 900-905.
65 Wallace J A, Bissada N F. Pulpal and root sensitivity rated to periodontal therapy. *Oral Surg Oral Med Oral Path* 1990; **69**: 743-247.
66 Chabanski M B, Gillam D G, Bulman J S, Newman H N. Prevalence of cervical dentine sensitivity in a population of patients referred to a specialist periodontology department. *J Clin Periodontol* 1996; **23**: 989-992.
67 Gorman W J. Prevalence and etiology of gingival recession. *J Periodontol* 1967; **38**: 316-319.
68 Lawson K, Gross K B, Overman P R, Anderson D. Effectiveness of chlorhexidine and sodium fluoride in reducing dentine hypersensitivity. *J Dent Hyg* 1991; **65**: 340-344.
69 Bissada N F. Symptomatology and clinical features of hypersensitive teeth. *Arch Oral Biol* 1994; **39** Suppl: 31-32.
70 Dachi S F. The relationship of pulpitis and hyperaemia to thermal sensitivity. *Oral Surg* 1965; **19**: 776-779.
71 Kuroiwa M, Kodaka T, Kuroiwa M, Abe M. Dentine hypersensitivity: Occlusion of dentinal tubules by brushing with and without an abrasive dentifrice. *J Periodontol* 1994; **65**: 291-296.
72 Absi E G, Addy M, Adams D. Dentine hypersensitivity: uptake of toothpastes onto dentine and effect of brushing, washing and dietary acid, SEM *in vitro* study. *J Oral Rehabil* 1995; **22**:175-182.
73 McAndrew R, Kourkouta S. Effects of toothbrushing prior and/or subsequent to dietary acid application on smear layer formation and the patency of dentinal tubules: an SEM study. *J Periodontol* 1995; **66**: 433-448.
74 Nunn J, Shaw L, Smith A. Tooth wear — dental erosion. *Br Dent J* 1996; **180**: 349-352.
75 Milosevic A. Toothwear (Management). *Dent Update* 1998; **25**: 50-55.
76 Shaw L, Smith A. Erosion in children. An increasing clinical problem? *Dent Update* 1994; **21**: 103-106.
77 Kleier D J, Aragon S B, Aberback R E. Dental management of the chronic vomiting patient. *J Am Dent Assoc* 1984; **108**: 618-621.
78 Newton J T. Dentist/patient communication: a review. *Dent Update* 1995; **22**: 118-122.
79 Trowbridge H O, Silver D R. A review of current approaches to in-office management of tooth hypersensitivity. *Dent Clin North Amer* 1990; **34**: 561-582.
80 Addy M, Dowell P. Dentine hypersensitivity – a review: clinical and *in vitro* evaluation of treatment agents. *J Clin Periodontol* 1983; **10**: 351-363.
81 Collaert B, Speelman J. The treatment of dentine hypersensitivity. *Rev Belge De Med Dent* 1991; **46**: 63-73.
82 Jackson R J, McDonald F E. A comparison of dentifrices for the treatment of dentine hypersensitivity. *Archs Oral Biol* 1994; **39**(Suppl):133-136.
83 Kanapka J A. Over the counter dentifrices in the treatment of tooth hypersensitivity: review of clinical studies. *Dent Clin North Amer* 1990; **34**: 545-560.
84 West N X, Addy M, Jackson R J, Ridge B D. Dentine hypersensitivity: review and discussion of controls and the placebo response. A comparison of the effect of strontium acetate and potassium nitrate toothpastes on dentine hypersensitivity. *J Clin Periodontol* 1997; **24**: 209-215.
85 Yates R, West N, Addy M, Marlow I. The effects of a potassium citrate, cetylpyridinium chloride, sodium fluoride mouthrinse on dentine hypersensitivity, plaque and gingivitis. A placebo controlled study. *J Clin Periodontol* 1997; **25**: 813-820.
86 Yates R, Owens J, Jackson R, Newcombe R G, Addy M. A split mouth placebo controlled study to determine the effect of amorphous calcium phosphate in the treatment of dentine hypersensitivity. *J Clin Periodontol* 1998; **25**: 687-692.