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Advances in the treatment of root dentine sensitivity: mechanisms and treatment principles

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There are limited studies specifically on the prevalence of root dentine hypersensitivity or root sensitivity *per se*; most of the published information relates to the prevalence of dentine hypersensitivity (DH). Several investigators have suggested that there may be some justification on the basis of differing pathologies of distinguishing between those individuals complaining of DH who have relatively healthy mouths with those who complain of DH as a result of periodontal disease and/or its treatment. It is generally recognized that those individuals diagnosed with periodontal disease and having periodontal therapy including scaling procedures may have a higher prevalence than those who present with healthy mouths and evidence of gingival recession. The availability of a vast array of treatments, however, would indicate either that there is no one effective desensitizing agent for completely resolving the discomfort or that the condition, due to its highly subjective nature, is difficult to treat irrespective of the available treatment options. The importance of implementing preventative strategies in identifying and eliminating predisposing factors in particularly erosive factors (e.g. dietary acids) cannot be ignored if the practitioner is going to treat this troublesome clinical condition successfully. This paper will review the published literature and provide information as to the prevalence of the condition, its etiology and causal factors, as well as recommendations for the clinical management of the problem.

Definition

According to Addy et al. (1), dentine hypersensitivity (DH) is characterized by 'pain derived from exposed dentine in response to chemical, thermal, tactile or osmotic stimuli which cannot be explained as arising from any other dental defect or pathology.' A recent modification to this definition has been made to replace the term 'pathology' with the word 'disease' (2) presumably with a view to avoid any confusion with other conditions such as atypical odontalgia. Traditionally, the term dentine hypersensitivity was used to describe this distinct clinical condition; however, several authors have also used the terms cervical dentine sensitivity (CDS) or cervical dentine hypersensitivity (CDH) or dentine sensitivity (DS), and root dentine sensitivity (RDS)/root dentine hypersensitivity (RDH) (1, 3–8). While accepting there may be justification for some of these terms to describe the condition, Addy (3) advocates the retention of the term dentine hypersensitivity for traditional reasons. Addy (3) also believes that there may be some justification in distinguishing between those individuals complaining of DH who have relatively healthy mouths from those who complain of DH as a result of periodontal disease and/or its treatment. Recently, the term root (dentine) sensitivity (RS/RDS) or root dentine hypersensitivity (RDH) has been used (6–8) to describe sensitivity arising from periodontal disease and its treatment. The rationale is that sensitivity following periodontal therapy may be a distinct condition from that of DH occurring after hydrodynamic stimulation (6–8). However, if the pain from RDS is provoked by hydrodynamic stimuli, then one could argue that DH and RDS are essentially the same condition. Addy (9) also posed a question as to whether DH is a tooth wear phenomenon with toothbrushing and toothwear (dental tribology[1]) as etiological factors in the localization and initiation of DH. As a result, he recommended

[1]Tribology. The study of wear that investigates the relationship between lubrication, friction, and wear.

that these factors should be taken into account when formulating a management strategy for the treatment of DH.

Prevalence studies (evidence from the available literature)

Clinical examinations (Table 1) and patient or consumer questionnaires (Table 2) indicate the prevalence of DH to be 4–69% (2, 8, 10–33). Females appear to suffer more than males presumably due to their overall health care and better oral hygiene awareness (8). The prevalence of the condition appears to peak at the end of the third decade and the beginning of the fourth (1). There are very few published studies on the prevalence or incidence of RDS or RS following periodontal therapy (Table 3) (34–38), although the study by Tammaro et al. (36) is one report that deals specifically with the incidence of RDS following periodontal treatment. Generally, those individuals complaining of DH as a result of periodontal disease and/or its treatment have provided higher prevalence values in the region of 60–98% depending on the type of assessment undertaken (5, 30, 34–38). Several investigators have also investigated the association of non-carious cervical lesions (NCCL) and abfraction with DH/RDS (25, 39). For example, according to Aw et al. (25), the prevalence of NCCL may range from 5% to 85% depending on the population investigated. Most of the NCCL examined in this study exhibited some degree of dentine sclerosis and were associated with no or mild sensitivity (inverse relationship). A 17-year retrospective study observed by Coleman et al. (39) also observed a positive association between cervical DH and abfraction lesions. These investigators found that 57% of molars and 31% of premolars exhibited DH on the buccal aspects of teeth and relatively few sites on the lingual surfaces (6% molars, 2% premolars).

Periodontal therapy may also be a contributory factor of DH (5, 15, 31, 34), although to what extent is difficult to ascertain. For example, Taani & Awartani (23) reported that the prevalence of DH was higher in the periodontal speciality (60.3%) than the general dental clinic (42.4%), Kontturri-Narhi (34) also observed that there was a difference between reported symptoms to various stimuli between those who previously had periodontal surgery (76.5%) to those who had undergone conventional periodontal therapy (63.5%). 35.7% (PS) and 24.5% (NS) group who had reported their symptoms to their dental practitioner claimed that they had been treated. Only 23.2% (PS) and 15.9% (NS) of these patients suffering from RDS claimed to have used a desensitizing agent for DH/RDS and would appear to suggest that the condition was not a major problem for them. This observation appears to be supported in the published literature (30–33). Several investigators have also suggested that patients are more at risk of having RDS if they have had periodontal surgery in the last 6 months (5, 30, 34).

Dental practitioners' perceptions

There have also been a number of recent studies or reviews that have indicated that dentists are uncertain about the condition and its effective management (2, 40, 41). Indeed, the evidence from the Schuurs et al. (40) and Gillam et al. (41) studies (Table 4) suggested that a majority of patients do not seek desensitizing treatment because they do not perceive DH as a severe oral health problem. According to Addy (8), of those complaining of DH only 48% actually complained to their dentist and only half of these individuals had any treatment for the condition recommended by the dentist. Schuurs et al. (40) also reported that dentists believe that DH presents a severe problem for only 1% of their diagnosed patients. From these studies, there appears to be a lack of awareness among dental professionals of the importance of implementing prevention strategies to eliminate the etiological causes of DH/RDS. It is also important to note that the number of patients who perceive DH/RDS to cause serious pain may still present a significant clinical challenge for the dental practitioner.

Etiology

The etiology of the condition (DH/RDS) is multifactorial and not completely understood, although it has been demonstrated (scanning and transmission electron microscopy) by several investigators that the structure of dentine in the affected (sensitive) areas of a tooth is altered, containing a larger number of patent dentine tubules with a wider tubular diameter than unaffected areas (non-sensitive) (42–44). These observations would appear to be consistent with Brännström's hydrodynamic theory (45), which suggests that DH is due to hydrodynamic fluid shifts occurring across exposed dentine with open tubules and that in

Table 1. Summary of prevalence studies on dentine hypersensitivity (Clinical)

Authors	Country	Setting	Study type	*N*	Prevalence (%)
Abel (10)	USA				c25
Jensen (11)	USA	University	Clinical	3000	30
Graf & Galasse (12)	Switzerland	Practice	Clinical	351	15
Flynn et al. (13)	Scotland	University	Clinical	369	18
Orchardson & Collins (14)*	Scotland	University	Clinical	109	74
Fischer et al. (15)	Brazil	University	Clinical	635	17
Lussi et al (16)	Switzerland	Community	Clinical	391	34.8
Chen et al. (**)	USA	University	Clinical	184	50
Chabanski et al. (5)	UK	University	Clinical	51	73
Duncan et al. (17)	USA	Practice	Clinical	764	53
Liu et al. (18)	Taiwan	University	Clinical	780	32
Verzak et al. (19)	Yugoslavia	University	Clinical	40	52.5 (32.5% questionnaire)
Rees (20)	UK	Practice	Clinical	3593	3.8
Al-Wahadni & Linden (21)	Jordan	Practice	Clinical	126	Case control study
Taani & Awartani (22)	Saudi Arabia	University	Clinical	302	52.6
Taani & Awartani (23)	Saudi Arabia	Practice	Clinical	144	42.4
Taani & Awartani (23)	Saudi Arabia	University	Clinical	151	60.3
Rees & Addy (24)	UK	Practice	Clinical	4841	4.1
Aw et al. (25)	USA	University	Clinical	57	171 teeth 73% (125 none or mild 0–3), 14% (24 moderate 4–6), 13% (22 extreme 7–10) VAS
Gillam et al. (26)*	UK	University	Clinical	117	49.8% of teeth evaluated
Rees et al. (27)	Hong Kong	University	Clinical	226	67.7

*Non-prevalence studies.
**as cited in Rees (20).
Acknowledgement: adapted from Rees (20) and Orchardson 2005 (unpublished data of 21 studies conducted between 1958 and 2003).
VAS, visual analogue scales.

turn mechanically activates the nerves situated at the inner ends of the dentine tubules or in the outer layers of the pulp.[2] According to Dababneh et al. (46), the pattern of the development of a 'hypersensitive' lesion may be a result of two processes: lesion localization and lesion initiation. This suggestion does offer the possibility of discriminating between traditional DH and RDS if the processes involved are different. Further investigation, however, is required to ascertain whether

[2]It should be noted from a clinical perspective that not all exposed dentine is necessarily sensitive.

Table 2. Summary of prevalence studies on dentine hypersensitivity (questionnaire)

Authors	Country	Setting	Study type	*N*	Prevalence (%)
Murray & Roberts (28)	Indonesia	Market Research	Questionnaire	1000	27
Murray & Roberts (28)	USA	Market Research	Questionnaire	1000	18
Murray & Roberts (28)	Japan	Market Research	Questionnaire	1000	16
Murray & Roberts (28)	France	Market Research	Questionnaire	1000	14 (Winter) 9 (Spring)
Murray & Roberts (28)	Germany	Market Research	Questionnaire	1000	13
Murray & Roberts (28)	Australia	Market Research	Questionnaire	1000	13
Irwin & McCusker (29)	UK	Practice	Questionnaire	250	57
Chabanski et al. (30)	UK	University	Questionnaire	507	84
Gillam et al. (31)	UK	Practice	Questionnaire	277	52
Gillam et al. (32)	UK/Korea	Practice	Questionnaire	557	52 (UK) 55.4 (Korean)
Claydon et al. (33)	UK	Practice	Questionnaire	228	50
The Chapman Group Ltd (2)	Canada	National sample	Questionnaire	683	30
Research Quorum (8)	North America	Consumer survey	Questionnaire	11 000	37
Research Quorum (8)	Europe	Consumer survey	Questionnaire		45
Research Quorum (8)	Others	Consumer survey	Questionnaire		52 Overall mean estimate 36

Acknowledgement: adapted from Rees (20) and Orchardson 2005 (unpublished data of 21 studies conducted between 1958 and 2003).

Table 3. Summary of prevalence studies on dentine hypersensitivity: dental practitioner awareness (questionnaire)

Authors	Country	Setting	Study type	*N*	Prevalence (%)
Schuur et al. (40)	The Netherlands	Practice	Questionnaire (postal)	259	9.8
Gillam et al. (41)	UK	Practice	Questionnaire (postal)	181	25
Canadian Advisory Board on DH (2)	Canada	Practice-based dentists/hygienists	Questionnaire (postal)	542	Prevalence was underestimated by respondents

this would be a valid distinction between the two conditions or simply a developmental pattern common to both with similar etiological factors. A number of reviews (47–50, 52) over the last 20 years have provided a degree of information that may be helpful to the dental practitioner. These reviews have indicated that exposure of the dentine may be a result of one of the following processes:

(1) anatomical characteristics in the region of cementum–enamel junction (CEJ),
(2) removal of the enamel covering the crown of the tooth, and

Table 4. Prevalence of dentine hypersensitivity/root dentine sensitivity following non-surgical and/or surgical periodontal treatment (selected studies)

Authors	Country	Setting	Study type	*N*	Prevalence/Incidence (%)
Kontturi-Närhi (34)	Finland	University	Questionnaire	388	76.5 (Periodontal surgery) 63.5% (conventional periodontal treatment)
Kontturi-Närhi (35)	Finland	University Dental Clinic	Development of clinical methods of assessment and evaluation of DH	23	352 teeth. 68% responded to thermal or evaporated stimuli
Fischer et al. (35)	Brazil	Urban Clinical Practice	Test group: supra and subgingival scaling. No control group	13 (11 completed study)	9% (1/11) of patients sensitive at baseline. 55% (6/11) of patients sensitive at 1 week after therapy
Tammaro et al. (36)	Sweden	Periodontal Department University Hospital	Clinical, split-mouth design and a 4-week follow-up. Included OHI, scaling, and root planning with a control group (OHI only)	49 (35 completed study)	23% (8/35) of patients sensitive at baseline. 54% (19/35) of patients sensitive at 1 week after therapy. Severity of DH increased after scaling but reduced in intensity over the 4-weeks
Tonetti et al. (37)	Europe	Multicentered European Study 12 centers in 7 Countries	Test Group with advanced chronic periodontitis with at least 1 infra bony defect $\geq$ 3 mm. Papillary preservation flap procedure with enamel matrix derivatives (EMD). Control. Flap procedures as in Test group but no EMD applied	172	No significant difference between test and control were observed. Reported sensitivity peaked at 3 weeks 45% and 35% respectively for test and control. Diminishing to below baseline values at week 6
Vaitkevičienė et al. (38)	Lithuania	Department of Dental and Oral Diseases, University Hospital	Flap surgery was performed involving 641 teeth. Patients divided into test (light-cured resin-based sealer) and placebo (water) groups	67 (62 completed study)	Significant differences noted after 30 days between test and placebo as measured (VAS) 69.9% (test) and 31.1% (placebo) of teeth responded to the test stimulus (air for 2 s). The perceived discomfort was relatively mild

DH, dental hypersensitivity; VAS, visual analogue scales.

(3) denudation of the root surface due to loss of cementum and overlying periodontal tissues.

Denudation of the root surface can be due to gingival recession increasing with age (51), chronic periodontal disease (52), periodontal surgery, and chronic trauma from patient's habits (53). Recession may also dictate the localization of lesions and sensitivity is initiated only when erosive factors expose the dentine tubule openings. Erosion occurs due to an excessively acidic environment. Sources of acid may be occupational, medicinal, due to illness (bulimia, gastric regurgitation), acidic diet (e.g. carbonated drinks, fruit), and mouthwashes that can act alone or in combination. DH/RDS may also be due to iatrogenic damage for example inadequate cervical coverage by a crown (54). Occlusal abnormalities may also lead to attrition, vigorous toothbrushing with an abrasive toothpaste may cause abrasion, and dietary acids can result in erosion. Abrasion may also have the potential to enhance tooth wear by toothbrushing following prior exposure to acidic food drink or low pH mouthrinses, which can be explained by a softening process that parallels the actual bulk loss of hard tissue (9). According to Addy (9), evidence from both *in vitro* and *in situ/ex vivo* studies indicates that abrasion and erosion may act either in an additive or a synergistic manner in the tooth wear process. More recently, the term abfraction has been added to the list of factors and it has been postulated that as the cervical fulcrum area of a tooth may be subjected to unique stress, torque and moments resulting from occlusal function, bruxing, and parafunctional activity (55), these flexural forces can act to disrupt the normal ordered crystalline structure of the thin enamel and underlying dentine by cyclic fatigue leading to cracks, chips, and ruptures. Stress corrosion and piezoelectric effects have also been theorized to have an effect (56). All these mechanisms, alone or in combination, may induce removal of the enamel (57). These etiological and predisposing factors are summarized in Table 5.

Table 5. Etiological and predisposing factors associated with dentine hypersensitivity/root dentine sensitivity

Etiological and predisposing factors
Loss of enamel
Denudation of cementum
Gingival recession
Attrition
Abrasion
Abfraction
Erosion (intrinsic and extrinsic)
Tooth malposition
Thinning, fenestration, absent buccal alveolar bone plate
Periodontal disease and its treatment
Periodontal surgery
Patient habits

Acknowledgement: adapted from Chabanski & Gillam (52).

Clinical features

The pain arising from DH/RDS is extremely variable in character, ranging in intensity from mild discomfort to extreme severity. The degree of pain varies in different teeth and in different persons. It is related to the patient's pain tolerance as well as to emotional and physical factors. It may emanate from one tooth or several teeth and it is sometimes felt in all quadrants of the jaws (10). Most patients describe the pain arising from DH as being rapid in onset, sharp in character, and of short duration (58–61). Patients also report a wide variety of pain-producing conditions and a large combination of stimuli has been recorded in the literature (13, 15, 30–34, 58, 61). The external stimuli eliciting dentinal pain can be thermal, osmotic, chemical, physical, or mechanical in nature. The thermal stimuli include hot and cold food and beverages and warm or cold blasts of air entering the oral cavity. Osmotic stimuli include sweet food and beverages. Acid stimuli include grapefruit, lemon, acid beverages, and medicines. Common mechanical stimuli are toothbrushes, eating utensils, and dental instruments (10, 57). The use of cold air blasts from a dental air syringe, cold water, and suction from a dental aspirator tip (physical) may also cause discomfort (34).

The clinical features of DH/RDS have been well documented in a number of reviews (14, 47–50). These reviews have primarily dealt with DH rather than

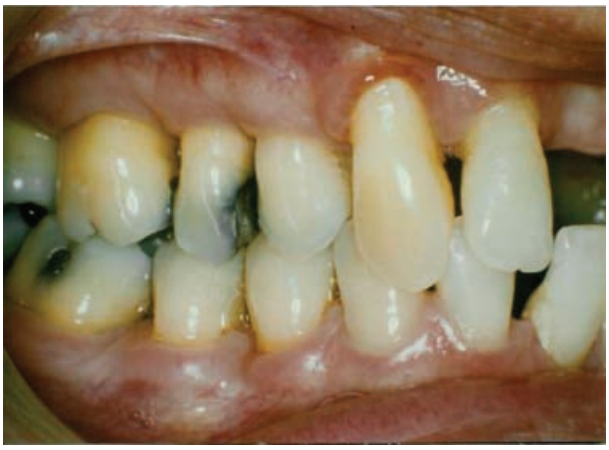

Fig. 1. Patient aged approximately 45 years old with a previous history of periodontal therapy including surgery with evidence of buccal recession particularly on the upper canine tooth but also affecting the other teeth in the posterior quadrant.

RDS *per se*; however, it is likely that some of the predisposing factors and clinical features will be the same (Fig. 1). The study of intra-oral distribution of DH by Fischer et al. (15) demonstrated that incisors and premolars (buccal surfaces) were the teeth most commonly sensitive to air and probe stimuli. Orchardson & Collins (14) (a non-prevalence study) observed that premolars (37.8%) were the first most sensitive, followed by incisors (25.9%) and canines (24.6%). Graf & Galasse (12) also recorded that the first premolars were the most sensitive. A recent study by Gillam et al. (26) reported that of those teeth responding to the stimuli used to evaluate DH, 477 (30.6%) were premolars, 437 (28%) incisors, 415 (26.8%) molars, and 232 (14.9%) canines. These results were similar to those reported by Chabanski et al. (5) and Coleman et al. (39), although the latter study failed to detect any anterior teeth with DH/RDS. These studies are of interest in that a higher prevalence of molar teeth with DH/RDS was recorded. This might reflect the population studied, for example Chabanski et al. (5) reported prevalence in referred periodontal patients, whereas Gillam et al. (26) reported from a generalized patient base, albeit with a history of routine dental treatment including scaling procedures. A further point worth noting from studies examining both lingual and buccal surfaces (5, 39) is the minimal response to the test stimuli on the lingual surface and this would tend to support Addy's assertion that DH is primarily a condition in clean mouths (3). One of the problems, however, in interpreting the results from these studies is the wide variation in the occurrence of DH/RDS in different tooth types. This may be due to a number of factors including the populations assessed and the methodology used to evaluate DH/RDS in these populations (52, 61).

Differential diagnosis

One of the difficulties facing the dental practitioner when confronted with a patient complaining of tooth pain is that there are a number of clinical conditions that may elicit the same clinical symptoms as DH and they have to be eliminated before a correct diagnosis of DH is made. The importance of the definition as suggested by Addy (1, 3, 8) and from the Canadian consensus document (2) is that it provides a very useful clinical description of the condition and suggests the need to exclude other forms of tooth pain or sensitivity. With regard to differentiating the term DH from RS, Addy (8) has suggested that as RS (RDS) may have a different etiology associated with bacterial penetration of the dentinal tubules in the root (62), as it does not fit the traditional definition of DH. The term 'root sensitivity' has also been adopted by the European Federation of Periodontology to describe the sensitivity of teeth associated with periodontal disease before and after non-surgical and surgical procedures (6, 8). The relationship between bacterial penetration, pulpal inflammatory changes, and symptoms arising from DH/RDS, however, remains unclear (34). For example, several investigators have shown that bacteria from plaque metabolites overlying exposed dentine have been able to penetrate the open dentinal tubules and elicit an inflammatory response in the pulp (63–65) that may in turn increase the responsiveness of the sensory pulpal nerves and subsequent development of hypersensitivity or hyperalgesia (34). There is evidence, however, to suggest that pain may occur independently of the state of the pulp (66). Seltzer et al. (67) also failed to find any correlation between clinical pathology and the histological state of the pulp, although from an inflammatory perspective it should be noted that leukocytes do not stimulate nerve fibers directly but via the release of inflammatory mediators such as substance P, bradykinin, etc. It should, however, be noted that some of the earlier studies were based on inflammatory changes following experimental cavity

preparation and caution is needed in extrapolating these results in coronal dentine to cervical dentine and symptoms associated with DH/RDS (68). Furthermore, it has been suggested that the presence of bacteria in itself does not necessarily indicate that a mechanism of stimuli transmission other than Brännström's hydrodynamic theory is responsible (34, 69). There is also the possibility of direct activation of pulp nerves and bypassing the hydrodynamic system (70).

There are also a number of clinical conditions that may provide clinical features similar to that of DH or RDS and it is important to distinguish between these in order to provide a correct diagnosis and successful management of the problem (Table 6). One condition, however, may be more problematic than some of the conditions, namely atypical odontalgia, which may be a variant of atypical facial pain and has been defined as pain and hypersensitive teeth in the absence of detectable pathology. This type of pain is typically indistinguishable from pulpitis or periodontitis but is aggravated by dental intervention (71).[3] A good history of the problem presented by the patient is essential and questioning by the clinician may help elicit the relevant information in order to treat the condition. According to Scully & Felix (71), when dealing with the problem of orofacial pain, it is essential to determine key points about the pain such as location, character, duration, frequency and periodicity, precipitating, aggravating and relieving factors, and any associated features. It should be noted that the major symptom of DH/RDS is pain characterized by rapid onset, sharpness, and short duration. Occasionally, pain arising from DH/RDS may persist as a dull or vague sensation in the affected tooth after removal of the stimulus (72). Traditionally, dental practitioners have used a dental explorer probe and air from a triple air syringe to identify any sensitive areas on the exposed root surface in order to elicit a response from the patient. A simple measure to quantify the response would be the use of a rating score such as a visual analogue scales (VAS; 0–10) and this would give the clinician an indication of how the patient rates his/her own pain. Other means of testing are also available (Table 7) but some of these may be more relevant in testing for pulp vitality than for DH/RDS.

[3]For further information on the treatment of Orofacial pain, please refer to the article by Scully and Felix (71).

Table 6. Differential diagnosis of dental pain that may conflict with an accurate diagnosis of dentine hypersensitivity/root dentine hypersensitivity

Etiological and predisposing factors
Cracked tooth syndrome
Fractured restorations
Fractured teeth
Dental caries
Post-operative sensitivity
Acute hyperfunction of teeth
Atypical facial odontalgia
Palatal-gingival groove
Hypoplastic enamel
Congenitally open cementum–enamel junction
Improperly insulated metallic restorations

Assessment of DH/RDS in clinical trials and application in the clinical situation

According to Gillam et al. (73), DH has been mainly subjectively evaluated on the basis of the individual patient's subjective response to the presenting stimulus for example, in the form of verbal rating and VAS and questionnaires. Recent recommendations by Holland et al. (74) suggest that DH may be evaluated either in terms of the stimulus intensity required to evoke pain (stimulus-based assessment), or as the subjective evaluation of the pain produced by a stimulus (response-based assessment). Stimulus-based methods usually involve the measurement of a pain threshold; response-based methods involve the estimation of pain severity. The presenting stimuli can be grouped into five main categories: mechanical, chemical, electrical, evaporative, and thermal (Table 7). It should be pointed out, however, that tactile testing may not be suitable for assessment of resin-based materials and consequently this method of assessment may be substituted for a second thermal or evaporative stimulus. The use of an explorer probe and an air blast

Table 7. Stimuli used to assess dentine hypersensitivity/root dentine sensitivity in the clinical setting

Mechanical (tactile) stimuli
Explorer probe
Constant pressure probe (Yeaple)
Mechanical pressure stimulators
Scaling procedures
Single-tufted brush
Chemical (osmotic) stimuli
Hypertonic solutions, for example, sodium chloride, glucose, sucrose, and calcium chloride
Electrical Stimulation
Electrical pulp testers
Dental pulp stethoscope
Evaporative stimuli
Cold air blast from a dental air syringe
Yeh air thermal system
Air jet stimulator
Temptronic device (microprocessor temperature-controlled air delivery system)
Thermal stimuli
Electronic threshold measurement device
Cold water testing
Heat
Thermo-electric devices (e.g. Biomat Thermal Probe, Eastman Dental Institute, London, UK)
Ethyl chloride
Ice-stick

NB: Hydrostatic Pressure evaluation has also been reported in the literature, but may be considered impractical for use in clinical studies.
Acknowledgement Gillam et al. (73).

from a dental air syringe would be an appropriate and relatively inexpensive method of evaluating DH/RDS in the dental practice. This examination may take up to 5–10 min but would prove invaluable in obtaining information on the extent of the problem and would therefore give an indication as to whether the condition is localized (to one or two teeth) or generalized (affecting part or all the standing teeth). By having this type of information, the clinician would be able to provide the relevant management of the condition. Furthermore, the practitioner should assess the 'global' severity of the pain experienced by the patient with a simple question 'on a scale of 0–10 [0 = no pain, 10 = extreme pain] how would you rate your pain today?' This helps to establish the severity of the condition to normal, everyday stimuli.

Postoperative hypersensitivity arising from dental treatment

It is important for the practitioner to discriminate between the various sources of dental pain arising from treatment procedures as well as other potentially conflicting conditions. In the context of the present paper, postoperative sensitivity from periodontal therapy will be addressed; however, it is worthwhile for the practitioner to consider that the effects of restorative and bleaching procedures may also impact on the patient's well-being in addition to creating difficulties for the practitioner in managing the problem of DH/RDS.

Periodontal procedures

Role of oral hygiene

The relationship between the effectiveness of plaque control and the etiology of DH/RDS is controversial (3, 72, 75, 76). Several investigators have identified the paucity of information regarding the prevalence, incidence, and severity of RDS as distinct from DH (7, 36). There have been a number of clinical studies (76–78) that have provided some information of the various clinical variables in the development of DH although as both Troll et al. (7) and Tammaro et al. (36) have indicated that the information may be inconclusive due to a number of features, study design, insufficient teeth, methodology, etc. There is certainly disagreement between investigators regarding the importance of plaque control in the development of the condition (3, 72, 75, 76) that has lead some investigators to suggest that there may be two distinct etiologies. For example, Addy et al. (79) have indicated

that DH is not initiated by poor plaque control but rather as a result of meticulous and perhaps overzealous oral hygiene procedures[4] whereas a number of other investigators (72, 75, 76) have previously suggested that poor plaque control leads to plaque accumulation and subsequent DH, which in turn would prevent patients from cleaning their teeth. Results from a number of questionnaire studies, however, do not appear to suggest that patients stop brushing their teeth if they experience discomfort from DH/RDS (5, 30–33). However, what is not in doubt is the importance of the patient maintaining good oral hygiene in a closely monitored maintenance program for a successful outcome of periodontal surgery. Indeed, there is evidence to suggest that plaque is not a major etiological factor as DH/RDS can occur post-surgery irrespective of any instituted oral hygiene standards (80). These investigators failed to demonstrate any statistical differences in pain values following surgery when patients used either a chlorhexidine mouthwash or saline control.

The rationale for the successful treatment of periodontal disease(s) has been referred to by Tammaro et al. (36) and can be accomplished through good oral hygiene measures by the patient and by professionally performed non-surgical mechanical debridement and by surgical procedures such as flap procedures. However, as these authors point out, these procedures may have unwanted side-effects including gingival recession, exposure of the underlying dentine following root cementum denudation with the risk of experiencing DH/RDS to tactile and thermal stimuli as well as (in the anterior region) esthetic problems. It should, however, be mentioned that root dentine may also become non-sensitive as a result of a number of factors; scaling itself may create a smear layer that could be supplemented by the natural mineralization processes in the mouth. Natural occlusion within the dentine tubules has been demonstrated in an *in situ* model using partial dentures (81, 82). An acidic environment encouraged by acidic food and drinks has the ability to dissolve the newly created smear layer (83) and this may be one reason why DH/RDS is cyclic in nature.

Several published studies have reported that RDS is a common occurrence following periodontal surgery and root scaling/debridement although with well-controlled oral hygiene procedures, DH appears to resolve over time (36). Troll et al. (7), in a systematic review of the topic, however, were only able to quote a small number of studies that fulfilled the entry criteria to be included in the review (although a number of recent studies have been included in this current review; (Table 4)). According to these various studies, prevalence rates for RDS of 9–27% before and 54–55% after periodontal therapy were observed and the reported intensity from RDS increased 1–4 weeks following therapy, after which it decreased back toward baseline values (34–38) (Table 4). A number of studies have also indicated that a small number of patients (1.3–7%) complain of severe RDS following treatment of infrabony defects with an enamel matrix derivative (84–86). Tammaro et al. (7) also reported that there was a significant difference in the levels of pain between the quadrants where only oral hygiene instruction was performed and scaling/root planning after 2–3 weeks. It would appear from these published studies that the intensity of RDS decreases 1–4 weeks after therapy (78, 87), although other studies indicate that RDS may last up to 2 months and 5 years (35, 88). Fardal et al. (89) reported that very low levels of discomfort were associated with both non-surgical and surgical periodontal treatment and that in comparison with other forms of dental discomfort, e.g., previous experience of conventional dental treatment (crowns/restorations), their perception of periodontal therapy was associated with less discomfort. Patients were able to distinguish between postoperative discomfort and postoperative sensitivity. From these observations, it would appear that any postoperative sensitivity was mild in nature and for the majority of patients lasted no more than a few days, which is in keeping with other studies (30–33, 68). The precise level of discomfort from these types of non-surgical and surgical procedures, however, is difficult to assess as a number of studies report either benefits in terms of improvement from the type of surgical procedure or implant material used. The reasons for the differences between the various published studies may be due to several factors such as the effect of time taken in root planing and handling of the soft tissues (86). Al-Hamdan et al. (90), in a meta-analysis review that included 40 papers for analysis, acknowledged that the indications for

[4]There is anecdotal evidence, however, that patients who are super efficient in their plaque control following periodontal treatment rarely suffer from DH/RDS Gunnar Bergenholtz (personal communication).

initiating root coverage procedures included DH/RDS[5], although no details were provided in the review to determine the prevalence or extent of the problem. Pagliaro et al. (91) also conducted a critical review of the effectiveness of root coverage procedures and suggested that while DH/RDS was considered one of the main indications for surgical root coverage, there were limited data available. For example out of the 90 accepted papers, DH/RDS was generally identified as being either present or absent (19 [21.1%]) and only nine articles (10%) recorded any pre- and post-treatment data; only two of these studies appeared to quantify DH/RDS on a 10-point scale.

Management of DH/RDS

Management of DH/RDS should be based on a correct diagnosis of the condition by the practitioner, who should be aware of other clinical conditions that are similar in their presenting features (1, 47) as well as the severity of the condition (localized/generalized). Irrespective of the cause of DH/RDS, it is important that the relevant advice is provided in order to prevent/minimize further damage to the exposed root surface. This may involve counselling patients with regard to their intake (especially frequency) of acidic fruits and beverages with low pH, particularly in relation to when the teeth are brushed (before/after meals) as well as information on correct brushing procedures (and type [texture] of brush). There have been a number of reviews over the last 10 years on the management of DH/RDS and these reviews may also provide sufficient information on the efficacy of the products used in the management of the condition (57, 92–97). A number of clinical algorithms have been published (2, 95, 98) to aid the busy practitioner in the management of DH/RDS and encourage the user to adopt an active rather than a passive role in monitoring the condition (see Fig. 2). Currently, there are two main approaches for the treatment of DH/RDS, namely, (a) tubule occlusion, (b) blocking nerve activity through direct ionic diffusion (increased potassium ions' concentration acting on the pulpal sensory nerve activity) (93). Historically, desensitizing agents have been classified, according to their mode of action (99), whether they are applied over-the-counter (OTC) or In-Office (P), or on their chemical or physical properties (100) or more recently as to whether they are reversible and non-reversible in nature (101). Generally speaking, these products may be in the form of dentifrices or gels and mouth rinses or in the form of topically applied agents such as resins, varnishes, primers, dentine bonding agents as well as periodontal grafting procedures and laser application. According to Pashley (97), In-Office restorative products are broadly defined as those treatment products that do not polymerize such as varnishes and precipitants and those agents that undergo setting or polymerization reactions such as conventional and resin-reinforced glass ionomer cements and adhesive resin primers. Other forms of treatment have also been reported in the literature such as the use of homoeopathic remedies (such as Plantago major) (102), propolis[6] (103), and the use of hypnosis (104) but information on the efficacy of these products from well-controlled clinical studies is sparse. Paine et al. (105), in a review of the literature on fluoride use in periodontal therapy, indicated that following routine scaling and root planing some dentists would advocate the use of fluoride application with a view to alleviate patient discomfort. These authors also suggested that there was evidence to support that the use of the home delivery of fluoride solutions in the form of dentifrice and mouth rinses such as potassium nitrate and strontium acetate with fluoride could benefit periodontal patients in reducing DH/RDS and in caries prevention/reduction of dentine solubility. Several investigators (106, 107) have incorporated the use of fluoride with iontophoresis, although the effectiveness of this technique in reducing DH/RDS has been questioned (61, 108). One should also note, however, that despite the widespread use of fluoride dentifrices in most western countries, there does not appear to be a drastic reduction in DH/RDS.

The advantages of using an OTC product readily available for the treatment of DH/RDS by the consumer compared with attending a practitioner for treatment include ease of access, expense, etc. One disadvantage is that OTC desensitizing products may take up to 2–4 weeks to relieve symptoms whereas in theory a practitioner-applied therapy ideally may provide immediate relief of discomfort. The availability

[5]Most periodontal studies still refer to DH rather than RDS.

[6]A mixture of resin, essential oils, and waxes mixed with bee glue as well as amino acid, minerals, ethanol, vitamin A, B complex, E, pollen, and bioflavenoid.

of an OTC desensitizing toothpaste to provide faster relief than currently available toothpastes (i.e. within 2 weeks) would, however, appear to be a significant advancement in the treatment of DH/RDS. For generalized sensitivity involving several teeth, the use of OTC toothpastes such as potassium nitrate and strontium-containing products has been shown to be clinically effective in well-controlled clinical studies and are readily available to the consumer (94, 96, 109), although a meta-analysis undertaken by Poulsen et al. (110) on a limited number of accepted studies (eight studies accepted, four used for analysis) meeting their criteria indicated that the efficacy of potassium nitrate to reduce DH is not strongly supported by the literature. Further studies, however, have been reported in the literature since 2000 that would appear to support the clinical efficacy of potassium-containing salts (111–114). It would also be appropriate for the dentist to recommend an OTC product for the patient to use for 3–4 weeks and then review the situation if the pain has not resolved sufficiently for the patient to enjoy some 'quality of life.' Subsequent treatment could be in the form of a more invasive therapy, e.g., restorations, periodontal grafts, etc. although in some situations, pulpal extirpation or extraction of the offending tooth may be the treatment of choice (115). Periodontal grafts and guided tissue regeneration (GTR) procedures have also been described in the literature for the treatment of gingival recession with RDS and are predictable procedures and might be the treatment of choice for many patients as they may provide a good esthetic as well as palliative solution to their clinical problem (92). However, as indicated earlier in this review, while there is an abundance of information in the published literature regarding these root coverage procedures, only limited evidence-based data are available on the extent of the problem of DH/RDS before and following the procedure(s). Drisko (92) also suggested that if the root coverage is not completely successful in relieving DH/RDS, then the remaining exposed cervical dentine could be treated with a more invasive restorative material. It is also imperative that practitioners should avoid placing subgingival restorations whenever possible in order to prevent plaque retention as well as maintaining the biological width when placing crowns (92) (Table 8). Several investigators have also advocated the use of a lidocaine 25 mg/g+prilocaine 25 mg/g anesthetic gel in reducing RDS following periodontal procedures (116, 117). The use of a postsurgical application of a 6.8% ferric oxalate sealant (118) or a 3% potassium oxalate topical application following subgingival scaling and root planing procedures (119) has also been reported to be effective in reducing RDS. The use of plastic inserts for scaling procedures may also reduce RDS (120). Lasers have also been recommended for treating DH/RDS; a review by Kimura et al. (121) suggested that the effectiveness of lasers ranges between 5.2% and 100% depending on the laser type

Table 8. Guidelines on management of dentine hypersensitivity/root dentine sensitivity

History and examination to establish diagnosis
Identification of cause
Treatment based on severity of problem
Incorporation of preventive measures- remove etiological and predisposing factors (Dietary and Oral hygiene advice)
Review the patient regularly for signs of attrition, abrasion, erosion, and abfraction
Give dietary advice in line with current thinking particularly in view of the potential effect of erosive materials (food and fizzy drinks) and brushing immediately after meals.
Give oral hygiene instruction and recommend an atraumatic toothbrushing technique to avoid potential damage to both hard and soft tissues
Mild generalized sensitivity-use of OTC desensitizing products (toothpastes gels, etc)
Localized moderate to severe sensitivity-use of In-office products (primers, varnishes, sealants, etc)
Avoid placing subgingival restorations that may retain plaque
Avoid violating the biological width when placing crown margins
Use of periodontal flap surgery (including GTR) in the treatment of exposed root dentin
In severe cases, pulpal extirpation and extraction may be the treatment of choice
Review on an appropriate basis and reassess if pain persists

GTR, guided tissue regeneration; OTC, over-the-counter.
Acknowledgement: adapted from Drisko C (92).

and parameters used to assess the condition. It would appear according to these authors that lasers are more effective in treating DH/RDS than other treatment modalities although in severe cases of DH/RDS, lasers are less effective and they recommended that the severity of DH/RDS should be assessed before undertaking any use of laser therapy. A 6-month study by Schwartz et al. (122) also compared the desensitizing effects of a Er:YAG (erbium-doped, yttrium, aluminum, and garnet) laser with a Dentin Protector® (Ivoclar Vivadent, Ellwangen, Germany) desensitizing system and reported that the laser treatment was significantly more effective in reducing DH/RDS over the study period than the Dentin Protector®. It may also be possible that by removing any predisposing etiological factors as well as treating the dentine with a laser could significantly reduce DH/RDS (123). Recently, a light-cured resin-based sealer was evaluated following flap surgery and was observed to reduce RDS over a 30-day period (38). Currently, the mechanism of the laser treatment in treating DH/RDS is unclear (121), although according to Pashley (97) it may be through the coagulation and precipitation of plasma proteins in dentinal fluid or through alteration of the intradental nerve activity. McCarthy et al. (124) also suggested that reduction of DH/RDS may be as a result of creating an altered surface layer on the root physically occluding the tubules (smear layer creation), although this action may be inconsistent with areas of unaffected open dentine tubules perhaps due to the restrictions of producing uniform laser treatment with a hand-held light-guide with lasers operating in a pulsing mode (97, 124). Further research is, however, required before this technique can be recognized as an acceptable treatment for this condition; indeed, several investigators (124, 125) have noted safety concerns that due to the apparent destruction of the dentine surface, lasers such as the neodymium–yttrium, aluminum garnet (Nd–YAG) at moderate or therapeutic power levels may be inappropriate for the treatment of DH/RDS. There is also a possibility particularly with the application of soft lasers that a strong placebo effect occurs when using lasers (126).

It should be noted as indicated in this review that while DH/RDS is a common occurrence following periodontal surgery and root scaling/debridement, it is generally mild in nature and with well-controlled oral hygiene procedures appears to resolve over time (36). In practical terms, however, it is important to follow-up patients who have undergone non-surgical and/or surgical procedures within a routine periodontal maintenance program and intervene at an appropriate time (Fig. 2).

The evidence from the published literature would appear to suggest that most if not all of the OTC products achieve their clinical effectiveness from the blocking of the dentine tubules by deposition of the dentifrice ingredients, e.g., silica, fluoride rather than through the blocking of the nerve activity. That does not mean that one should exclude the possibly of products containing potassium from exerting an effect through blocking nerve activity in the clinical situation but more information is required to demonstrate this effectively in humans (94). In-Office restorative materials such as resins, varnishes, and sealants would appear to act by tubular occlusion and subsequent reduction in dentine permeability (flow rate) as demonstrated by a number of *in vitro* scanning electron microscopy (SEM) and hydraulic conductance studies (127–135) (Fig. 3). Evidence is also available in the published literature that would provide support for the clinical efficacy of these products in reducing DH/RDS (136–147).

Finally, a cautionary note should be made that despite the plethora of products that are available to the practitioner claiming to be clinically effective in the treatment of DH/RDS, the evidence from the published literature would appear to indicate that no one desensitizing agent (OTC/In-Office) could be considered to be the ideal panacea in providing relief from DH/RDS. Indeed, one of the problems in evaluating the results from the published studies on the efficacy of products used in the treatment of DH/RDS particularly with the dentifrice and laser studies is the strong influence of the placebo and non-placebo (e.g. Hawthorne) effects exerted during the duration of the study (148–150). Currently, there does not appear to be a globally agreed gold standard product for comparative purposes in the clinical trial setting for the evaluation of new desensitizing agents.

Recent developments in the treatment of DH/RDS

Generally speaking, it should also be noted that over the last decade or so, toothbrush technology has also brought in improvements to the standards of safety,

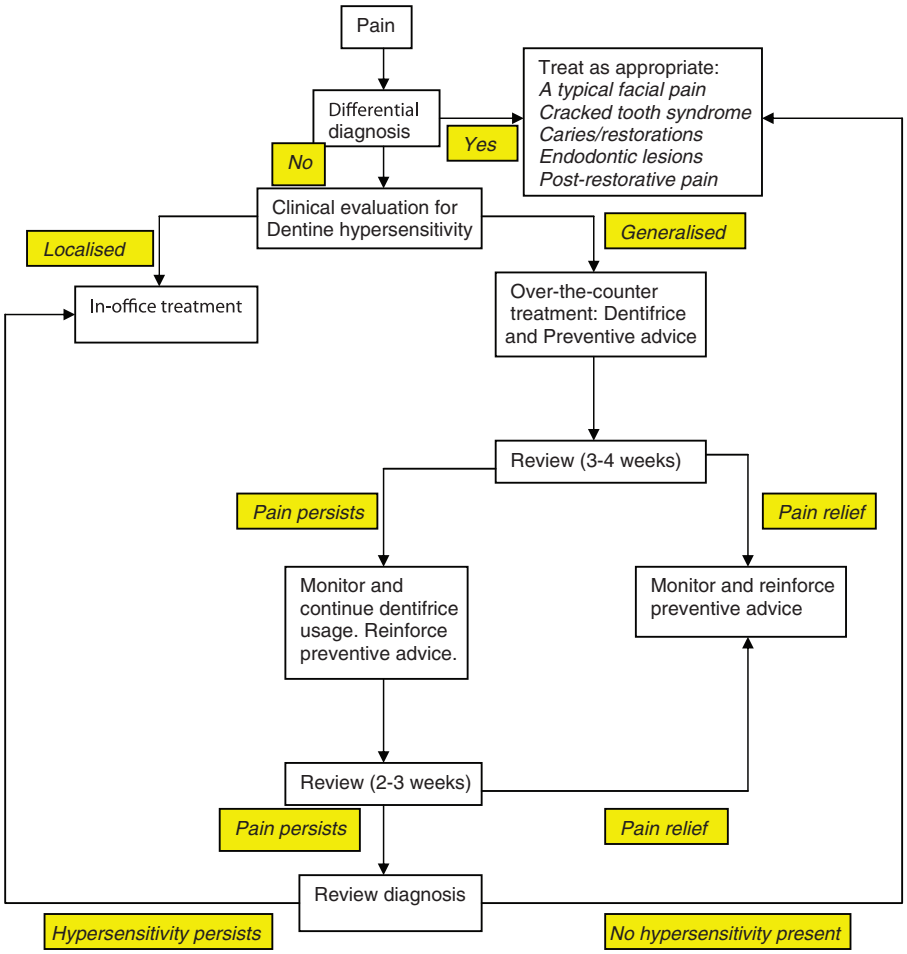

Fig. 2. Algorithm for the treatment of Dentine Hypersensitivity/Root Dentine Sensitivity by Dental Practitioners. (Reproduced from Dental Update ISSN 0305–5000, with permission from George Warman Publications, UK (99)).

design, texture, type of filament, etc. which, together with a dentifrice formulation, has both oral health and cosmetic benefits (9). Advancement in dentifrice technology has enabled the number of ingredients and compatible flavors to be included in dentifrices that not only act as a desensitizer but also claim to be anti-plaque and anti-caries. For example, historically, Sensodyne Original (GSK Consumer HealthCare, Jersey City, NJ, USA) did not have fluoride as an ingredient; this was probable due to the interaction of strontium with Fluoride. An alternative formulation (Macleans Sensitive, GSK Consumer HealthCare, Brentford, UK) incorporated fluoride using strontium acetate that, it was claimed, enabled the active ingredients to be delivered to the tooth surface without interaction between the strontium and fluoride. A dentifrice-containing potassium nitrate, in combination with fluoride, a copolymer, and anti-calculus [tartar] ingredients, has also been successfully formulated to reduce DH/RDS (151). Other products have recently used dual-tube technology to deliver the active ingredients that may interact if placed in the same tube onto the tooth surface, for example Colgate Sensitive (Colgate–Palmolive Company, Piscataway, NJ, USA) that incorporates potassium nitrate and stannous ions (111, 112). Most dentifrice products on the market now contain potassium salts (nitrate, chloride, or citrate) with fluoride and an anti-plaque ingredient such as triclosan, although there has been concern raised over possible interactions of some of these ingredients with the desensitizing activity (113). Improvements in the abrasive (e.g. artificial silica) with low Radioactive Dentin Abrasivity (RDA) values and detergent systems (anionic/non-ionic) have also occurred over the last decade or so and these are important particularly for removing plaque and stain, etc. However, it is possible that some of the anionic detergents systems such as sodium lauryl sulfate may

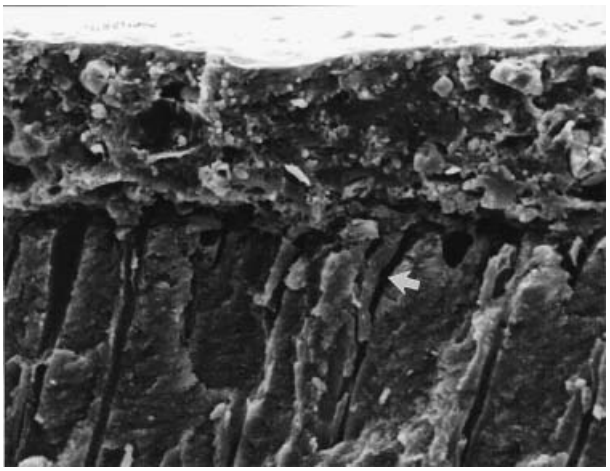

Fig. 3. Scanning electron micrograph of a fractured dentine disc treated with a bifluoride product. Tubule penetration of the product is observed occupying most of the tubule lumen (arrow) (× 1420). Acknowledgment Nicky Mordan.

affect the attachment of artificial silica to dentine possibly by ionic competition; an alternative non-detergent system such as tego betaine may be used to prevent this interaction with the artificial silica (9). The extent to which dentifrice ingredients may react with one another and possibly interfere with the delivery and potential efficacy of the active ingredient in the clinical setting still needs to be investigated.

Intra-oral fluoride releasing devices (152, 153), bio-adhesive potassium nitrate 5/10% gels (154), and application of 3% potassium oxalate or 6% ferric oxalate (118–119, 134, 155), re-mineralization toothpastes and novel silica formulations have also been developed (156). The combination of casein phosphopeptides (CPP) and amorphous calcium phosphate (ACP) Recaldent™ [CPP–ACP] (GC America Inc., Alsip IL, USA.) has been marketed and claimed to reduce DH (157). ACP has also been used in bleaching trays to reduce DH during the bleaching process and RDS (158–160). Products have also been developed from bioactive and biocompatible glasses that are known to induce osteogenesis in physiological systems and may offer suitable materials for surface reactivity that could theoretically occlude tubules (161–163). NovaMin® (calcium sodium phosphosilicate) is a new product formulation found in a variety of dental products such as Nucare Prophy Paste (Sunstar Butler, Chicago, IL, USA) and Oralief™ Therapy for Sensitive teeth (NovaMin Technology Inc., Gainsville, FL, USA) (164) and would appear to be based on Bioglass technology that was initially used in periodontal procedures (165) and a desensitizing dentifrice formulation (162, 166, 167). Other recent innovations include chewing gums containing potassium chloride (168) and mouthrinses containing potassium citrate and potassium nitrate solutions (3%) (4, 149, 169) or gels (10%) in a mouthguard (170), although the efficacy of these products has been varied. More recently, potassium nitrate has been used in bleaching trays to reduce DH/RDS during and following the bleaching process (101). Advances in restorative material technology (such as multi-bottle to single-bottle applications e.g., glutardaldehyde) (171) have also enabled the practitioner to use a vast array of dentine bonding agents, varnishes, sealants, etc. to treat DH/RDS in a very simple and efficient manner. Despite this development, some concerns have been raised over the lack of adequate clinical evaluation before their commercial availability (172), although the efficacy of some of these products used in the treatment of DH/RDS has been subsequently published (138, 139, 142, 145, 147). Several investigators have also raised concern regarding biocompatibility and possible cytotoxic effects of some of the dentine desensitizers (69, 173).

The advancement in periodontal grafting procedures with a range of new products and techniques such as bio-absorbable membranes to treat localized gingival recession with DH/RDS may also enable the skilled practitioner to treat DH/RDS successfully (92). There is also the possibility that advances in gene therapy may influence the response of sensory nerve fibers in the pulp following restorative procedures as well as in non-surgical and surgical procedures that may initiate DH/RDS (92).

Concluding remarks

Addy, in a recent review (8), suggested that DH and by implication RDS was undiagnosed and undertreated by the dental practitioner and argued that failure to consider the causation of the condition could result in recurrence and possible failure of prescribed treatment. From this statement and other information (2, 40, 41), we can assume that there may be a general lack of understanding by practitioners about the condition and its effective management despite the availability of an abundance of articles in the published literature.

Management of DH/RDS should be based on a correct diagnosis of the condition by the practitioner, who should also be aware of other clinical conditions that are similar in their presenting features (1, 47) as well as on the severity of the condition (localized/generalized). The dental practitioner should be aware of the importance of a preventative strategy, particularly with a view to the removal of any etiological factors and minimizing the effects of erosion and altering the timing of toothbrushing relative to meals and snacks, etc. (9). Furthermore, one should be aware of the importance of the patient maintaining good oral hygiene in a closely monitored maintenance program for a successful outcome of periodontal surgery. The goal of treatment of DH/RDS ideally should be the restoration of the original impermeability of the dentinal tubules and the relief of DH/RDS experienced by the patient or at least to reduce the level of discomfort to enable the patient's quality of life to be maintained. Although there is a plethora of products available to the practitioner claiming to be clinically effective in the treatment of DH/RDS, the evidence from the published literature would appear to indicate that no one desensitizing agent (OTC/In-Office) could be considered to be the ideal panacea in providing relief from DH/RDS. The practitioner should therefore use their clinical judgment in determining the appropriate agent based on the severity of the condition and monitor the patient's progress over time within the constraints of the practice environment.

One of the difficulties that the authors faced when writing this review was the lack of clarity between what a number of investigators called the condition when reporting the findings of their treatment. This was particularly true when reviewing the periodontal therapy-based papers whose authors generally used the traditional term DH rather than RDS or RS as recently adopted by the European Federation of Periodontology (6, 8). Historically, a number of the earlier efficacy studies were on patients who had a periodontal condition and experienced DH; the findings of these studies were based mainly on the results of treatment effects (109, 118), whereas today studies specifically designed for DH studies evaluating dentifrices, etc. generally exclude patients who had periodontal surgery or therapy (scaling/root debridement) within a specific timeframe (174). It should also be noted that the different prevalence rates from the various studies included for review may have included both individuals who would be categorized as having either DH or RDS (as recently suggested by Addy (8)) and it would be useful to obtain information on a longitudinal basis to determine whether DH and RDS are actually separate clinical conditions with differing etiologies or the same condition exacerbated by treatment such as periodontal therapy.

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