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DENTIN HYPERSENSITIVITY:

Consensus-Based Recommendations for the Diagnosis & Management of Dentin Hypersensitivity

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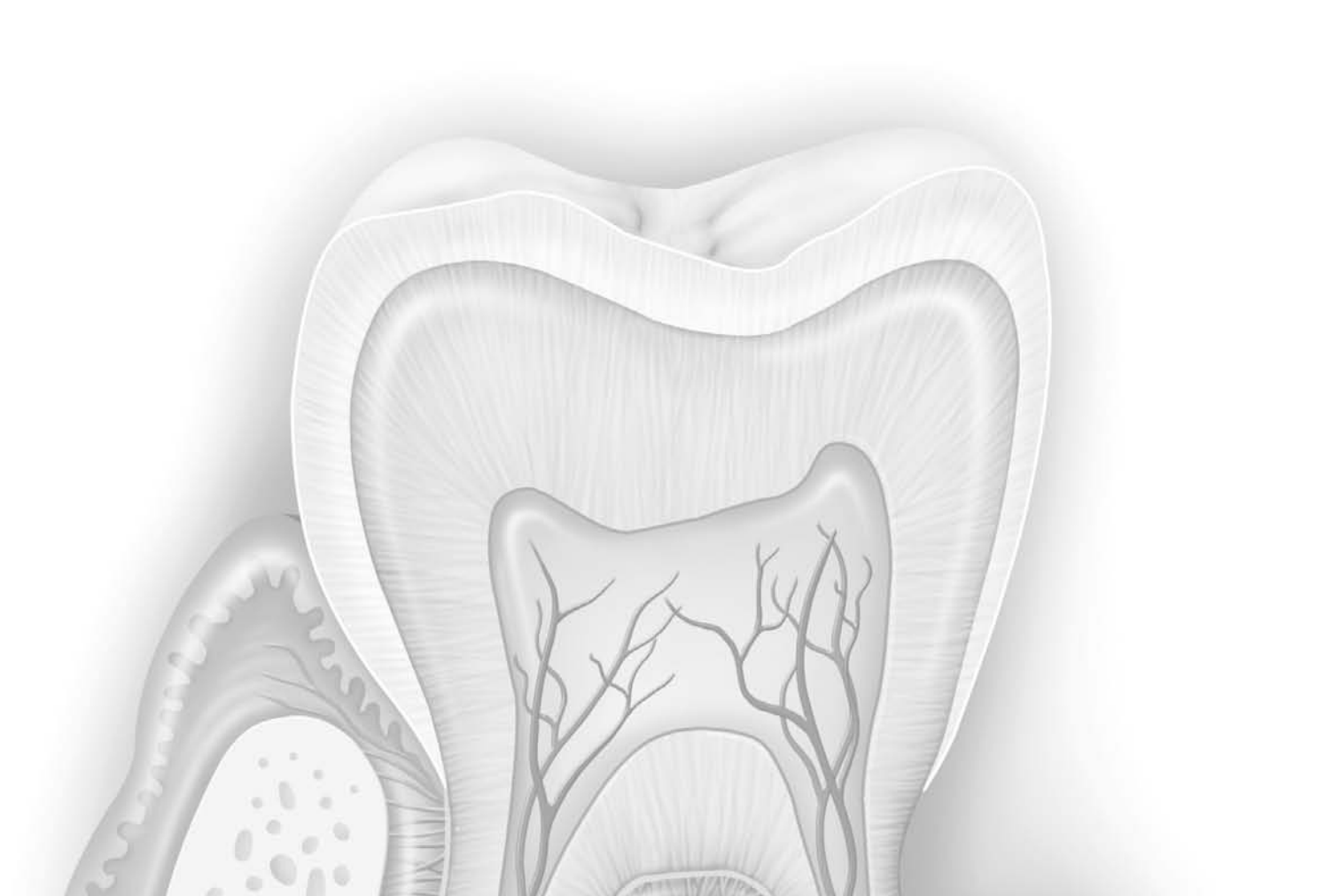

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Consensus-Based Recommendations for the Diagnosis and Management of Dentin Hypersensitivity

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Introduction

Pain and discomfort have many causes in dentistry, and are typically one of the major reasons for unscheduled or emergency dental visits. As American dentistry has strived to bring the diseases of infectious origin, caries and periodontitis, under control, a new set of conditions is emerging—as a consequence of the 21st century lifestyle. Gingival recession may well be caused by overly enthusiastic oral hygiene, and acid wear may be becoming more prevalent in all ages due to the modern acid-containing diet. Both of these conditions lead to exposed dentin, which under the right circumstances, leads to the initiation of dentin hypersensitivity—their sole and common symptom. The final common path for dentin hypersensitivity is the activation of pulpal nerves. The successful use of potassium-containing products to treat both types of tooth sensitivity justifies the thorough treatment of the clinical condition. This consensus monograph purposes to help clinicians define, diagnose, and treat a condition that appears to be increasing in incidence. Furthermore, as all forms of vital bleaching—a treatment of ever-increasing commonality in the age of esthetic dentistry—are associated with some level of tooth sensitivity, that condition and its management are addressed as well.

Dentin hypersensitivity is best defined as a "short, sharp pain arising from exposed dentin in response to stimuli typically thermal, evaporative, tactile, osmotic, or chemical, and which cannot be ascribed to any other form of dental defect or pathology."[1] Dentin hypersensitivity is a common oral condition that affects as many as 57% of patients.[2-8] It has been described as the "common cold of dentistry"[9] by some and "toothbrush disease" by others, when it occurs in the presence of gingival recession.[10]

Dentin hypersensitivity is often episodic and the strategies for managing the condition are remarkably varied. Recognizing these issues, the Medical College of Georgia School of Dentistry, under the leadership of Dean Connie Drisko and her world-renowned team of category experts—Drs. Pashley, Tay, Haywood, and Collins—undertook to evaluate the condition and present a consensus statement regarding definition, etiology, diagnosis, and treatment. In August 2008, a consensus panel recommendation, peer-reviewed by the associate dean for research, Gerard Kugel, and assistant dean of international relations, Noshir Mehta, of Tufts University School of Dental Medicine, was produced.

METHODS

The panel considered data from an extensive literature search and recognizes that dentin hypersensitivity is complex and is best diagnosed by a process of elimination by a thorough dental screening, examination, and dental history. Conditions that must be excluded include cracked-tooth syndrome, fractured restorations, chipped teeth, dental caries, gingival inflammation, post-restorative sensitivity, marginal leakage, pulpitis, and palatogingival grooves. The panel brought together all of its considerations to form a set of consensus recommendations to guide practitioners through diagnosis and case management. The consensus recommendations are supported by an algorithm (Figure 1) for quick reference by the practitioner to the elements and critical steps required in making diagnostic decisions and the appropriate action based upon the findings.

DATA COLLECTION—LITERATURE SEARCH

An extensive computer (MEDLINE) and hand search of the literature identified original articles and reviews for the period 1966 to 2008.

DEFINITION

Dentin hypersensitivity is characterized by short, sharp pain arising from exposed dentin in response to stimuli—typically thermal, evaporative, tactile, osmotic, or chemical—that cannot be attributed to any other dental defect or pathology.

PRESENTING SIGNS AND SYMPTOMS

Physiology

- The presence of tubules renders dentin permeable to fluid movement. The number of tubules per unit area varies depending on location, because of the decreasing area of the dentin surface in a pulpal direction. Dentinal tubules follow a sinuous course from the amelodentinal junction and from the cementodentinal junction and are conical, being wider at the pulpal end than at the periphery. These tubules are interconnected by an intricate and profuse system of canaliculi that branch off from the main tubules at different angles.
- The three essential features required for dentin hypersensitivity to occur are:

  1) the presence of exposed dentin surfaces;
  2) open tubule orifices on the exposed dentin surface; and
  3) patent tubules leading to a vital pulp.

- The short, sharp pain arising from exposed dentin in response to thermal, evaporative, tactile, or osmotic stimuli is a result of minute inward or outward movement of dentinal fluid inside tubules that activate pulpal nerve fibers.
- Occlusion of dentin tubular orifices by a smear layer created during tooth brushing or by silica particles in a dentifrice result in reduction of fluid movements within the dentinal tubules. This physical blockade may partially account for the effectiveness of desensitizing dentifrices.
- Changes occur in the dentin as a result of age or trauma. Secondary dentin is deposited throughout life, and the formation of peritubular dentin and/or deposition of intratubular whitlockite (ie, magnesium-substituted tricalcium phosphate) crystals may ultimately result in partial or complete obturation of the dentinal tubules, producing dead tracts and areas of sclerotic dentin. Traumatic injuries to the tooth may result in the deposition of an irregular layer of tertiary dentin that has fewer tubules. As these newly deposited tubules are not continuous with those in primary dentin, they provide an effective barrier to diffusion and rapid fluid movements and contribute to the reduction of dentin hypersensitivity.

Mechanism of Action

By far, the most widely accepted theory for dentin hypersensitivity is the hydrodynamic theory. When dentinal tubules in vital teeth are exposed after cementum or enamel is lost due to erosion, abrasion, dental manipulation, or a tooth defect, fluid within the dentinal tubules may flow in either an inward or outward direction depending on pressure differences in the surrounding tissue. A cold stimulus causes the tissue fluid volume to shrink slightly, and heat causes it to expand. Strongly osmotic sugar or sour solutions cause fluid to be drawn out of the tubules. An air blast on the tooth dries a tiny portion of fluid at the distal end of the tubule, causing a significant outer flow of fluid in the tubule. Touching the tooth with a dental instrument or periodontal cleansing aid forces a small amount of fluid into the tubule. These intratubular fluid shifts, in turn, activate mechanoreceptors in intratubular nerves or in the superficial pulp, and are perceived as pain by the patient.

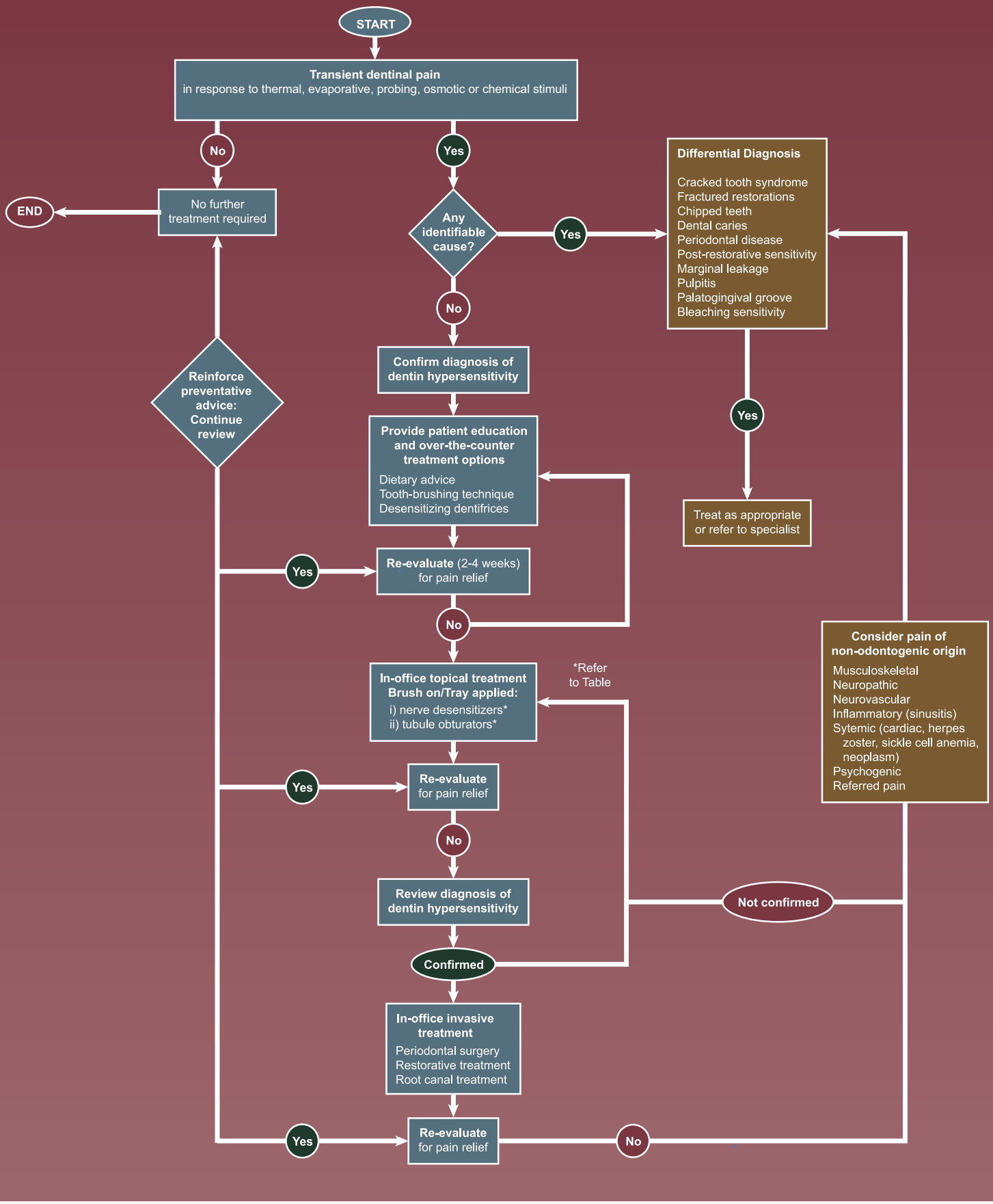

Figure 1 Flowchart showing the clinical management of dentin hypersensitivity.

Diagnosis

- Differential diagnosis is essential in order to exclude other conditions with similar symptoms where dentin is exposed and sensitive, such as chipped teeth, fractured cusps, cracked teeth, caries, and restorations with marginal deficiencies/leakage. Arriving at a correct differential diagnosis requires careful clinical and radiographic examinations and a thorough dental history (Figure 1).

Etiology

- The most important factor in the etiology of dentin hypersensitivity is exposed dentin as a result of gingival recession associated with exposure of root surfaces and/or as a result of loss of enamel associated with tooth wear or trauma; followed by opening of the dentinal tubules (ie, loss of cementum or removal of any smear layer).
- Traumatic tooth brushing in an otherwise healthy dentition is often undiagnosed in adolescents and early adults. Subclinical soft and hard tissue abrasion lesions are most likely a precursor of gingival recession and tooth wear, and thus dentin hypersensitivity.
- Tooth wear refers to the irreversible loss of tooth structure and includes conditions such as abrasion, erosion, attrition, and abfraction. Development of wedge-shaped cervical lesions is often associated with abrasion and occlusal hyperfunction. Some lesions may be located subgingivally, out of reach of a toothbrush, and have been referred to as abfraction to describe the mechanism associated with the cervical loss of enamel and dentin.
- Although there are many causes of noncarious cervical lesions of dentin, improper brushing, especially in the presence of an acidic diet, is a major cause.

Epidemiology

- The prevalence of dentin hypersensitivity varies, but averages about 57% and peaks between 20 to 40 years of age.
- The loss of enamel in the absence of gingival recession can involve any location on the tooth and is usually a result of the combined actions of attrition, abrasion, erosion, and abfraction.
- The individual sites around the mouth with the highest prevalence of dentin hypersensitivity are associated with gingival recession and are located on the facial surfaces of canines > premolars > incisors > molars.
- Use of a highly abrasive dentifrice may also cause additional soft tissue damage and tooth wear leading to hypersensitivity, although this phenomenon is not well-documented in the literature.
- Dentin hypersensitivity peaks in the first few days after scaling and root planing or periodontal surgery and usually is substantially reduced by 8 weeks, but can vary from months to more than 30 years.

Clinical Trials

- Clinical trials on dentin hypersensitivity should use randomized group assignments, be double-masked and contain a placebo product that is identical to the test product except that it does not contain the active ingredient.
- It is critical to evaluate the placebo effect, which can be very strong in such studies.
- Conclusions derived from early studies on dentin hypersensitivity using single-masked methods, or inappropriate stimuli, such as electric pulp testing, should be viewed with caution.
- Limited evidence would indicate tooth brushing without dentifrice lowers hypersensitivity scores (promotes the formation of a smear layer) while brushing with a dentifrice increases dentin hypersensitivity scores (removes the smear layer) unless the dentifrice contains a potassium-containing desensitizing agent.
- Although a recent meta-analysis of six clinical trials using potassium-containing desensitizing dentifrices demonstrated reductions in the patients' perceived symptoms of dentin hypersensitivity compared to control dentifrices, the scientific evidence supporting the use of potassium salts to reduce nerve activity is based largely on in vivo animal studies and one recent human in vivo study.

Vital Bleaching Sensitivity

- All forms of vital bleaching are associated with some level of sensitivity.
- The history of sensitive teeth as well as the patient's response during examination to explorer touch or air can be a reasonable predictor that sensitivity will occur during bleaching.
- Tooth sensitivity is the single most significant factor in noncompliance with, or failure to complete, a bleaching regimen, and must be understood to be able to manage treatment.
- Tooth sensitivity is the most common side effect of bleaching, and may be caused primarily by the peroxide penetration to the pulp.
- Treatment of bleaching sensitivity involves many possible options. Twice daily use of a potassium nitrate-containing toothpaste for 2 weeks before and during the regimen can reduce or avoid sensitivity from bleaching.
- Further alleviation of bleaching sensitivity can be achieved

by using bleaching materials containing potassium nitrate, or by applying a dentifrice or professional product containing potassium nitrate in a well-fitted tray.

Quality of Life

- Early identification of risk factors and appropriate intervention, including changing destructive habits, are essential in preventing the onset of dentin sensitivity and ensuring long-term success in managing dentin hypersensitivity.
- Quality of life, esthetic concerns, and chronic pain are strong motivators for patients to seek treatment for their dentin hypersensitivity.
- Quality of life is significantly affected by the symptoms of hypersensitivity. Sufferers may no longer enjoy their favorite foods and beverages, allowing hot drinks to cool and putting less or no ice in cold drinks, using a straw to deflect cold drinks away from certain teeth. Many sufferers are only 'somewhat bothered' and about one third are 'very bothered'.
- The two conditions, gingival recession and tooth wear, most commonly leading to dentin hypersensitivity alter the appearance and visual appeal of the mouth, and thus facial expression and perceived beauty.

Treatment (Figure 1)

- Prevention is the most cost-effective treatment option.
- The first recommendation by the dentist or dental hygienist should include cessation of predisposing destructive habits and the twice-daily use of a desensitizing dentifrice.
- Tray application of potassium nitrate can be an effective episodic treatment for sensitivity according to limited practice-based evidence.
- Clinical trials have shown that twice-daily use of desensitizing dentifrices improves hypersensitivity and increases in effectiveness over time.
- If, after using a desensitizing dentifrice, the patient's dentin hypersensitivity remains a problem, clinicians should re-evaluate the differential diagnosis and consider in-office treatments beginning with topically applied desensitizing agents. After the diagnosis is reconfirmed, including the elimination of other causative factors such as undiagnosed caries or cracked teeth, other methods of treatment including gingival grafting may need to be considered.
- A periodontist should be consulted before placement of restorative materials on the roots to assess the potential for future use of gingival grafts for root coverage, as placement of any bonded restoration prior to grafting may diminish the success rate of such procedures.

Maintenance

- Clinicians should take careful histories of their patients' dietary habits and make patients aware of the importance of erosive influences.
- Current in-office treatment modalities include use of professionally applied desensitizing agents (see Table and Figure 2 through Figure 5 in "Dentin Hypersensitivity: Current State of the Art and Science").
- Evidence-based recommendations regarding consumer products for management of dentin hypersensitivity should be communicated to patients, including use of potassium nitrate-containing, low-abrasivity dentifrice and soft toothbrushes.
- Patients should demonstrate their tooth brushing technique to the dental hygienist or dentist at every appointment until they have mastered proper brushing techniques.
- At-risk patients should avoid whitening toothpastes, and other dentifrices with high abrasivity values as these tend to contribute to removal of the smear layer and further tooth wear.

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Table: Professionally Applied Dentin Desensitizers

PRODUCT CLASS	MODE OF ACTION	BRAND NAME	MEASURES	CLINICAL RESULTS
Oxalate products	Tubule occlusion (see Figure 2)	BisBlock (Bisco), Protect (SunStar), SuperSeal (Phoenix), D'Sense Crystal, (Centrix Direct)	Air blast, tactile, *in vivo*	Camps & Pashley, 2003; 3% potassium oxalate reduced clinical dentin sensitivity ($p<0.01$) to air or tactile over water controls.
		Sensodyne Sealant (GlaxoSmithKline),Pain-Free (Parkell), MS-Coat (Sun Medical)	Patient self-reports	Pillon *et al.*, 2004; 3% oxalate reduced sensitivity compared to placebo gel.
Calcium phosphate desensitizers	Tubule occlusion (see Figure 3)	D/Sense 2 (Centrix Direct), Quell Desensitizer (Pentron Clinical Technologies)	Quantitative permeability reductions, in vitro	Kolker *et al.*, 2002; This *in vitro* study reported that the largest permeability reductions were obtained with SuperSeal (oxalate) > Hurri Seal = Gluma = D/Sense 2 = Seal & Protect. No clinical reports available.
5% NaF varnish	Tubule occlusion	Durphat (Colgate Oral Pharm.), Duraflor (Pharmascience)	Tactile, cold, air blast	Merika *et al.*, 2006; In a 4 week study, both Duraphat and Super Seal, an oxalate product were effective at reducing dentin sensitivity.
		Cavity Shield (OMNI Oral Pharm.), Fluor Protector (Ivoclar Vivadent), AllSolutions Fluoride Varnish (Dentsply)	Air blast, ice	Ritter *et al.*, 2006; Both Duraphat and AllSolutions Fluoride Varnish were effective in reducing dentin sensitivity for 24 weeks.
Glutaralde-hyde-HEMA	Protein precipitation/ tubule occlusion (see Figure 4)	Gluma Desensitizer (Heraeus Kulzer), MicroPrime G (Danville), Glu/Sense (Centrix Direct), HemaSeal G (Germiphene)	Tactile, air blast	Kakaboura *et al.*, 2005; This clinical trial compares the desensitizing effects of Gluma vs. One-Step adhesive. Both were effective immediately and at 8 weeks but Gluma gave better desensitization after 9 months compared to a water control.
		Gluma vs. Fuji VII	Air blast	Polderman *et al.*, 2007; compared Gluma to Fuji VII GIC in a split mouth design. Although both treatments reduced sensitivity, Fuji VII was better after 24 months.
HEMA + other	Protein ppt./tubule occlusion	Hurri-Seal Dentin Desensitizer (Beutlich Pharm.), HemaSeal & Cide (Germiphene), MicroPrime B Desensitizer (Danville)	Quantitative permeability reductions, in vitro	Kolker *et al.*, 2002; This in vitro study found HurriSeal to be as effective as Gluma or D/Sense 2 or Seal & Protect.
NaF, SnF_2	Tubule occlusion	DentinBloc (Colgate Oral Pharm.)	Air blast Tactile	Thrash *et al.*, 1992; In this doubleblinded clinical trial, Dentin Bloc was compared to Gel Kem, both applied 2 X/day vs. a water control group.The DentinBloc treated teeth were less sensitive after 2 weeks than the other two groups. An additional group revealed that Dentin Bloc could desensitize in 15 min. participants in the new dentifrice group demonstrated statistically significant improvements ($p<0.05$) in tactile and air blast sensitivity, as compared to those using the positive and negative control dentifrices.
		DentiBloc (Colgate Oral Pharm.) vs. Pain-Free (Parkell)	Air blast, tactile	Morris *et al.*, 1999; Compared clinical effects of DentinBloc vs.Pain-Free. Both products and the placebo decreased dentin sensitivity for up to 3 months. No significant differences were found between the 3 groups.

	PRODUCT CLASS	MODE OF ACTION	BRAND NAME	MEASURES	CLINICAL RESULTS
	Light-cured adhesives	Tubule occlusion (see Figure 5)	Seal & Protect (Dentsply)	Air blast	Baysan *et al*., 2003; Seal & Protect decreased dentin sensitivity for 19 months but no controls were used.
			One-Step (Bisco) vs. Gluma	Tactile, air blast	Kakaboura *et al*., 2005; Both treatments reduced DH immediately and after 8 weeks compared to water treatment placebo.
			Single Bond (3M-ESPE) vs. MS Coat (Sun Medical)	Air blast, cold, tactile	Prati *et al*., 2001; After 4 weeks, both treatments were equally effective at reducing sensitivity using all stimuli. This was a randomized, double-blind study.
			Prime & Bond 2.1 (Dentsply)	Air blast, cold	Swift Jr *et al*., 2001; Application of Prime & Bond 2.1 significantly reduced sensitivity compared to pretreatment controls for up to 24 weeks.
				Quantitative permeability	Tagami *et al*., showed that many adhesives reduce dentin permeability by ppt. plasma proteins.
	Glass-ionomer cements	Tubule occlusion	Vitrabond-like (3M-ESPE) vs. Gluma (Heraeus Kulzer)	Tactile, cold	Tantbirojn *et al*., 2006; A Vitrabond-like GIC was compared to Gluma over a 12 month study. Both treatments significantly reduced dentin sensitivity, but the GIC material was generally more effective.
			Fuji VII (GC) vs. Gluma (Heraeus Kulzer)	Air blast	Polderman *et al*., 2007; compared Fuji VII GIC with Gluma over 25 months. Although both materials were effective, the GIC was more effective at every time period.
	Desensitizing dentifrices	Lower nerve sensitivity	5% KNO_3 (Sensodyne)	Air blast, cold, tactile	Nagata *et al*., 1994; Relief of subjective symptoms throughout the 12 weeks' examination was noted in 67% of the subjects in patients using a 5% KNO3-containing dentifrice; Schiff et al., 2000; After 4- and 8-weeks' use of a 5% KNO3-containing dentifrice, participants in the new dentifrice group demonstrated statistically significant improvements ($p < 0.05$) in tactile and air blast sensitivity, as compared to those using the positive and negative control dentifrices.
	Potassium-containing products	Lower nerve sensitivity	3-5% KNO_3 radent Products, 3%), Relief (Discus Dental, 5%), Desensitize (Den Mat, 5%)	Self-reports	Haywood *et al*., 2001; In a clinical trial of tray bleaching, 16 of 30 patients developed tooth sensitivity. Of the 16 sensitive patients, 12 used 5% KNO_3 gel containing 1000 ppm F for 10-30 min to reduce sensitivity. Eleven of the 12 obtained relief. No placebo gels were used.

Dentin Hypersensitivity: Current State of the Art and Science

The consensus definition of dentin hypersensitivity is tooth pain that is characterized by brief, sharp, well-localized dentin pain in response to thermal, evaporative, tactile, osmotic, or chemical stimuli that cannot be attributed to any other dental diseases. The use of clinical descriptors (ie, brief, sharp, well-localized pain) distinguishes dentinal pain from pulpal pain that is prolonged, dull/aching, and poorly localized and lasts far longer than the applied stimulus. This is very important as the treatment of these two types of pain is very different. The definition of dentin hypersensitivity also requires a differential diagnosis as there are many other clinical conditions where dentin is exposed and sensitive, such as chipped teeth, fractured cusps, caries, and restorations with marginal gaps with leakage. Indeed, it is possible to have sensitive cervical dentin and sensitive marginal gaps in class II restorations in the same teeth. Arriving at a correct differential diagnosis requires careful clinical and radiographic examinations.

Inclusion criteria for the diagnosis of dentin hypersensitivity are 1) the presence of exposed dentin surfaces; 2) open tubule orifices on the exposed dentin surface; and 3) patent tubules leading to a vital pulp. The exposure of dentin often occurs as a result of removal of cervical cementum during scaling and root planing, during the finishing of restorations, or by excessive toothbrushing by the patient, especially after application of acidic food and drinks to exposed dentin.[1] Patency of tubules and vitality of the pulp can be determined by blowing a gentle air stream on the tooth in question for 0.5 to 1 second while covering the adjacent teeth with gloved fingers. Nonvital teeth or impermeable dentin do not respond to air blasts.

EPIDEMIOLOGY OF DENTIN HYPERSENSITIVITY

The prevalence of dentin hypersensitivity varies widely as a result of the use of widely different methods of evaluations. Those reports that rely on questionnaires risk inappropriate self-reports of dentin hypersensitivity by patients who may have tooth sensitivity from many different etiologies. When surveys have been published where clinicians have examined the patients, the prevalence of dentin hypersensitivity usually ranges from 4% to 57%. Although the age range for dentin hypersensitivity varies from 15 to 70 years, the peak incidence is between 20 to 40 years. Generally, dentin hypersensitivity is thought to appear with gingival recession in the third to fourth decade of life. The apparent decrease in dentin hypersensitivity in older patients may reflect reductions in dentin permeability reported in aged teeth[1] and reductions in innervation density with age.

The highest incidence of dentin hypersensitivity has been reported on the buccal cervical area of teeth. The teeth most commonly affected are canines > premolars > incisors > molars. Interestingly, a significantly higher proportion of left vs right contralateral teeth was reported in right-handed patients with dentin hypersensitivity. Addy and his colleagues[2] reported that all sensitive teeth have very low plaque scores, suggesting that toothbrushing with dentifrice may facilitate the development of dentin hypersensitivity. Others report that there is a positive correlation between dentin hypersensitivity and plaque scores. However, brushing without dentifrice lowers dentin hypersensitivity scores, while brushing with toothpaste increases them, and argues in favor of toothpastes contributing to dentin hypersensitivity, presumably because of their abrasiveness.

HISTOPATHOLOGY OF DENTIN HYPERSENSITIVITY

To delineate the possible correlation between clinical symptoms of hypersensitive dentin and changes in pulpal histology, Brännström[3] ground through the enamel and into midcoronal dentin of premolars in children scheduled for extraction for orthodontic treatment to expose cross-sectioned dentinal tubules. When he tested the tactile sensitivity of the vital dentin initially, and then again after the ground dentin had been left exposed to saliva for 1 week, he found that their sensitivity had increased substantially. These teeth were then extracted and the pulps examined histologically. The pulpal region just below the cut tubules was found to be infiltrated with acute inflammatory cells. Brännström attributed the increase in dentin sensitivity to the presence of acute inflammatory cells. We now know that the ground dentin

was initially covered with a smear layer that disappears from dentin surfaces within 1 week. Thus, the increase in sensitivity reported by Brännström may have been a result, in part, of the loss of smear layers making the dentin hyperconductive, as well as a result of the action of inflammatory mediators to make pulp sensory nerves more sensitive.

The effectiveness of saliva as a pulpal irritant in Brännström's study encouraged Lundy and Stanley[4] to include a wider range of patient ages and follow changes in pain responses and pulpal histopathology over a much longer time. Their classical study provided extremely valuable observations on the relationship between dentin hypersensitivity and histopathologic pulpal reactions. The authors recruited patients that had clinically asymptomatic teeth scheduled for extraction and cut deep class-V cavity preparations into dentin of those vital teeth. The empty cavities were left exposed to saliva for 1 to 120 days. Just before extracting the teeth, the pulpal responses to hot, cold, and electric pulp tests, plus the dentinal responses to probing and air blasts, were recorded. The teeth were then extracted and processed for light microscopy. When they correlated their clinical tests with histopathologic tests, the degree of dentin sensitivity to probing and air blasts increased profoundly during the first week but then fell rapidly over time. The teeth with hypersensitive dentin were associated with acute inflammation in the pulpal region just below the cut tubules. The subjective symptoms and histologic reactions were completely different at the longer time periods. The patients no longer reported sensitivity to hot or cold foods even though the cavities remained open. The histologic appearance of these pulps was mild to moderate chronic inflammation. The authors speculated that the permeability of the exposed dentin decreased between 7 to 11 days, resulting in a reduction of pulpal inflammation. Later work confirmed that dentin permeability does not remain constant, but decreases rapidly and spontaneously in vivo.[5]

Generally, cervical root dentin sensitivity does not develop such profound inflammatory reaction after periodontal treatment, because the permeability of root dentin is much lower than that of coronal dentin. However, severe reactions occur with enough frequency to make some patients reluctant to have their periodontal surgical treatments completed. Clinicians must be prepared to deal with dentin hypersensitivity to remain credible to their patients. While most cases of dentin hypersensitivity associated with periodontal treatments resolve in 7 to 10 days, severe hypersensitivity is extremely unpleasant and should be aggressively treated as soon as it appears.

CAUSATIVE THEORIES OF DENTIN HYPERSENSITIVITY

Innervation of Dentin

In the 19th century, restorative dentists knew that as soon as their burs passed through enamel and touched dentin, their patients began to feel sharp, intense pain. They deduced that there must be sensory nerves that pass from the pulp, out to the dentinal tubules, to the dentinoenamel junction (DEJ). When histologists stained teeth with special silver stains used to identify nerves, although they saw the nerve plexus in the pulp just below the odontoblast layer, they could not identify nerves passing more than 100 µm into peripheral dentin. This was very confusing. How could the DEJ be so sensitive without nerves?

The pulpodentin complex is innervated by myelinated (Aβ and Aδ) and unmyelinated C-fiber sensory nerves. Dentin sensitivity (ie, hydrodynamically stimulated dentin) is a result of the activation of Aβ and Aδ sensory nerves in dentinal tubules and near the dentin-pulp junction. Their distribution is not uniform, being most numerous (innervating 40% of tubules) over pulp horns. They are

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progressively less frequent near cervical dentin and least prevalent in root dentin (ca 3.5%). The majority of the nerves in teeth are unmyelinated C-fibers that are responsive to capsaicin and inflammatory mediators such as histamine and bradykinin but not to hydrodynamic stimuli. C-fibers contain neuropeptides such as substance P, CGRP, and neurokinin A. These fibers are in the pulp but not the dentin.

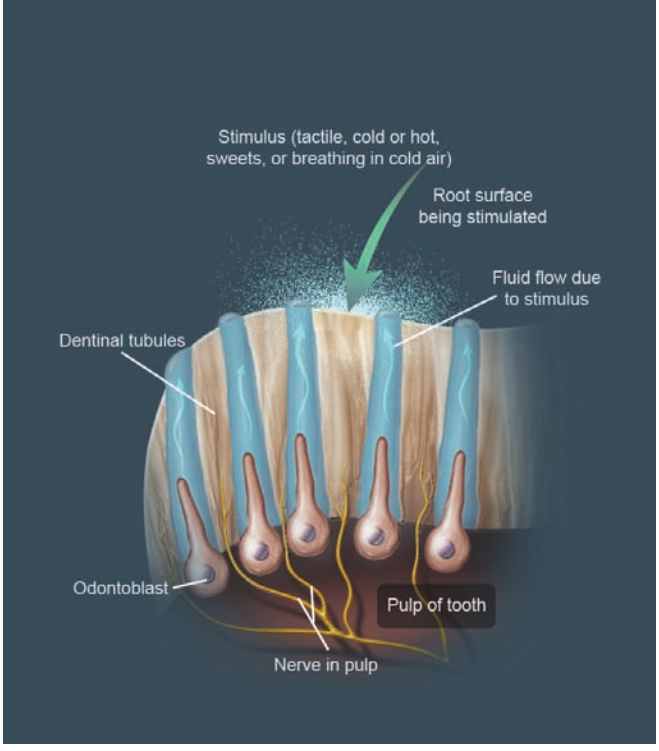

Figure 1 Gysi and later Brännström postulated that painful stimuli move fluid in or out of dentin, and that this fluid activates intradental or pulpal nerves to cause pain.

HISTORICAL PERSPECTIVE

In 1955, Kramer[6] proposed the "hydrodynamic theory" as follows: "The dentinal tubules contain fluid or semi-fluid materials and their walls are relatively rigid. Peripheral stimuli are transmitted to the pulp surface by movements of this column of semi-fluid material within the tubules." However, it was Brännström who correlated a series of in vivo experiments on painful stimulation of human teeth by negative pressure, evaporative air blasts, and chemical stimuli with measurements of fluid shifts across dentin in vitro in response to these stimuli. He popularized the hydrodynamic theory of dentin hypersensitivity through his many publications and lectures around the world.

The evidence supporting the hydrodynamic mechanism of Aδ nerve activation is based both on in vivo studies in human subjects and experimental animals. The results of human experiments confirm that open dentinal tubules are required for exposed dentin to be sensitive. A positive correlation was reported between the degree of dentin sensitivity and the density of open dentinal tubules seen in microscopic replicas of human dentin. Yoshiyama developed a method for taking biopsies of insensitive vs sensitive root surfaces using miniature diamond-encrusted coring drills and then examining the retrieved cores by scanning or transmission electron microscopy. Sensitive dentin surfaces had more and larger open tubules than insensitive areas. In vitro measurements of fluid flow through dentin disks revealed that open tubules exhibited high hydraulic conductances, but blockage of these same tubules reduced fluid flow. Thus, tubule occlusion is the basis for many professionally applied dentin desensitizing agents.

The Odontoblast Transducer Mechanism

The second possible explanation for sensitivity of the DEJ in the absence of peripheral nerves would be if the odontoblast process could serve as a sensory receptor. This would require that pulpal sensory nerves form synaptic junctions with odontoblasts. However, destruction of odontoblasts did not cause dentin to be insensitive. If odontoblasts served as sensory receptors, such dentin should be insensitive. In fact, such dentin was even more sensitive than normal. Careful transmission electron microscopy of nerves touching odontoblasts failed to demonstrate the required modifications to the plasma membranes of the nerves and the odontoblasts if they were true synapses. Thus, the hypothesis that odontoblasts serve as sensory receptors and contribute to dentin sensitivity has largely been rejected. Through a process of elimination, the third mechanism responsible for dentin sensitivity is the hydrodynamic theory of fluid flow through dentinal tubules that acts as the coupling or transducing mechanism that activates intradental nerves.

The Hydrodynamic Theory of Fluid Flow

Although the so-called "hydrodynamic stimuli" include hot and cold, tactile, evaporative, and osmotic, their final common path is fluid movement within dentinal tubules that, in turn, activate mechanoreceptors in intratubular nerves or in the superficial pulp (Figure 1). Thus, the true physiologic stimulus is inward or outward fluid shifts. This has been most difficult to measure because it involves nanoliter or picoliter fluid shifts.

Much debate has involved the question over whether exposed dentin is "sensitive" or "hypersensitive." Pain perception is a personal, subjective sensation that is influenced by a patient's previous experience, emotional state, and cultural

traditions. The two essential elements of the hydrodynamic mechanism involve the dentinal tubules and mechanosensitive nerves in the pulp. The first important element is fluid flow through dentinal tubules.

The elegant research of Matthews et al[7] over the past 15 years has added much critical understanding of stimulus-response coupling in the pulpo-dentin complex. In a series of experiments in animals and humans, Matthews and his colleagues measured the rate of spontaneous outward fluid flow in exposed dentin in cats and humans and demonstrated how this could be altered experimentally by applying positive or negative hydrostatic pressures to dentin surfaces. By manipulating positive or negative pressures of known magnitudes, Matthews and Vongsavan[7] could induce action potentials in single nerves dissected from the inferior alveolar nerve in cats and record multi-unit nerve activity from the exposed human dentin.

The second element in the hydrodynamic mechanism is the pulpal sensory nerves. They fall into the category of myelinated Aβ and Aδ, and unmyelinated C-fibers. The sharp, well-localized pain of dentin sensitivity is thought to be due primarily to Aδ nerves. All nerves have thresholds for firing. Under normal conditions, these thresholds are relatively constant. However, in patent dentinal tubules, bacterial products from plaque slowly diffuse from outside into the pulp where they may induce varying degrees of inflammation (acute and chronic). Cytokines and mediators associated with inflammation are thought to down-regulate normal sodium channels (sensitive to Tetrodotoxin [TTX]) and up-regulate the expression of TTX-resistant sodium channels such as Navl.[8] channels.[8] We speculate that mild pulpal inflammation beneath patent sensitive dentinal tubules may induce the expression of hypersensitive sodium channels that respond to smaller intratubular fluid shifts than normal sodium channels, making this dentin truly "hypersensitive." This provides the rationale for clinicians who insist that their patients maintain good plaque control. In the absence of plaque, it is thought that there is less permeation of bacterial products across dentin to induce localized, mild pulpal inflammation that is thought to be responsible for inducing hypersensitive dentin.

Finally, some extreme cases of dentin hypersensitivity may be a result of stimulation of hyperconductive dentinal tubules innervated by pulpal nerves with extra low thresholds. In the presence of localized pulpal inflammation, pulpal nerves can sprout or branch, thereby increasing the receptive field of each nerve,[9] making larger surface areas of exposed dentin more sensitive.

ETIOLOGY OF DENTIN HYPERSENSITIVITY

Gingival Recession

Perhaps the most important factor in the etiology of dentin hypersensitivity is gingival recession because it causes exposure of root surfaces. The causes of gingival recession were reviewed by Addy[10] and include the anatomy of the buccal plate of alveolar bone. As the buccal alveolar bone provides much of the local blood supply for buccal gingivae, loss of the underlying bone is associated with loss of buccal gingivae. For instance, thin, fenestrated, or absent alveolar bone predisposes someone to gingival recession. Tooth anatomy or position also affects alveolar bone thickness. Often, orthodontic tooth movement results in inadvertently moving teeth through the buccal plate that can make such sites more likely to develop gingival recession.

Poor oral hygiene may cause gingival recession indirectly by allowing for the development of periodontal disease.

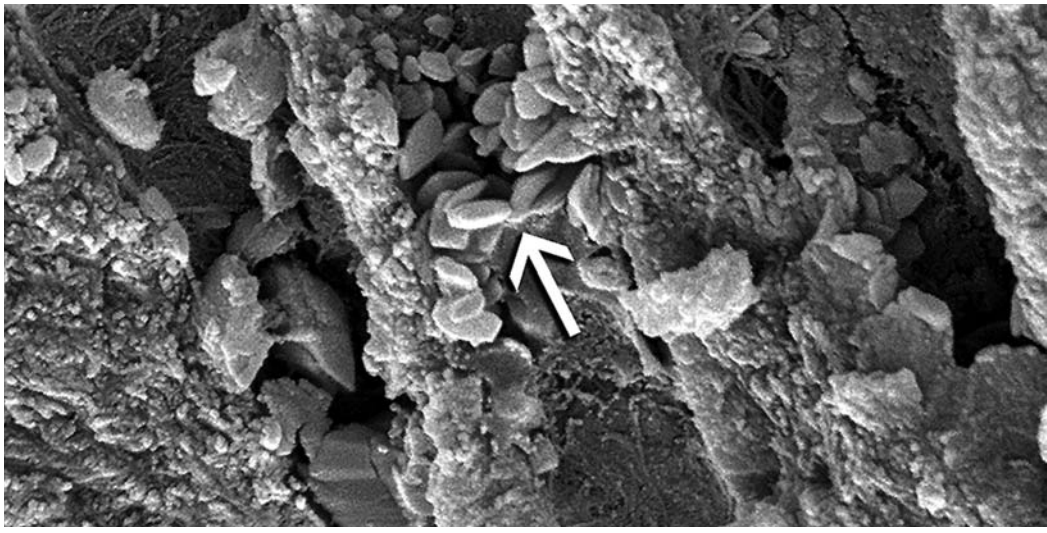

Figure 2 Scanning electron micrograph showing occlusion of dentinal tubules with calcium oxalate crystals (arrow) after the application of a slightly acidic potassium oxalate solution to acid-etched dentin.

However, gingival recession resulting from periodontal bone loss seldom occurs on buccal-cervical sites. Clinical studies have reported more gingival recession with good oral hygiene or improved oral hygiene. Indeed, the most brushed teeth with the lowest plaque scores exhibited the most gingival recession. This has led to the description of gingival recession/dentin hypersensitivity as "toothbrush disease." Because toothbrushing alone (without toothpaste) has no abrasive or erosive action on dentin, the loss of dentin is a result of the abrasivity of toothpastes. Once gingival recession has exposed root surfaces, the cementum is rapidly lost from brushing with toothpaste and/or professional cleaning.

Periodontal Disease

Dentin hypersensitivity is seen more frequently in patients with periodontitis. The prevalence of dentin hypersensitivity has been estimated to be between 60% and 98% in patients with periodontitis. Several studies have investigated changes in root dentin sensitivity after periodontal surgery. Nishida and colleagues[11] followed dentin sensitivity for 8 weeks after periodontal surgery. The highest sensitivity occurred 1 week after surgery. Immediately after surgery, the proportion of sensitive teeth increased from 21% to 36.8%. In many cases, by 8 weeks postoperatively, the sensitivity had largely resolved. The teeth of young patients (aged 19 to 29 years) showed a higher incidence and degree of postoperative hypersensitivity than did an older group (aged 40 to 61 years), and the spontaneous decrease in hypersensitivity required a longer time in the young group. During the first 2 postoperative weeks, the degree of sensitivity correlated with the width of the exposed root surfaces. This correlation was lost as many of the teeth became less sensitive over time.[11]

In another clinical study, there was a more than 100% increase in dentin hypersensitivity after periodontal surgery. After 8 weeks, the control group that received no treatment showed a 34% reduction in hypersensitivity, but it remained above the preoperative level. Wallace and Bissida[12] reported the results of periodontal surgery on dentin hypersensitivity. In a study on 10 patients with 42 periodontally treated teeth and 42 contralateral control teeth, root sensitivity was directly related to the extent of root surface exposure after surgery. Scaling and root planing had no significant effect on immediate root sensitivity. However, 1 day after scaling and root planing, there was a significant increase in hypersensitivity that continued for 2 to 3 days but decreased after 5 days or longer. In another study, six out of 11 patients

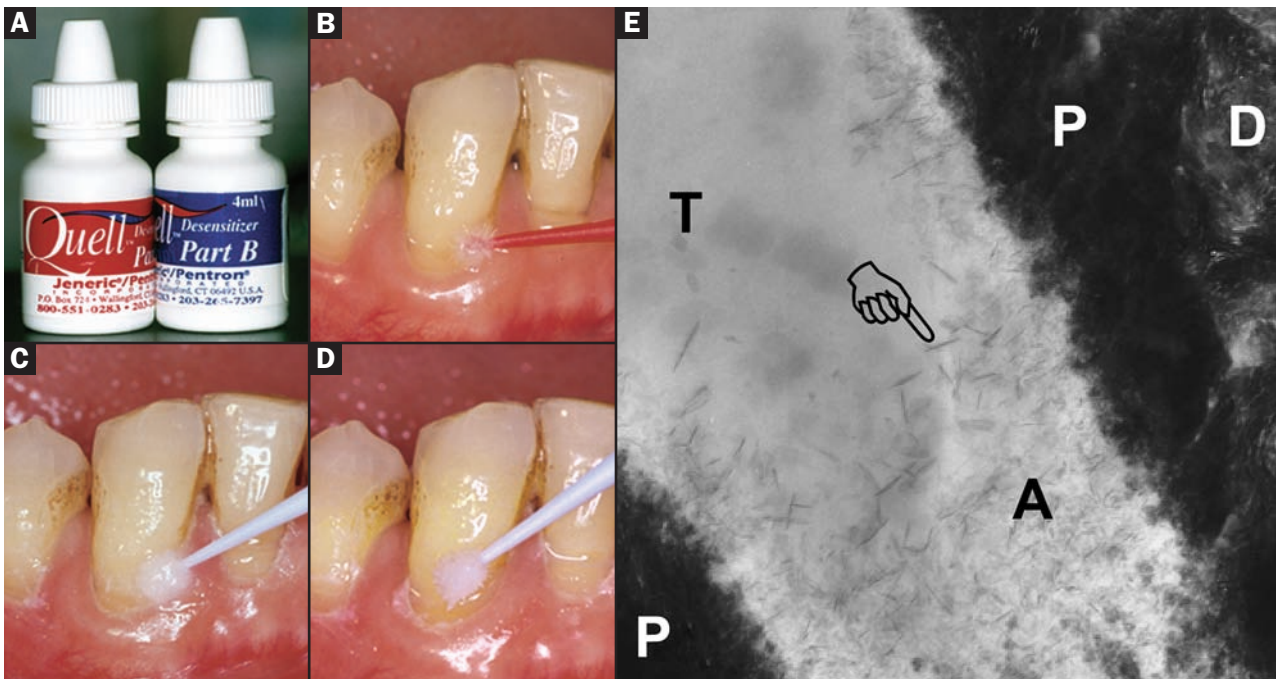

Figure 3 **(A)** Two-step, two-bottle type calcium precipitating solutions have been developed to occlude open dentinal tubules during in-office treatment. **(B)** Topical application of a phosphate-containing Solution A. **(C)** This was followed by the topical application of a calcium-ion containing Solution B. **(D)** Formation of a white calcium-phosphate precipitate could not be identified clinically from the dentin surface, but was apparent along the adjacent buccal gingivae. **(E)** Transmission electron micrograph showing the occlusion of a patent dentinal tubule (T) with needle-shaped apatite crystals (pointer). P: peritubular dentin; D: intertubular dentin.

with periodontally involved mandibular incisors showed increased dentin sensitivity to probing and air blasts after periodontal treatment. The greatest increase in sensitivity occurred 1 week after subgingival root planing. However, by 8 weeks, the increased sensitivity was reduced in five of the six patients.[13] Thus, to summarize, transient to long-term dentin hypersensitivity may occur after deep scaling, root planing, or periodontal surgery.

If the hydrodynamic mechanism is correct, cases of persistent hypersensitivity must be a result of either local pulpal inflammation that causes persistent nerve sprouting or lowering of nerve thresholds, or that some dentinal tubules remain hyperconductive.

Loss of Enamel

Peripheral dentin is covered by cementum on root surfaces, and enamel on coronal surfaces. Thus, loss of enamel can expose dentin, placing it at risk of developing dentin hypersensitivity. The loss of enamel in the absence of gingival recession can involve any location on the tooth and is usually a result of the combined actions of attrition, abrasion, and erosion. Attrition is the loss of enamel resulting from tooth-to-tooth contact such as bruxism. It is usually found on incisal edges and occlusal surfaces. Abrasion involves loss of enamel by physical mechanisms not involving tooth-to-tooth contact, such as the cervical areas of teeth. It often results in angular wedge-shaped cervical lesions, generally on the buccal surfaces of maxillary canines and premolars, although such lesions can be found on the lingual surfaces of molars. Many have attributed the development of these lesions to excessive and improper toothbrushing technique, but some lesions are located subgingivally where toothbrush trauma cannot occur. In such cases, clinicians have used the term "abfraction" to describe the mechanism associated with loss of enamel and dentin.[14]

Erosion is defined as the loss of tooth structure by chemical dissolution resulting from extrinsic or intrinsic acids. Extrinsic acid exposure is a result of dietary sources of acids (citrus fruit and drinks, acidic wines, carbonated drinks) (see Zero and Lussi[15] for review). Intrinsic acids are largely gastric acid (0.1 N HCl) from inadvertent gastroesophageal reflux disease, from psychogenic vomiting syndromes (bulimia, etc) or from the side effects of drugs that irritate the gastric mucosa or cause nausea and vomiting. Erosive tooth wear, or acid wear, is a two-stage process where acids soften the surface (3 µm to 5 µm) through demineralization in a process that only takes seconds. Although these softened surfaces may reharden through the action of saliva and fluoride the process will take 1 to 2 hours. If during the vulnerable period the softened enamel is subject to frictional or abrasive forces the surface will be permanently removed resulting cumulatively over time as an erosive lesion.

Enamel erosive lesions appear dull, smooth, rounded, and without surface contour. That is, there is blunting of cusp tips or lingual cingulae in the early stages followed by their complete loss in advanced stages. As enamel is lost, the yellow color of dentin becomes dominant through the thinner enamel and the surface begins to appear concave. This is especially apparent with maxillary incisors. The enamel along the gingival margin often remains intact, perhaps as a result of outward gingival fluid flow, leaving the appearance of a crown or veneer preparation. On the occlusal surface, the loss of enamel cusps and exposure of underlying dentin creates the potential of abrasion lesions, which are often described as cupping. With further erosion into exposed dentin, the loss of tooth structure increases rapidly. The exposed dentin is often very sensitive and involves a large cumulative surface area of the involved teeth. Once mineralized tissues have been softened by repeated exposure to acids, they become more susceptible to combinations of attrition and abrasion. Treatment is difficult until the exposure to acids can be controlled. Enamel has the propensity to remineralize and thus reharden, albeit slowly, but dentin does not. The surface of enamel will become softened with acids at pH 5.5 and below. Dentin is demineralized with pHs as high as 6.5. Not all acids share the same softening abilities for the same pH. Softening is determined by the type of acid and whether or not it possesses chelating properties (citric acid), the buffering capacity, and the presence of calcium, phosphate, and fluoride in the acidic food or beverage. A contradiction is evident in yogurt, typically at pH 4.0, which is unable to soften the surface at all as it is saturated with respect to calcium. Therefore, no matter what the pH, it is not possible to remove additional calcium from the tooth and into the yogurt solution surrounding it.

Cracked Tooth

Patients with cracked teeth often complain of a long history of pain which has been difficult to diagnose and treatment which has failed to relieve their symptoms. They tend to have erratic pain on mastication, especially with release of biting pressure. Generally, there is no pain to percussion and radiographs are usually inconclusive. In addition, there may be a variable degree of sensitivity to temperature changes.

Such diversity in the presentation of clinical signs and symptoms is a result of the presence of five types of tooth cracks now recognized by the American Association of Endodontists: craze line, fractured cusp, cracked tooth, split tooth, and vertical tooth fracture.[16]

The type of tooth crack that is most likely to be associated with dentin hypersensitivity is the early stages of a cracked tooth. The most frequently involved teeth are the mandibular molars, followed by maxillary premolars and maxillary first molars. Such a crack extends from the occlusal surface of the involved tooth apically, without separation of the two segments. The crack may cross one or both marginal ridges and is most often oriented mesiodistally.

The signs and symptoms of a cracked tooth vary significantly depending on the progress of the crack. In its early stages, a crack may involve only the coronal dentin without extending into the pulp chamber. Clinically, such a cracked tooth may exhibit acute pain on mastication and sharp, brief sensitivity to cold with the pain disappearing on removal of the stimulus, with the pulpal diagnosis of reversible pulpitis. A recent clinical study showed that if a cracked tooth with reversible pulpitis is identified early enough, it may be salvaged with a crown and that root canal treatment will only be necessary in 20% of these cases within a 6-month period. Progression of interproximal periodontal defects associated with the crack (ie, resulting in a split tooth) was found to occur in only 4% of all the cases examined.[17]

If the crack has progressed to involve the pulp or periodontal tissue, the patient may experience thermal sensitivity that lingers after removal of the stimulus, or slight to very severe spontaneous pain that is consistent with the diagnosis of irreversible pulpitis, pulpal necrosis, or symptomatic apical periodontitis. There may even be pulp necrosis with periradicular pathosis. Under such circumstances, root canal treatment will be necessary.

CLINICAL RESEARCH ON DENTIN SENSITIVITY

Measurement of Dentin Hypersensitivity

In clinical trials of dentin sensitivity, most authorities recommend that two different stimuli be used to evaluate the sensitivity. These can be either variable stimuli to a constant response, or a constant stimulus to a variable response. In the first case, one would apply a tactile stimulus, for instance, with a dental explorer that has been modified to display the force applied from 5 g to 150 g or centiNewtons (cN). Kleinberg and his colleagues used the scratchometer, which is a hand-held analog load gauge that has a dental explorer welded to the scratch tine.[18] The scratchometer is applied perpendicular to the sensitive surface and scratched across the surface using 10, 20, 30, 40, etc, cN of force. Insensitive dentin can withstand 80 cN to 100 cN.

If one blows compressed air on an exposed dentin surface while covering the two adjacent teeth with gloved fingers at full force for 1 second at a distance of 5 cm, one can ask the patient to rate their pain perception on a visual analog scale that ranges from 0 mm to 100 mm, with zero being no pain and 100 being unbearable pain. An air blast is an evaporative stimulus, causing rapid outward fluid flow. The use of electrical stimuli, championed by Kleinberg's group, has been largely abandoned because they rely on devices that vary in voltage instead of current. A more detailed discussion of the use of electrical stimuli to measure changes in dentin sensitivity, or the details of how thermal, osmotic, and hydrodynamic stimuli have been used to test dentin hypersensitivity, can be found in Gillam's article.[19]

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Design and Clinical Trials on Dentin Hypersensitivity

Most experts agree that clinical studies should use randomized group assignments, be doubled-blinded, and contain a placebo product that is identical to the test product except that it does not contain the active ingredient. It is critical to evaluate the placebo effect, which can be very strong in such studies.[20] Many of the early studies on dentin sensitivity did not include appropriate placebos or were only single-blinded or used inappropriate stimuli (electric pulp testing). These will not be reviewed.

Jackson reviewed the results of six "modern" clinical trials (1992-1996) on strontium-containing desensitizing toothpastes.[21] With a number of qualifications, Jackson concluded that in none of these studies was there a consistent, significant improvement in the patients' symptoms of dentin hypersensitivity for strontium-containing toothpastes compared with negative control toothpaste (ie, placebo). That is, all toothpastes gave some relief that increased over time as a result of either creation of a smear layer by the toothpaste or the placebo effect. He concluded that strontium salts appear to have only a minimal effect in reducing the symptoms of dentin hypersensitivity.

Jackson also reviewed the efficacy of eight potassium-containing desensitizing toothpaste clinical trials.[21] All of those studies (done between 1992 and 1997) demonstrated reductions in the patients' perceived symptoms of dentin hypersensitivity that increased over time, compared to control toothpastes. However, two of the eight studies failed to show any benefit for toothpaste-containing potassium compared to conventional potassium-free toothpastes.

Two clinical trials compared potassium-containing desensitizing mouthrinses compared to control mouthrinses. Although there were significant improvements in the patients' symptoms, there was no difference between the tests vs control mouthrinses.[21] This led Jackson to conclude that the effects of potassium-containing desensitizing products were marginally effective, but were not significantly different from placebos. Similar conclusions were reached in the more recent Cochrane Collaboration report on the efficacy of potassium-containing toothpastes for dentin hypersensitivity.[22] That review of 38 studies (over the period of 1994 to 2000) only accepted six studies as being valid clinical trials for various reasons. Jackson further opined[21] that any agent (ie, strontium or potassium salts) thought to be effective in reducing dentin hypersensitivity should be effective as a simple aqueous solution, but that no such well-controlled clinical study had been reported. By delivering potassium in dentifrices, its efficacy may be due, in part, to the creation of a smear layer or partial occlusion of tubules by silica fillers in the toothpastes. The major problem in these clinical trials is that the concentration of the active ingredient (5% potassium salts) is only marginally more effective than the placebo effect (see Curro[20] for an excellent discussion of placebo effects for over-the-counter drugs). Patients have difficulty in deciding if their sensitivity to given stimulus has changed over time.

SENSITIVITY RESULTING FROM BLEACHING

Tooth whitening has become an extremely popular procedure that has left the dental office and gone "over-the-counter" as many different consumer products have been marketed. All of these products contain either hydrogen peroxide or compounds that break down to hydrogen peroxide (ie, sodium perborate or carbamide peroxide). While the popularity of tooth bleaching is expanding exponentially, a common side effect of external tooth bleaching is tooth sensitivity.

This sensitivity can be severe enough to cause patients to discontinue home bleaching.

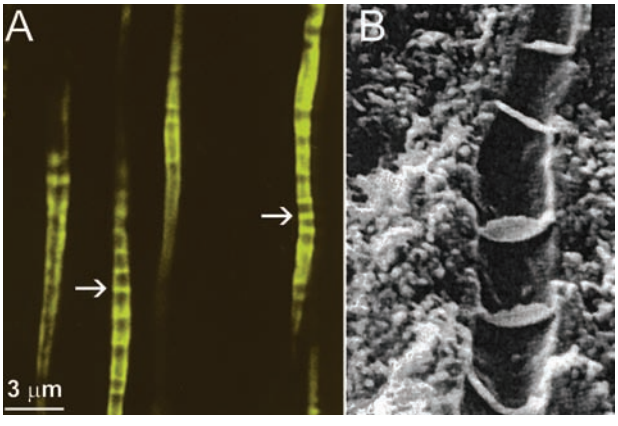

Figure 4 Fluorescence microscopy (A) and scanning electron microscopy (B) showing precipitations of plasma proteins derived from the dentinal fluid as intratubular septa (arrow) after the topical application of an aqueous solution of 35% hydroxyethyl methacrylate and 5% glutaraldehyde. These intra-tubular septa reduce the permeability of dentinal tubules to fluid movement and contribute to the reduction of dentin hypersensitivity (reprinted from Schüpbach et al, 2007, with permission from the publisher).

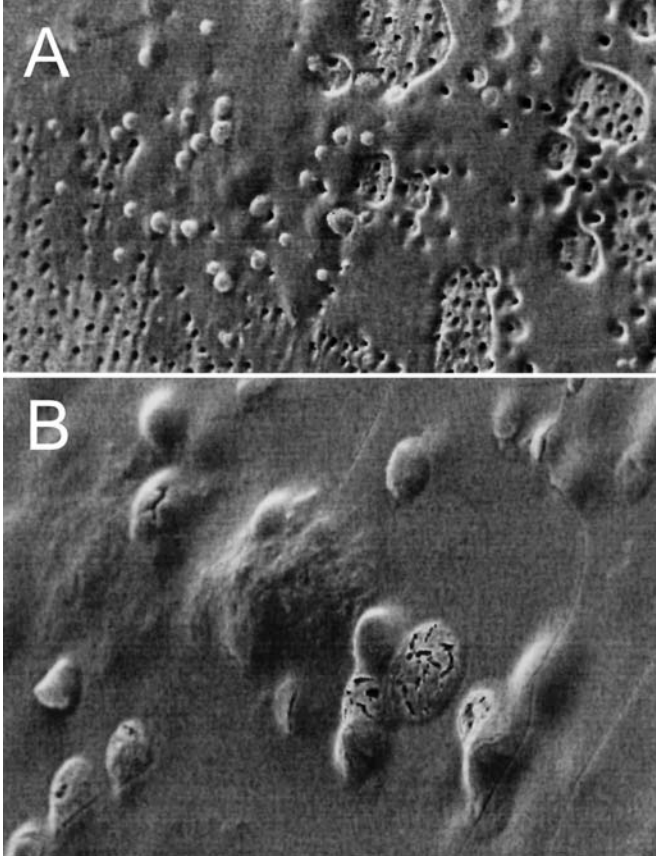

Figure 5 (A) Attempts to seal open dentinal tubules with adhesive resins can fail if the resin film is too thin and becomes saturated with atmospheric oxygen that consumes all of the free radicals generated during light-curing. The co-monomers never polymerize and the film is incomplete, leaving many tubules open. **(B)** Even thicker films can be displaced by water seeping from dentin during bonding. These water blisters represent unbonded areas that can be removed by toothbrushing (Courtesy of Dr. Stephan Paul).

The authors speculate that the dental therapeutic use of hydrogen peroxide or hydrogen peroxide-generating compounds allows hydrogen peroxide to permeate through enamel and dentin to reach the pulp's soft tissues faster than it can be inactivated by pulpal glutathione peroxidase and catalase. This may be responsible for the tooth sensitivity that is commonly associated with mouth guard bleaching.[23]

One final mechanism does involve the hydrodynamic mechanism of fluid movement, and is based on the observation that bleaching gels are all hypertonic. Those authors measured the osmolarity of a number of commercial bleaching gels using a freezing-point osmometer. The osmolarities varied from 4,900 mOsm/kg to 55,000 mOsm/kg. Because plasma and extracellular fluids have osmolarities of 290 mOsm/kg, these bleaching gels are all extremely hypertonic and would tend to osmotically draw water from pulp, through dentin and enamel, and into the bleaching gels. This might hydrodynamically activate intradental nerves. Although many regard enamel as being impermeable, several studies have shown that enamel has a low but significant permeability to water and hydrogen peroxide. The molecular weight of water is only 18 g/L, and for hydrogen peroxide it is only 34 g/L. This small size makes hydrogen peroxide very permeable.

Mechanism of Action of Potassium Ions to Reduce Dentin Hypersensitivity

Potassium nitrate penetrates the enamel and dentin to travel to the pulp and creates a calming effect on the nerve by affecting the transmission of nerve impulses.[24] As potassium ions diffuse to the nerve, they cause the nerve to depolarize once in response to a painful stimulus. However, it cannot re-polarize, so the excitability of the nerve is reduced. Potassium nitrate has an almost anesthetic effect on the nerve.

Desensitization with Potassium Ions

The scientific evidence supporting the use of potassium salts to lower sensitivity is based largely on in vivo animal studies,[25] where the intradental nerve activity of cat teeth could be reduced by potassium but not sodium salts. Later in vitro work using rat spinal nerves revealed that if the medium potassium ion (K^+) concentration was increased from the normal value of 4 mEq/L to between 8 and 64 mEq/L, action potentials of nerves fell in a dose-response manner. The action was reversible, as when the high K^+ concentrations were returned to normal, the sensitivity of the nerves returned. Thus, clearly elevations in K^+ could block nerve conduction, but there was no evidence that the K^+, which diffused into dentin during brushing with K^+-containing dentifrices, would maintain dentinal fluid K^+ concentrations high enough to block nerve conduction. Mathematical modeling predicted that the potassium levels would not remain high enough between brushings to maintain nerve blockage.

However, in a recently published paper, Matthews' group tested the ability of filtering 3.7 wt% sodium chloride (KCl) across human dentin, in vivo, on pain sensations evoked by probing or air blasts in human volunteers.[26] In young patients (aged 17 to 30 years) scheduled for extraction of premolars for orthodontic treatment, the buccal cusps were flattened to expose dentin that was then etched to remove the smear layer and make the dentin conductive. After filtering 3.7 wt% (500 mM) KCl across the dentin using a 150 mm Hg hydrostatic pressure for 4 minutes, they tested the sensitivity of the dentin to air blasts and probing for 2, 10, 20, and 30 minutes. The pain responses

to probing and air blasts were significantly reduced during the first 10 minutes. This is the first time potassium salts have been shown to decrease tooth sensitivity through relatively thick dentin in humans in vivo, using a controlled experimental design and visual analog pain scales that can be statistically evaluated. They confirm the results of Hodosh,[27] who used topical applications of up to 15% solution of potassium nitrate to desensitize hypersensitive teeth. Although everyone now uses 5% KNO_3 or potassium nitrate, Hodosh reported that the best results were obtained with 35%. He relied on the diffusion of K^+ rather than using a hydrostatic pressure. The amount of potassium that can diffuse across dentin from a 35% solution of KCl may be similar to the amount of KCl that reaches the pulp after filtration of a 3.7% KCl solution.

MANAGEMENT OF DENTIN HYPERSENSITIVITY

Dentin hypersensitivity cannot be properly managed unless the etiology of the condition is identified and eliminated (see Figure 1 in the Introduction). For instance, if excessive eccentric occlusal contact has induced cervical abfraction on one or more teeth that have become hypersensitive, careful evaluation and correction of the occlusion may not only cure the hypersensitivity, it may prevent its reoccurrence. If the hypersensitivity was a result of bulimia, it is unlikely that any treatment of dentin will produce a lasting effect until the bulimia is first managed. By far, the most common etiologies of dentin hypersensitivity are dietary acids (citrus fruits and drinks, sports drinks, acidic wines) followed by improper toothbrushing or overly frequent, aggressive toothbrushing with toothpaste. Thus, clinicians should take careful histories of their patients' dietary habits and make patients aware of the importance of erosive influences coupled with improper toothbrushing. Patients should demonstrate their toothbrushing technique to their hygienist or dentist after every appointment until they have mastered proper technique.

Clinical trials have shown that daily use of desensitizing toothpastes twice daily requires 2 to 4 weeks to show any significant desensitization.[28] If, after using a desensitizing toothpaste, the patient's dentin sensitivity remains a problem, clinicians should re-evaluate the differential diagnosis and consider in-office treatments beginning with topically applied desensitizing agents (Table).

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Dentin Hypersensitivity and Gingival Recession

Dental patients of all ages are increasingly concerned about their smile and overall appearance, and are particularly unhappy when they have esthetic issues and dental pain associated with exposed roots. Recession seen in healthy patients is mainly due to oral care that is "too much of a good thing" where recession seen in periodontally diseased patients is likely due to a chronic inflammatory process that may represent years of "doing too little of a good thing." Regardless of the frequency and intensity of oral hygiene, the symptom that brings periodontally healthy and unhealthy patients with recession to the office is a very unpleasant side effect common to both groups—dentin hypersensitivity.

ETIOLOGY

Perhaps the most important factor in the etiology of dentin hypersensitivity is the exposure of root surfaces from gingival recession.[1,2] There is general consensus that gingival recession usually precedes dentin hypersensitivity and is perhaps the most significant predisposing condition of dentin hypersensitivity.[3-5] Other significant factors contributing to dentin hypersensitivity include loss of the cervical enamel and dentin as a result of excessive oral hygiene habits, or tooth wear that can be attributed to brushing shortly after consuming erosive dietary foods and drinks.[6,7] The pathophysiology of gingival recession is not well understood. However, limited evidence gleaned from early histologic studies in the rat and monkey models[8,9] showed that periodontal inflammation and epithelial proliferation are essential to the formation of cleft defects in animals, subsequently leading to loss of gingival integrity and gingival recession.

ANATOMIC FACTORS

While gingival recession is largely preventable, it is known to be exacerbated in the presence of certain anatomic factors:

- Root prominence in the presence of thin mucosa
- Dehiscences and fenestrations in the underlying alveolar bone
- Frenum pulls
- Orthodontic movement of teeth/roots outside the alveolar housing

Because the buccal alveolar bone provides much of the local blood supply for buccal gingivae, loss of the underlying bone is associated with loss of buccal gingivae.[10] Thin, fenestrated, or absent alveolar bone predisposes the site to gingival recession, a phenomenon that frequently occurs on the labial surfaces of canines and premolars and the mesial root of molars. Tooth anatomy or position also affects alveolar bone thickness, with facially positioned teeth more likely to be located within or outside of thin alveolar bone. During therapy, orthodontic tooth movement may inadvertently reposition teeth outside the buccal plate, putting such sites at risk to develop gingival recession. Crowding in the lower anterior segment increases the risk of gingival recessions.[11,12]

ORAL HYGIENE HABITS AND RECESSION

Overzealous Toothbrushing

Clinical studies have reported more gingival recession with

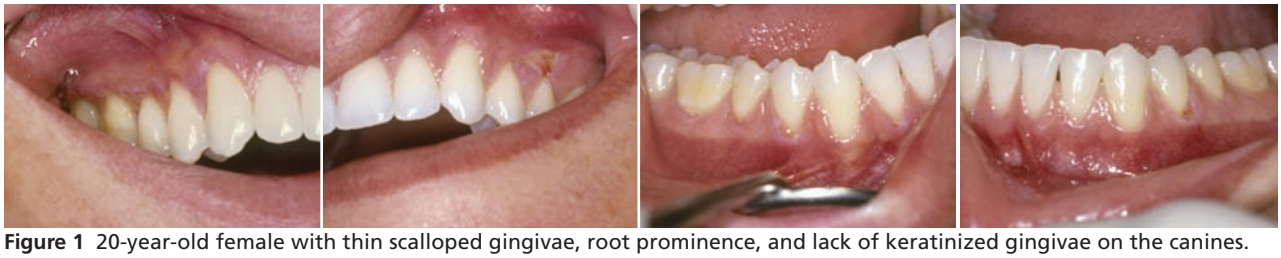

Figure 1 20-year-old female with thin scalloped gingivae, root prominence, and lack of keratinized gingivae on the canines. This patient is at risk for future additional gingival recession. Recession lesions are in the early stages of development on the first premolars. Photograph courtesy of Dr. Terry Rees.

good oral hygiene[13] or improved oral hygiene.[14] Indeed, the most brushed teeth with the lowest plaque scores exhibited the most gingival recession.[15] This has led to the description of gingival recession/dentin hypersensitivity as "toothbrush disease."

Toothbrushing is also known to produce subclinical traumatic lesions of the cervical area in both the soft and hard tissues. In dentally healthy young adults, one study reported frequent signs of inflammation, namely fluid exudate and distortion of gingival contour changes as a result of swelling immediately after the students performed their normal oral hygiene procedure.[16] This was a particularly significant finding, as few if any of the corresponding areas in their mouths had clinically visible gingival damage. Based on this study, it seems reasonable to assume that a significant degree of subclinical gingival inflammation, abrasion, and early exposure of cervical dentin after toothbrushing frequently occurs, undetectable to the naked eye. Because subclinical lesions appear to precede any clinically visible signs associated with destructive oral hygiene habits, early diagnosis and intervention remain challenging aspects in the prevention of gingival recession.

Another well-designed longitudinal clinical study of dental students demonstrated the presence of increased recession over 5 years in the healthy, highly motivated, oral hygiene-compliant population. Progressive gingival recession occurred despite intensive oral hygiene instruction in the students' first year of dental school that was subsequently reinforced over time. Instruction was aimed at replacing harmful oral hygiene habits in favor of more acceptable self-care techniques.[17] It is apparent from this study that "too much of a good thing" is at work in many instances of gingival recession with persons practicing what they perceive as meticulous oral hygiene. Instead, their goal of achieving optimal oral health may lead to over-brushing certain areas of their mouths, ultimately enhancing the frequency and severity of gingival recession in an otherwise healthy dentition (Figure 1).

It has also been reported that recession will increase over time with the use of hard-bristle brushes, excessive force, and frequency of brushing.[18] However, not all sites exhibiting gingival recession and exposed dentin are hypersensitive. When SEM replica models from 28 teeth in ten patients exhibiting hypersensitivity symptoms after acid-etching were compared to non-sensitive sites, an amorphous smear layer was frequently seen coating the area in 88% of unetched non-sensitive specimens, where only 9.3% of specimens showed a few patent narrow dentinal tubules.[19] In the non-sensitive dentin, the acid-etching failed to remove or only partially removed the smear layer, whereas in the hypersensitive dentin, acid-etching always removed the smear layer. This study suggests that the smear layer plays an important role in reducing permeability of exposed dentin in patients with dentin hypersensitivity. Avoidance of oral hygiene products that remove the smear layer, such as some tartar-control or highly abrasive dentifrices, could ultimately benefit patients and is of particular importance in helping them choose their daily homecare products.

Dentifrice Abrasivity

Although the relationship is not well-documented in the literature, dentifrices that contain higher levels of abrasive ingredients may also cause soft tissue damage and tooth wear leading to hypersensitivity.[20] Toothbrushing alone has no abrasive or erosive action on dentin; loss of dentin may be a result of the abrasivity of toothpastes.[21] Once gingival recession has exposed root surfaces, the cementum is rapidly lost as a result of brushing with toothpaste and/or professional tooth cleaning. It has been speculated that in some subjects or on some tooth surfaces, defective cementum formation or its

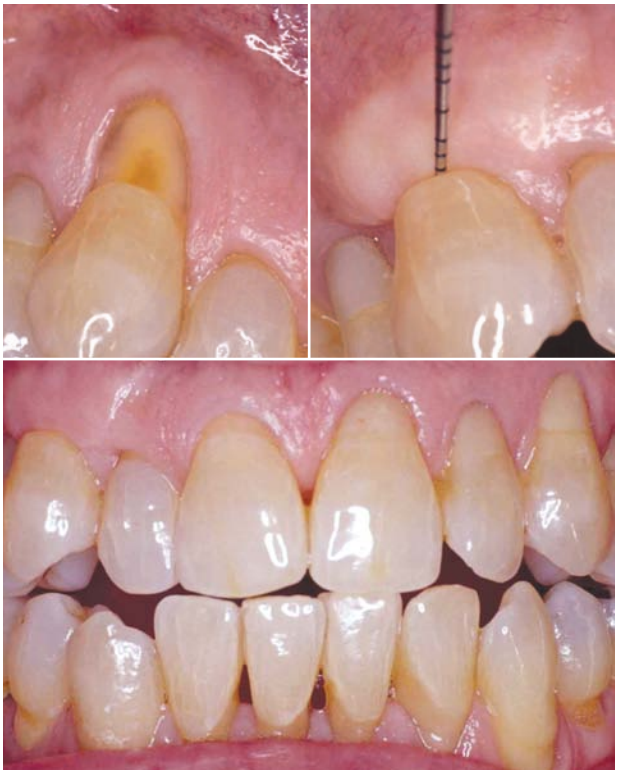

Figure 2A and Figure 2B Six-mm recession on tooth No. 6 (upper right canine) was treated with a connective tissue graft to cover the root and treat the dentin hypersensitivity. Similar grafting techniques were contraindicated on the facials of teeth Nos. 5, 10, 11, and 12 due to the previous placement of root surface restorations.

partial absence at the enamel-cementum junction could predispose some patients to dentin hypersensitivity.[22]

Periodontal Disease

Dentin hypersensitivity is seen more frequently in patients with periodontitis.[23] The prevalence of dentin hypersensitivity is between 60% and 98% in patients with periodontitis. Poor oral hygiene may cause gingival recession indirectly by allowing for the development of periodontal disease. Chronic inflammatory periodontitis seldom causes recession on buccal cervical sites until it is well-advanced. However, as the inflammatory disease progresses apically, the facial and lingual marginal bone and gingivae eventually recede as well. Several studies have investigated changes in root dentin sensitivity after periodontal surgery. Nishida et al[24] followed dentin sensitivity for 8 weeks after periodontal surgery. The highest sensitivity occurred 1 week after surgery. In many cases, by 8 weeks postoperatively, the sensitivity had largely resolved. The teeth of young patients (aged 19 to 29 years) showed a higher incidence and degree of postoperative hypersensitivity than did an older group (aged 40 to 61 years), and the spontaneous decrease in hypersensitivity required a longer time in the young group. During the first 2 postoperative weeks, the degree of sensitivity may be correlated with the width of the exposed root surfaces; however, this correlation is lost as many of the teeth became less sensitive over time.

In another clinical study,[25] there was a more than 100% increase in dentin hypersensitivity after periodontal surgery. After 8 weeks, the control group that received no treatment showed a 34% reduction in hypersensitivity, but it remained above the preoperative level. Some report that root sensitivity is directly related to the extent of root surface exposure after surgery but find no significant effect on immediate root sensitivity from scaling and root planing.[26] Conversely, Sim and Han[27] reported that 1 day after scaling and root planing there was a significant increase in hypersensitivity that continued for 2 to 3 days but decreased after 5 days or more. Periodontally involved mandibular incisors showed increased dentin sensitivity 1 week after root planing. However, by 8 weeks, the increased sensitivity was reduced in five of the six patients.[28] It would appear from most studies that the majority of sensitivity after scaling and root planing disappears or is significantly reduced after 8 weeks.

Scaling and root planing in moderate to deep pockets causes approximately 1.25 mm to 2 mm of gingival recession respectively. Surgical pocket elimination or other surgical access procedures also routinely cause more recession that non-surgical procedures; however, after non-surgical therapy, residual pocket depths will generally be deeper and more difficult to maintain than surgically treated sites.[29]

Scaling and root planing creates a smear layer on root dentin that occludes the orifices of the tubules of exposed dentin.[30] The presence of smear layers is known to lower the hydraulic conductance of dentin.[31] Several studies have shown that smear layers dissolve in vivo within 7 days.[32,33] This permits the dentin to increase its hydraulic conductance that is associated with increases in dentin hypersensitivity. Generally, over the next 10 to 14 days, the sensitivity spontaneously decreases as salivary mineral deposits partially occlude the tubules.[33] However, some teeth remain hypersensitive for years after periodontal treatment. The duration of hypersensitivity after periodontal surgery was reported to range from 2 to 3 months to up to 30 years.[34,35]

It is generally shown that transient to long-term dentin hypersensitivity will likely occur after deep scaling, root planing, or periodontal surgery.[36] Therefore, steps should be taken to prevent the sensitivity if at all possible before or in close proximity to the periodontal therapy. Because open dentin tubules are a prerequisite for hypersensitivity, the use of desensitizing dentifrices in preparation for and during periodontal therapy is advised. Additionally, over-instrumentation of exposed roots for the sole purpose of stain

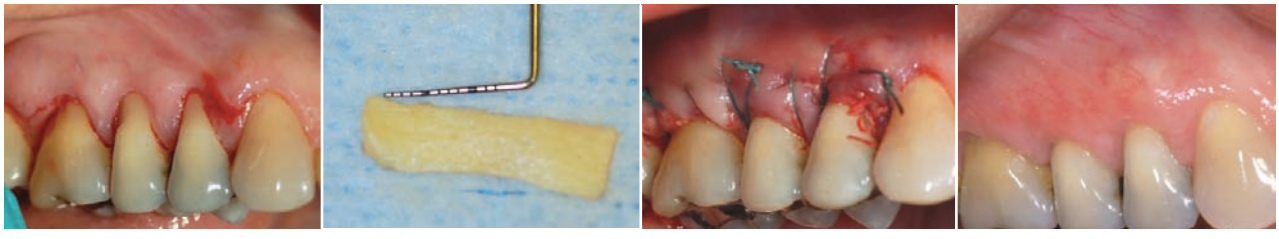

Figure 3 **(A)** Initial incision maxillary right premolar. **(B)** Connective tissue graft taken from palate. **(C)** Connective tissue graft sutured. **(D)** Four weeks postsurgery results in nearly 100% root coverage on teeth Nos. 3 and 4.
**Photographs courtesy of Dr. Poulos, Medical College of Georgia Periodontal Residency Program.*

removal is not recommended in susceptible patients without concomitant use of desensitizing agents as needed.

Other Causes

Some recession may be iatrogenic, through self-inflicted wounds with the chronic use of fingernails, toothpicks, or other sharp objects to clean the teeth, or by the placement of tongue or lip jewelry and studs that strip the buccal and lingual gingivae away from the surfaces of the mandibular teeth. Early studies have also identified the increased risk of gingival recession with over-instrumentation of healthy sulci during periodontal therapy by the dentist or dental hygienist.[37] Practitioners are cautioned to resist scaling and root planing in shallow pockets (≤ 4 mm) lest they cause an additional 0.3 mm of gingival recession in shallow healthy sites over time.

PREVALENCE

Albandar and Kingman[38] reported epidemiological data on 9,689 subjects 30 to 90 years of age in the United States and projected that more than 23.8 million people have one or more tooth surfaces with gingival recession of at least 3 mm or more. They found that the prevalence of recession of 1 mm or more in this population was 58% and that it increased with age.[11,39] Woofter[40] found gingival recession in the third to fourth decade of life. In addition, males have been found to have increased recession compared to females,[41,42] and black males have more recession than Caucasian males.[38] Frequency of gingival recession increases with age and is greater in men than in women of the same age.[13] Recession was also associated with labially positioned teeth in 40% of patients 16 to 25 years of age and the prevalence increased to an impressive 80% of patients between 36 and 86 years of age.

Receding gums are also a common manifestation of periodontal disease. In a study of 1,460 subjects in an urban Brazilian population, more than half (51.6%) of individuals and 17% of their teeth had ≥ 3mm of recession associated with chronic periodontitis. In addition, 22% of individuals had ≥ 5mm of recession in 5.8% of their teeth[39] and was also shown to be associated with age. Males ≥ 30 years of age showed the highest extent, prevalence, and severity of recession in this study. Using a multivariable model approach to the analysis, smoking and the presence of supragingival calculus were also factors most often associated with both localized and generalized recession. Significant associations between gender, socioeconomic status, dental visits, and gingival recession differed among different populations. Therefore, etiologic factors and risk factors may vary across countries and cultures and must be taken into consideration when looking at epidemiologic data relative to gingival recession.

LOCATION OF GINGIVAL RECESSION

The most common location of recession is on the facial aspect of canines, premolars, and molars.[43-47] The teeth most commonly affected are canines > premolars > incisors > molars.[44,45,47,48] Interestingly, a significantly higher proportion of left vs right contralateral teeth was reported in right-handed patients with dentin hypersensitivity.[15] Other studies report significant differences in severity between the right and left halves of the mouth depending on the dominant hand of the brusher. In the case of right-handed people, more recession is usually seen on the left half of the upper and lower facial surfaces according to Addy et al,[15] but conversely, on the right side according to Tezel et al.[41] Regardless of the side affected, recession is highly associated with vigorous and overzealous or improperly executed brushing.

TREATMENT IN THE PRESENCE OF GINGIVAL RECESSION

Nonsurgical Therapy

Hypersensitivity with minimal exposure of dentin is usually the most easily treated symptom of gingival recession. Dentifrices containing potassium nitrate are shown to be effective in reducing dentinal sensitivity within the first few weeks of daily use in a large segment of the population with mild or transient hypersensitivity. Specific lasers used at specific settings have also been shown to be nearly or equally as effective for treating hypersensitive roots as dentifrices.[49] Laser treatment is significantly more expensive than a dentifrice, but may be indicated for immediate relief of severe sensitivity in localized areas. Restorative materials can also be used to block the open tubules or to restore the contour of lost tooth structure. However, placement of restorative materials in the root surface complicates the ability of the surgeon to cover the roots by a graft procedure in the future time. The materials are designed to block the tubules and may prevent the formation of a new epithelial or connective tissue attachment to the root without first removing the part of the root surface impregnated with the filling material (Figure 2A and Figure 2B).

Long-term relief of moderate-to-severe dentinal sensitivity associated with gingival recession more than 1 mm is more difficult to achieve and may require multiple surgical interventions to cover the exposed root. In addition, exposed dentin

may increasingly become more sensitive, and recession may present significant esthetic problems, prompting the patient to seek more invasive solutions, including surgical root coverage, that are capable of addressing both concerns.

Surgical Correction of Gingival Recession

A variety of root-coverage techniques are available that have been shown to be highly successful over time.[50] Recent meta analyses of certain root-coverage techniques show 95% to 100% success over 5 years.[50] The most common procedure used by dentists worldwide for root coverage is the connective tissue graft (Figure 3A through Figure 3D).

Selection of a particular surgical technique is routinely based on the depth and width of the recession according to the Miller Classification system,[51] which also considers the height of the interproximal bone, a strong predictor of the potential root coverage in each classification of recession defects.[51] Other considerations in selecting a particular surgical technique are based on the number of teeth with recession, the width and thickness of the keratinized gingiva at the recession site, and availability of host tissue that may be transplanted from one area of the mouth to another. When several adjacent teeth are in need of root-coverage procedures, multiple surgical procedures may be needed to treat large recession areas. Fortunately, within the last few years, acellular dermal allograft material has become available[52] that may be used in lieu of autogenous grafts harvested from distant donor sites within the mouth. Although use of the acellular dermal allograft material is highly technique-sensitive, success rates are similar to autogenous grafts.[53,54]

CONCLUSION

- Excessive oral hygiene and plaque control habits produce gingival abrasion lesions that may ultimately lead to short- and long-term recession in an otherwise healthy mouth.
- Dentin hypersensitivity associated with gingival recession is very common in periodontitis patients and is usually more acute immediately after periodontal therapy but may persist for weeks to months or even years.
- Periodontal evaluation to determine the feasibility of root-coverage procedures should precede the placement of restorative materials on the root surfaces to reduce dentin hypersensitivity.
- Treatment of sensitive roots with restorative materials may negatively influence the success of future gingival grafting or root-coverage procedures.
- More research is needed to specifically guide the practitioner in the selection of appropriate desensitizing products for their patients with hypersensitivity in the presence of recession.
- Minimal recession of 1 mm or less with transient dentin hypersensitivity can often be treated by the recognition and correction of destructive oral hygiene habits in conjunction with the use of a desensitizing dentifrice.
- Early diagnosis and intervention would likely prevent the subsequent development of recession associated with dentin hypersensitivity in the majority of cases.

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Considerations for Managing Bleaching Sensitivity

Tooth sensitivity is the single most significant deterrent to bleaching, and must be understood to be able to manage the treatment of patients. All forms of vital tooth bleaching are associated with some level of sensitivity.[1-6] Hence, the dental office and the patient must be prepared for the possibility of sensitivity during bleaching treatment.

PREVALENCE AND CAUSE

The three major classes of bleaching—in-office, tray, and over-the-counter (OTC)—all demonstrate some prevalence of sensitivity. Typical bleaching ingredients are either hydrogen peroxide or carbamide peroxide. For comparison, a 10% carbamide peroxide product is approximately 3.5% hydrogen peroxide. Generally, the higher the concentration of the peroxide, the greater the chance of sensitivity.[7] In-office bleaching uses the highest concentration of peroxide (15% to 35% hydrogen peroxide), and has a range of sensitivity from 10% to 90%, with some sensitivity being so severe as to require analgesics posttreatment.[8-10] Typically, multiple in-office visits are required for maximum whitening,[11] and those visits should be spaced at least 1 week apart to allow for reduction of sensitivity caused by treatment.[12] It is also recommended to pre-medicate patients with non-steroid anti-inflammatory drugs to reduce the incidence of sensitivity.[12] The second highest concentration of peroxide is found in the OTC products. These products typically range from 6% to 15% hydrogen peroxide. Although they have a shorter treatment time due to the limited efficacy of hydrogen peroxide (30 to 60 minutes), they still generate tooth sensitivity as well as gingival irritation. Even shorter treatment times of OTC strips with higher concentrations have exhibited greater sensitivity than lower concentrations with longer treatment times.[13]

The classic tray bleaching treatment involves 10% carbamide peroxide or 3.5% hydrogen peroxide. Incidences of 25% to 75% are reported,[14,15] although differences in study design influence data in all treatment options. Generally, sensitivity occurs in the first 2 weeks of treatment, often in the first few days.[16] The more recent addition of potassium nitrate to bleaching materials has reduced, but not eliminated, sensitivity. It is important to note that the presence of sensitivity is the most probable cause for persons discontinuing bleaching, with one report of 14% termination of bleaching due to sensitivity.[17]

A recent report on double-blinded, placebo-controlled clinical trials has provided evidence that the addition of low levels of potassium nitrate and/or potassium nitrate and fluoride significantly reduce postoperative sensitivity relative to products that do not contain either agent.[3,5]

Whereas all of the typical causes of dentin hypersensitivity generally involve the hydrodynamic theory of fluid flow, the sensitivity associated with bleaching seems to have a different origin. In bleaching situations, the teeth may be in excellent condition, with no cracks, exposed dentin, or deep restorations, but after a few days of bleaching, the tooth may experience severe sensitivity. This seems to be related to the easy passage of hydrogen peroxide

Figure 1 Tray application of a potassium nitrate-containing desensitizing material is a very effective approach to treatment of sensitivity.

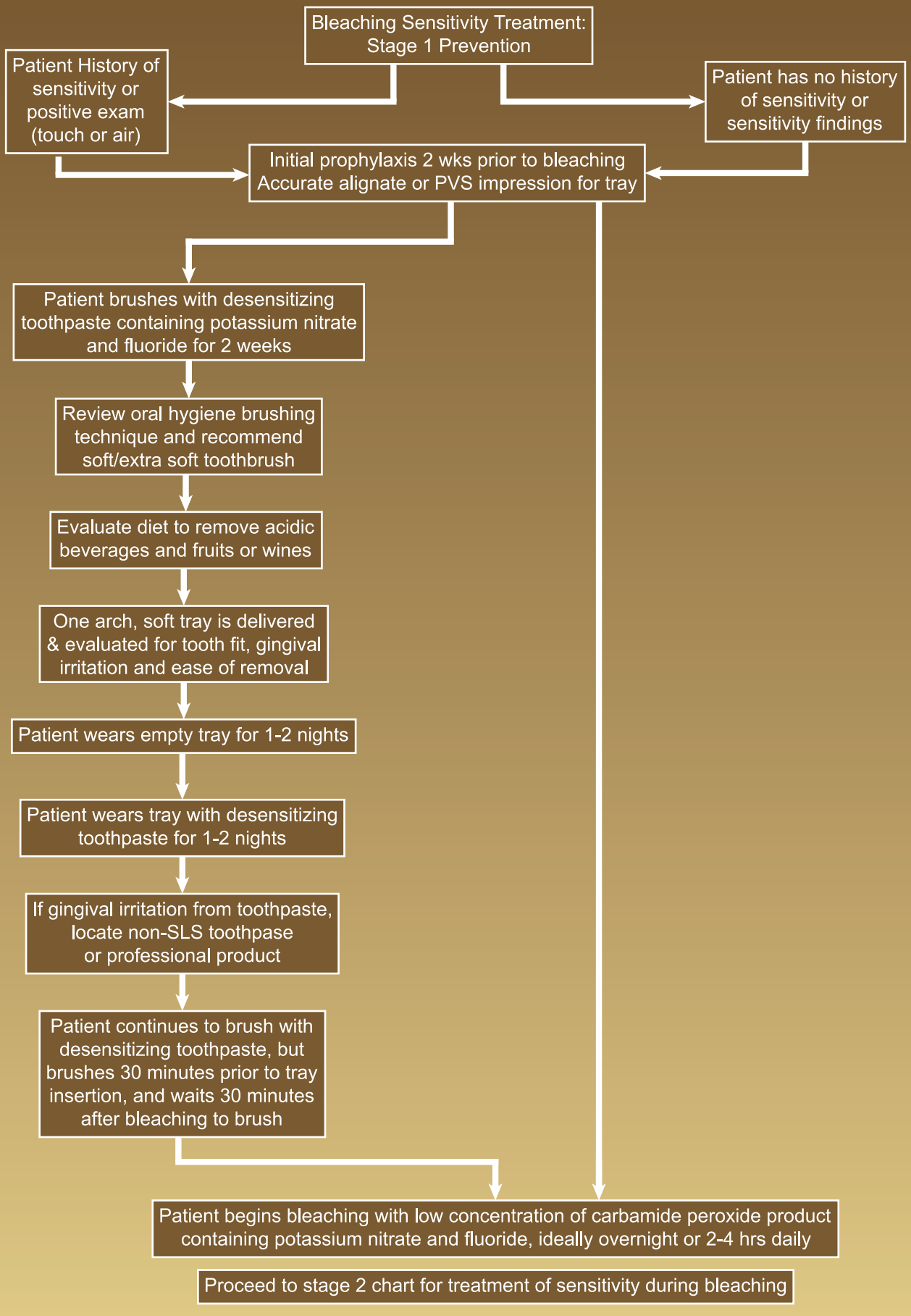

Figure 2 Bleaching Sensitivity Treatment: Stage 1 Prevention options in patients with existing sensitive teeth.

and urea through the intact enamel, through the dentin in the interstitial spaces into the pulp within 5 to 15 minutes.[18] In effect, the tooth is a semipermeable membrane that is quite open to certain-sized molecules. Once it is understood how easily the peroxide penetrates the tooth, the resultant pulpal response of sensitivity may be considered a reversible pulpitis. Tooth sensitivity is the main side effect of bleaching, and may be caused primarily by the peroxide penetration to the pulp, and secondarily by the mechanical pressure of an improperly fitting tray or occlusion on the tray. The other side effect recorded is gingival irritation, which may be related to an improperly fitted tray, occlusion on the tray, or chemical irritation from higher concentrations of hydrogen or carbamide peroxide.

PREVENTION

Because tooth sensitivity mainly depends on inherent patient sensitivity, frequency of application, and concentration of the material, a history of sensitivity should be determined during the examination.[14,19] Patients generally will report or should be asked if their teeth are sensitive to cold. Additionally, existing sensitivity can be determined from the preoperative exam by simple methods of explorer contact with areas on the teeth, or air blown on the teeth. Patients can be counseled in the frequency of application and the appropriate concentration of bleaching agent, with instructions that applications more than once a day or higher concentrations of bleaching agent increase the likelihood of sensitivity.[3,4,20-22] All other delineators, such as pulp size, exposed dentin, cracks, gingival recession, caries, sex or age of the patient, or other physical characteristics are not predictive of who would have sensitivity.

Most reports of sensitivity occur within the first 2 weeks, regardless of how long the patient may treat their teeth. Often, these reports are a single day of sensitivity, followed by no problems the next day. The tooth's response to bleaching is very individualistic, and can only be determined by beginning treatment. However, the history of sensitive teeth by the patient, as well as their response during examination to explorer touch or air, can be a reasonable predictor.

Because bleaching tends to produce some tooth sensitivity under ordinary circumstances, patients with pre-existing tooth sensitivity must be cautioned that increased sensitivity, albeit transitory, may occur, and that management of the sensitivity may require a longer time span for bleaching as a result of the additional time to treat the sensitivity.

Other contributors to sensitivity include rigid tray materials, the base vehicle composition and viscosity, flavoring agents, or patient habits such as clenching or bruxism. The short-term pulpal response varies from patient to patient and even from tooth to tooth. Although penetration of peroxide through the tooth to the pulp can produce sensitivity, the pulp remains healthy and the sensitivity is completely reversible when treatment is terminated. No long-term sequelae remain after the sensitivity has abated.[23-25] Research also has shown that patients have tooth sensitivity even when using a non-bleaching agent in a tray, or just wearing a tray alone. Hence, it is not possible to have all patients be sensitivity-free because of the mechanical forces of materials and occlusion, and some plans must be made to address potential problems.

TREATMENT RECOMMENDATIONS

Most of the earlier treatments for sensitivity involved tray bleaching, as the ease of use of this system and universal popularity made it the most commonly used system for tooth bleaching.[26,27] The passive approach for treating sensitivity was first used. This involved a reduction in wear time, or in frequency of application. Sensitivity treatment could also involve temporary interruption of the bleaching treatment. After the interruption, treatment can often be

A recent report on double-blinded, placebo-controlled clinical trials has provided evidence that the addition of low levels of potassium nitrate and/or potassium nitrate and fluoride significantly reduce postoperative sensitivity relative to products that do not contain either agent.

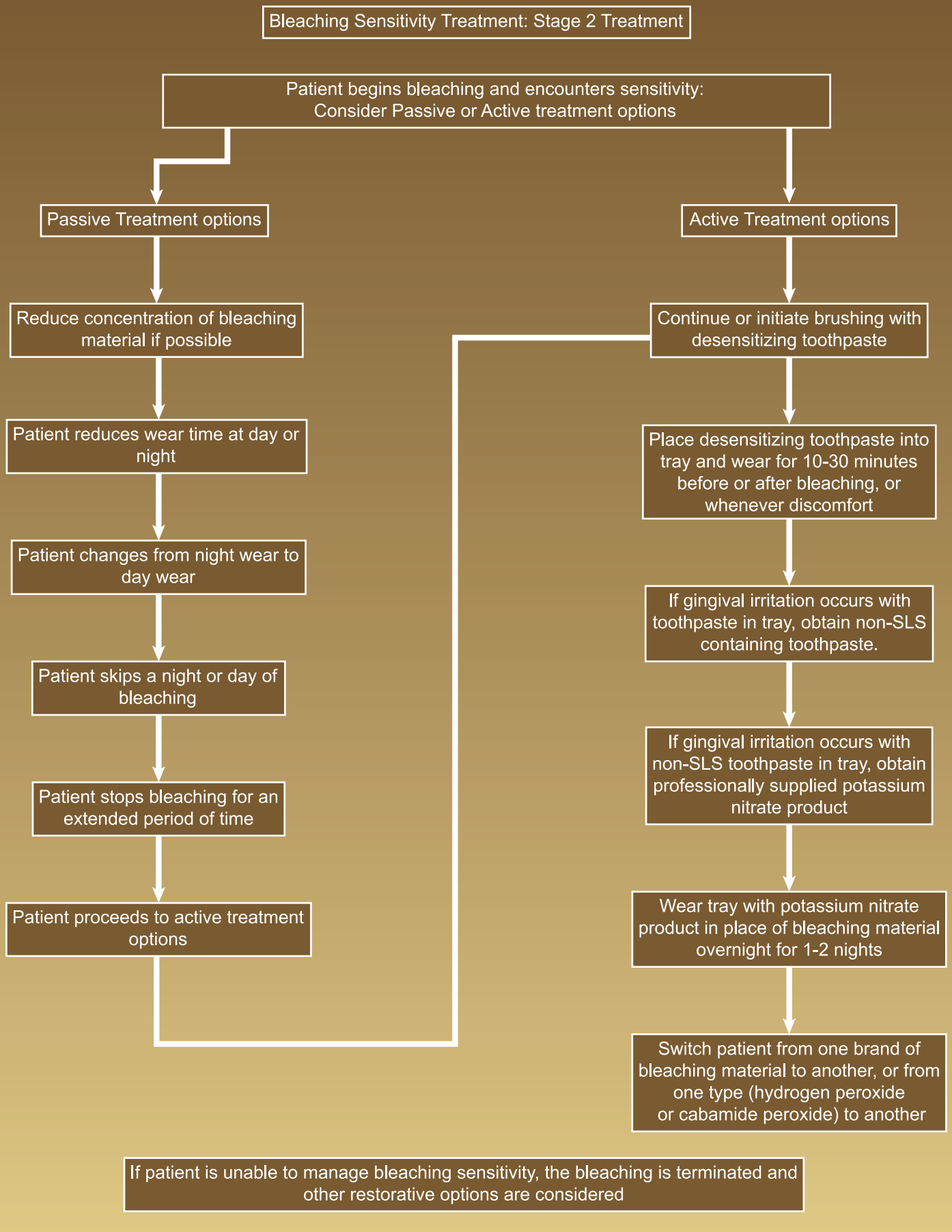

Figure 3 Bleaching Sensitivity Treatment: Stage 2 Treatment options for patients who experience sensitive teeth during bleaching.

resumed without any further sensitivity. Cessation of treatment results in no lingering sensitivity. Although the passive approach has some success, patients and dentists prefer to have a more active approach. The active approach involves the use of either fluoride, potassium nitrate, or both in combination. Traditionally, fluoride has been used as a method of reducing sensitivity. The primary mechanism for action is to occlude dentinal tubules or increase the hardness of enamel, which impedes the flow of materials to the pulp. However, the peroxide molecule is so small that it can travel in the interstitial spaces between the dentinal tubules. Hence, fluoride has not been particularly beneficial in treating bleaching sensitivity.

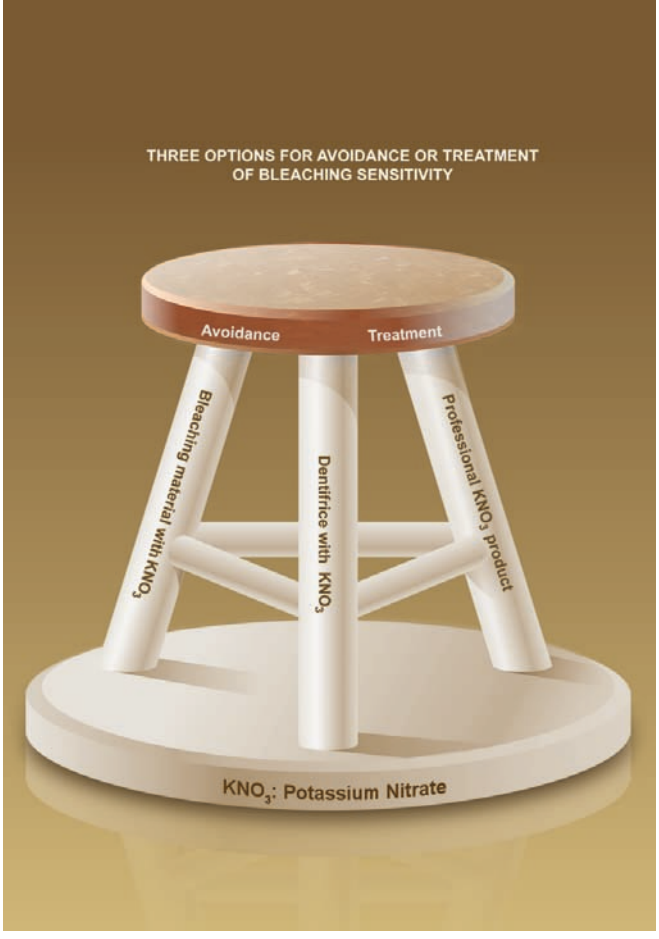

Figure 4 The three options for avoidance or treatment of bleaching sensitivity involve the application of potassium nitrate products either in the bleaching tray or topically.

Potassium Nitrate Use in Bleaching

Potassium nitrate has a completely different mechanism of action than fluoride. Potassium nitrate penetrates the enamel and dentin to travel to the pulp and creates a calming effect on the nerve by affecting the transmission of nerve impulses. After the nerve depolarizes in the pain stimulus-response, it cannot re-polarize, so the excitability of the nerve is reduced. Potassium nitrate almost has an "anesthetic-like effect" on the nerve.

One study demonstrated that applying potassium nitrate for 10 to 30 minutes in a bleaching tray could be successful in reducing sensitivity in more than 90% of the patients, and allow them to complete the bleaching procedure successfully.[28] This technique was originally used by Jerome to treat tooth sensitivity after periodontal surgery in non-bleaching patients.[29] He placed desensitizing toothpaste into soft trays that covered the now-exposed root surfaces of the teeth, and achieved good results. For patients with chronic sensitivity unrelated to bleaching, the toothpaste gives them an OTC product that they can use whenever they need it with tray application, even before a prophylaxis. This approach was extended by Haywood to include patients experiencing sensitivity during bleaching.[28] Tray application could be used either before or after the bleaching treatment (Figure 1). Because the pain can occur remotely from the bleaching treatment, the potassium nitrate could be used as needed during the day or night. In severe situations, the potassium nitrate could be substituted for the bleaching material on alternating nights of wear.

The more readily available source of 5% potassium nitrate in the United States is desensitizing toothpastes that contain 5% potassium nitrate. Five percent is the maximum amount of potassium nitrate approved by the US Food and Drug Administration, and is the primary ingredient for sensitivity treatment allowed in OTC toothpaste. Based on the tray application study, desensitizing toothpaste can be placed in the tray for 10 to 30 minutes whenever sensitivity occurs. The only caution with toothpaste application is that some patients may experience a gingival reaction to the foaming ingredient sodium lauryl sulfate. This reaction is not caused by the potassium nitrate. The reaction generally produces a tissue burn or reddening of the gingiva. If this irritation occurs with one brand or flavor of toothpaste, the clinician may have to experiment with various OTC formulations for certain patients. Initially there was only one toothpaste available which had potassium nitrate, but not sodium laural sulfate, and that was the original "Pink packaged" Sensodyne. More recently, the advent of "Pronamel Sensodyne" has provided a new option for a non-sodium laural sulfate, potassium-nitrate containing toothpaste to be used in brushing or in the tray for treatment of sensitivity.

If suitable toothpaste cannot be found for the patient, then the clinician should use the professionally available products containing 3% to 5% potassium nitrate and fluoride.

Several companies provide 3% to 5% potassium nitrate in a syringe for application in the bleaching tray as needed. The syringe materials, which must be purchased from the companies, may be more appropriate for episodic sensitivity associated with the bleaching itself where the toothpaste was not acceptable because of the gingival response. There are also disposable trays containing potassium nitrate which may be helpful, especially if there is no bleaching tray available for in-office techniques being used alone.

Once research determined that potassium nitrate in the tray was successful, the next step was to incorporate this material in the bleaching material rather than require a separate application. First attempts were not too chemically successful, but now most manufacturers have their bleaching product containing both fluoride and potassium nitrate. Examples of this would be Opalescence PF (Ultradent Products, Inc, South Jordan, UT), NiteWhite® Excel and NiteWhite® ACP (Discus Dental, Culver City, CA), Contrastpm® (Spectrum Dental, Corpus Christi, TX) , GC TiON™ (GC America), and Opalescence® Treswhite™ Supreme (Ultradent Products). Early concerns were that either the fluoride or the potassium nitrate would interfere with the bleaching, but one study has indicated that bleaching efficacy is not reduced.[30] Certainly, if there is any reduction in efficacy or increase in time of treatment, it is minor, and much better than termination of bleaching resulting from unmanageable sensitivity.[31] Having the potassium nitrate in the material could also minimize the effects of mechanical irritation from an improperly fitting tray or occlusion causing movement of the tray and resultant tooth sensitivity.[5]

Pre-Brushing with Potassium Nitrate for Sensitivity Avoidance

Even though tray application of potassium nitrate was very effective, and the incorporation of potassium nitrate into the bleaching material has helped, these advances do not totally eliminate sensitivity. Relief from sensitivity requires brushing with potassium nitrate for approximately 2 weeks to be effective.[32] A recent study[33] compared patients who pre-brushed with the toothpaste containing potassium nitrate (Sensodyne) for 2 weeks before initiating bleaching to another group that used conventional fluoride-containing toothpaste. The group that pre-brushed with the potassium nitrate-containing toothpaste had less sensitivity overall, less sensitivity in the first 3 days, and more sensitivity-free days before a first occurrence. Results of patient surveys showed that the switch to a potassium nitrate-containing toothpaste was easy and well-accepted.

Recommended Treatment

Bleaching sensitivity may result from a combination of the patient's pre-existing tooth and gingival conditions, the chemical nature of the peroxide, and the mechanical nature of the tray. The dentist should determine if the patient has pre-existing sensitive teeth that require a protocol to minimize sensitivity during bleaching. If the patient has no pre-existing sensitivity, a proactive protocol should be developed to address sensitivity should it occur. Figure 2 and Figure 3 offer this information in two treatment options, one for patients with a history of sensitivity, and one for patients with no pre-existing sensitivity. They also explain the options for passive or active treatment of sensitivity that occurs once the bleaching process is initiated.

CONCLUSION

Treatment of bleaching sensitivity involves many possible options (Figure 4). Prebrushing with a potassium nitrate-containing toothpaste can reduce or avoid sensitivity from bleaching. Tray application of potassium nitrate can be an effective episodic treatment for sensitivity. Other treatment time variations, use of different concentrations of material, and varying tray designs can all be part of a sensitivity management program. It is far better to try to avoid or minimize the sensitivity with the above steps than to treat sensitivity after it occurs. Even with all these options for sensitivity avoidance and treatment, there are still some patients who cannot manage their sensitivity and elect to terminate bleaching. Sensitivity seems to be a multi-factorial event which cannot be entirely controlled in every patient. However, the majority of patients, after a proper dental examination, history, and radiographs, can find an appropriate method with adjustment of treatment time and material, brushing with a desensitizing toothpaste containing potassium nitrate, or tray application of potassium nitrate, to minimize any sensitivity they may encounter, and proceed to a successful completion of the bleaching process.

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The Role of the Dental Hygienist in the Management of Dentin Hypersensitivity

The role of the dental hygienist is pivotally important in the prevention and management of dentin hypersensitivity. Prevention of hypersensitivity is the most cost-effective treatment option for patients. Through promotion of good oral hygiene practices, nutritional counseling, nonsurgical periodontal therapy, and application of desensitizing agents, dental hygienists are uniquely placed to be a first line of defense in the prevention of dentin hypersensitivity and its major predisposing conditions.

Patients may be reluctant to report symptoms of dentin hypersensitivity to the dentist during the comprehensive or periodic oral examinations. The first discussion is frequently with the dental hygienist during the dental prophylaxis, when hypersensitive areas may be stimulated.

When symptoms of hypersensitivity first become apparent to the dental hygienist, it is important that a thorough health questionnaire is completed and that the sites of sensitivity are documented, including duration, onset, and the nature of stimuli (if any) initiating the symptom. All contributory and predisposing factors and conditions should be explored, such as gingival recession, tooth wear, oral hygiene, and any harmful or factitious habits.

Due to the common nature of symptoms of hypersensitivity, a differential diagnosis is essential (see Figure 1 in Introduction). The dentist, as diagnostician, should follow the appropriate protocol to ensure that the most appropriate restorative or surgical treatment is rendered.

TOOTH WEAR

Tooth wear may be a result of mechanical (attrition and abrasion) or chemical (erosion) activity or, quite commonly, both (chemical softening of the surface prior to its mechanical removal). Attrition is wear resulting from tooth-to-tooth contact during normal mastication and abrasion is mechanical wear by forces other than mastication. Erosion is a loss of tooth substance by chemical processes unrelated to bacterial action, most commonly, dietary acids. With all three types of tooth wear, dentin hypersensitivity commonly results when enamel is lost and dentin is exposed.

An aspect of patient education, within the scope of dental hygiene practice, is nutritional counseling and referral. The dental hygienist must thoroughly assess the nutritional habits of patients with dental history that includes intake of soft drinks/acidic beverages or eating disorders (bulimia/anorexia nervosa, GERD) that may lead to dental erosion. With the rapidly increasing changes in lifestyles and consumption of acidic beverages, chemical tooth wear (erosion) of enamel and dentin may inevitably result in more tooth hypersensitivity for many patients.[1]

Dietary modifications should include limiting foods and beverages that cause hypersensitivity such as citrus fruits, acidic beverages, pickled foods, and ciders, as well as incorporation of foods and beverages into the diet immediately after an acid exposure that encourage saliva secretion and remineralization (eg, milk, cheese, yogurt). Some erosive tooth wear is caused by chronic vomiting related to pregnancy or bulimia. Patients should be instructed not to brush immediately after vomiting to allow the acidity of the oral cavity to decrease. Patients with these conditions should be referred for medical and psychological evaluation.

Mechanical tooth wear from abnormal habits (abrasion) and wear from normal occlusion (attrition) also can contribute to dental hypersensitivity.[2] The dental hygienist should frequently assess these behaviors during recare and periodontal maintenance appointments that follow active periodontal/restorative therapy.[3]

ORAL HYGIENE INSTRUCTION

One of the most important roles of dental hygienists is to effectively communicate individualized oral hygiene instructions to all patients. Patients should demonstrate their routine brushing technique while the dental hygienist actively observes.

Traditionally, it has been concluded that overzealous brushing and using a hard-bristled toothbrush could cause or worsen gingival recession.[4] However, in a recent systematic review of several studies assessing this correlation,

Rajapakse et al[5] concluded that data to support or refute an association between toothbrushing and gingival recession are inconclusive. This is echoed by Drisko.[6] Although the evidence is inconclusive, brushing duration and frequency are the most-cited causes of toothbrush-related gingival recession. Other factors studied are brushing technique, brushing force, toothbrush age, and hardness of toothbrush bristles.[4]

Patients should be advised to brush at least 2 minutes, twice per day.[7] Toothbrushes should be discarded and replaced every 3 months or sooner when the patient experiences a transmissible infection or when bristles begin to fray.[7] To reduce brushing force, patients with normal dexterity should be advised to use a finger grip on their toothbrush handles as opposed to a palm grip. Brushing with the non-dominant hand may also alleviate destructive brushing since studies have reported a higher proportion of sensitive teeth on the left side of the mouth versus the right side in right-handed patients.[7] Because hard bristled toothbrushes may contribute to tooth and gingival wear, a soft-, sensitive-, or extra-soft bristled brush should be recommended to all patients, especially those experiencing sensitive teeth.[1,4,8]

Marginal biofilm can cause gingival recession to worsen so brushing technique should be routinely emphasized. Manual toothbrush bristles should be adapted at a 45° angle toward the sulcular area. Then, the patient should be instructed to gently brush back and forth, progressing around the arch in small increments. Once the gum line brushing has been done on the facial and lingual surfaces of both arches, the patient can be instructed to then brush the teeth surfaces. Redirecting patient brushing habits from toothbrushing to sulcular brushing will effectively remove harmful bio-film and promote firmly attached and resilient marginal gingiva.

The dental hygienist should be clear in explaining that how the toothbrush is used is more important than the toothbrush design. This is true whether the toothbrush is powered or manual. In a few studies, the results obtained using a power toothbrush were superior to manual toothbrushing.[9,10] Since those investigations, power toothbrushes have undergone much innovation with enhanced features of particular benefit for patients with hypersensitivity, which include visual timers, brush guide location by quadrant, and visual pressure indicators to alert patients when they are brushing too hard.[11]

Clinicians and patients must realize the importance of meticulous oral hygiene in suppressing and preventing periodontal disease, regardless of tooth hypersensitivity. Children and patients with poor manual dexterity will often benefit from a powered brush because of the larger handles and it being less technique dependent than manual toothbrushing. The supply and demand for more convenient and efficient oral care has sparked rapid advancements in several manual and powered brush designs. Innovation and product development may possibly eradicate the factors that were/are thought to link toothbrushing with gingival recession.

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DESENSITIZING AGENTS

The hydrodynamic theory is widely accepted as the mechanism of action of dentin hypersensitivity. This theory states that the hypersensitivity or pain is caused by various stimuli (temperature, pressure, touch, chemical) which can lead to changes in the movement of fluids in and out of exposed dentinal tubules leading to changes in pressure or flow around the mechano-receptors found in the nerve endings surrounding the odontoblastic processes.[12] The mechanism of action for most desensitizing agents is either to desensitize the nerve so that the fluid flow and resulting changes in pressure do not cause the mechanoreceptors to fire; or to block exposed tubules

so there can be no fluid movement at all.

Potassium salts (nitrate most commonly, but also chloride and citrate) are found in desensitizing toothpastes and have been proven safe and effective in several clinical trials. Potassium delivered in the form of toothpaste is the most clinically evaluated desensitizing agent. Office-prescribed potassium nitrate has been shown to be effective in patients experiencing hypersensitivity from vital teeth bleaching. Potassium nitrate is thought to work by depolarizing the nerve and preventing pain signals from reaching the brain.[13] Custom-tray application of potassium nitrate before bleaching has provided relief for many patients experiencing teeth hypersensitivity.[14]

Other desensitizing agents work typically as dentinal tubule blockers. Several professionally applied agents are available with various levels of clinical evaluation (see Table in Introduction). These include high-concentration fluorides, various oxalate salts, protein precipitants, and physical agents such as filled and unfilled resins and glass ionomers.

Patients should be instructed to use OTC desensitizing agents exclusively for maximum results. They should be advised that the full desensitization effect may not occur immediately (2 or more weeks) and be encouraged to use the dentifrices continually.[8] Patients with dentin hypersensitivity and high caries or erosion risk should select a desensitizing dentifrice that also has high fluoride availability and demonstrated fluoride uptake.[15]

NONSURGICAL PERIODONTAL THERAPY

Traditionally, the objective of mechanical therapy was to aggressively root plane the tooth surface to achieve a surface that was glassy smooth to a lightly held dental explorer. This aggressive debridement with sharp periodontal instruments was found to create or worsen tooth hypersensitivity. A contemporary objective is to remove all calculus deposits and cementum contaminated with endotoxins with the least amount of effective lateral pressure. After hard-deposit removal, a root-surface debridement technique follows. Root-surface debridement is a paradigm shift from traditional dental hygiene practice in that light pressure using fine finishing curettes is advocated to gently debride root surfaces and remove harmful endotoxins.[16]

The use of power scalers is another important step in reducing hypersensitivity caused during dental hygiene therapy. Ultrasonic and sonic scalers enable dental hygienists to debride hard deposits with minimal lateral pressure applied to the tooth surface. Additionally, the lavage action of power scalers assists with removal of endotoxins.

Removal of extrinsic stains is another dental hygiene treatment that can promote or aggravate tooth hypersensitivity. Tenacious deposits traditionally have required repeated application of lateral pressure using periodontal instruments. Advances in air power-polishing technology have drastically reduced the need for this method of debridement.[16]

For patients who do not require extensive power scaling, prophy pastes are often used to remove biofilm and light stains from the teeth. When using abrasive polishing agents, such as coarse-grit prophy paste, damage in the form of surface scratching and loss of enamel and cementum may result. Fine-grit prophy paste is recommended to reduce this potential damage.[7,16]

Recent advances in prophy paste include formulas that deliver amorphous calcium phosphate (ACP), an agent shown to desensitize dentin by depositing ACP into the tubules.[15] Another innovation is a prophy angle that embeds the paste within the prophy cup and claims to reduce enamel abrasiveness by 50% when compared with using a prophy cup and medium-grit paste separately. As a general rule, when selecting a prophy paste, the least abrasive will be the paste of choice, and it should be deployed with the least amount of pressure commensurate with removing the stain and leaving a smooth and minimally scratched surface.[7,16]

Innovations in nonsurgical periodontal instrumentation for the removal of hard deposits, biofilm, and extrinsic stains are continuously providing dental hygienists with greater armamentarium for preventing and managing dentin hypersensitivity.

CONCLUSION

The dentist is responsible for the diagnosis and initial therapy in the treatment of sensitive teeth. Concurrently, the dental hygienist must become actively involved in the suppression of symptoms and prevention of severity as the patient returns for recare appointments. Based on the patient's oral health status and oral hygiene habits, appropriate intervals for recare should be established. Current treatment modalities in nonsurgical periodontal instrumentation, in-office and OTC desensitizing medicaments, and oral hygiene products have provided dental hygienists with effective means of managing patients with dentin hypersensitivity. New knowledge and products are rapidly developing. It is critical for dental hygienists to engage in lifelong learning through continuing education and review of the published literature regarding advancements in evidence related to dental hypersensitivity. Contributions by dental

Innovations in nonsurgical periodontal instrumentation for the removal of hard deposits, biofilm, and extrinsic stains are continuously providing dental hygienists with greater armamentarium for preventing and managing dentin hypersensitivity.

hygienists to the body of knowledge through participation in research, publication, and presentation are equally vital to the dental profession and to the patients they serve.

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RECOMMENDED READING

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Schiff T, He T, Sagel L, Baker R. Efficacy and safety of a novel stabilized stannous fluoride and sodium hexametaphosphate dentifrice for dentinal hypersensitivity. *J Contemp Dent Pract*. 2006;7(2):1-8.

Jackson R. Potential treatment modalities for dentine hypersensitivity: home use products. In: Addy M, Embery G, Edgar WM, Orchardson R. *Tooth Wear and Sensitivity: Clinical Advances in Restorative Dentistry*. London: Martin Dunitz, 2000; 327-338.

Addy M, Hunter ML. Can tooth brushing damage your health? Effects on oral and dental tissues. *Int Dent J*. 2003;53 (Suppl 3):177-186.

Lindhe J, Nyman S, Karring T. Scaling and root planing in shallow pockets. *J Clin Periodontol*. 1982;9(5):415-418.

Silverman G, Berman E, Hanna CB, et al. Assessing the efficacy of three dentifrices in the treatment of dentinal hypersensitivity. *J Am Dent Assoc*. 1996;127:191-201.

Haywood VB, Cordero F, Wright K, et al. Brushing with potassium nitrate dentifrice to reduce bleaching sensitivity. *J Clin Dent*. 2005:16(1):17-22.

Research Panel

David H. Pashley

David H. Pashley earned a DMD degree from the Oregon Health Sciences University in 1964 and then moved to Rochester, New York, to earn a PhD in physiology from the University of Rochester School of Medicine and Dentistry in 1970. That same year, he joined the faculty at the Medical College of Georgia as an assistant professor and is currently Regents' Professor (1987-present) of Oral Biology in the School of Dentistry and professor of Physiology and Endocrinology in the School of Medicine.

His research interests include pulp biology, the structure and function of dentin, dentin sensitivity and its treatment, dentin bonding, the mechanical properties of dentin and their modification, adhesive dentistry, bonding and dentin hypersensitivity. His research activities have been supported by the NIDCR since 1973. Dr. Pashley has published more than 500 papers in peer-reviewed journals, about two thirds of which deal with the structure and function of dentin. He has held various offices in the Pulp Biology Group of the IADR and received the Pulp Biology Research Award (one of the IADR Distinguished Scientist Awards) in 1990 for his research and contributions to the field of pulp biology. He received the Hollenbach award from the Academy of Operative Dentistry in 1998. In 2001, he received the Wilmer Souder award for this work in Dental Materials.

Dr. Pashley serves on several editorial boards, lectures nationally and internationally, and participates in international conferences in his area of research.

Franklin R. Tay

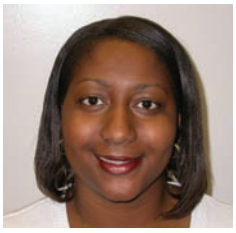
Franklin R. Tay received his BDSc with first-class honors from the University of Queensland, Australia, his PhD from the University of Hong Kong, China, and his Certificate of Endodontics from the Medical College of Georgia. He received his Board Certification from the American Association for Endodontists in 2008 and is a Diplomate of the American Board of Endodontics. He is currently an associate professor in the Department of Endodontics, School of Dentistry, Medical College of Georgia, and honorary professor, Faculty of Dentistry, The University of Hong Kong.

Dr. Tay's areas of research include: dental materials related to endondontics, dentin structure and function, dentin permeability, dentin bonding, regional bond strength measurements, and mechanical properties of dentin, collagen and resins, and biomimetic remineralization of dentin using nanotechnological approaches.

Van B. Haywood

Van B. Haywood is a professor in the Department of Oral Rehabilitation, School of Dentistry, Medical College of Georgia. A 1974 alumni of MCG, he was in private practice for 7 years in Augusta, Georgia, and taught at the University of North Carolina School of Dentistry in operative and prosthodontics for 12 years before returning to MCG in 1993. He teaches in the castings course, the occlusion course, and the esthetics course, as well as in student clinics, and is the director of continuing education for the School of Dentistry. He also sits on the editorial boards of several peer-reviewed publications.

In 1989, Dr. Haywood co-authored the first publication in the world on nightguard vital bleaching (at-home bleaching) with Dr. Harald Heymann, and in 1997 co-authored the first article on extended treatment (6 months) of tetracycline-stained teeth using this technique. He has completed further research and more than 90 publications on the nightguard vital bleaching technique and the topic of bleaching and esthetics, including the first papers on treating bleaching sensitivity with potassium nitrate, direct thermoplastic tray fabrication, and bleaching primary teeth.



Marie A. Collins

Marie A. Collins is chair and associate professor, Department of Dental Hygiene, and associate professor, Department of Periodontics, at the Medical College of Georgia School of Allied Health Sciences and School of Dentistry. She earned her BS (1994) and MS (1998) degrees from the University of North Carolina at Chapel Hill and her EdD (2006) from Georgia Southern University. She teaches periodontal instrumentation, research design, pharmacology, and basic life support. Since 2001, Dr. Collins has served as a director of the annual Dental Hygiene Symposium, one of the largest continuing education programs for dental hygienists. She was the recipient of the 2005 Outstanding Faculty Award from the MCG Greenblatt Library and is currently serving on two editorial review boards. Dr. Collins is a consultant for the Joint Commission on Dental Accreditation. Her publications include three chapters in dental hygiene textbooks and several refereed journal articles.

Connie Drisko

Connie Drisko was a practicing dental hygienist for 16 years before receiving a dental degree from the University of Missouri-Kansas City and completing a hospital-based general practice residency at the Veterans Administration Medical Center, Leavenworth, Kansas. Dr. Drisko received her certificate in periodontics and became a Diplomate of the American Board of Periodontology in 1990, was elected as a director in 2000 and served as chairman in 2006. She is a Fellow of the American College of Dentists, the International College of Dentists, and the Pierre Fauchard Academy. Dr. Drisko serves as chairman of the ADEA Women's Advisory Committee. In 2001, she was selected as a Fellow for the Executive Leadership Academic Medicine (ELAM) program and remains active in ELUM. She is currently Dean and Merritt Professor at the Medical College of Georgia School of Dentistry in Augusta, Georgia. Dr. Drisko has published more than 60 papers and monographs and presented continuing education programs on non-surgical periodontal therapy and dentin hypersensitivity nationally and internationally. She has participated in numerous symposia and expert panels on dentin hypersensitivity. Dr. Drisko's clinical research interests include local drug delivery, non-surgical therapies, and oral-health systemic diseases.

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