Dentine hypersensitivity
From diagnosis to treatment

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Dentine hypersensitivity is defined as pain arising from an exposed dentine in response to thermal, chemical, tactile or osmotic stimuli, which cannot be explained as arising from any other form of dental defect or pathology (Addy & Urquhart 1995). Such hypersensitivity is common: in a 1987 survey of a group of patients in the UK, Orchardson found that 74% suffered from some form of hypersensitivity. He also noted a very early peak of prevalence between 20 and 25 years. However, Addy (1992) reported a peak between 20 and 40 years, and Fisher (1992) reported a peak between 40 and 49. The teeth most frequently affected were the canines and upper first molars. It was also reported that in patients with periodontal disease, molars were more frequently affected.

It has been suggested that the incidence of hypersensitivity will increase as humans’ longevity increases and they retain their teeth until even more advanced ages; however, this is not supported by epidemiological studies. It is true that gingival recession and loss of cementum are more common in older individuals, but aged dentine is also less permeable, owing to the deposition of sclerotic and secondary dentine. Dentine hypersensitivity, while not a serious dental problem, can be removed by aggressive tooth brushing or root planning. One can explain the high incidence of sensitivity after periodontal treatment with exposed dentine due to both gingival recession and aggressive root planning. Another reason for exposed dentine is a developmental anomaly in the cervical region when enamel and dentine do not meet during the development stage. However, dentine hypersensitivity is often the result of a combination of the above factors.

It is a common belief that enamel is an impermeable tissue; however, a study of intra-tubular structure shows that it contains space around the crystals and prisms (Fig. 5), which is normal.

Neural theory
Nerve endings are present in the dentinal tubules, and mechanical and chemical stimuli trigger the pulpal nerve endings directly.

Hydrodynamic theory
This theory was proposed by Brannstrom and co-workers and is the most supported theory. The dentinal tubules are fluid filled and the fluid is disturbed by thermal, physical and osmotic changes on the surface of dentine, the baroreceptors are stimulated, which leads to discharge of nerve endings. Accordingly, dentine hypersensitivity tends to stand out is poor oral hygiene and subsequent plaque accumulation on root surfaces. It could be possible that stagnant plaque leads to demineralisation with a smear layer and opening of dental tubules. These early demineralised areas tend to be softer and more discoloured than the surrounding areas (Fig. 4).

Treatment options
The treatment options for dentine hypersensitivity can be broadly grouped into the desensitisation of nerve endings and the plugging or covering of the dentinal tubules.

Table 1. The high prevalence of dentine hypersensitivity led to the development of a surprisingly large number of products designed to alleviate this clinical problem. All available treatments appear to work; however, in recommending a treatment to patients, dental professionals should consider the needs of the individual, in order to maximise compliance.

Nerve desensitisation
There is much evidence to indicate that products containing potassium nitrate are effective in controlling dentine hypersensitivity. Tarbet et al. (1980; 1981; 1982) demonstrated in well-conducted clinical trials that, with daily use, a toothpaste containing 5% potassium nitrate was effective in desensitising for up to four weeks and that potassium nitrate does not induce changes in the pulp. It was suggested that the potassium (K) ion blocks nerve impulses by interfering with the sodium (Na) pump and depolarisation of nerve cell walls.

In the case of potassium oxalate, it is postulated that in addition to the effect of K there is also some plugging of the dentinal tubules with calcium oxalate.

These options with their relevant active ingredients are listed in Table 1. The high prevalence of dentine hypersensitivity led to the development of a surprisingly large number of products designed to alleviate this clinical problem. All available treatments appear to work; however, in recommending a treatment to patients, dental professionals should consider the needs of the individual, in order to maximise compliance.

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FIG. 1: A single step procedure in a dentinal tubule (this process is normally located in extracellular fluid).

FIG. 2: Dentine hypersensitivity can only occur when dentinal tubules are exposed to the external environment.
calcium oxalate salt, which decreases dentine’s permeability.

Nerve desensitisation works best in patients with generalised, mild to severe dentine hypersensitivity. As the active ingredient is built into a toothpaste, compliance is normally high because no extra step is added to the daily oral-care routine.

Dentine surface cover

When dentine hypersensitivity is severe and localised, patients may not be able to brush the affected areas well. It is important to protect these areas temporarily with a physical barrier, such as a varnish or thin glass ionomer, in order to desensitise and allow better cleaning. This method should also be combined with the use of a product containing potassium nitrate.

The use of a dentine bonding agent has been advocated, as it can provide short-term relief, but the seal provided by a dentine bonding agent, especially the single-bottle version, does not last long. Dentine bonding agents are not designed to be exposed to the oral environment and should not be utilised in an off-label fashion.

CPP-ACP-containing products, such as Tooth Mousse or MI Paste, are also good desensitisers. These products work instantly after direct application to the affected areas. The mechanism of action has not been elucidated, but it has been suggested that the protein component of CPP-ACP, casein phosphopeptide, forms a protective physical barrier over exposed dentine.

Plugging of dentinal tubules

There is evidence to support the use of the various active ingredients listed under the minerals/salts sub-heading. Most of the products containing these ingredients are for daily home use, and compliance is thus an issue that clinicians will need to manage.

The application of formaldehyde or glutaraldehyde should be done with caution, as these are strong tissue fixatives and much safer alternatives for the treatment of dentine hypersensitivity are available.

Conclusion

Dentine hypersensitivity is a common dental problem that can be managed successfully, using a wide range of in-office procedures and at-home products. When it is mild and generalised, the condition can be treated using toothpaste containing potassium nitrate or potassium oxalate as the active ingredient. Potassium nitrate, stannous fluoride and strontium chloride are active ingredients specifically designed for dentine hypersensitivity. Only in severe and localised cases should in-office procedures be used in complement at-home treatment.

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