Laser Treatment of Hypersensitive Dentin: Comparative ESEM Investigations

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Purpose: To determine by environmental scanning electron microscopic (ESEM) examination whether KTP, diode, and CO_2 lasers are able to seal dentinal tubules with and without prior application of different types and concentrations of fluoride.

Materials and Methods: For in vitro environmental scanning electron microscopic (ESEM) examinations, human teeth were irradiated either with a laser alone or with a prior application of a 0.4% stannous fluoride gel, a 1% amino fluoride fluid or a 5% sodium fluoride varnish. Irradiation was performed with a KTP Laser (532 nm) at 0.7 W, a diode laser (810 nm) at 0.8 W, or a CO_2 laser (10.6 µm) at 0.3 W for 30 s.

Results: Only the combination of the stannous fluoride gel with CO_2 laser irradiation revealed complete closure of the dentinal tubules. Other fluorides combined with the CO_2 laser or any combination with the diode laser showed only partly occluded tubules. Fluoridation and KTP laser irradiation attained occlusion of most tubules. No closure of the dentinal tubules was observed in teeth which were treated with fluorides only.

Conclusion: CO_2 laser irradiation through a layer of stannous fluoride causes a highly resistant protective layer on sensitized dentin. This layer, induced by physical and chemical bonding mechanisms, provides a superior defense against external stimuli.

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Dentinal tubules, which are open to both the oral cavity and the pulp cavity, provide a connection between the oral environment and the sensitive nerve endings of the tooth pulp. The dentinal tubules are filled with long odontoblastic processes, also referred to as Tomes' fibers, and with interstitial hard-tissue fluid, termed dentinal liquor. Several stimuli can cause unpleasant sensations on exposed dentinal surfaces. Dentin hypersensitivity is characterized by 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.[1,2] Essentially, exposure of the dentin results from one of two processes, either removal of the enamel covering the crown of the tooth, or denudation of the root surface by loss of cement and overlying periodontal tissues. Removal of the enamel may result from attrition relating to occlusal abnormalities, toothbrush abrasion, dietary erosion, habits, or a combination of these factors. Etiologically important are also gingival recessions which increase in severity with advancing age, chronic periodontal disease, and certain forms of periodontal treatment.[3-8]

Areas of sensitive cervical dentin display patent dentin tubules.[9] Hypersensitive teeth demonstrate tubular diameters that are significantly wider than those of nonsensitive teeth, so it would appear that treatment focused on decreasing the radius is a prerequisite for effective desensitization.[10] Thus, in those individuals where no symptoms arise from dentin exposure, occlusion of tubules may have resulted from the formation of secondary dentin, or the development of sclerotic dentin.[11,12] However, blockage of the tubules at the dentin surface by other means may occur and may include dentifrice ingredients and oral debris.[13]

The prevalence of this discomfort ranges between 4% and 57%.[14-22] In patients with periodontitis, the prevalence of dentin hypersensitivity is between 60% and 98%.[23] The severity of the pain, or the patient's interpretation of this, appears to determine whether treatment is sought.[24-26] Since pain upon stimulation may arise where the pulp is normal,[27-29] inflamed or necrotic, the patient's experience of pain, or lack of it, is not a satisfactory indication of the pulp condition.[30] The very subjective measurement of pain arising from exposed dentin, which may be further modified by psychological factors, makes an accurate assessment of the extent of the problem difficult.[31] Nevertheless, dentin hypersensitivity – besides directly causing patient discomfort – may indirectly pose other problems, in particular those associated with reduced oral hygiene.[13] The failure to practice satisfactory plaque control has well-established consequences with respect to gingival health.[32] Furthermore, in those patients where dentin surfaces are exposed after gingival surgery, the success of treatment in the long term may be compromised.[33,34]

The most widely accepted theory for the transmission of stimuli to the pulp is by a hydrodynamic mechanism,[35-37] with a rapid movement of extracellular fluid within the dentinal tubules.[38-39] The walls of dentinal tubules were found to be considerably more mineralized than the rest of the dentin,[40] and the fluid contained therein would obey the same physical laws as liquids in glass capillaries.[37] Scanning electron microscopic investigations of human dentinal tubules demonstrated numbers of approximately 45,000/mm^2 at the pulp, 29,500/mm^2 in the middle dentin, and 20,000/mm^2 peripherally, with the diameter of tubules decreasing from 2.5 µm at the pulp to 0.9 µm peripherally.[41] Interestingly, odontoblast processes were seen only in the tubules near the pulp.

Pain would appear to be produced by the rapid displacement of the tubular contents at the pulp dentinal border, as opposed to the slow outward fluid flow, which seems to occur normally.[37] It was estimated that pain-producing stimuli created an outward fluid flow in tubules of 2 to 4 mm/s.[42,43] Rapid flow in the pulpal part of the dentinal tubule can be expected to result in deformation not only of the cellular processes but also of nerve fibers which might be present in the dentinal tubules or adjacent pulp.[44] Pain receptors of the tooth are not chemoreceptors, but rather mechanoreceptors.[45,46] The application of heat, however, produces an inward movement of the tubular contents at the pulp-dentin border.[47]Interestingly, the pain produced by the prolonged application of heat is of a dull nature, totally different from the sharp pain elicited by cold or an air blast.[48]

The pulp has several natural defenses to protect itself from irritating stimuli.[49] Pulpal calcification and the formation of secondary dentin, peritubular dentin, and dentinal sclerosis have been demonstrated.[50,51] This natural occlusion of the peritubular dentin by calcium crystals is the tooth's physiological response to dentinal sensitivity. The tooth may naturally desensitize itself with peritubular dentin mineralization. Another defense mechanism that may decrease dentinal sensitivity is the formation of plaque in the acquired salivary pellicle material, coupled with salivary occlusion.[52] Electron microscopic studies on teeth with incisor attrition revealed partially or completely obliterated dentinal tubules. Sclerotic zones beneath the region of attrition were occluded by peritubular, dentin-like material.[12]

In 1935, Grossman suggested the following requirements for a satisfactory material for the treatment of dentin hypersensitivity, which would appear to still be valid today: nonirritating to the pulp, relatively painless on application, easily applied, rapid in action, effective for a long time, nonstaining, and consistently effective.[53]

Conventional Treatment Alternatives

Treatment options for desensitizing hypersensitive teeth are desensitization of the nerve, coverage of the dentinal tubules, or as the final consequence, endo-dontic treatment (Table 1, modification of Jacobsen [54]).

Common therapies[49,55] employed to relieve pain have relied upon the astringent or coagulating effects of various agents, the occluding properties of others, or the ability to render calcium less soluble.[56] Among the most common agents now being used, the literature contains references to the efficacy of strontium chloride, sodium monofluorphosphate, sodium fluo-

Table 1 Treatment options

1. Desensitization of the nerve
potassium nitrate
low level laser therapy
Nd:YAG Laser
neural therapy (infiltrations with local anesthetics)
laser acupuncture
2. Coverage of dentinal tubules
1. Ions/salts
stannous fluoride
Na fluoride/stannous fluoride combination
potassium oxalate
ferrous oxide
strontium chloride
in combination with an adhesive
2. Precipitates – proteins/amino acids
a. glutaraldehyde
3. Resins
a. dentin sealers
b. methyl methacrylate
4. Laser treatment
periodontal surgery/grafting
composite/GIC restoration
crown placement
plugging (sclerosis) of dentinal tubules
3. Endodontics

ride,[57-59] calcium hydroxide, calcium phosphate,[60-62] potassium nitrate, potassium citrate,[63-67] formaldehyde, sodium citrate-pluronic gel, stannous fluoride, glucocorticoids, adhesives, bonding agents and resins,[68-79] glass-ionomer cement,[30,31] bioactive and biocompatible glasses,[32-35] and oxalate-containing products.[86-93]

Laser Application for Dentin Hypersensitivity Treatment

Conventional treatment methods as described before have the great disadvantage of having to be repeated regularly to achieve continuous pain relief.[94-96] Because acids contained in food or aggressive tooth brushing cause gradual removal of precipitations and superficial coatings,[97] the treatment agent must be applied repeatedly. The use of lasers might open up new dimensions in the treatment of dentin hypersensitivity.

The lasers used for the treatment of dentin hypersensitivity are divided into two groups: low output power or low-level lasers (He-Ne or diode lasers) and middle output power lasers (argon, KTP, diode, Nd:YAG, Er:YAG, ErCr:YSGG, and CO_2 lasers).[98-101]

Recurrence of hypersensitivity varies with each laser and treatment protocol. Laser effects are considered to be due to the effects of sealing the dentinal tubules, nerve analgesia, laser acupuncture, or a placebo effect. Only the sealing effect is considered to be durable.[6]

Low-level Lasers[6]

The He-Ne laser (wavelength 633 nm) was used for the treatment of dentin hypersensitivity by several investigators at an output power of 6 mW for 0.5 to 5 min.[102-106] There are two irradiation modes: pulsed (5 Hz only) and continuous wave (cw) mode. Wilder-Smith et al found a treatment effectiveness of only 5.2% to 17.5%.[107]

Three wavelengths (780, 830 and 900 nm) of GaAlAs (diode) lasers have been used for the treatment of dentin hypersensitivity. GaAlAs lasers at 780 nm were used at an output power of 30 mW in cw mode. Irradiation time ranged from 0.5 to 3 min.[108-111] 830-nm diode lasers were used at an output power of 20 to 60 mW, cw. Irradiation time ranged from 0.5 to 3 min.[112-120]

It is postulated that low output power lasers mediate an analgesic effect related to depressed nerve transmission. This effect is caused by blocking the depolarization of C-fiber afferents.[121-123] GaAlAs laser irradiation at a maximum power of 60 mW does not affect the enamel or dentin surface morphologically, but a small fraction of the laser energy is transmitted through enamel or dentin to reach the pulp tissue.[124]

Other areas of laser irradiation (laser acupuncture) for cervical dentin hypersensitivity treatment induce the nerve fibers related to the symptomatic region and acupuncture of sites, such as musculus adductor pollicis and lobulus auriculae.[125] Treatment effectiveness is dependent on the area irradiated.

Nd:YAG Laser, Diode Laser, Ho:YAG Lasers

Several authors used Nd:YAG lasers for the treatment of dentin hypersensitivity.[126-134] The output power ranged from 0.3 to 10 W, but 1 or 2 W output was most common. Irradiation methods were dependent on the laser powers and varied from 0.3 W for 90 s in noncontact mode to 2 W for 0.5 s on black ink in contact mode. When using Nd:YAG laser irradiation, the use of black ink as an absorption enhancer can prevent deep penetration of the Nd:YAG laser beam through the enamel and dentin and excessive effects in the pulp.[135] The use of black ink for enhancing the effects of Nd:YAG laser irradiation to treat dentin hypersensitivity has been reported to be effective.[115,118,119]

The mechanism of Nd:YAG laser effects on dentin hypersensitivity is thought to be the laser-induced occlusion or narrowing of dentinal tubules[136] as well as direct nerve analgesia. Laser energy at 1064 nm is transmitted through dentin,[137] producing thermally mediated effects on microcirculation[138] and pulpal analgesia via its innervation.[139] The mechanism of desensitization can also be regarded as a denaturation of the odontoblastic processes or as an overheating of the dentinal liquor.[133,134]

As mentioned, there is a significantly high correlation between the morphology[140] and the number[141,142] of open dentinal tubules and dentin hypersensitivity. During the irradiation of root dentin, a fusing of the dentinal tubules has been described, along with a vitrification of the dentinal surfaces.[143] Otherwise, no complete closure of the dentinal tubules can be observed in the dental neck region. In contrast, even adverse effects, such as an increase in color penetration, have been reported.[144] The absence of a smear layer can be regarded as the reason for this effect.

The sealing depth achieved by Nd:YAG laser irradiation at 30 mJ/pulse and 10 Hz on dentinal tubules was measured to be less than 4 mm.[145] However, in an in vitro study by Goharkhay et al,[146] scanning electron microscopic and stain penetration tests revealed topographically only incomplete closure of dentinal tubules with an inhomogeneous dentin surface when irradiated with the Nd:YAG laser at 0.2 or 0.5 W, 10 Hz, with and without prior application of a stannous fluoride gel. Higher energy resulted in a greater number of closed tubules with an increased removal of dentin.

When using laser in vivo, thermal effects on pulpal tissues are a concern. Compared to other lasers, the Nd:YAG laser beam penetrates deeply through dentin,[109] bone, and nonpigmented soft tissues.[147] Irradiation causing temperature rises exceeding the threshold of pulpal tolerance will cause thermal injury to the pulp. Previous studies have demonstrated that healthy pulp tissue is not injured thermally if the laser equipment is used at the correct parameters and the temperature increase to the pulp remains under 5.5°C.[148] Irradiation at 2 W and 2 Hz for 10 s induced pulpal temperature rises of 13.4°C through 2 mm of remaining dentin thickness.[149]

A decrease in permeability of 19% was observed in vitro.[150] However, Lier et al[151] concluded that the effect of treatment of hypersensitive teeth with Nd:YAG laser is not different from treatment with a placebo. The observed effects seemed to last for at least 16 weeks. Furthermore, no significant difference in the occluding effect of Nd:YAG laser and Sensodyne® toothpaste could be found in a recent study.[152]

In contrast to favorable congress contributions, no scientific publications proving the efficiency of the diode and Ho:YAG lasers are available. The diode laser application in combination with a fluoride gel could be advantageous due to the continuous wave or "chopped" working mode of this device. Irradiation with a diode laser (810 nm) at 0.2 or 0.5 W, 10 Hz, showed no closure in the cervical region, and Ho:YAG laser (2940 nm) treatment achieved partial sealing of the dentinal tubules at a lower power setting (0.2 W), but not at a higher setting (0.5 W). The dentin surface demonstrated a nonhomogeneous surface structure.

Furthermore, bacteria also seem to play an important role in the sensitivity of teeth. The pain threshold of the nerve fibers seems to be lowered in presence of inflammation mediators.[153] In this context, it is important to point to the results of previous studies, which have shown the high bactericidal potential of middle output power lasers.[154-162]

Er:YAG Laser

Desensitizing effects of an Er:YAG Laser (wavelength 2940 nm) have been reported by Schwarz et al.[163] Irradiation occurred at an energy level of 80 mJ/pulse, 3 Hz with water irrigation in a defocused manner for 2 min/tooth by scanning in an overlapping pattern. Significant improvement immediately after treatment was found to persist at the same level at the 6-month follow-up.

The energy setting used is lower than the ablation thresholds of dental hard tissues. The high absorption of the Er:YAG laser emission wavelength in water may result in an evaporation of the dentinal fluid and the smear layer. In a comparative study, the Er:YAG laser was the most effective tool in removing the smear layer from root canal walls.[164] Thus, it could be suggested that a deposition of insoluble salts in the exposed tubules are responsible for an obturation of the dentin tubules. In vitro, a decrease of permeability of 26% was achieved.[150]

Investigations at the University of Vienna resulted in the splitting off of dentinal hard tissue without sealing of the dentinal tubules, when irradiated with two different Er:YAG lasers, even at the lowest possible power settings of 0.2 and 0.5 W, with and without prior application of a stannous fluoride gel. The treated surfaces did not show any melted areas.[146]

The Er:YAG laser shows the lowest limitation due to thermal side effects because of its thermomechanical ablation mechanism and the high absorption of its wavelength by water.[165-168] An Er:YAG laser has a water absorption characteristic approximately 15 times greater than that of the CO_2 and even 20,000 times greater than the Nd:YAG laser.[169,170] The resulting penetration depth of the Er:YAG laser is in the μm range.

CO_2 Laser

The CO_2 Laser (10.6 μm) is the most frequently discussed laser for the treatment of dentin hypersensitivity. The impact of this laser is based on a closure or stricture of the dentinal tubules. In principle, there are two possibilities to utilize the CO_2 laser: either CO_2 laser alone, ie, the exposed dentin is directly irradiated (direct method), or as first described by Moritz et al[171-174] in combination with a fluoride gel (indirect method). In this case, stannous fluoride gel is first applied to the cleaned dental cervical area and the laser irradiation is carried out through the gel layer. Output powers described in literature of 0.5 and 1 W and the cw mode were used for both methods. Irradiation time ranged from 0.5 to 5 s, and irradiation was repeated 5 to 10 times.[175] CO_2 laser irradiation can also melt a DP-bioglass paste and create about 10 microns of sealing depth. There have been no reports on nerve analgesia by CO_2 laser irradiation.[176]

Using the CO_2 laser directly at moderate energy densities, mainly sealing of dentinal tubules is achieved, as a reduction of permeability due to the occlusion or narrowing of dentinal tubules.[177] CO_2 laser irradiation, like other wavelengths, may also cause dentinal desiccation, yielding temporary clinical relief of dentinal hypersensitivity.[178] The sealing depth achieved by CO_2 laser irradiation at 0.3 W for 0.1 s on dentinal tubules is usually measured to be 2 to 8 μm.[179] The long-term success rate of the sole laser application seems to be questionable: A treatment success of only 50% was reported.[180]

Moritz et al first described the indirect method: the CO_2 laser irradiation through a thin layer of stannous fluoride.[171-174] This procedure was developed based on combining the advantages of laser and fluoride therapy to thus achieve long-lasting treatment success. Combined use of laser irradiation with the chemical agent stannous fluoride,[218,219] which is incorporated into the dentinal surface for several years, guarantees freedom from pain for a long period of time. Comprehensive studies were carried out in vitro to document the safety and efficacy of this treatment method.

Stain penetration and ESEM (environmental scanning electron microscopy) investigations revealed that at present, the continuous wave CO_2 laser at a power output of 0.3 W in combination with prior application of a thin layer of stannous fluoride gel is the only laser to achieve optimum closure of dentin surfaces with a very homogenous surface structure, as seen with the scanning electron microscope.[146]

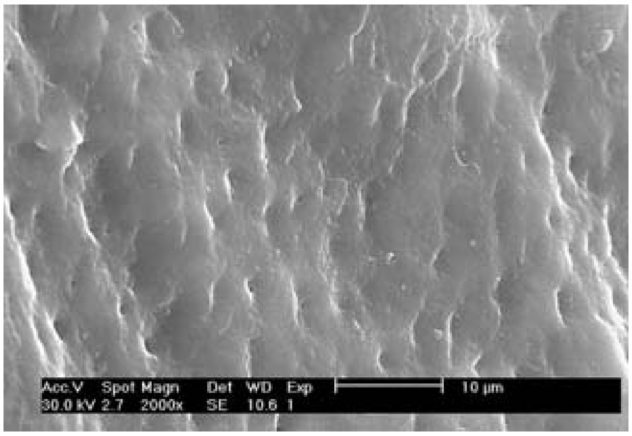

Fig 1 ESEM, 2000X, 30 kV, stannous fluoride gel and CO_2 laser irradiation, a continuous layer of closed tubules is formed.

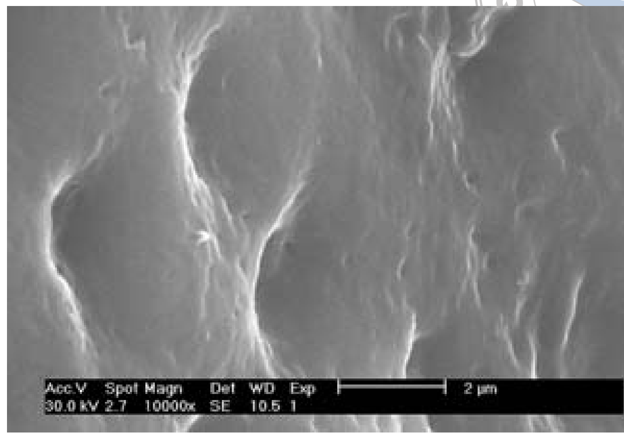

Fig 2 ESEM, 10,000X, 30 kV, detail from Fig 1, closed orifices.

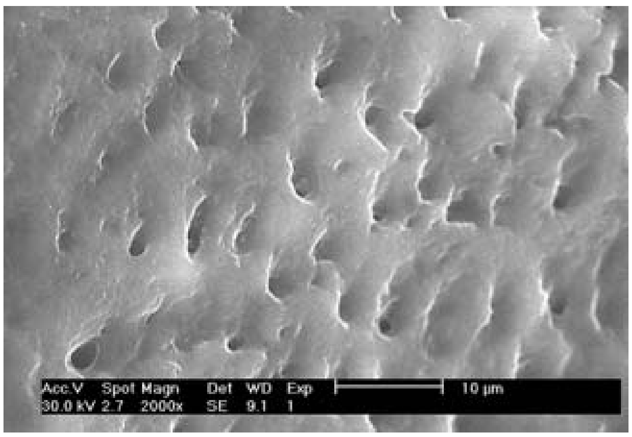

Fig 3 ESEM, 2000X, 30 kV, aminofluoride gel and CO_2 laser irradiation, tubules only partly closed, no homogeneous layer.

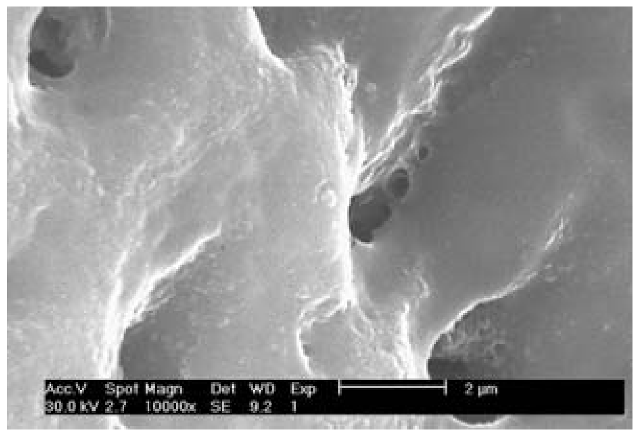

Fig 4 ESEM, 10,000X, 30 kV, detail from Fig 3, partly obstructed tubules.

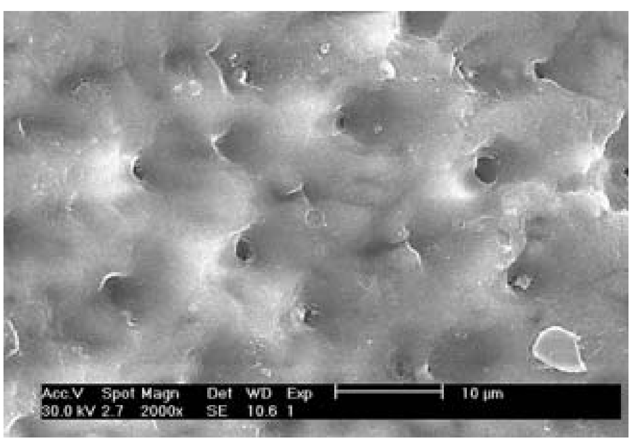

Fig 5 ESEM, 2000X, 30 kV, stannous fluoride gel and KTP laser irradiation, major obstruction of tubules.

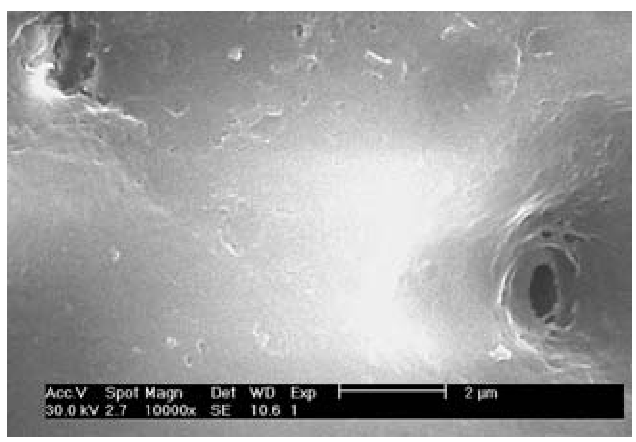

Fig 6 ESEM, 10,000X, 30 kV, detail from Fig 5, obstructed and constricted tubules.

To exclude thermal damage of the dental pulp after CO_2 laser irradiation, temperature measurements were performed. With the CO_2 laser, the enamel and dentin surfaces reach very high temperatures, but only low temperatures are measured in the pulp chambers.[135] At parameters of 0.5 or 1 W, an intrapulpal temperature rise below 1°C was measured.[181] This is related to the very high absorption and low penetration of light in hard dental tissues at this wavelength. Intermittent lasing of 6 times for 5 s with 20 s breaks excludes thermal damage to the pulp because the maximum temperature rise at 0.5 W does not exceed 2.5 C°.[171,172] The safety of CO_2 laser therapy is confirmed by these findings to a high extent.

The purpose of this in vitro study was to determine by environmental scanning electron microscopic (ESEM) examination whether KTP, diode, and CO_2 lasers are able to seal dentinal tubules with and without prior application of different types and concentrations of fluoride.

MATERIALS AND METHODS

For in vitro environmental scanning electron microscopic (ESEM) examinations, 32 extracted human premolars and molars were divided into 12 treatment and 4 control groups. Only caries-free teeth with intact enamel surfaces were used, which were extracted for periodontal or orthodontic reasons. Further inclusion criteria were no to minimal fillings and low levels of plaque. To avoid dehydration, the teeth were stored in physiological saline solution immediately after extraction. To achieve optimal surface conditions, the teeth underwent a cleaning procedure with polishing and a final ultrasonic bath followed by visual control under the optical microscope. The teeth were irradiated in a previously marked area of the dentin-enamel junction with different treatment protocols. Lasing occurred either with a KTP, diode, or CO_2 laser alone or with a prior application of a 0.4% stannous fluoride gel (gel kam, Colgate Palmolive, USA), a 1% amino fluoride fluid (elmex, GABA, Lörrach, Germany), or a 5% sodium fluoride varnish (duraphat, Colgate Palmolive, USA). Irradiation was performed with 3 different lasers: a Smart Lite KTP Laser (DEKA Dental Laser Systems, Florence, Italy) with 532 nm and a bleaching handpiece with a diameter of 5.7 mm at an output power of 1 W in continuous wave mode (effective output power measured with a Watt meter: 0.7 W); a LD 15 diode laser with 810 nm (Dentec Laser Systems, Bremen, Germany), 1.5W, cw and a handpiece diameter of 4.5 mm (effective output power: 0.8 W); or a Lasersat CO_2 laser with 10.6 µm (SATELEC, France), 0.5W, cw (effective output power: 0.3W). The teeth were lased at several intervals with permanent movement of the handpiece; 5 s of irradiation were followed by a 20-s break. This procedure was repeated 6 times, so that each sample was exposed to laser irradiation for 30 s. The control teeth were only fluoridated for 24 h in a humidity chamber. After treatment, the teeth were brushed off, cleaned as described above, and examined under the ESEM (Philips XL30 ESEM, Amsterdam, The Netherlands).

RESULTS

Environmental scanning electron microscopy revealed complete closure of the dentinal tubules only in teeth treated with stannous fluoride gel combined with CO_2 laser irradiation. A combination of aminofluoride fluid or sodium fluoride varnish with the CO_2 laser or any combination with the diode laser showed only partly obstructed tubules. CO_2 laser irradiation alone produced partially melted and fused areas. Fluoridation and KTP laser irradiation attained occlusion of most tubules (Figs 1 to 6). No closure of the dentinal tubules was observed in teeth which had been treated with fluorides only (Table 2).

DISCUSSION

In an in vivo study, the combination of stannous fluoride gel (indirect method) and the CO_2 laser showed a success rate of 94.5%; when marked pain relief was included in the definition of treatment success, 98.6% of the patients were treated successfully. Treatment of the control group with conventional dental cervix fluoridation resulted in no marked improvement. All patients showed identical perfusion indices immediately before and after CO_2 laser treatment at 0.5 W in the cw mode for 6 x 5 s with 20-s intervals, as well as 1 week after treatment.[172] Other authors also found no change in pulpal blood flow due to laser treatment.[182]

To assess the treatment method's exact mode of action, detailed physical examinations (AAS, XPS, EPMA, x-ray diffraction) were carried out at the Institute of Solid State Physics at the Technical University of Vienna. The examined teeth had been irradiated with the CO_2 laser and stannous fluoride in vivo and were extracted 18 months after the treatment for orthodontic reasons.[183] AAS (atomic absorption spectroscopy ex-

Table 2 ESEM evaluations

	no fluoridation	stannous fluoride	amino fluoride	sodium fluoride
control	-	-	-	-
CO_2 laser	-*	++	~	~
diode laser	~	~	~	~
KTP laser	~	+	+	+

++ closed tubules, homogeneous surface layer
+ tubules mostly obstructed, no homogeneous layer
~ tubules partly obstructed, no homogeneous layer
- open dentinal tubules
* partial melting

aminations) revealed traces of tin (Sn) in very small dentin samples (in the g range), indicating that stannous fluoride had been integrated into the dentin surface and that it had remained there for the observed duration.

X-ray photoelectron spectroscopy (XPS) revealed no shifts in phosphorus, calcium, or tin within the samples, while a marked difference in the bonding energy of fluorine was observed. These findings indicate a change in the bonding characteristics of fluorine. Bonding of fluorine to dentin at the outermost surface (approximately 1 nm) of the tooth is obviously improved when CO_2 laser treatment is combined with the application of SnF_2 gel. Therefore, a chemical bond between the stannous fluoride gel and the dental cervical surface can be assumed.

Electron probe microanalysis (EPMA) and X-ray energy dispersed images of the cross sections of lased teeth showed a 2- to 3-µm thick layer deficient in calcium and phosphorus and enriched with tin. The results of XPS and EPMA indicate that there are physical and chemical bonds between stannous fluoride gel and the treated dental cervical surface following irradiation with the CO_2 laser.

The amazingly swift improvement of the clinical situation as well as the encouraging long-term effect and the high rate of acceptance achievable by the combined treatment scheme emphasize the usefulness of stannous fluoride gel and the CO_2 laser in the field of hypersensitive dental necks. Due to the previously mentioned physical properties and the specific wavelength of the CO_2 laser, a highly resistant protective layer on sensitized dentin can be generated. This layer induced by physical and chemical bonding mechanisms provides a superior defense against external stimuli.

CONCLUSION

Until now, no other treatment combination has resulted in a comparably sealed and homogenous dentin surface. The presented ESEM investigations did not reveal complete closure of the tubules with either the KTP or the diode laser. Moreover, CO_2 laser alone or the combination with aminofluoride fluid or sodium fluoride presented no satisfactory results.

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