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
- 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 per cent 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 pressure 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 symptoms:

- caries
- pulpsitis
- marginal leakage

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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 include:26

• periodontal disease
• gingival recession
• para-functional (abstractions)
• 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 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.27

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 fluoride varnishes, tissue sealants, casein phosphates, strontium tri fluoroaluminate and strontium chloride in proportions to be used in toothpaste formulations. These products are effective in occluding the dentinal tubules, and may be used as a band-aid approach while waiting for the desensitising agent to work.28

Fluorides - Fluoride varnishes, tissue sealants, casein phosphates, strontium tri fluor aluminate and strontium chloride can be used in the concentrations shown in Table I:29-32

Table I. Treatment of dentine hypersensitivity

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Power of action</th>
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<tbody>
<tr>
<td>Fluorides</td>
<td>Reduce fluid movement in the dentinal tubules</td>
</tr>
<tr>
<td>Casein phosphates</td>
<td>Reduce fluid movement in the dentinal tubules</td>
</tr>
<tr>
<td>Strontium tri fluor aluminate</td>
<td>Reduce fluid movement in the dentinal tubules</td>
</tr>
<tr>
<td>Strontium chloride</td>
<td>Reduce fluid movement in the dentinal tubules</td>
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</table>

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.33 Different fluorides show differing efficacies. Stannous fluoride is more effective than sodium fluoride in occluding dentinal tubules (Figs 1 & 2).34

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Bioresorbable barrier coatings

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**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)

**Fig. 2.** Fluid shifts in the dentinal tubules activate pain receptors to cause pain. (Courtesy of Procter & Gamble)

**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. (Courtesy of Tannock Brothers)

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.29,30 The effect is cumulative up to about six weeks and then stabilises.

2. AC-P: AC-P forms a protective mineral barrier of hydroxyapatite that occludes the exposed dentinal tubules (Fig. 3a & b).31 AC-P 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.32,33 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).34
Potassium nitrate can be delivered in several effective ways to counteract whitening sensitivity:

1. Pre-brushing with five per cent 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.  

2. Whitening tray delivery of a potassium nitrate toothpaste for ten to 50 minutes during whitening treatment: This appears to be very effective for more acute sensitivity. 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.

Conclusion  
Treating dentine hypersensitivity is a simple, clear process. It starts with a differential diagnosis, ruling out other possible aetiologies like caries, pulpsitis, cracks, marginal leakage, etc. Next, an attempt is made to eliminate predisposing factors such as periodontal disease, parafunction, 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.

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. Desensitising 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.