Dentin Hypersensitivity: Prevalence, Etiology, Pathogenesis, and Management

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Cor van Loveren, Patrick R. Schmidlin, Luc C. Martens, and Bennett T. Amaechi

Abstract

Dentin hypersensitivity is simply defined as a short sharply painful reaction of the exposed and innervated pulp-dentin complex in response to stimuli being typically thermal, evaporative, tactile, osmotic, or chemical and which reaction cannot be attributed to any dental defect or pathology. To be hypersensitive, dentin must be exposed and the exposed tubules must be open and patent to both the oral cavity and the pulp. Exposure of dentin through the loss of gingival and periodontal tissue may be caused by either too meticulous or by neglected oral hygiene. Exposure of dentin by the loss of the protecting enamel is mainly caused by erosion, abrasion, and abfraction or a combination thereof. Clinical examination for dentin hypersensitivity would include a pain provocation test by a tactile stimulus, an evaporative air stimulus, or a cold stimulus. A number of other dental conditions can give rise to pain symptoms, which may mimic those of dentin

C. van Loveren, DDS PhD (✉)
Department of Preventive Dentistry, Academic Center for Dentistry Amsterdam (ACTA), University of Amsterdam and VU University Amsterdam, Gustav Mahlerlaan 3004, Amsterdam 1081 LA, The Netherlands
e-mail: c.van.loveren@acta.nl

P.R. Schmidlin
Clinic of Preventive Dentistry, Periodontology and Cariology, Centre of Dental and Oral Medicine, University of Zurich, Plattenstrasse 11, Zurich 8032, Switzerland
e-mail: patrick.schmidlin@zzm.uzh.ch

L.C. Martens, DDS, MSc, PhD
Department Paediatric Dentistry & Special Care Dentistry, Dental School of Ghent, De Pintelaan 185 (P8), Ghent B-9000, Belgium
e-mail: Luc.Martens@UGent.be

B.T. Amaechi, BDS, MSc, PhD
Department of Comprehensive Dentistry, University of Texas Health Science Center at San Antonio, 7703 Floyd Curl Drive, San Antonio, TX 78229-3900, USA
e-mail: amaechi@uthscsa.edu

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hypersensitivity. Therefore, careful examination is necessary to exclude the conditions, which need different treatment options. When the patients do suffer from dentin hypersensitivity, there is broad range of treatment options comprising home-use and professional approaches. It is advised to start with the less invasive home-use therapies and only expand to professional in-office treatments when the home-use treatments are not effective. When decided to continue with in-office treatments, again one should start with the least invasive ones. The working mechanisms fall under two basic categories being nerve desensitization (potassium salts and guanethidine) and occlusion of exposed dental tubules (chemically: strontium, fluoride, stannous, oxalate, calcium phospho silicate, arginine calcium carbonate, nano-hydroxyapatite, and glutaraldehyde; mechanically: pumice paste, glassionomers, dentin bondings, and resins; laser therapy). Regenerative mucogingival therapy also remains an alternative, where hard and soft tissue conditions allow.

15.1 Prevalence

Dentin hypersensitivity (DHS) is simply defined as a short sharply painful reaction of the exposed and innervated pulp-dentin complex in response to stimuli being typically thermal, evaporative, tactile, osmotic, or chemical. An important part of the definition is that the reaction cannot be attributed to any dental defect or pathology [1].

Studies report a wide range of prevalence rates varying form 3 up to 98 %, which can be explained in part by different evaluation methods and different patient populations, but, generally, patients display higher degrees immediately after periodontal treatment [2, 3]. In a large European study in which over 3000 18–35 years old patients from general dental practices in France, Spain, Italy, United Kingdom, Finland, Latvia, and Estonia were enrolled, the self-reported prevalence of dentin hypersensitivity was approximately 27 %, while 42 % of the patients reported pain upon cold air stimulation of exposed dentin surfaces [4]. There was a significant heterogeneity regarding this prevalence data between the countries. The differences in prevalence between self-reported sensitivity and clinical elicited sensitivity may reflect that patients develop coping strategies to avoid incitement of the pain.

The exposure of dentin of the root or crown is essential. For root dentin exposure, it is important to acknowledge that mainly localized attachment loss due to anatomically predisposing factors or periodontal disease is probably the most widespread and relevant factor leading to root surface denudation and subsequent dentin hypersensitivity. Several predisposing factors of gingival recessions have been identified, e.g., dehiscency or fenestration of the alveolar bone and soft tissue morphotypes, but triggering pathological, therapeutic, or iatrogenic factors are also crucial for its development [5, 6]. At the (mainly cervical aspects of) crowns, loss of the protecting enamel is considered an alternative pathway of dentin exposure and is mainly caused by erosion, abrasion, and abfraction or a combination thereof [5]. These

processes may also increase the patency of tubules depending on the specific etiologic factor. West and coworkers [4] found significant associations between the elicited dentin hypersensitivity and erosive tooth wear. This study showed a significant association between fresh fruit, isotonic/energy drinks, but less clearly fruit/vegetable juices with the increased dentin hypersensitivity. A significant association was found for dentin hypersensitivity in patients reporting frequent heartburn and gastric reflux and to a lesser extent frequent vomiting. These associations are consistent with increased erosive tooth wear and the impact on dentin hypersensitivity response by removing the dentin smear layer and opening tubules [6].

This clear implication of a topically acidic environment explains why dentin hypersensitivity is discussed in a book on tooth wear.

15.2 Etiology and Mechanisms

The currently accepted hypothesis is the hydrodynamic theory, first suggested by Gysi [7] and later substantiated by Brännström [8] (Fig. 15.1). Dentin hypersensitivity is caused by movement of the dentinal tubule contents which may exert a shear force and a so called "streaming potential" exciting intradental A-type nerve fibers causing a sharp, shooting pain [9]. An increased outward movement may be more painful than an inward flow of the tubule fluid. This explains that cold, which causes an outward stream, generally triggers more dramatic pain than heat, which causes a fluid retreat [10]. The characteristic sharp pain experienced with dentin hypersensitivity can persist as a dull, throbbing ache for variable periods of time. The nerves causing this pain are not excited by the hydrodynamic mechanism. Hypersensitivity may sometimes persist despite of blocking the tubules, which also indicates that some other mechanisms may operate in the nerve activation instead of, or in addition to the hydrodynamic one. Inflammation may sensitize the nerve endings to such an extent that smaller fluid shifts would be sufficient for nerve activation or, for example, thermal stimulation may activate the nerves by a direct effect

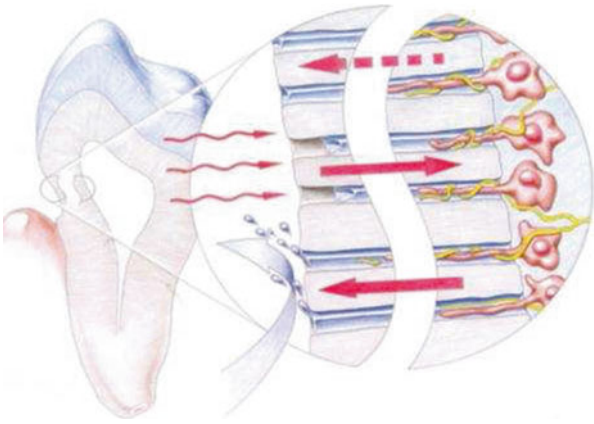

Fig. 15.1 Schematic drawing of the hydrodynamic mechanisms causing dentin hypersensitivity. Excitation at the dentin surface (i.e., root surface) causes an inward or outward flow of the intratubular fluid exciting the pulp nerve fibers (Courtesy of Dr. Luc M Martens)

[11]. In cases of interdental dentin hypersensitivity occurring in periodontally involved teeth, microorganisms invading the root dentin have also been discussed [12]. This condition may be of different etiology but results in similar pain symptoms. This type of dentin hypersensitivity is often referred to as root sensitivity.

Regarding the hydrodynamic mechanism, dentin will only be sensitive if the tubules are patent from the pulp to the oral environment (Fig. 15.2). Sensitive teeth have up to eight times more and up to two times wider tubules at the buccal cervical area as compared to nonsensitive teeth [13]. It has also been shown that smear layers in sensitive dentin are thinner and less calcified as compared to those of nonsensitive dentin [14]. As the patency will change with production and removal of the smear layer, episodic conditions are possible [13]. Spontaneously occurring changes in the exposed dentin, which in many cases seem to block the tubules, may reduce the responses to hydrodynamic stimulation and, thus, have an alleviating effect on dentin sensitivity.

15.2.1 Predisposing Factors

15.2.1.1 Gingival Recession Exposing Dentin

To be hypersensitive, dentin must be exposed and the exposed tubules must be open and patent to both the oral cavity and the pulp [10, 15]. Exposure of dentin through the loss of gingival and periodontal tissue may be caused by either too meticulous or by neglected oral hygiene (Fig. 15.3). The exact mechanism by which too meticulous oral hygiene causes loss of tissue is not very well understood and often implies brushing force and brush bristle characteristics. Several studies have shown the injury potential of sharp nonrounded filament tips on gingival abrasion [16, 17]. Surprisingly there is no information on the role of toothpastes in this process. Such a role could be both physical, through abrasion, and chemical, through cytotoxicity of ingredients such as detergents to the soft tissues [18]. In any case, modifying

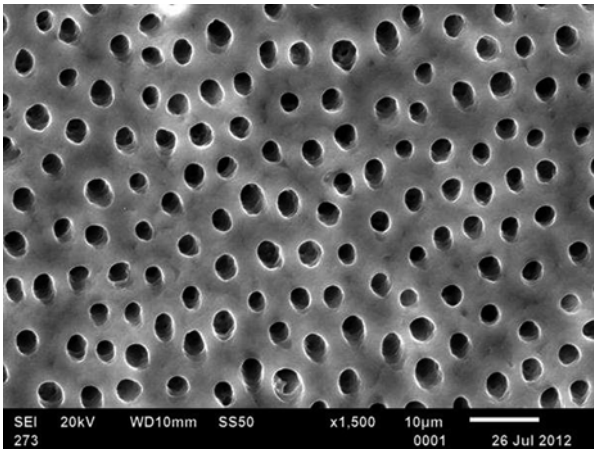

Fig. 15.2 Open tubules are a prerequisite for dentin hypersensitivity in most cases (Courtesy of Dr. Bennett T. Amaechi)

factors like inserting frenula, thin gingival biotypes, a lack of keratinized gingiva, or absence of the buccal bone may be implicated and should be considered. The mechanism by which neglected oral hygiene causes recessions runs through acute and chronic periodontal diseases and nonsurgical and surgical treatments.

15.2.1.2 Loss of Hard Tissue Exposing Dentin

Exposure of dentin by the loss of enamel is often ascribed to abrasion. However, most abrasives are softer than enamel and it must be concluded that toothpaste abrasion alone would play a clinically insignificant role in exposure of dentin [18]. In contrast, acids from intrinsic or extrinsic sources are more harmful for enamel by dissolution and by softening. The softened enamel is subsequently abraded away by mechanical forces. Shear forces of the oral soft tissues may be sufficient to abrade the softened enamel [19], but toothbrushing surely will as will grinding and clenching [20]. So when there is exposure of dentin as a result of loss of enamel, the patient's history should reveal the role of intrinsic or extrinsic acids (Table 15.1).

Since nonsensitive dentin reveals few if any open dentinal tubules at the surface [13], it is assumed that the tubules are covered by a "smear layer," consisting of protein components and calcium phosphate deposits derived from saliva [23] (Fig. 15.4). To initiate dentin hypersensitivity this layer has to be removed, and in vitro and in situ studies implicate erosive wear, as the smear layer is sensitive to acids [24, 25]. When acids have softened the smear layer and dentin, the materials

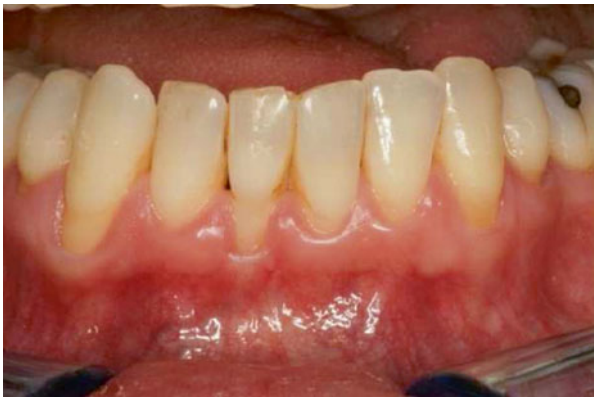

Fig. 15.3 Exposure of dentin through the loss of gingival and periodontal tissue may be caused by either too meticulous or neglected oral hygiene (Courtesy of Dr. Luc M Martens)

Table 15.1 Patient history

Ask patient to describe pain (look for description of pain as short, sharp)
Ask patient to identify pain-inciting stimuli (thermal, tactile, evaporative, osmotic, chemical)
Determine patient's desire for treatment
Probe for lifestyle habits/practices, intrinsic and extrinsic acid (citrus juices and fruits, carbonated drinks, wines, ciders)
Obtain detailed dietary information including dietary intake relevant to medical problems
Probe for gastric acid reflux and excessive vomiting

Canadian Advisory Board on Dentin Hypersensitivity, 2003; Martens, 2013 [21, 22]

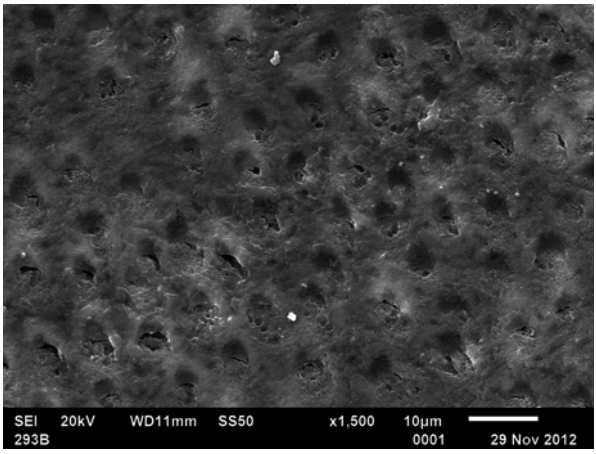

Fig. 15.4 Dentin surface of Fig. 15.2 covered by a smear layer (Courtesy of Dr. B Amaechi)

are more susceptible to physical forces, such as toothbrushing. Clinical data suggest that physical forces alone are not a key factor in removing the smear layer and opening exposed dentin tubules [10]. Also toothpaste will remove the smear layer [25, 26] probably by a combined abrasive and detergent action. Moore and Addy [27] have suggested that certain "mild" surfactants and "gentle" abrasives might have advantages over their more traditional counterparts in toothpastes marketed for the relief of dentin hypersensitivity [27]. However, this hypothesis does not appear to have been clinically validated in well-designed clinical studies [15]. One study showed no difference in desensitizing effect after elicitation using the evaporative method when using four desensitizing toothpastes different in abrasivity with RDA 60, 108, 150, or 210, respectively [28].

Subsequent to tubule exposure, toothpaste may reduce patency by secondary abrasive smearing or deposition of toothpaste constituents onto the dentin surface and into tubules. This makes the role of toothpaste without active ingredients to reduce dentin hypersensitivity inconclusive, even of fluoride containing pastes. Additionally it suggests that when using desensitizing toothpastes application with a fingertip or cotton swab after brushing may be beneficial.

15.3 Clinical Assessment

Clinical examination for dentin hypersensitivity would include a pain provocation test. However, the patient's perception of dentin hypersensitivity is subjective and clinical evaluation based on any scoring or rating system regarding its severity is challenging. Nevertheless, it is important to detect, rate, and monitor the pain as accurately as possible in order to define the baseline status and to observe any changes in due course and after therapy. Ideally, the latter ends in a status where "no pain" can be attested, but this ideal dichotomous treatment goal is still difficult to achieve.

Provocation tests are most frequently used to simulate pain and to assess the immediate reaction:

1. Tactile stimulus. This is the use of a probe, which is used as a "scratch" test on the exposed dentin, preferably with a standardized pressure. The use of probes would be contraindicated in evaluating treatments that use adhesive restorative materials, or other barrier methods. In such cases, the use of controlled air stimuli, graded cold water, or contact cold probes would be more appropriate [29].
2. An evaporative air stimulus. The Schiff Cold Air Sensitivity Scale is frequently used to assess the subject response to the air blast hypersensitivity [30]. This scale is scored as follows:
   0 = Subject does not respond to air stimulus.
   1 = Subject responds to air stimulus but does not request discontinuation of stimulus.
   2 = Subject responds to air stimulus and requests discontinuation or moves from stimulus.
   3 = Subject responds to air stimulus, considers stimulus to be extremely painful, and requests discontinuation of the stimulus.
   Noteworthy, the teeth on either side of the tooth under investigation should be isolated so that no referred pain is detected.
3. A cold stimulus, which can be graded cold water or contact cold probes.

After this pain induction, either a scoring system such as "Dental Pain Scale (DPS)" rates the pain answer or a visual analogue scale (VAS) can be used to "quantify" the severity in millimeter (mm) (Fig. 15.5).

Often individuals will not respond to all types of stimulus or may respond differently to different stimuli [31–33], so it is recommended that at least two

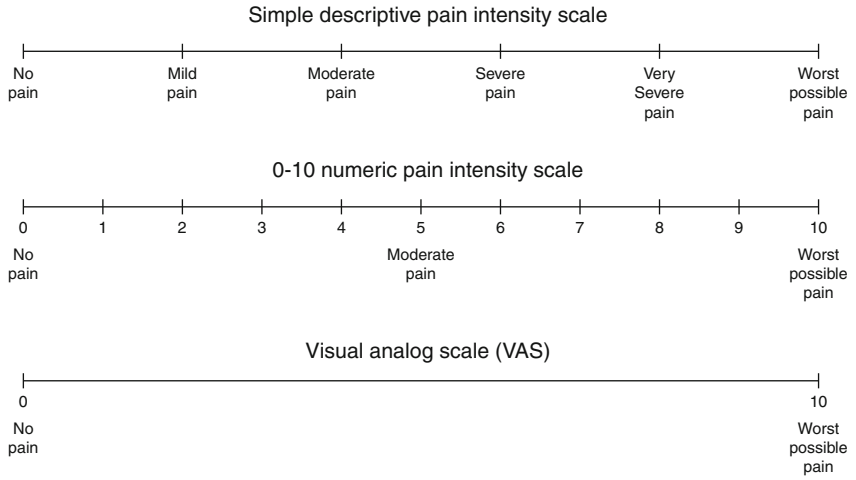

Fig. 15.5 Frequently used scales for pain intensity measurement

hydrodynamic stimuli should be used. The interval between stimulus applications should be of sufficient duration to minimize interactions between stimuli. If multiple stimuli are used to help to achieve a diagnosis, the order of application should be that which causes the least to the most amount of pain [34]. Repeated testing should be avoided as it is not known how long it takes to reach threshold evaluation. In case of a negative provocation test, any dentin hypersensitivity therapy becomes needless.

It is also questionable to what extent therapies should be performed in cases where patients display with no self-reported pain but show the typical signs of dentin hypersensitivity during a routine clinical examination. In fact, prophylactic measures, which protect the exposed surfaces against cariologic and wear challenges, may be considered, but the patient's awareness of nonexisting subjective pathologic conditions should not be stimulated.

15.3.1 Oral Health-Related Quality of Life

Dentin hypersensitivity may disturb the patient during eating, drinking, toothbrushing, and sometimes even breathing. The resulting restrictions on everyday activities can have an important effect on the patient's quality of life [35]. Oral health-related quality of life (OHRQoL) is a relatively new concept in dentistry. It is an aspect of dental health addressing the patient's perception of whether his/her current oral health status has an impact upon his/her actual quality of life [35]. Therefore, OHRQoL may provide a new perspective when looking at a patient, by measuring treatment efficacy in terms of patient satisfaction. There is only little research into the relevance of the various quality of life questionnaires in the treatment of dentin hypersensitivity, yet it may be very valuable to the patient to evaluate the treatment according to these values. Boiko et al. [36] developed, based on in-depth and focus group interviews, a dentin hypersensitivity experience questionnaire (Table 15.2) to capture subjective impacts on patients. The questions can be phrased like "Having the sensations in my teeth takes a lot of the pleasure out of eating and drinking" after which the patients can indicate to what extent he agrees or disagrees.

15.4 Differential Diagnosis

A number of other dental conditions can give rise to pain symptoms, which may mimic those of dentin hypersensitivity. Therefore, careful examination is necessary to exclude the following conditions, which need a variety of different treatment options [6, 22, 37, 38]:

- Cracked tooth syndrome
- Incorrect placement of dentin adhesives in restorative dentistry, leading to nanoleakage
- Fractured restorations and incorrectly placed dentin pins

- Inappropriate application of various medicaments during cavity floor preparation
- Lack of care while contouring restorations so the tooth is left in traumatic occlusion
- Pulpal response to caries and recent restorative treatment
- Palatogingival groove and other enamel invaginations and defects
- Chipped/fractured teeth causing exposed dentin
- Tooth bleaching
- Acute periodontal infections (e.g., necrotizing gingivitis/periodontitis or abscesses)

Table 15.2 The items of the dentin hypersensitivity experience questionnaire developed by Boiko et al. [36] to determine the impact of dentin hypersensitivity on a patient's quality of life

		1 disagree strongly	2 agree a little	3 agree	4 agree moderately	5 agree strongly
Restrictions	Pleasure out of eating					
	Cannot finish meal					
	Longer to finish meal					
	Problems with eating ice-cream					
Adaptation	Modification of eating					
	Careful when breathing					
	Warming food/drinks					
	Cooling food/drink					
	Cutting fruit					
	Putting a scarf over mouth					
	Avoiding cold drinks/ foods					
	Avoiding hot drinks/ foods					
	Avoiding contact with certain teeth					
	Change toothbrushing habits					
	Biting in small pieces					
	Avoiding other food					
Social	Longer than others to finish					
	Choose food with others					
	Hide the way of eating					
	Unable to take part in conversations					
	Painful at the dentist					

(continued)

Table 15.2 (continued)

		1 disagree strongly	2 agree a little	3 agree	4 agree moderately	5 agree strongly
Emotions	Frustrated not finding a cure					
	Anxious of eating contributes					
	Irritating sensations					
	Annoyed with myself for contributing					
	Guilty for contributing					
	Annoying sensations					
	Embarrassing sensations					
	Anxious because of sensations					
Identity	Difficult to accept					
	Different from others					
	Makes me feel old					
	Makes me feel damaged					
	Makes me feel unhealthy					

15.5 Preventive Strategies

Prevention is always better than cure. Thus, primary prevention represents the first line of defense against dentin exposure, i.e., the formation of gingival recession and dental hard tissue deterioration. Careful oral hygiene instructions and dietary advices are crucial. When dentin is already exposed, patients should be instructed in order to minimize the risk of opening the tubules and, thus, increasing the patency. Suggestions for patients and the dental professionals to avoid aggravating behavior or iatrogenic damage developed by Martens [22] are given in Table 15.3.

15.6 Treatment Strategies

When the patients do suffer from dentin hypersensitivity, there is broad range of treatment options comprising home-use and professional approaches. It is advised to start with the less invasive home-use therapies and only expand to professional in-office treatments when the home-use treatments are not effective. When decided to continue with in-office treatments, again one should start with the least invasive ones.

Table 15.3 Suggestions for patients and the dental professionals to avoid aggravating behavior or iatrogenic damage developed by Martens [22] based on Chu et al. [39] and Drisko [40]

Suggestions for patients
Limit dietary acids
Use soft-medium toothbrush and adequate brushing technique
Use additional topical fluorides
Avoid picking, scratching at the gingival margins
Avoid excessive flossing or improper use of toothpicks
Suggestions for dental professionals
Avoid overinstrumentation of the root surfaces during scaling
Avoid excessive polishing of exposed dentin during stain removal
Avoid burning the gingival tissues during in-office bleaching
Advise patients to be careful during home-bleaching
Avoid harmful instruments and materials

Home-use products have several benefits including ease of use, convenience of self-application, and easier access but may require several weeks before taking effect. In-office treatments are generally more invasive and more effective under specific conditions and can provide instant relief, e.g., an adhesive sealing or restoration.

A remaining aspect is the placebo effect, which is an important and potentially beneficial side effect when dealing with pain and its treatment and management. Using arthritis of the knee as an example in the medical field, it has been impressively shown that sham endoscopic interventions lead to the same reduction of pain and symptoms as conventional treatment modalities [41]. In addition, prescription of differently colored pills resulted in significant differences in pain reduction [42]. Whereas a red placebo tablet, for instance, showed comparable pain relief as the best antirheumatic test pill used, the blue equivalent showed the least effect. Thus, improved psychological cotherapeutic strategies may one day become an important auxiliary aspect in dentin hypersensitivity management, especially when it comes to changing patients' expectations of treatment outcomes and confidence. The psychological training of dental professionals still has some room for development.

15.6.1 At-Home Therapy

For home use, both toothpaste and mouthrinses are available. There are a few studies on chewing gum but the results are not very reliable [43, 44]. The working mechanisms fall under two basic categories, being nerve desensitization and occlusion of exposed dental tubules (Table 15.4).

15.6.1.1 Nerve Desensitization

Potassium salts and, to a lesser extent, strontium and calcium [45] are agents that may have a direct desensitizing action on the nerves located at the pulpal side of the

Table 15.4 Active ingredients in products for at-home therapy

						Calcium/hydroxyapatite carriers		
Active ingredient	Potassium	Strontium	Fluoride	Stannous salts	Oxalates	Calcium phospho silicate (Novamin)	Arginine calcium carbonate (Pro-arginin)	Nanohydroxyapatite
Mechanism of action	Nerve Desensibilization	Precipitative occlusion of dentin tubules						
Compatible with Fluoride	Yes	SrCl not SrAc yes	Yes	Yes	Yes	MonoFluoro Phosphate (MFP)	MFP Sodium Fluoride	Yes Caveat: some pastes do not contain F

tubules. Therefore, the ions must be able to pass through the dentinal tubules against the dentin fluid flow and build up a sufficiently high concentration to desensitize the nerves at the interface of the inner dentin surface and the pulpal chamber. A concentration of 8 mM might be necessary needing a lag time of several weeks before pain relief is experienced. Once at the nerve site, potassium alters the cell's electrical potential, resulting in depolarization, making the cell less responsive to stimuli. When people stop using the product, the potassium will diffuse away, and sensitivity reestablishes. Strontium as other divalent cation may operate by a different mechanism from potassium, such that the membrane of the nerve cell is stabilized but the potential of the cell remains unchanged [45].

Mainly potassium nitrate (5 %), citrate (5.5 %), and chloride (3.75 %) have been formulated into toothpastes as each of the salts provides 2 % potassium, which is needed for relief. In the United States of America, desensitizing toothpastes typically contain 5 % potassium nitrate, to meet FDA regulations. Many manufacturers have a potassium-based desensitizing product, suggesting it being (or having been) the "golden standard." A recent Cochrane review included six studies in a meta-analysis, which showed a statistically significant effect of potassium nitrate toothpastes on air blast and tactile sensitivity tests at 6–8 weeks follow-up, respectively. The subjective reports of the patients on dentin hypersensitivity, in contrast, failed to show a significant effect at the respective time points (Table 15.5) [46].

15.6.1.2 Tubule Occlusion

As mentioned previously, tubules must be patent in order to allow for fluid movements. Blocking or occluding these patent tubules, therefore, seems a simple and conceptually effective way of decreasing sensitivity. There are several mechanisms by which products for home use can occlude exposed dentinal tubules. Mechanical formation of a natural smear layer by burnishing dentin induces tubule occlusion. Topically applied compounds, which form insoluble materials that precipitate in the tubules and on the surface, are also effective (Fig. 15.6). Such compounds include abrasive particles, strontium, stannous, arginine calcium carbonate, oxalate, or bioactive glasses.

Addy and Mostafa [47] examined in vitro three artificial silica abrasive based toothpastes, two with strontium acetate alone or combined with fluoride and one without. The study showed that these formulations coated the dentin surface and occluded the tubules. The analysis revealed that the occluding agent was the artificial silica, which was not water or acid labile. A parallel clinical study also showed all three artificial silica formulations to be effective in the treatment of dentin hypersensitivity [48]. Many of today's toothpaste contain similar artificial silica abrasives, but are not as effective in reducing dentin hypersensitivity. One explanation may lay in the use of sodium lauryl sulphate (SLS) as detergent in most tooth pastes, which would compete with silica for the adsorption to dentin. The experimental toothpaste in the study of Addy and Mostafa [47] did not contain SLS. It is important to note, however, that the dentin hypersensitivity benefits attributed to the presence of silica abrasive in these strontium toothpastes have not been reproduced in other studies, which included silica-based toothpastes [49, 50].

Table 15.5 Results of a systemic review and meta-analysis on the effect of potassium-containing toothpastes on dentin hypersensitivity

Outcome and comparison	No. of studies	No. of participants	Effects size (95 % CI) (std mean difference[a])
Tactile	*5*		*1.19 (0.79, 1.59)*
Potassium nitrate no F Versus No potassium nitrate no F	1	110	0.72 (0.33, 1.11)
Potassium nitrate plus F Versus No potassium nitrate plus F	4	246	1.34 (0.97, 1.71)
Air blast	*6*	*392*	*−1.25 (−1.65, −0.85)*
Potassium nitrate no F Versus No potassium nitrate no F	2	146	−1.18 (−1.88, −0.48)
Potassium nitrate plus F Versus No potassium nitrate plus F	4	246	−1.30 (−1.88, −0.72)
Subjective	*3*	*206*	*−0.67 (−1.44, 0.10)*
Potassium nitrate no F Versus No potassium nitrate no F	2	146	−1.01 (−1,53, −0.49)
Potassium nitrate plus F Versus No potassium nitrate plus F	1	60	0.10 (−0.41, 0.60)

From Poulsen et al. 2006 [46]

[a]The standardized mean difference is used as a summary statistic in meta-analysis when the studies all assess the same outcome but measure it in a variety of ways (i.e., the use of different scales). In this circumstance, it is necessary to standardize the results of the studies to a uniform scale before they can be combined. The standardized mean difference expresses the size of the intervention effect in each study relative to the variability observed in that study

Strontium chloride was introduced more than 50 years ago. Today, most products contain strontium acetate due to its improved clinical efficacy and its compatibility with fluoride and potassium nitrate. But there are still strontium chloride toothpastes on the market that do not contain fluoride. Several mechanisms are hypothesized by which strontium would reduce dentin hypersensitivity: (1) precipitation of particles on the tooth surface, (2) incorporation in the dentin matrix making it less soluble, and (3) stabilization of the membrane of dental nerves [45]. There is very little scientific evidence to support any of these mechanisms, but the first one has been proposed to be the most likely one [51]. A recent review on clinical studies found insufficient data for making any absolute conclusions about the efficacy of strontium treatment due to the diversity of testing methods used in the studies [15].

Stannous salt solutions precipitate onto dentin and may block tubules. The deposits are water and acid resistant and may even provide a protective effect against acid erosion [52]. Clinical studies reported efficacy of stannous fluoride gel or

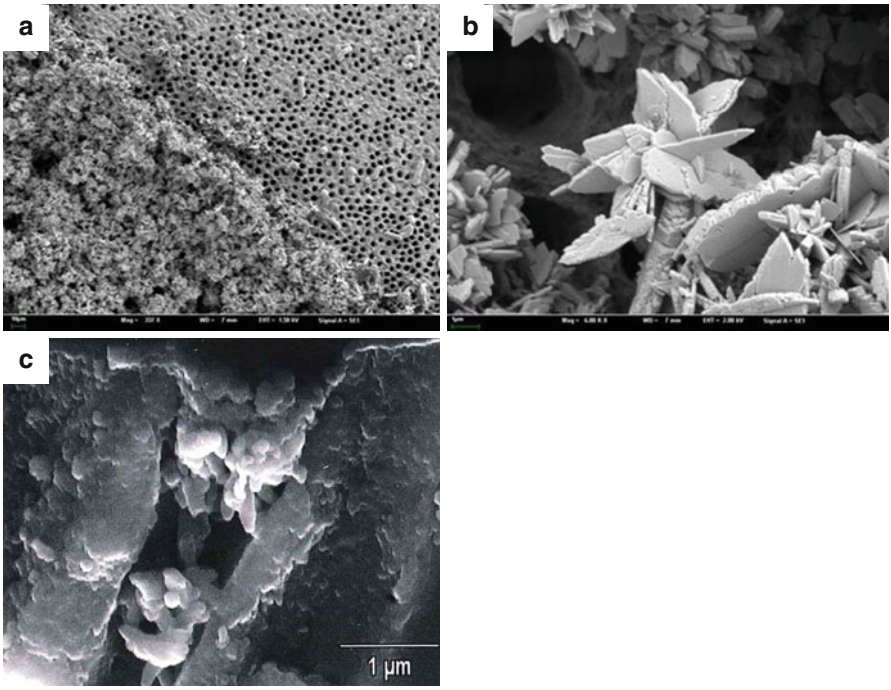

Fig. 15.6 SEM image of dentin treated with a precipitating agent: (**a**) shows treated (*bottom left*) and untreated (*top right*) areas. However, despite clear evidence of crystallite deposition, uncovered dentin areas and tubule entrances can be seen (**b**). A plug precipitated in the orifice of a dental tubule (**c**). (Courtesy of Dr. L.M. Martens)

solutions in the treatment of dentin hypersensitivity [53–55]. More recently, randomized controlled trials have reported that hexametaphosphate stabilized stannous fluoride toothpaste provided some immediate relief, as well as after 4 and 8 weeks [56–59].

Recently, a mouthrinse containing 1.4 % potassium oxalate has also been introduced. Soluble oxalate salts have been shown to occlude tubules by reacting with naturally occurring calcium ions in the oral fluids to precipitate as insoluble calcium oxalate crystals [60]. This precipitate blocks fluid flow in the dentinal tubules, leading to decreased hypersensitivity. The precipitates of oxalates are relatively resistant to dissolution in acidic environments, increasing their durability [61].

Bioactive glass consists of specific proportions of SiO_2, Na_2O, and P_2O_5 (calcium sodium phosphosilicate). Bioglass in solution or toothpaste interacts on the dentin surface and forms a hydroxyapatite-like silica deposit over the dentin and in the tubules [62]. This tubule blocking deposit appears water and acid insoluble and mechanically resistant. A number of randomized controlled trials extending up to 8 weeks showed significant benefits for the CSPS product in the treatment of dentin hypersensitivity [63–66].

Arginine, an amino acid naturally present in saliva, works in conjunction with calcium carbonate and phosphate to create a plug in dentinal tubules that prevents fluid flow [15]. The hypothesized mechanism of action suggests that the positively charged arginine is attracted to negatively charged dentin. The alkaline pH promotes deposition of calcium, phosphate, arginine, and carbonate on the dentin surface and inside the dentin tubules [67]. Several studies have shown 8 % arginine toothpaste and 0.8 % arginine mouth washes to be effective against dentin hypersensitivity [68–71].

Nanohydroxyapatite (nHAP) in dentifrice promotes deposition of precipitate layer over and within the dentin tubules by acting as a calcium and phosphate reservoir, helping to maintain a topical state of supersaturation of these ions with respect to tooth minerals, and thus causing deposition on the surface of tooth tissue. In a double-blind randomized clinical trial comparing the efficacy in reducing DHS of a dentifrice formulation containing 15 % nHAP without fluoride, with fluoride dentifrice, and a placebo, a significant reduction of cold air sensitivity and tactile sensitivity were observed for the nHAP group at 2 and 4 weeks compared to baseline and the two comparison groups [72].

At-home product comparison reveals that the products should be considered equally effective and can be recommended for use [18]. The various modes of actions and different solubility of the various precipitates that form suggest that when one product does not give sufficient relief, it might be worthwhile to try another product. After an evaluation period of 4–6 weeks (for potassium salts maybe 8 weeks), another product may be tried before proceeding to the in-office treatments.

With regard to laboratory studies, which are frequently used to show occluding effects on dentin, one should critically amend that most of these studies were performed without the simulation of dentin fluid dynamics, i.e., a liquid outflow. Therefore, precipitation phenomena should not be overestimated. In addition, brushing and acid challenges are also not performed in most studies, which may additionally impair the long-term stability of any claimed layer formation.

15.6.2 In-office Treatment

In the dental office, comparable compounds are available as mentioned before, but, as said, in more powerful compositions. Products containing the following agents can be used:

- Dentin bonding agents
- Composite resins
- Fluoride varnishes
- NaF ionthophoresis
- Glutaraldehyde-based agents
- Remineralization promoting cements
- Laser therapy

Dentin bonding agents and composite resin materials exhibit long-term or permanent effects. These materials can effectively seal dentinal tubules by forming a hybrid layer, block tubules by forming tags, and create a covering layer [73].

Some primers contain glutaraldehyde, which can lead per se to protein coagulation within the dentinal tubules, while the adhesive resin materials form an occluding barrier on a more hydrophobic surface. Such materials have shown good results in dentin hypersensitivity management in clinical trials [29].

When using cements, there may be benefit from using the remineralization promoting cements, which are cements containing calcium and phosphates [74, 75], and recently a calcium silicate paste, derived from Portland cement, was shown to be effective in the occlusion of tubules in in vitro experiments [76].

Sgolastra et al. [77] systematically reviewed the literature on lasers for the treatment of dentin hypersensitivity. They identified several theories by which lasers may be effective. For low-intensity lasers (e.g., Gallium-Aluminum-Arsenide (GaAlAs)), the irradiation may have a photo-bio-modulating effect on cellular activity, increasing the deposition of tertiary dentin by odontoblastic cells [78]. Middle-output-power lasers (e.g., Erbium: Yttrium Aluminium Garnet (Er:YAG), Neodymium: Yttrium Aluminium Garnet (Nd:YAG), and Erbium, Chromium: Yttrium, Scandium, Gallium, Garnet (Er,Cr:YSGG)) may reduce or obliterate the dentinal tubules [79]. For Er:YAG and Er,Cr:YSGG, the efficacy in reducing dentin hypersensitivity is thought to be related to the thermo-mechanical ablation mechanism and to the high absorption of their wavelengths by water [80]. These effects may lead to the evaporation of the superficial layer of dentinal fluid, reducing the flow within the dentinal tubules. Due to exposure to Nd:YAG laser, dentin may be fused, solidifying into a glazed, nonporous surface [81]. Nd:YAG irradiation can also directly act at the nerve level by blocking C and Aβ fibers [82]. Sgolastra et al. [77] concluded that Er:YAG, Nd:YAG, and GaAlAs lasers appear to be efficacious in reducing dentin hypersensitivity. However, given the high heterogeneity of the included studies, future randomized controlled clinical trials are needed to confirm these results.

Recently Lin et al. [83] evaluated in-office treatments for dentin hypersensitivity in a systematic review with a network meta-analysis (Table 15.6). In this meta-analysis, articles were chosen that used evaporative air test to elicit dentin hypersensitivity. Forty studies were included. The standardized mean difference (the mean difference in each study divided by that study's standard deviation) between placebo and physical occlusion was 2.57 [95 % CI: 0.94–4.24], placebo versus chemical occlusion was 2.33 (95 % CI: 1.04–3.65), placebo versus nerve desensitization was 1.72 (95 % CI: 0.52–4.00), placebo versus laser therapy was 2.81 (95 % CI: 1.24–4.41), and placebo versus combined treatment was 3.47 (95 % CI: 5.99–0.96). The comparisons between the five active treatments showed no significant differences. Therefore, it was concluded that most active treatment options have a positive effect and show significantly better treatment outcomes than placebo treatment.

Table 15.6 Grouping of the included articles for network meta-analysis (Lin et al. [83])

Group	Group I Placebo	Group II Physical occlusion of dentinal tubules	Group III Chemical occlusion of dentinal tubules	Group IV Nerve desensitization	Group V Photobiomodulating action	Group VI Combined treatment
Treatment option	No treatment Water Not specified placebo desensitizing toothpaste	Pumice paste Sodium bicarbonate Hydroxyapatites Bioglasses Glass ionomers Dentin bonding agents Resins	Fluorides Oxalates Glutaraldehyde-based agents Calcium compounds Arginine bicarbonate calcium carbonate	Potassium nitrates Guanethidine	Laser therapy	Any combination of Groups II–V

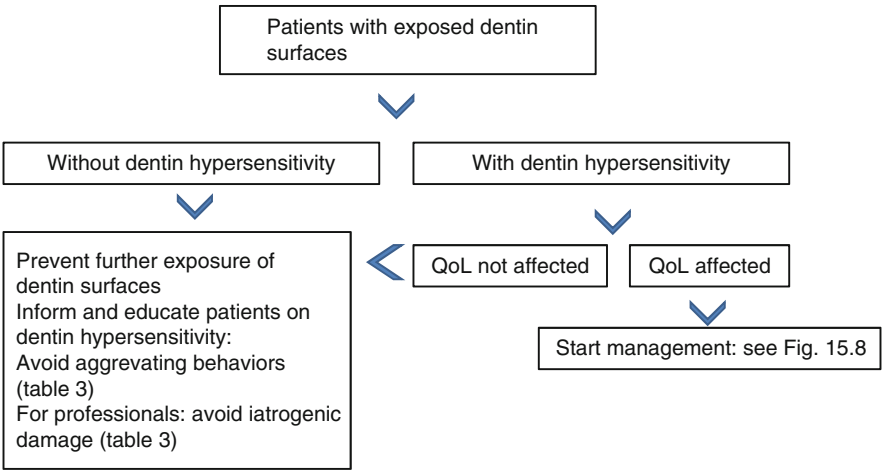

Fig. 15.7 Treatment decision tree for patients with exposed dentin surfaces (Adapted from Martens [22])

15.7 The Decision to Treat

Most of the patients who experience DHS wait to mention until the next recall visit and most of them do not specifically seek treatment for this problem, most likely because they do not view it as a significant dental health problem [35]. However, it is clearly shown that DHS can significantly be related to substantially impaired oral health–related quality of life [36, 84]. If the patient presents with exposed cervical dentin (ECD) combined with a complaint of DHS, one has to point out if this pain sensation affects the patient's quality of life (Qol). In this respect, the patient can be questioned as indicated in Table 15.2.

Figure 15.7 represents a flow diagram which can be followed for patients with exposed dentin surfaces [22]. If the patient has no dentin hypersensitivity, no treatment is required. However, a preventive strategy might be envisaged. The latter avoids further exposure of dentin surfaces and includes patient information and education, avoidance of aggravating behaviors that could induce dentin hypersensitivity, and, for the professionals, avoidance of iatrogenic damage (Table 15.3). This prevention program has also to be started in patients with dentin hypersensitivity without complaints of Qol. In addition, a desensitizing fluoride containing toothpaste may be advised. If the Qol is affected in patients with exposed dentin surfaces with dentin hypersensitivity, the treatment decision tree in Fig. 15.8 can be followed. A complete patient history especially focused on nutritional habits, oral hygiene habits, and the promoted diagnosis by exclusion has to be performed (Table 15.1). If there is no consistency between history and examination, causes other than dentin hypersensitivity must be identified and treated accordingly. If

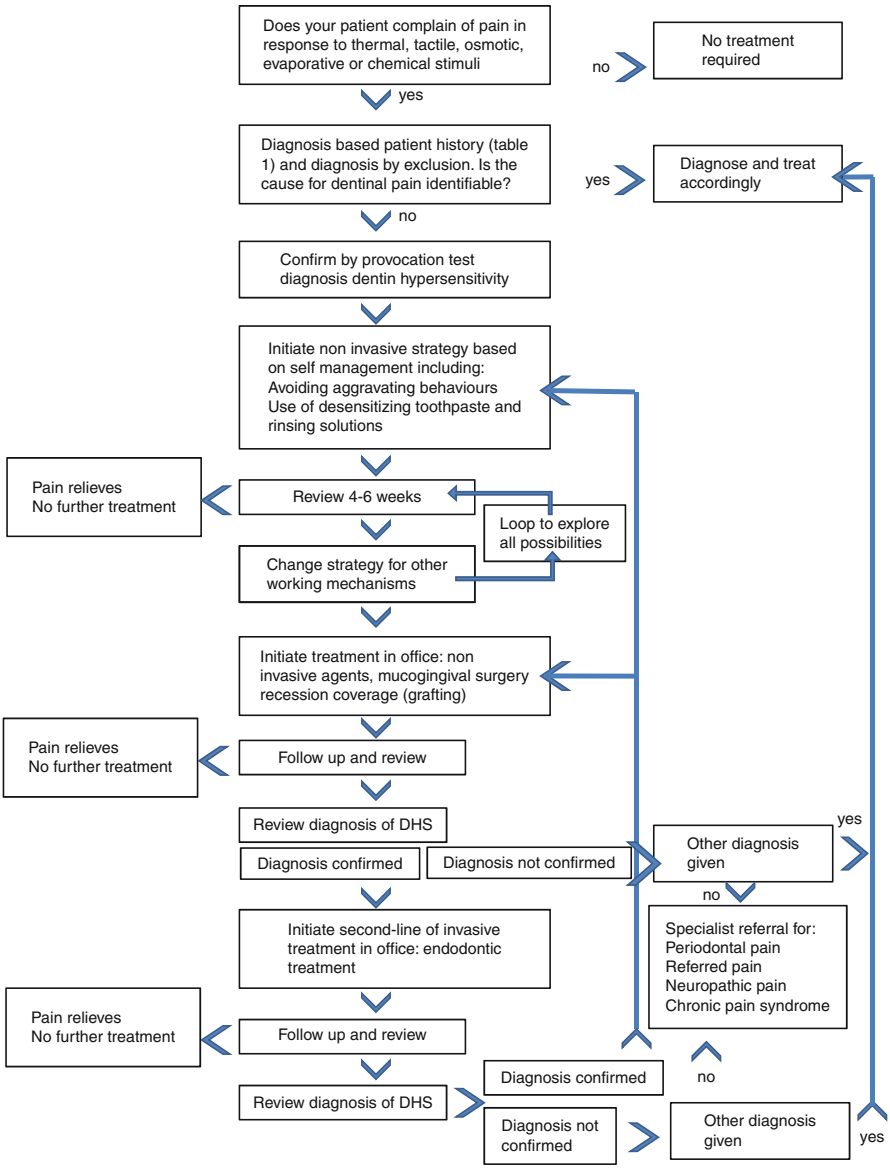

Fig. 15.8 Flow chart of the treatment strategy for dentin hypersensitivity (Adapted from Martens [22], Orchardson and Gillam [43] and the Special topic nr 6 on sensitive teeth by Colgate and Adelaide University (Adelaide University) [85])

consistency is present, management of dentin hypersensitivity must be initiated. The latter should be focused on suggestions for patients as well as for professionals (Table 15.3). Regarding the patients, dietary counseling and nonharmful oral hygiene habits are very important. This can be supported by the daily use of desensitizing toothpastes. If necessary, a less traumatic brushing method may also be introduced. While at-home treatment can be the first choice for generalized dentin hypersensitivity, when localized to one or two teeth or when immediate relief is required, practitioners may elect to use an in-office method as the first choice of treatment for dentin hypersensitivity [43]. Regarding the professionals, nonharmful professional dental care must be carried out. This must result in a well-considered choice and use of instruments and additional tools performing restorative dentistry. If, during follow-up typically 4–6 weeks, symptoms are relieved or disappeared, improving the patient's Qol, no further treatment is required. Regarding desensitizing toothpastes, two treatment approaches are well known: occluding dentinal tubules (plugging) or blocking the neural transmission to the pulp. For the occlusion of the tubules, various strategies are aimed at by the various pastes. Therefore, it is suggested if one product with a certain working mechanisms is not sufficiently effective to try a product based on another working mechanism.

If symptoms are confirmed, no pain relief present, or a further decrease of the patients' Qol is present, professional in-office treatments for DHS must be initiated. It is recommended to start with less invasive procedures first such as the use of topical fluorides and dentin bonding agents or laser therapy, which were presented in the Table 15.6. Still, all procedures can – or even should – be accompanied by the use of desensitizing toothpastes twice a day, i.e., concomitant at-home therapy as individually suggested. If treatment is carried out successfully, one should maintain and review the therapy on a regularly basis at given recall appointments.

The methods described above are indicated especially in cases with limited amounts of dental hard tissue loss, i.e., no classical abrasive or erosive defect characteristics. In cases where a class V restoration is indicated, an adhesive filling is a valid option (Fig. 15.9). Regenerative mucogingival therapy also remains an alternative, where hard and soft tissue conditions allow [86] (Fig. 15.10). A suggested strategy for dentin hypersensitivity management, taking morphological aspects into consideration, is depicted in Fig. 15.11.

If after all these treatment procedures still no pain relief can be achieved, one should start an advanced diagnosis based on exclusion before deciding to proceed to endodontic therapy, which really represents the last option of an actually failing therapy. If the diagnosis is not confirmed but revaluated, the patient should be treated accordingly. If the diagnosis is not confirmed and no other diagnosis can be given, the patient should be referred to a specialist to examine for acute periodontal infections, referred pain, neuropathic pain, or chronic pain syndrome.

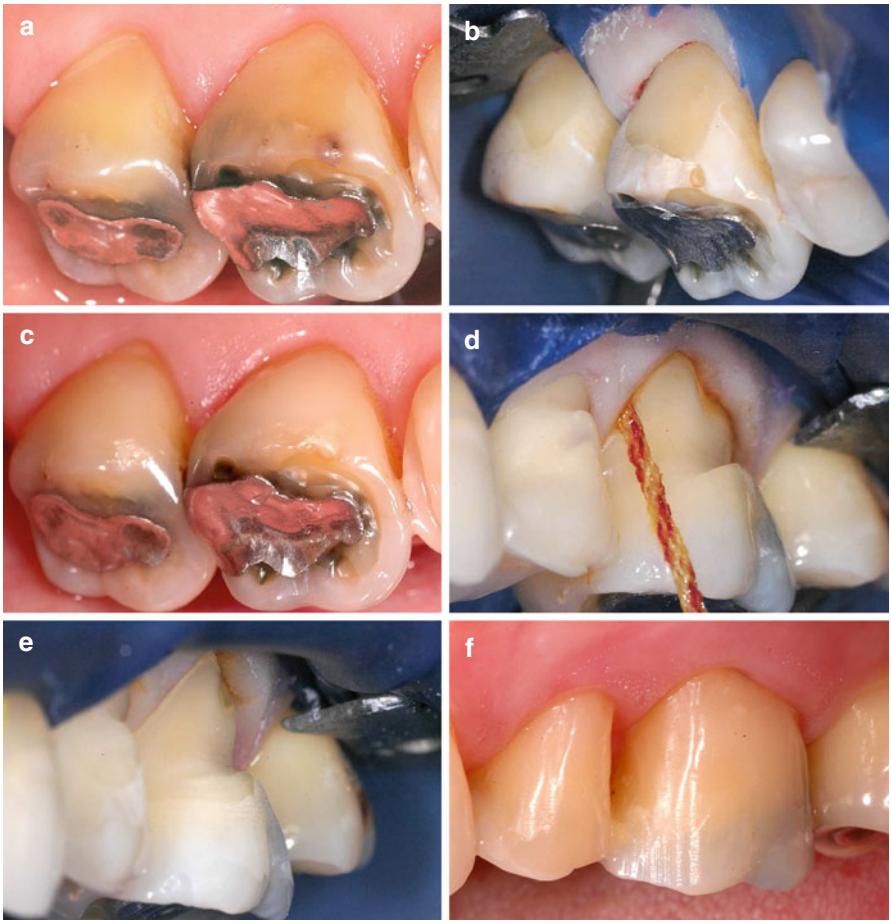

Fig. 15.9 Patient suffering from severe dentin hypersensitivity in the second quadrant (teeth 25, 26, and 27 with Schiff scores 2 and 3, respectively) and mixed defects (erosion and abrasion) at the palatal (**a**–**c**) and buccal (**d**–**f**) cervical aspects, which were treated with adhesively placed fillings. (**a**) Palatal aspect before treatment, (**b**) isolation with glued rubbed dam and retraction cords after etching, (**c**) restorations after 6 months, (**d**) buccal aspect, placement of a cord after rubber dam placement, (**e**) situation after cavity finishing and etching with phosphoric acid, and (**f**) restorations after 6 months. The pain was completely removed. Only one single aspect at tooth 25 buccally still displayed a Schiff 1 score after 6 months (Courtesy of Dr. P.R. Schmidlin)

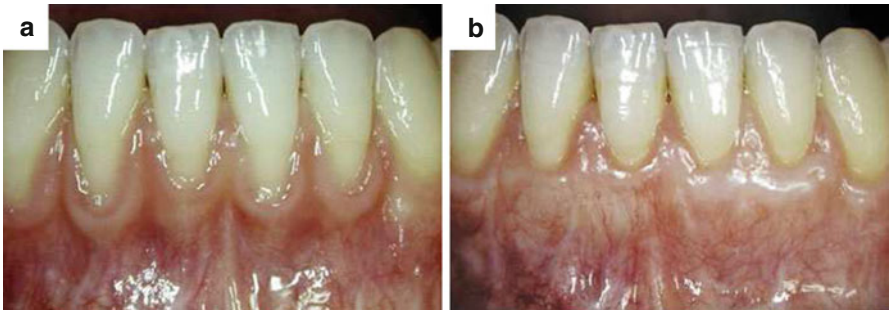

Fig. 15.10 Recession coverage using a connective tissue graft before it can be indicated, especially if the tooth substance loss is limited and the soft tissue morphology is adequate for a mucogingival approach (panel (**a**) before treatment and (**b**) 1 year after mucogingival surgery using a coronally advanced flap and connective tissue graft)

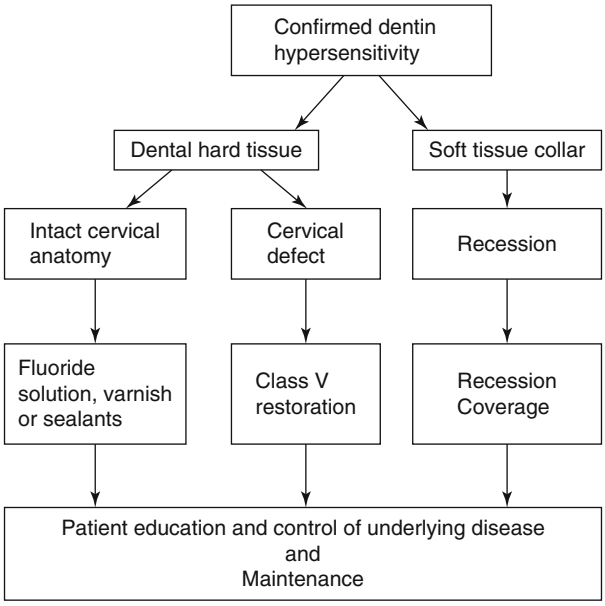

Fig. 15.11 Flow-chart of the decision-making process based on the underlying defect. Depending on the dental hard tissue damage and the morphology of the surrounding soft tissues, an adequate therapy can be initiated (Modified from Schmidlin and Sahrmann [87])

Conclusion

Dentin hypersensitivity is a problematic clinical entity that may become an increasing clinical problem for dentists to treat as a consequence of patients retaining their teeth throughout life and improved oral hygiene practices. For that, it is strongly recommended to screen routinely all dentate patients for exposed dentin surfaces and dentin hypersensitivity. In this respect, underdiagnosis of the condition will be avoided and the preventive management can be initiated early. Active management of dentin hypersensitivity usually will begin with at-home therapy of which brushing with desensitizing toothpastes is the most important. Complete management will usually involve a combination of at-home and in-office therapies.

References

1. Dowell P, Addy M. Dentine hypersensitivity – a review, aetiology, symptoms and theories of pain production. J Clin Periodontol. 1983;10(4):341–50.
2. Chabanski MB, Gillam DG. Aetiology, prevalence and clinical features of cervical dentine sensitivity. J Oral Rehabil. 1977;24(1):15–9.
3. Addy M. Etiology and clinical implications of dentine hypersensitivity. Dent Clin North Am. 1990;34(3):503–14.
4. West NX, Sanz M, Lussi A, Bartlett D, Bouchard P, Bourgeois D. Prevalence of dentine hypersensitivity and the study of associated factors; a European population-based cross-sectional study. J Dent. 2013;41(10):841–51.
5. West NX, Lussi A, Seong J, Hellwig E. Dentin hypersensitivity: pain mechanisms and aetiology of exposed cervical dentin. Clin Oral Investig. 2012;17 Suppl 1:S9–19.
6. West NX, Hughes J, Addy M. Erosion of dentine and enamel in vitro by dietary acids: the effect of temperature, acid character and concentration. J Oral Rehabil. 2000;27(10):875–80.
7. Gysi A. An attempt to explain the sensitiveness of dentin. Br J Dent Sci. 1900;43:865–8.
8. Brännström M. A hydrodynamic mechanism in the transmission of pain-produced stimuli through the dentine. In: Anderson DJ, editor. Sensory mechanisms in dentine. London: Pergamon Press; 1963. p. 73–9.
9. Vongsavan N, Matthews B. Fluid flow through cat dentine in vivo. Arch Oral Biol. 1992;37(3):175–85.
10. Addy M. Dentine hypersensitivity: new perspectives on an old problem. Int Dent J. 2002;52 Suppl 5:367–75.
11. Närhi M, Kontturi-Närhi V, Hirvonen T, Ngassapa D. Neurophysiological mechanisms of dentin hypersensitivity. Proceedings of the Finnish Dental Society. Suomen Hammaslaakariseuran Toimituksia. 1992;88(Suppl 1):15–22.
12. Adriaens PA, DeBoever JA, Loesche WJ. Bacterial invasion in root, cementum and radicular dentine of periodontally diseased teeth in humans – a reservoir of periodontopathic bacteria. J Periodontol. 1988;59(4):222–30.
13. Absi EG, 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(5):280–4.
14. Rimondini L, Baroni C, Carrassi A. Ultrastructure of hypersensitive and non-sensitive dentine. A study on replica models. J Clin Periodontol. 1995;22(12):899–902.
15. Cummins D. Recent advances in dentin hypersensitivity: clinically proven treatments for instant and lasting sensitivity relief. Am J Dent. 2010;23(Spec issue A):3A–13.
16. Alexander JF, Saffir AJ, Gold W. The measurement of the effect of toothbrushes on soft tissue abrasion. J Dent Res. 1977;56(7):722–7.

17. Breitenmoser J, Mörmann W, Mühlemann HR. Damaging effects of toothbrush bristle end form on gingiva. J Periodontol. 1979;50(4):212–6.
18. Addy M, West NX. The role of toothpaste in the aetiology and treatment of dentine hypersensitivity. In: Van Loveren C, editor. Toothpastes. Monogr Oral Sci, vol. 23. Basel: Karger; 2013. p. 75–87.
19. Amaechi BT, Higham SM, Edgar WM. Influence of abrasion in clinical manifestation of human dental erosion. J Oral Rehabil. 2003;30(4):407–13.
20. Addy M, Shellis RP. Interaction between attrition, abrasion and erosion in tooth wear. In: Lussi A editor. Dental erosion from diagnosis to therapy. Monogr Oral Sci. Basel Karger. 2006;20: 17–31.
21. Canadian Advisory Board on Dentin Hypersensitivity. Consensus-based recommendations for the diagnosis and management of dentin hypersensitivity. J Can Dent Assoc. 2003;69(4):221–6.
22. Martens LC. A decision tree for the management of exposed cervical dentin(ECD) and dentin hypersensitivity (DHS). Clin Oral Investig. 2013;17 Suppl 1:S77–83.
23. Pashley DH. Smear layer: physiological considerations. Oper Dent. 1984;9 Suppl 3:13–29.
24. Absi EG, Addy M, Adams D. Dentine Hypersensitivity. The effects of toothbrushing and dietary compounds on dentine in vitro: a SEM study. J Oral Rehabil. 1992;19(2):101–10.
25. Banfield N, Addy M. Dentine hypersensitivity: development and evaluation of a model in situ to study tubule patency. J Clin Periodontol. 2004;31(5):325–35.
26. Absi EG, Addy M, Adams D. Dentine hypersensitivity: uptake of toothpastes onto dentine and effects of brushing, washing and dietary acid. J Oral Rehabil. 1995;22(3):175–82.
27. Moore C, Addy M. Wear of dentine in vitro by toothpaste abrasives and detergents alone and combined. J Clin Periodontol. 2005;32(12):1242–6.
28. Curro FA, Hays RD, Stewart B, Masters JG. Clinical effects of increasing toothpaste abrasivity on tooth hypersensitivity. J Dent Res. 2008;87(Spec issue):(Abstr 1024).
29. Holland GR, Närhi MN, Addy M, Gangarosa L, Orchardson R. Guidelines for the design and conduct of clinical trials on dentine hypersensitivity. J Clin Periodontol. 1997;24(11):808–13.
30. Schiff T, Dotson M, Cohen S, De Vizio W, McCool J, Volpe A. Efficacy of a dentifrice containing potassium nitrate, soluble pyrophosphate, PVM/MA copolymer, and sodium fluoride on dentinal hypersensitivity: a twelve week clinical study. J Clin Dent. 1994;5(Spec No):87–92.
31. Närhi MV. The characteristics of intradental sensory units and their responses to stimulation. J Dent Res. 1985;64(Spec No):564–71.
32. Orchardson R, Collins WJ. Thresholds of hypersensitive teeth to 2 forms of controlled stimulation. J Clin Periodontol. 1987;14(2):68–73.
33. Orchardson R, Collins WJ. Clinical features of hypersensitive teeth. Br Dent J. 1987; 162(7):253–6.
34. Gillam DG, Newman HN. Assessment of pain in cervical dentinal sensitivity studies. A review. J Clin Periodontol. 1993;20(6):383–94.
35. Bekes K, Hirsch C. What is known about the influence of dentine hypersensitivity on oral health-related quality of life? Clin Oral Investig. 2013;17 Suppl 1:S45–51.
36. Boiko OV, Baker SR, Gibson BJ, Locker D, Sufi F, Barlow AP, Robinson PG. Construction and validation of the quality of life measure for dentine hypersensitivity (DHEQ). J Clin Periodontol. 2010;37(11):973–80.
37. Dowell P, Addy M, Dummer P. Dentine hypersensitivity: aetiology, differential diagnosis and management. Br Dent J. 1985;158(3):92–6.
38. West NX. Dentine hypersensitivity: preventive and therapeutic approaches to treatment. Periodontol 2000. 2008;48(1):31–41.
39. Chu CH, Lam A, Lo EC. Dentin hypersensitivity and its management. Gen Dent. 2011;59(2):115–22. quiz 123–4.
40. Drisko CH. Dentine hypersensitivity: dental hygiene and periodontal considerations. Int Dent J. 2002;52:385–93.

41. Moseley JB, O'Malley K, Petersen NJ, Menke TJ, Brody BA, Kuykendall DH, Hollingsworth JC, Ashton CM, Wray NP. A controlled trial of arthroscopic surgery for osteoarthritis of the knee. N Engl J Med. 2002;347(2):81–8.
42. Huskisson EC. Simple analgesics for arthritis. Br Med J. 1974;4(5938):196–200.
43. Orchardson R, Gillam DG. Managing dentin hypersensitivity. J Am Dent Assoc. 2006;137(7):990–8.
44. Gillam DG, Seo HS, Bulman JS, Newman HN. Perceptions of dentine hypersensitivity in a general practice population. J Oral Rehabil. 1999;26(9):710–4.
45. Markowitz K, Kim S. The role of selected cations in the desensitization of intradental nerves. Proc Finn Dent Soc. 1992;88 Suppl 1:39–54.
46. Poulsen S, Errboe M, Lescay Mevil Y, Glenny AM. Potassium containing toothpastes for dentine hypersensitivity. Cochrane Database Syst Rev. 2006;19(3), CD001476.
47. Addy M, Mostafa P. Dentine hypersensitivity II. Effects produced by the uptake in vitro of toothpastes onto dentine. J Oral Rehabil. 1989;16(1):35–48.
48. Addy M, Mostafa P, Newcombe RG. Dentine hypersensitivity: a comparison of five toothpastes used during a 6-week period. Br Dent J. 1987;163(2):45–51.
49. Pearce NX, Addy M, Newcombe RG. Dentine hypersensitivity: a clinical trial to compare 2 strontium desensitizing toothpastes with a conventional fluoride toothpaste. J Periodontol. 1994;65(2):113–9.
50. Gillam DG, Newman HN, Bulman JS, Davies EH. Dentifrice abrasivity and cervical dentine hypersensitivity. Results 12 weeks following cessation of 8 weeks' supervised use. J Periodontol. 1992;63(1):7–12.
51. Markowitz K. The original desensitizers: strontium and potassium salts. J Clin Dent. 2009;20(5):145–51.
52. Ganss C, Schulze K, Schlueter N. In: Van Loveren C editor. Toothpastes. Toothpaste and erosion. Monogr Oral Sci. 2013;23:88–99.
53. Blong MA, Volding B, Thrash WJ, Jones DL. Effects of a gel containing 0.4% stannous fluoride on dentinal hypersensitivity. Dent Hyg (Chic). 1985;59(11):489–92.
54. Snyder RA, Beck FM, Horton JE. The efficacy of a 0.4% stannous fluoride gel on root surface hypersensitivity. J Dent Res. 1985;62:201. abstr 237.
55. Thrash WJ, Dodds WJ, Jones DL. The effect of stannous fluoride on dentine hypersensitivity. Int Dent J. 1994;44 Suppl 1:107–18.
56. Schiff T, He T, Sagel L, Baker RJ. Efficacy and safety of a novel stabilized stannous fluoride and sodium hexametaphosphate dentifrice for dental hypersensitivity. J Contemp Dent Pract. 2006;7(2):1–8.
57. Schiff T, Saletta L, Baker RA, Winston JL, He T. Desensitizing effect of a stabilized stannous fluoride/sodium hexametaphosphate dentifrice. Compend Contin Educ Dent. 2005;26(9 Suppl 1):35–40.
58. He T, Barker ML, Qaqish J, Sharma N. Fast onset sensitivity relief of a 0.454% stannous fluoride dentifrice. J Clin Dent. 2011;22(Spec issue 2):46–50.
59. He T, Cheng R, Biesbrock AR, Chang A, Sun L. Rapid desensitizing efficacy of a stannous-containing sodium fluoride dentifrice. J Clin Dent. 2011;22(Spec issue 2):40–5.
60. Pashley DH. Dentin permeability, dentin sensitivity and treatment through tubule occlusion. J Endod. 1986;12(10):465–74.
61. Pashley DH, Galloway SE. The effects of oxalate treatment on the smear layer of ground surfaces of human dentine. Arch Oral Biol. 1985;30(10):731–7.
62. Earl JS, Topping N, Elle J, Langford RM, Greenspan DC. Physical and chemical characterization of dentin surface following treatment with Nova Min technology. J Clin Dent. 2011;22(Spec issue 3):62–7.
63. Du Min Q, Bian Z, Jiang H, Greenspan DC, Burwell AK, Zhong J, Tai BJ. Clinical evaluation of a dentifrice containing calcium sodium phosphosilicate (NovaMin) for the treatment of dentin hypersensitivity. Am J Dent. 2008;21(4):210–4.

64. Pradeep AR, Anuj SJ. Comparison of the clinical efficacy of a dentifrice containing calcium sodium phosphosilicate with a dentifrice containing potassium nitrate and a placebo on dentinal hypersensitivity. J Periodontol. 2010;81(8):1167–73.
65. Litkowski L, Greenspan DC. A clinical study of the effect of calcium sodium phosphosilicate on dentin hypersensitivity- proof of principle. J Clin Dent. 2010;21(Spec issue 3):77–81.
66. Salian S, Thakur S, Kulkarni S, LaTorre G. A randomized controlled clinical study evaluating the efficacy of two desensitizing dentifrices. J Clin Dent. 2010;21(Spec issue 3):82–7.
67. Petrou I, Heu R, Stranick M, Lavender S, Zaidel L, Cummins D, Sullivan RJ, Hsueh C, Gimzewski JK. A breakthrough therapy for dentine hypersensitivity: how dental products containing 8% arginine and calcium carbonate work to deliver effective relief of sensitive teeth. J Clin Dent. 2009;20(Spec issue 1):23–31.
68. Lavender SA, Petrou I, Heu R, Stranick MA, Cummins D, Kilpatrick-Liverman L, Sullivan RJ, Santarpia RP. Mode of action studies on a new desensitizing dentifrice containing 8 % arginine, a high cleaning calcium carbonate system and 1450 ppm fluoride. Am J Dent. 2010;3(Spec issue A):14A–9.
69. Hamlin D, Williams KP, Delgado E, Zhang YP, DeVizio W, Mateo LR. Clinical evaluation of the efficacy of a desensitizing paste containing 8.0 % arginine and calcium carbonate for the in-office relief of dentin hypersensitivity associated with dental prophylaxis. Am J Dent. 2009;22(Spec issue A):16A–20.
70. Elias Boneta AR, Galán Salás RM, Mateo LR, Stewart B, Mello S, Arvanitidou LS, et al. Efficacy of a mouthwash containing 0.8% arginine, PVM/MA copolymer, pyrophosphates, and 0.05% sodium fluoride compared to a commercial mouthwash containing 2.4% potassium nitrate and 0.022% sodium fluoride and a control mouthwash containing 0.05% sodium fluoride on dentine hypersensitivity: a six-week randomized clinical study. J Dent. 2013;41 Suppl 1:S34–41.
71. Hu D, Stewart B, Mello S, Arvanitidou L, Panagakos F, De Vizio W, et al. Efficacy of a mouthwash containing 0.8% arginine, PVM/MA copolymer, pyrophosphates, and 0.05% sodium fluoride compared to a negative control mouthwash on dentin hypersensitivity reduction. A randomized clinical trial. J Dent. 2013;41 Suppl 1:S26–33.
72. Vano M, Derchi G, Barone A, Covani U. Effectiveness of nano-hydroxyapatite toothpaste in reducing dentin hypersensitivity: a double-blind randomized controlled trial. Quintessence Int. 2014;45(8):703–11.
73. Orchardsen R, Gillam D. Managing dentin hypersensitivity. J Am Dent Assoc. 2006;137(7):990–7.
74. Thanatvarakorn O, Nakashima S, Sadr A, Prasansuttiporn T, Ikeda M, Tagami J. In vitro evaluation of dentinal hydraulic conductance and tubule sealing by a novel calcium-phosphate desensitizer. J Biomed Mater Res B Appl Biomater. 2013;101(2):303–9.
75. Endo H, Kawamoto R, Takahashi F, Takenaka H, Yoshida F, Nojiri K, Takamizawa T, Miyazaki M. Evaluation of a calcium phosphate desensitizer using an ultrasonic device. Dent Mater J. 2013;32(3):456–61.
76. Gandolfi MG, Silvia F, H PD, Gasparotto G, Carlo P. Calcium silicate coating derived from Portland cement as treatment for hypersensitive dentine. J Dent. 2008;36(8):565–78.
77. Sgolastra F, Petrucci A, Severino M, Gatto R, Monaco A. Lasers for the treatment of dentin hypersensitivity: a meta-analysis. J Dent Res. 2013;92(6):492–9.
78. Ladalardo TC, Pinheiro A, Campos RA, Brugnera Júnior A, Zanin F, Albernaz PL, Weckx LL. Laser therapy in the treatment of dentine hypersensitivity. Braz Dent J. 2004;15(2): 144–50.
79. Sgolastra F, Petrucci A, Gatto R, Monaco A. Effectiveness of laser in dentinal hypersensitivity treatment: a systematic review. J Endod. 2011;37(3):297–303.
80. Yilmaz HG, Kurtulmus-Yilmaz S, Cengiz E, Bayindir H, Aykac Y. Clinical evaluation of Er, Cr:YSGG and GaAlAs laser therapy for treating dentine hypersensitivity: a randomized controlled clinical trial. J Dent. 2011;39(3):249–54.

81. Birang R, Poursamimi J, Gutknecht N, Lampert F, Mir M. Comparative evaluation of the effects of Nd:YAG and Er:YAG laser in dentin hypersensitivity treatment. Lasers Med Sci. 2001;22(1):21–4.
82. Orchardson R, Peacock JM, Whitters CJ. Effect of pulsed NdYAG laser irradiation on action potential conduction in isolated mammalian spinal nerves. Lasers Surg Med. 1997;21(2):142–8.
83. Lin P-Y, Cheng Y-W, Chu C-Y, Chien K-L, Lin C-P, Tu Y-K. In-office treatment for dentin hypersensitivity: a systematic review and network meta-analysis. J Clin Periodontol. 2013;40(1):53–64.
84. Bekes K, John MT, Schaller HG, Hirsch C. Oral health-related quality of life in patients seeking care for dentin hypersensitivity. J Oral Rehabil. 2009;36(1):45–51.
85. Adelaide University special topic nr 6. Dentine hypersensitivity—is it an erstwhile problem or a modern day enigma? https://www.adelaide.edu.au/…/hypersensitivity/Sensit.
86. Chambrone L, Pannuti CM, Tu YK, Chambrone LA. Evidence-based periodontal plastic surgery. II. An individual data meta-analysis for evaluating factors in achieving complete root coverage. J Periodontol. 2012;83(4):477–90.
87. Schmidlin PR, Sahrmann P. Current management of dentin hypersensitivity. Clin Oral Investig. 2013;17 Suppl 1:S55–9.