

Dentine hypersensitivity: Simplified

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Fig. 1

Fig. 1 The essential components of dentine hypersensitivity are exposed dentine surfaces with open patent tubules leading to a vital pulp. (Image courtesy of GSK)

All dental practices have patients with dentine hypersensitivity. Many patients avoid dental treatment because of their hypersensitivity. Surprisingly, most practices do not have a systematic approach for diagnosing and treating this condition. This is simply because it seems too complicated. There is a multitude of products. What works? Why does it work? Many practitioners have had poor success in the past with sensitivity treatments and are reluctant to try again. Today's products are effective and easy to use. The following discussion will attempt to bring simplicity and clarity to the subject of diagnosis and treatment of dentine hypersensitivity.

Definition

Dentine hypersensitivity is defined as a short sharp pain arising from exposed dentine in response to:

- _ thermal change,
- _ evaporation of air,
- _ tactile stimulus,
- _ osmotic pressure or
- _ chemical stimulus

and cannot be ascribed to any defect or pathology.¹

The three essential components of dentine hypersensitivity are (Fig. 1)²:

1. exposed dentine surfaces;
2. open tubule orifices on the exposed dentine surfaces;
3. patent tubules leading to vital pulp.

Dentine hypersensitivity has been reported to affect up to 57 % of the general population.³⁻¹⁰ It occurs most frequently in patients of 30 to 40 years of age.¹¹ All teeth are susceptible but canines and premolars are the most affected.^{12,13}

A 2002 international survey of 11,000 adults revealed that only half of the affected individuals had talked to their dentist about their sensitivity and only half of this group actually received treatment recommendations.¹⁴ Many patients do not wish to burden the dentist with this problem, or they may feel that it may not be taken seriously.

Mechanism of action

The most widely accepted theory for the mechanism that causes dentine hypersensitivity is the hydrodynamic theory first proposed by Brännström in 1963.¹⁵ When dentinal tubules in vital teeth are exposed and open, the fluid in the tubules flows in an inward or outward direction, depending on pres-

Active ingredient	Benefits and indications	Delivery	Product examples
Fluorides	Some efficacy. Stannous fluoride best. For mild sensitivity where patient also has gingivitis.	Toothpaste/at home	Crest Pro-Health (Procter & Gamble)
Fluoride varnishes	Some efficacy. For mild sensitivity where patient also requires anti-caries effect.	Varnish/in office	Vanish XT Extended Contact Varnish Vanish 5 % Sodium Fluoride White Varnish (all 3M ESPE) X-PUR White Varnish (Oral Science)
Tissue fixatives (glutaraldehyde or HEMA)	Strong efficacy. Place away from gingiva.	Liquid/in office	GLUMA Desensitizer (Heraeus)
Oxalates	Strong efficacy. Fast onset. Lasting.	Liquid/in office	Super Seal (Phoenix Dental)
Novamin	Strong efficacy. Cumulative onset. Remineraliser.	Toothpaste/at home	X-PUR (Oral Science) Sensodyne Repair and Protect (GlaxoSmithKline)
Recaldent	Strong efficacy. Cumulative onset. Remineraliser.	Extra paste/at home	MI Paste MI Paste Plus (GC Europe)
Pro-Argin Technology	Strong efficacy. Fast onset. Lasting.	Paste/in office Toothpaste/at home	Sensitive Pro-Relief (Colgate)
5 % potassium nitrate	Strong efficacy. Cumulative onset. Best treatment for whitening sensitivity.	Toothpaste/at home Toothpaste/in tray/at home Gel/at home	Sensodyne (GlaxoSmithKline) Crest Sensitivity Protection (Procter & Gamble) Sensodyne (GlaxoSmithKline) Crest Sensitivity Protection (Procter & Gamble) Soothe desensitizer (SDI) Ultra EZ (Ultradent) Relief ACP (Discus Dental)

sure differences in the surrounding tissue. This fluid shift activates pain receptors in the intra-tubular nerves or superficial pulp and the patient feels pain (Fig. 2).²

Diagnosis

Prior to establishing the diagnosis of dentine hypersensitivity, one must first rule out other conditions that exhibit similar symptoms²:

- _caries;
- _pulpitis;
- _marginal leakage;
- _restoration fracture;
- _cracked tooth;
- _polymerisation shrinkage.

It is important to use specific clinical descriptors with the patient (like brief, sharp, localised) to differentiate dentine hypersensitivity from pulpal pain (which is prolonged, dull, aching, poorly localised and longer lasting).²

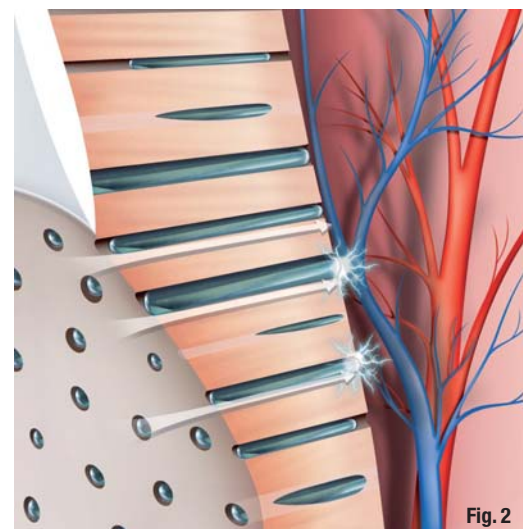
Risk factors for dentine hypersensitivity include¹⁶:

- _periodontal disease;
- _gingival recession;
- _para-function (abfractions);
- _acidic diet;
- _xerostomia;
- _bleaching.

These factors predispose the patient to the essential components of dentine hypersensitivity: exposed, open and patent dentinal tubules leading to vital pulp. There may also be passage of fluids through the enamel. The enamel may be thought of as a semi-permeable membrane that allows passage of fluids and small molecules through the organic defects between the enamel crystals. With time, the organic channels

Table 1 Treatment of dentine hypersensitivity.

Fig. 2 Fluid shifts in the dentinal tubules activate pain receptors to cause pain. (Courtesy of GSK)



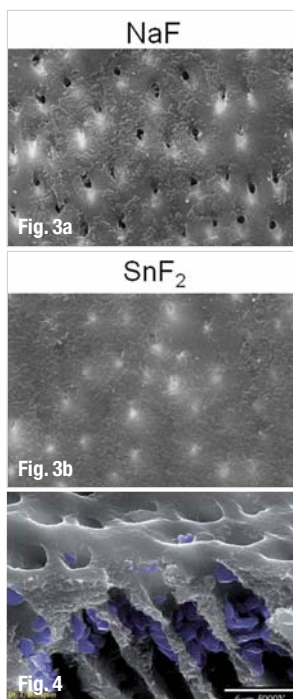


Fig. 3a & b In the concentrations used for toothpastes, stannous fluoride is more effective than sodium fluoride in occluding dentinal tubules. (Courtesy of Procter & Gamble)

Fig. 4 Super Seal forms a complex with the calcium of the peri-tubular dentine to create a crystal plug across the dentine tubules. (Courtesy of Tammy Bonstein)

become plugged owing to the formation of organic biofilm. When this occurs, the bidirectional flow of fluids stops and so does the pain. During bleaching, the organic plugs may be dissolved, reopening the enamel channels and causing sensitivity.¹⁷

Treatment

The first line of treatment for dentine hypersensitivity is of course prevention. All of the predisposing factors must be dealt with first. This may not be an easy task. Periodontal disease, recession, occlusal forces and diet present many challenges. The treatment of sensitivity is much simpler in comparison.

If we review the mechanism of action of dentine hypersensitivity, it is easy to understand the wide range of products available for treatment. The product must either block the movement of fluid in the tubules or stop the transmission of the pain response to the pulp. For added simplification, it is important to focus on the active ingredient, and not on the multitude of products (Table I).

Products are available for in-office or at-home application. Treatment should not be restricted to one option only. This is not a one-size-fits-all solution. Different treatments may be tried and modified based on the patient's response.

The first group of products works by occluding the open tubules and decreasing pulpal fluid flow. This group includes fluorides, fluoride varnishes, tissue fixatives, oxalates, remineralising agents and Pro-Argin Technology. The second group of products works by depolarising the nerve so that it cannot transmit the pain response.

Occlusion of dentinal tubules

Fluorides

Fluoride application is believed to work through a reaction between the fluoride ion and ionised calcium in the tubular fluid. This reaction forms an insoluble calcium fluoride precipitate in the tubule.¹⁸ Different fluorides show differing efficacies. Stannous fluoride is more effective than sodium fluoride in the concentrations used for toothpaste formulations (Fig. 3a & b).

Fluoride varnishes

Fluoride varnishes may be used for sensitivity relief but are chiefly indicated for caries control and remineralisation. The desensitisation effect is transient, since the material is abraded soon after placement. Many applications may be necessary for increased efficacy. It is thought that the benefit comes from the

physical blockage of the tubules by the varnish base rather than the fluoride itself.¹⁸

Tissue fixatives

Tissue-fixative desensitising products contain agents such as glutaraldehyde or HEMA. These agents bind to tissue fluid proteins in the dentinal tubules and the superficial cells of the subadjacent pulp and denature (coagulate) these proteins. These products cannot be placed near the gingival epithelium, since they may cause necrosis of the gingiva, as well as loss of the biological attachment.¹⁷

Oxalates

Desensitisers containing metallic salts, predominantly oxalates, form insoluble chemical precipitates in the peri-tubular dentine. No acid etch or light curing is needed. They cause no irritation of the gingival tissue. One example is Super Seal (Phoenix Dental). Super Seal forms a complex with the calcium-rich zone of the peri-tubular dentine to create a crystal plug. This effectively shuts down dentine sensitivity almost entirely (Fig. 4).¹⁹

Remineralising pastes

Remineralising pastes are used in the office or at home to restore the minerals that have leached out of patients' teeth owing to caries, diet, etc. These pastes have the added advantage of reducing sensitivity through tubule occlusion. Two active ingredients have been shown to be the most effective for this purpose:

1. Novamin (calcium sodium phosphosilicate bio-active glass) and amorphous calcium phosphate: Novamin-containing toothpastes have been shown to reduce dentine hypersensitivity significantly, with continued home use.^{20,21} The effect is cumulative up to about six weeks and then stabilises.
2. ACP: ACP forms a protective mineral barrier of hydroxyapatite that occludes the exposed dentinal tubules (Fig. 5a & b).²² ACP is most effective in the form called Recaldent (casein phosphopeptide-amorphous calcium phosphate) in which the casein portion (derived from milk) binds the ACP to the tooth surface, where it can do its job. Recaldent-containing pastes are placed on the affected areas after regular brushing.

Pro-Argin Technology

In healthy patients, saliva is normally very effective in reducing dentine hypersensitivity. Saliva provides calcium and phosphate, which over time occlude open dentine tubules. Pro-Argin Technology was developed based on this role that saliva plays in naturally reducing hypersensitivity.^{23,24}

The Pro-Argin formula contains arginine, an amino acid found in saliva. The positively charged arginine binds to the negatively charged dentine surface. This attracts a calcium-rich layer from the saliva to infiltrate and block the dentinal tubules (Fig. 6).¹⁶

This technology is available for in-office application, through a paste that is delivered by prophylaxis cup. There is also a toothpaste for at-home use. The in-office paste has been found to provide immediate and lasting relief of hypersensitivity for four weeks when it is applied as the final polishing step of a professional cleaning.²⁵ It has also been found to decrease dental prophylaxis discomfort when used prior to the procedure.²⁶

Depolarisation of the nerve

The second major group of desensitisation products works by depolarising the nerve that transmits the pain response. After the nerve has been depolarised, it cannot re-polarise and this diminishes its excitability. The ingredient that produces this effect is potassium nitrate.²⁷ According to the FDA, for a potassium nitrate toothpaste to claim to be desensitising it must contain 5% of the ingredient. Potassium nitrate penetrates the enamel and dentine to travel to the pulp and exerts a calming effect on the nerve. This effect can be thought of as anesthetic-like.²⁸

Potassium nitrate products are ideal for whitening sensitivity. Whitening sensitivity occurs due to the easy passage of peroxide through the enamel (a semi-permeable membrane) and dentine to the pulp. Desensitisation products that work by occluding the dentinal tubules are ineffective in preventing the passage of the tiny peroxide molecule, which can travel in the interstitial spaces between the tubules.²⁸

Potassium nitrate can be delivered in several effective ways to counteract whitening sensitivity:

1. Pre-brushing with a 5 % potassium nitrate toothpaste for two weeks pre-whitening and during whitening: It takes approximately two weeks for the potassium nitrate to be at peak desensitisation efficacy.²⁹
2. Whitening tray delivery of a potassium nitrate toothpaste for ten to 30 minutes during whitening treatment: This appears to be very effective for more acute sensitivity.²⁸ It is preferable to use a toothpaste without sodium lauryl sulphate, which is the primary ingredient in most toothpastes, and creates the effect of foaming. Sodium lauryl sulphate has been associated with increased gingival irritation, especially on prolonged contact.
3. Syringe delivery of potassium nitrate and fluoride: The material is applied as needed for specific areas of sensitivity.

4. Potassium nitrate incorporation into the whitening gel itself: Bleaching efficacy does not appear to be affected by this addition.³⁰

Conclusion

Treatment of dentine hypersensitivity is a simple, clear process. It starts with a differential diagnosis, ruling out other possible aetiologies like caries, pulpitis, cracks, marginal leakage, etc. Next, an attempt is made to eliminate predisposing factors such as periodontal disease, para-function, acidic diet and xerostomia.

At the same time, the patient is evaluated with respect to the potpourri of potential desensitisation ingredients and the products that contain them. It is essential for the dental practitioner to be familiar with these ingredients, their mechanisms of action, benefits and indications. Some patients may require more than one type of treatment. The treatment is fine-tuned until a successful solution is found. There is no longer a reason for any patient to endure dentine hypersensitivity. Simple answers have been found to this long-time problem, and the dentist has gained a patient for life.

Editorial note: A complete list of references is available from the publisher.

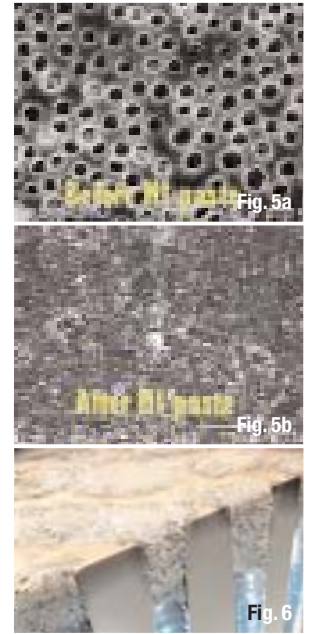


Fig. 5a & b ACP forms a mineral barrier of hydroxyapatite, which occludes the exposed dentinal tubules. (Courtesy of GC)

Fig. 6 The Pro-Argin formulation attracts a calcium-rich layer from the saliva to infiltrate and block the dentinal tubules. (Courtesy of Colgate)

about the author

cosmetic
dentistry



Dr Fay Goldstep has been a featured speaker in the ADA Seminar Series, and has lectured at the American Dental Association, Yankee, American Academy of Cosmetic Dentistry, Academy of General Dentistry, and the Big Apple dental conferences.

She has lectured nationally and internationally on Conservative dentistry, innovations in hygiene, dentist health issues, magnification and office design. She has served on the teaching faculties of the postgraduate programmes in Aesthetic Dentistry at SUNY Buffalo, University of Florida, University of Minnesota and University of Missouri-Kansas City. She has been a contributing author to three textbooks and has published more than 20 articles. She is a Fellow of the American College of Dentists, International Academy for Dental-Facial Esthetics and Academy of Dentistry International.

She sits on the editorial boards of the *Oral Health Journal* (healing/preventive dentistry) and *Dental Tribune US Edition*. She has been listed as one of the leaders in continuing education by *Dentistry Today* since 2002. Dr Goldstep is a consultant to a number of dental companies and maintains a private practice in Toronto, Canada.