Laser treatment of dentine hypersensitivity

An overview Part I

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_Introduction

More than two decades ago, laser applications in the treatment of dentine hypersensitivity were introduced to dentistry. Many clinical studies using different laser types have been published since. This overview summarises the basic and clinical aspects, including treatment protocols.

_The hypersensitive dental neck

Dentine hypersensitivity is a widespread painful condition in dentistry (Orchardson et al. 2006; Schwarz et al. 2002). Unfortunately, this clinical condition is often poorly understood (Pesevska et al. 2010). It is important to understand this condition in light of dental therapy because the need for treatment is increasing (Gerschman et al. 1994; Miglani et al. 2010). Hypersensitivity occurs when tooth necks are exposed. Brannström's hydrodynamic theory describes these mechanisms. Chemical, osmotic, physical or mechanical stimuli induce movement of fluid in dental tubules, which activates pain fibres—mainly A-δ fibres—at the pulp-dentine border (Brannström 1992). Afflicted patients describe an intense and sharp pain of short duration that cannot be ascribed to any other form of dental defect or disease (Canadian Advisory Board on Dentin Hypersensitivity 2003). In a huge number of cases, these symptoms are triggered by cold and are not related to restorative or caries therapy. The well-being of patients who suffer from dentine hypersensitivity is often affected. Daily stimulation, for example eating or teeth brushing, could induce considerable pain (Rösing et al. 2009; Tengrungsun et al. 2008).

Today, more than 30 % of the adult population of industrialised nations is thought to be affected (Ritter et al. 2006; Gillam et al. 1997), but the actual prevalence is still unknown (Rösing et al. 2009). The reported incidence mainly depends on the population under examination and the methodology of the studies, which vary a lot. Middle-aged patients are most often affected (Al Sabbagh et al. 2004), but a growing number of young persons are affected by dentine hypersensitivity (Sykes 2007). The higher frequency of hypersensitivity, even in middle-aged adults, could be explained by increasing exposure of tooth necks, which occurs increasingly early on in young people (Dowell et al. 1983). The main reasons for exposure are erosion, abrasion and attrition. However, erosion is most likely to be the main factor (Addy et al. 1994; Gillam et al. 1997). This comes from an increasing awareness of dental hygiene, which has resulted in well-intentioned but incorrect brushing (Fig. 1). For these reasons, sensitive tooth necks frequently occur opposite the hand that is brushing. The loss of the thin cementum layer in the tooth-neck area is responsible for more than 90 % of the hypersensitive surfaces (Orchardson et al. 1987). As a result, dentine hypersensitivity can be characterised as a tooth-wear phenomenon (Bamise et al. 2008). Restorations, functional overload and bleaching of vital teeth are also causes of dentine hypersensitivity (Brannström 1992; Jacobsen et al. 2001; Haywood 2002). Nowadays, one therefore speaks of a multifactorial genesis of dentine hypersensitivity associated with tooth wear.

Despite intensive research, the precise physiological mechanisms of pain production and transmission in the dental pulp have not been sufficiently determined (He et al. 2011; Bal et al. 1999; Zhang et al. 1998). The surface texture of dentine, that is the number of open tubules at the root neck, is of great relevance to the process of dentine hypersensitivity (Absi et al. 1987; Markowitz 1993; Fig. 2). The extent of hypersensitivity depends on



Fig. 1_Clinical situation of a patient with gingival recession. The occurrence of gingival recession is a precursor to the loss of cement, owing to poor oral hygiene, the exposure of tooth necks and dentine hypersensitivity.

the number of exposed tubules (Ngassapa 1996). Clinical studies (Narhi et al. 1992) and SEM examinations (Absi et al. 1987) have demonstrated that the dentine of hypersensitive teeth has a considerably higher number of exposed dental tubules (eight times the number) with a considerably greater aperture (twice the size) compared with teeth with no hypersensitivity symptoms (Absi et al. 1987). Since dentine hypersensitivity can occur even after the closure of most of the dental tubules, other factors, like inflammatory mediators, are assumed to be involved in nerve stimulation and pain (Narhi et al. 1992; Ngassapa 1996) in addition to the hydrodynamic theory.

_Conventional therapy

Currently, a multitude of desensitising agents are available on the market. Normally, the therapeutic mechanism is based upon the hydrodynamic theory. By occluding dentinal tubules, movement of fluid should be reduced. Furthermore, the reduction of pulpal pain-nerve fibre firing should contribute to reduced sensitivity.

The spectrum of applications ranges from agents for topical application, dentine adhesives, light-curing primer systems, mucogingival surgery for soft-tissue management, conventional fillings, to endodontics as ultima ratio. Topical applications are the most important for desensitisation; among them are fluorides, potassium and calcium compounds, hydroxyapatite, bioactive glass and glass-ceramics, oxalates, glutaraldehyde, formalin and chlorhexidine. These ingredients are mostly found in toothpastes, mouth rinses, gels, suspensions, varnishes or pastes for topical application. They can also be applied via chewing gum or iontophoresis. Today, the application of nano-sized particles of different mixtures is also very popular.

In 1935, Grossman formulated a set of criteria that desensitising agents are to fulfil. These criteria are still valid today (Renton-Harper et al. 1992):

– no irritation of pulp tissue;
– painless application;
– easy to apply;
– rapid in effect;
– sufficient efficiency;
– no side-effects; and
– long-term effectiveness.

The predominant number of compounds lead to a rapid resolution of symptoms, but often they are only short-lived. In most cases, a long-term occlusion of dentinal tubules cannot be attained. Thus far, no desensitising substance or method of application has fulfilled all the criteria Grossmann postulated in 1935. There has been a constant improvement in terms of the available means of treatment and active substances, but the reports still demonstrate difficulty in relieving the pain (Romano et al. 2011). Until recently, no universally applicable substance was available (Mitchell et al. 2011; Tirapelli et al. 2010; Dabahne et al. 1999).

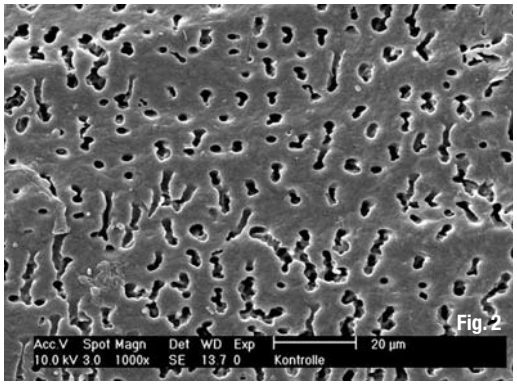


Fig. 2_SEM examination of a human cervical dentinal surface with open tubules, after experimental removal of the smear layer with 50 % citric acid for one minute (1,000x magnification).

_Laser application

Precipitates and surface coverings disintegrate after some time, for example owing to the influence of acid or toothbrush abrasion (Addy et al. 1983). Conventional desensitising treatments had to be applied repeatedly at regular intervals to ensure a durable analgesia (Gillam et al. 1992; Cuenin et al. 1991). The application of laser beams of different wavelengths and energy levels, alone or in combination with topical application, for example fluoride, is an alternative method for the treatment of dentine hypersensitivity.

_Studies on the effects of laser application

In vitro and *in vivo* studies document high effectiveness rates and comparatively long-lasting pain relief. The most commonly used types of lasers for the treatment of dentine hypersensitivity already determined *in vivo* can be divided into two groups. Those lasers with a lower output power, low-level lasers (He-Ne and GaAlAs diode lasers), are applied to biostimulation and are distinguishable from laser types with a middle-output power, middle-output power lasers, which have the ability to morphologically alter dental hard tissue.

The middle-output power lasers include the Nd:YAG, Er:YAG laser and CO_2 lasers (Dederich et al. 1984; Melcer et al. 1985; Featherstone et al. 1987). The effect of these laser types can probably be attributed to the sealing of the dentinal tubules, nerve analgesia and placebo effects. The sealing has been observed to be of long-lasting effect, whereas in

the case of nerve analgesia and placebo effects, the effects are not durable (Kimura et al. 2000a). Despite the precise mechanisms of laser action, the long-term value of laser therapy in treating dentine hypersensitivity is uncertain; current evidence is based upon a slight superiority compared with conventional topical applications (He et al. 2011).

Laser therapy with low-output power has been applied to humans since the 1970s and was originally used for wound healing (Kimura et al. 1991, 1993, 1997). In the 1980s, the inhibition of inflammation (Karu 1988, 1989) and the stimulation of nerve cells were demonstrated (Kimura et al. 1993; Jarvis et al. 1990).

_Low-level lasers: GaAlAs laser

Thus far, GaAlAs diode lasers of three wavelengths have been used clinically to treat dentine hypersensitivity: 780, 830 and 900 nm. The 780 nm wavelength has been used with good clinical success (30 mW in continuous wave mode [cw] and an application time of about 0.5 to three minutes; Matsumoto et al. 1985a). The 830 nm wavelength for the treatment of dentine hypersensitivity was first described by Matsumoto in 1990. With an energy setting of 20 to 60 mW in cw mode and an application time of 0.5 to three minutes, the effectiveness of therapy was 30 to 100 %, depending on the energy level (Matsumoto et al. 1990). The effectiveness of therapy using the 900 nm wavelength and an output power of 2.4 mW at 1.2 Hz for 2.5 minutes was approximately 73 to 100 % (Kimura et al. 2000b). Yamaguchi et al. (1990) compared the application of a GaAlAs diode laser of a wavelength of 790 nm and an output power of 30 mW and a placebo in vivo. Significant differences between laser application and the placebo group were found. At two hours, one day and five days after laser application, a significant improvement was detected (Yamaguchi et al. 1990). Overall, compared with the placebo, an improvement of 60 to 22 % was achieved. GaAlAs laser application with a maximal energy output of 60 mW did not damage enamel or dentine surfaces morphologically (Watanabe et al. 1991).

According to another in vitro study, the application of GaAlAs laser diodes with energy levels of 30 mW (cw, 780 nm), 60 mW (cw, 830 nm) and 10 W (pulsed, 900 nm) did not lead to a significant increase in the temperature in the dental pulp (Arrastia et al. 1994). Gerschman et al. (1994) accomplished a clinical double-blind study with GaAlAs lasers compared with a placebo. During the therapy, the apical and cervical regions of the tooth were irradiated for one minute; this procedure was repeated in one-week, two-week and eight-week intervals. Dentine hypersensitivity evoked by tactile and thermal stimuli was measured at each control and compared with the placebo group. A significant difference concerning dentine hypersensitivity was detected during the analysis. It was therefore deduced that the GaAlAs laser is an effective method for dentine desensitisation (Gerschman et al. 1994).

_Combined application of GaAlAs laser and fluoride

In another clinical study, a laser with an energy density of 3 and 5 J/cm^2 was applied up to six times in an interval of 72 hours after each application (Marsilio et al. 2003). Dentine hypersensitivity was measured initially after each application, and at 15 and 60 days after application. In more than 85 % of the teeth treated, an improvement was detected. Side-effects were not observed (Marsilio et al. 2003). In a comparative study of GaAlAs laser application (630–670 nm) at energy levels of 15 mW in contact mode for 20 seconds and fluoride application (Fluor Protector, Ivoclar Vivadent), significant and complete relief from pain after three sessions was achieved in 86.6 % of the cases compared with 26.6 % after fluoridation only (Pesevska et al. 2010). The clinical findings of Noya et al. (2004) proved that just a single application of GaAlAs laser (670 nm, 15 mW, 5 J/cm2) yields a statistically significant reduction in sensitivity to mechanical and thermal stimuli and that two applications are enough to reduce sensitivity to air. No other application led to any additional benefit (Noya et al. 2004).

A significant reduction in VAS scores was also detected with laser application for 160 seconds at an energy density of 4 J/cm^2 per dental element at 24 hours and seven days after application compared with a placebo application (Orhan et al. 2010). All teeth remained vital and no adverse effects were found radiographically, but the results were not significantly different from any conventional desensitiser (Gluma Desensitizer, Heraeus Kulzer; Orhan et al. 2010).

A combined application of GaAlAs laser light with sodium fluoride (NaF) at an energy output of 15 mW and density of 4 J/cm^2 led to no statistically significant differences between laser application and combined laser and fluoridation treatment (Corona et al. 2003).

Whether a combined application of GaAlAs laser and fluoride is effective in treating dentine hyper-



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sensitivity is questionable. Results are contradictory, often masked by placebo effects and highly dependent on laser parameters and the given circumstances for each patient. Both methods of treatment, GaAlAs laser application and fluoridation, are suitable for dentine hypersensitivity therapy.

_Biostimulative mechanisms of GaAlAs lasers

The GaAlAs laser mechanism is thought to be biostimulative rather than effecting morphological changes in dentine. It is assumed that the laser energy is transmitted to the dentine–pulp complex, interacts with the pulp tissue and causes a photobiomodulating effect, increasing the cellular metabolic activity of the odontoblasts and obliterating the dentinal tubules by means of tertiary dentine production (Ladalardo et al. 2004; Walsh 1997).

Tengrungsun et al. (2008) compared the efficiency of a GaAlAs laser (790 nm, 30 mW, 1 min) with a dentine bonding agent (Scotchbond, 3M ESPE) and found a statistically significant reduction immediately and 15 days after treatment in both of the two groups. No additional reduction in the level of hypersensitivity 30 days after treatment was observed. The dentine bonding agent was significantly superior to the GaAlAs laser within all measuring periods, thus confirming the assumption that laser light may act in a different way from occluding dentinal tubules (Tengrungsun et al. 2008).

The immediate laser effect is assumed to be triggered by placebo effects as a result of endorphin release induced by the activation of the pain inhibition system of the organism. This leads to the release of endorphins by the central nervous system, possibly controlling the painful stimulus at the periphery, and causing biostimulative effects that happen gradually within a few days (Tuner et al. 2002; Kimura et al. 2000b; Trowgridge et al. 1990). Another assumption is the blocking of the depolarisation of C-fibre afferences (Wakabayashi et al. 1993). The exact mechanism of action of low-intensity lasers in dentine hypersensitivity is not thoroughly understood (Orhan et al. 2010).

_Infra-red wavelengths and high-power density

Laser diodes, some of which have already been tested *in vitro* and *in vivo*, with wavelengths in the infra-red area and a high-power density are easily available on the market. Because of the laser–tissue interaction of this laser type with high energy per surface density, its results are comparable to that of an Nd:YAG laser. The sealing ability of this laser type was observed with the parameters 810 nm, 2 W and 30 ms, and was found to be less than that of CO_2, Er,Cr:YSGG lasers and Nd:YAG lasers (Gholami et al. 2011).

Clinical findings with the use of a GaAlAs laser of a wavelength of 810 nm at 1.5 to 2.5 mW for one minute resulted in a rapid reduction of discomfort, 15 minutes and 30 minutes after laser irradiation (37 % after 15 minutes and 41 % after 30 minutes) compared with placebo laser application with a reduction of 9 %. The improvement remained stable until two weeks (72 %) and two months (66 %) after application compared with placebo laser application (a 28 % reduction at two weeks and 26 % at two months) and the application of 10 % potassium nitrate gel (a 36 % reduction at two weeks and 30 % at two months; Sicilia et al. 2009).

_Different effects in different age groups

Ladalardo et al. (2004) evaluated the clinical desensitising effect of red and infra-red GaAlAs laser light application (660 nm and 830 nm, 4 J/cm^2, cw, 114 seconds, contact mode) in adult patients of different age groups (25–35 years and 36–45 years) as an immediate (15 minutes and 30 minutes after application; four sessions with intervals of seven days between each session) and late therapeutic effect (15, 30 and 60 days after the conclusion of treatment). Significant desensitising levels were only found in patients aged between 25 to 35 with the 660 nm red laser, which was found to be more effective than the 830 nm infra-red laser immediately after and in the follow-up periods at 15, 30 and 60 days after the conclusion of treatment.

In the group of 36- to 45-year-old patients, the effect of the red laser was only moderate, with recurrence of sensitivity, and the infra-red laser was ineffective. The authors concluded that the desensitising effects immediately after laser irradiation resulted from a suppression of evoked potential of the pulp nociceptive nerve fibres, with a better tissue response in the younger group. Pathological processes, regressive or atrophic alterations of the odontoblasts and the dentine pulp complex may lower the reaction potential in older patients, with reduced effectiveness of the laser biostimulation (Ladalardo et al. 2004).

A significant clinical improvement was also observed after the combined application of GaAlAs laser (808 nm, cw, contact mode, 25 seconds) and

desensitiser toothpaste compared with the toothpaste alone (Dilsiz et al. 2010a).

_GaAlAs laser and acid resistance

Recently, the GaAlAs diode laser was tested to establish enhancement of the acid resistance of dentine surfaces as a preventive method for hypersensitivity. A degree of improvement in the acid resistance of dentine specimens after laser irradiation (808 mn, cw, 60 J/cm^2) and erosive challenge (1 M hypochlorous acid for five minutes) was found without creating thermal or structural damage (De-Melo et al. 2010). However, the exact mechanism of action remains unclear. Laser treatment can be a useful tool for rapid reduction in pain, and possibly in the improvement of acid resistance of dentine. Nevertheless, more basic research and long-term clinical trials are needed to evaluate the long-term efficiency of this method and to explain the exact mechanism of action.

_Low-level lasers: He-Ne laser

The He-Ne laser has a wavelength of 633 nm (Moritz et al. 2006) and is a low-level laser. The first application of the He-Ne laser in the therapy of dentine hypersensitivity was described by Senda et al. (1985), who used an output power of 6 mW and chose two different modes (cw and pulsed, 5 Hz). Effectiveness ranged from 5 to 100 %. In another study, an effectiveness of 5 to 18 % was achieved (Wilder-Smith 1988). To date, the mechanism of action of the He-Ne laser has not been explained completely (Moritz et al. 2006). An energy level of 6 mW does not alter the enamel or dentinal surface structure morphologically; however, laser energy transmits through enamel and dentine and reaches the pulp tissue (Watanabe 1993). Physiological experiments have shown that the He-Ne laser application does not influence nociceptors of circumferential A-δ- or C-fibres (Jarvis et al. 1990), but it does influence electrical activity (action potential), which in healthy nerves is enhanced by about 33 % after application (Rochkind et al. 1986). The effect is long-lasting and leads to an increase in the action potential of nerve fibres for more than eight months after laser application (Rochkind et al. 1986).

_He-Ne laser compared to Nd:YAG laser

In a clinical study (Gelsky et al. 1992), which was aimed at testing the effectiveness and confidence of the Nd:YAG laser in the therapy of dentine hypersensitivity in vivo (see below), the He-Ne laser was applied to one group and the He-Ne laser and the Nd:YAG laser were applied to the other group (30–100 mJ, 10 pps increments of ten to 40 seconds, total application time < two minutes, without local anaesthesia). Dentine hypersensitivity was measured mechanically (dental probe) and thermally (stream of cold air) with the aid of a VAS. Additionally, the pulpal sensibility was evoked electrically. Initially and after three months, X-rays were taken to identify possible side-effects of the laser application. Directly after laser application and three months later, a reduction in the level of discomfort was observed. After three months, dentine hypersensitivity evoked by thermal stimuli was scaled back to 63 % and hypersensitivity evoked by mechanical stimulation was scaled back to 61 %. Compared with the combined application of He-Ne and Nd:YAG lasers, there was not much difference (reduction of dentine hypersensitivity evoked by thermal stimulation to 58 %; evoked by mechanical stimulation to 61 %). All of the teeth were vital after application and no side-effects or complications could be detected.

If dentine hypersensitivity is conditioned by the hydrodynamic mechanism exclusively, (thermal) effects of Nd:YAG laser application should reduce dentine hypersensitivity primarily (Gelsky et al. 1992). In contrast, He-Ne laser application should not lead to important effects. Thus, it is assumed that a surficial modification is not the only desensitising factor, but that there is apparently also a neurophysiological component (Gelsky et al. 1992), which is affected by biostimulative effects.

In a study with a similar design, the reduction of dentine hypersensitivity after He-Ne laser application alone, as well as after combined He-Ne laser and Nd:YAG laser application, was detected. It remained stable for six weeks (Halket et al. 1996)._

Editorial note: To be continued in our next issue of Laser. A list of references is available from the publisher.

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