Current Research and Future Dreams: The Second Generation of Hard Tissue Lasers

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Core Message

While all commercially available dental lasers can be clinically used for soft tissue procedures, the quest continues for the ideal instrument to safely and efficiently interact with dental hard tissue. This chapter will describe several years of research that has resulted in the second generation of hard tissue lasers. The interaction of carbon dioxide laser photonic energy with tooth enamel and dentin has been studied for several decades, with first publications in the 1960s. Carbon dioxide lasers at 9.3 µm and 9.6 µm wavelength show the highest absorption of all dental lasers in dental hard tissues. Laboratory studies have shown that these short-pulsed CO_2 lasers can efficiently be used to render enamel caries resistant by transforming the originally carbonated apatite into the much acid less soluble hydroxyapatite. Adding fluoride after the laser treatment additionally reduces the acid solubility of enamel and creates the desired least acid-soluble fluorapatite. Irradiation with the 9.3 µm laser wavelength can reduce mineral loss by 55% over untreated enamel. Safety studies have shown that without harmful effects to the pulpal tissue these lasers can efficiently and safely be used on vital teeth. The first in vivo clinical study engaging an orthodontic bracket model showed over 4 weeks a 46% and over 12 weeks a 87% reduction in mineral loss. The first in vivo occlusal caries prevention by pulsed CO_2 laser and additional fluoride varnish application demonstrated that a microsecond-pulsed 9.6 µm CO_2 laser with additional fluoride varnish applications significantly inhibited the formation of carious lesions in fissures of molars in vivo in comparison to a non-irradiated control tooth in the same arch over a 1-year observation interval. Using ICDAS and the SOPROLIFE daylight and fluorescence assessment tools proved the reduction in caries. Moreover, the 9.3 and 9.6 µm CO_2 µs short-pulsed lasers are very efficient in cutting dental hard and soft tissue. Results of shear-bond strength testing with multiple bonding agents to such laser cuts are promising.

17.1 Rendering Enamel Caries Resistant: Laboratory Work

During the creation of tooth mineral, a pure hydroxyapatite [$Ca_{10}(PO_4)_6(OH)_2$] is actually not formed. In fact, the mineral portion of the enamel and dentin is best called a highly substituted carbonated apatite [1]. The mineral is closely related to hydroxyapatite, but in acid it is much more soluble. Carbonated apatite is deficient in calcium (sodium, magnesium, zinc, etc. replaces the calcium) and contains between 3% and 6% carbonate by weight. In the crystal lattice, carbonate mostly replaces phosphate ions [2–4]. The mineral of enamel and dentin can be described by the formula of carbonated hydroxyapatite [$Ca_{10-x}(Na)_x(PO_4)_{6-y}(CO_3)_z(OH)_{2-u}(F)_u$].

Numerous laboratory studies in the past have shown that increasing resistance to enamel demineralization may be achieved by microsecond-pulsed CO_2 laser irradiation [5, 6]. The most strongly absorbed wavelengths in dental enamel are the 9.3 and 9.6 µm CO_2 laser wavelengths [7, 8] (Fig. 17.1).

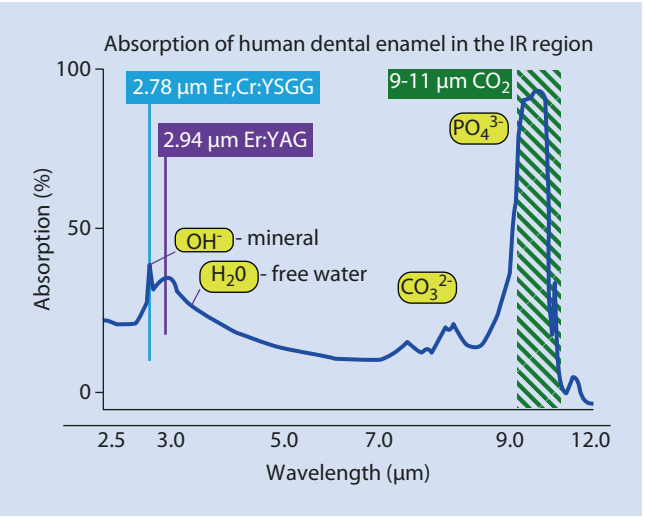

Fig. 17.1 Absorption of human dental enamel in the infrared (IR) spectral region showing the position of the primary absorbers, namely, phosphate (PO_4^{3-}), carbonate (CO_3^{2-}), hydroxyl (OH–), and water (H_2O), overlapped by the positions of the Er, Cr:YSGG, Er:YAG, and carbon dioxide (9.3, 9.6, 10.3, and 10.6 µm) emission wavelengths (the curve is a simplified transformation from an infrared transmission spectrum of dental enamel; significantly modified from Refs. [5, 8, 9])

Table 17.1 Selected optical properties of enamel and dentin, tabulated from references mentioned as sources, from Ref. [9]

Absorption coefficients (μ_a) of light in dental enamel			
Wavelength		**μ_a cm^{-1}**	**Source**
Visible			
450–700 nm		3–4	Fried et al./Ten Bosch
Near IR			
Nd:YAG	1.06 µm	<1	Fried et al.
Mid IR			
Ho:YAG	2.10 µm	<20	estimate
Er:YSGG	2.79 µm	450	Zuerlein et al.
Er:YAG	2.94 µm	770	Zuerlein et al.
CO_2	9.3 µm	5500	Zuerlein et al.
	9.6 µm	8000	
	10.3 µm	1125	
	10.6 µm	825	

At 9.3 and 9.6 µm wavelengths, the enamel absorption coefficient is ten times higher compared to the 10.6 µm CO_2 laser wavelength [7, 8]. This is demonstrated in Table 17.1.

Due to the irradiation heat, the carbonate phase loss from the enamel crystals is responsible for the reduced dissolution of enamel in acid [10] due to transforming the carbonated

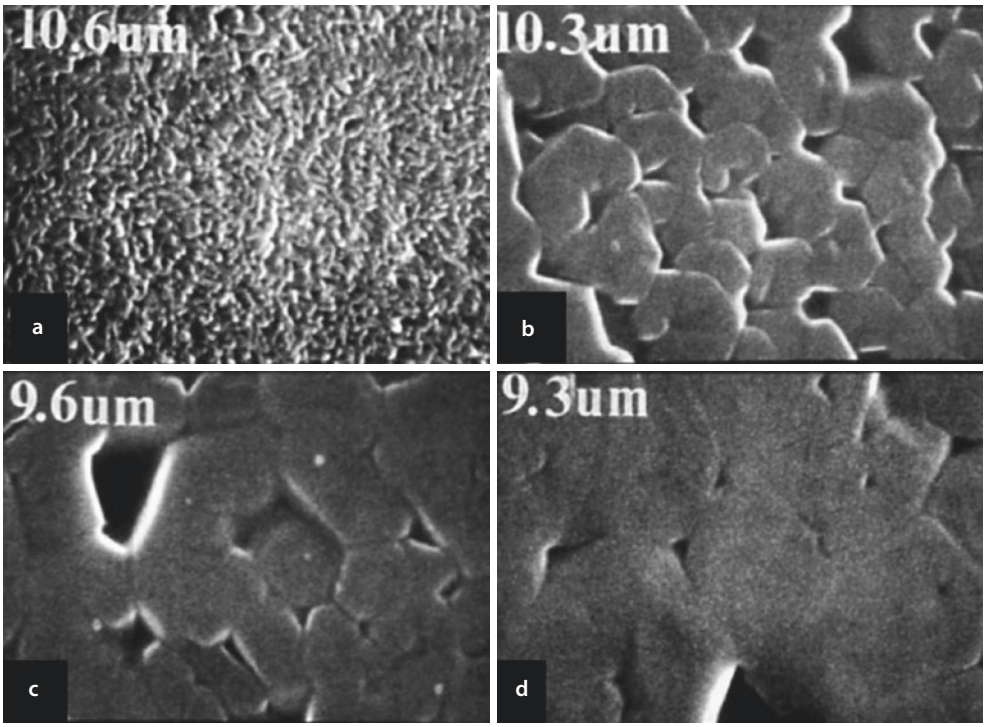

Fig. 17.2 SEM pictures of unpolished enamel after irradiation with CO_2 laser wavelength **a** 10.6 µm, **b** 10.3 µm, **c** 9.6 µm, and **d** 9.3 µm, all 50 µs pulse duration; melting of the enamel prisms occurs at 10.3, 9.6, and 9.3 µm but not at 10.6 µm (Photos from Ref. [16]. Used with permission from the Journal of Dental Research)

hydroxyapatite into the more acid-resistant hydroxyapatite. If at this point in time fluoride is added, fluorapatite is created, which is even less acid soluble than hydroxyapatite [11].

Fluoride works predominantly via topical mechanisms, including [1] demineralization inhibition at the crystal surfaces inside the enamel, [2] remineralization enhancement at the surfaces of the crystal (the newly created remineralized layer is very resistant to acid attack), and [3] finally inhibition of bacterial enzymes [12].

Topical fluoride in solution in the oral cavity boosts remineralization by accelerating the growth of a new surface on top of the partially demineralized subsurface crystals in the carious tooth structure. The newly formed veneer-like surface layer on top of the crystal is like fluorapatite, exhibiting much lower acid solubility than the original carbonated apatite mineral [13, 14].

Initial investigations postulated that melting of enamel was required to accomplish caries resistance. Surface enamel melts at about 1100 °C and fuses at or above the hydroxyapatite melting point of about 1280 °C [15]. The goal in basic research was to determine parameters that will selectively melt and/or chemically alter crystals near the surface to a depth that will provide the greatest efficacy for caries prevention [16]. Consequently, McCormack et al. in 1995 irradiated bovine and human enamel by a tunable, pulsed CO_2 laser (Fig. 17.2) [16]. This specific laser prototype could be tuned to the wavelengths 9.3, 9.6, 10.3, and 10.6 µm. To irradiate the samples, 5, 25, or 100 pulses were used, at fluences of 2, 5, 10, or 20 J/cm^2 with pulse widths of 50, 100, 200, and 500 µs, respectively. The authors observed crystal fusion at fluences of 5 J/cm^2 with the 9.3, 9.6, and 10.3 µm wavelength but never with the 10.6 µm wavelength [16].

Fried et al. in 1996 showed when using a 9.6 µm CO_2 laser at 100 µs pulsed duration and a fluence of 4 J/cm^2 that they achieved a 800 °C enamel surface temperature, which was just not melting enamel. Surface temperatures of 800 °C and up to 1200 °C, achieved at 6 and 8 J/cm^2, respectively, caused the mineral to melt [17]. Applying 10 J/cm^2 resulted in vaporization of the enamel (Fig. 17.3).

As consequence of irradiation with a 9.6 µm carbon dioxide laser with 100 µs pulse duration, Featherstone et al. in 1997 showed using fluences of 0–3 J/cm^2 did not or only slightly reduced the carbonate content of enamel. In contrast, irradiation with fluences of 4, 5, or 6 J/cm^2 eliminated the carbonate from enamel surfaces. Measurements were done by Fourier Transform Infrared Reflectance (FTIR) spectroscopy (Fig. 17.4) [9, 18].

Lately a short-pulsed carbon dioxide laser emitting at a wavelength of 9.3 µm became available on the US market for use in dental offices (Solea, Convergent Dental, Inc., Natick, MA). The CO_2 gas of the laser medium is «radio-frequency excited,» and thus the direct-pulsed laser can emit extremely short laser pulse durations as short as a 3 µs minimum pulse duration.

In order to test the caries preventive potential of the 9.3 µm short-pulsed CO_2 laser, five different pulse durations between 3 and 7 µs were used irradiating enamel samples in a laboratory study. The consequently delivered pulse energies ranged from 1.49 mJ/pulse up to 2.9 mJ/pulse, resulting in fluences between 3.0 and 5.9 J/cm^2. Non-irradiated samples served as control in this study. In addition a series of samples received additional fluoride treatment. After a 9-day pH cycling period, when using cross-sectional microhardness testing, this study showed by laser treatment without

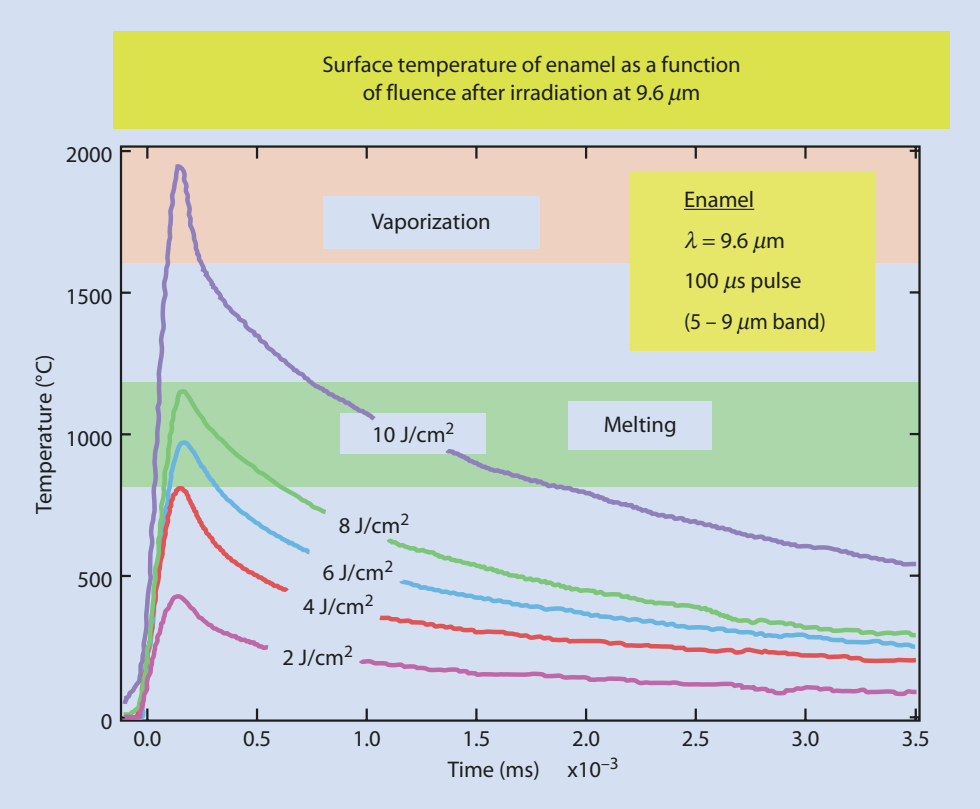

Fig. 17.3 Plot of temperature at the surface of dental enamel versus time following irradiation by a carbon dioxide laser at 9.6 μm, over a range of fluences, and with a pulse duration of 100 μs (Adapted from Ref. [17])

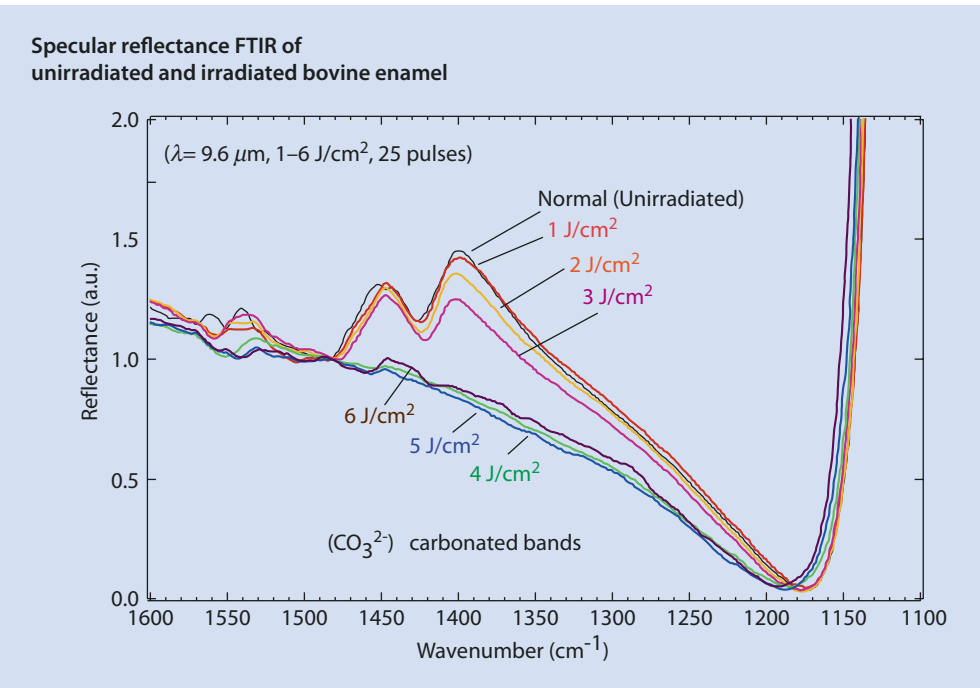

Fig. 17.4 Fourier Transform Infrared Reflectance (FTIR) spectrum showing the carbonate bands following surface treatment of dental enamel after applying a range of fluences with a 9.6 μm CO_2 laser (Adapted from Ref. [18]. Used with permission: SPIE, Bellingham, WA)

additional fluoride the average mineral loss of the test samples was already significantly reduced by 53 % ± 11% (Fig. 17.5a). When additional fluoride applications were used without any laser treatment, the mineral loss of these controls was already reduced in average by more than 50 %. Adding laser irradiation, the average mineral loss was significantly reduced by 55 % ± 9% (Fig. 17.5b) [19] on top of the already gained resistance when using fluoride alone.

As mentioned above, it had been reported that enamel surface temperatures of 800 °C and above caused the mineral

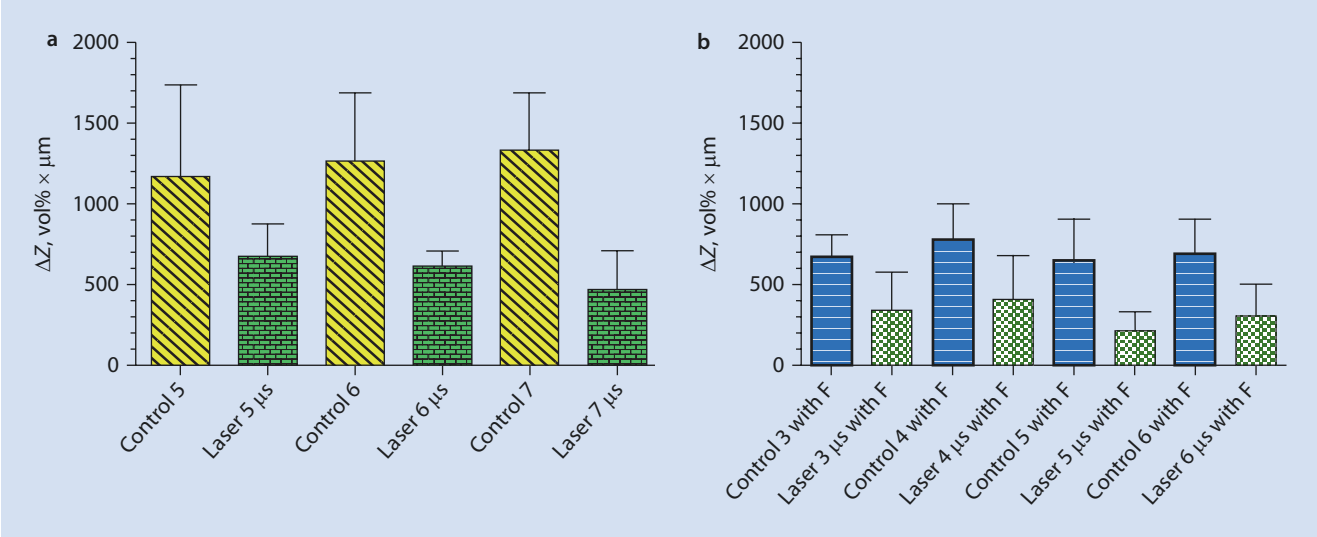

Fig. 17.5 **a** Mean relative mineral loss DZ for the laser-treated enamel and for the control groups, three different laser energies, after 9 days of pH cycling with no additional fluoride use resulting on average in 53% reduction in mineral loss for the laser-treated teeth (statistically significant reduced mineral loss with $P < 0.0001$; error bars represent standard deviations. **b** Mean relative mineral loss DZ for the laser-treated and for the control groups, four different laser energies, after 9 days of pH cycling with additional fluoride, showing on average 55% reduced mineral loss for the laser-treated teeth (statistically significant reduced mineral loss with $P < 0.0001$; error bars represent standard deviations) (Adapted from Ref. [19])

melting and the mineral transformation into less acid-soluble mineral after cooling [15, 20, 21]. Other work has established that temperatures of 400 °C and above are necessary to decompose the carbonate inclusions in the enamel and transform the carbonated hydroxyapatite to the much less acid-soluble hydroxyapatite [21, 22]. As seen below, the scanning electron microscope of the Rechmann et al. study in 2016 revealed that the lowest applied energies (pulse durations) did not produce very noticeable surface modifications, besides some minor areas with insignificant melting (Fig. 17.6). Nevertheless, the cross-sectional microhardness testing after simulating caries demineralization and

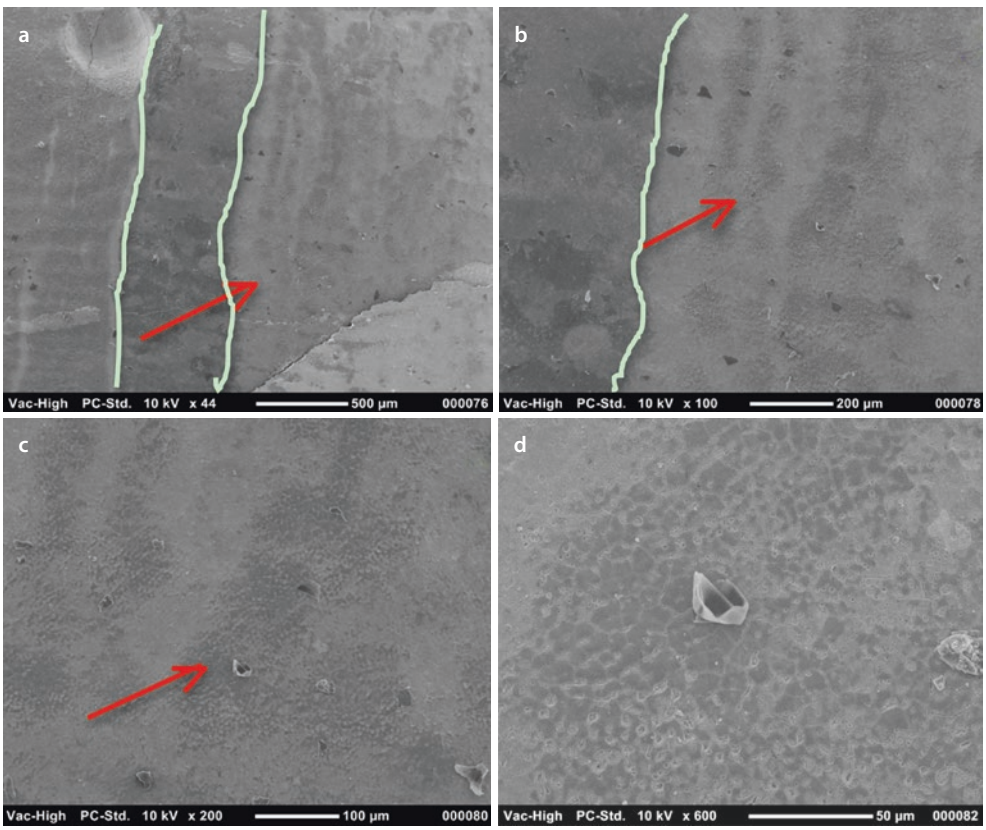

Fig. 17.6 SEM pictures of enamel after 3 µs pulse duration irradiation; only minor or no changes are visible with a few molten areas at the highest magnification (*lines* are drawn between irradiated and non-irradiated surfaces; *arrows* indicate area showed at the next higher magnification) (Adapted from Ref. [19])

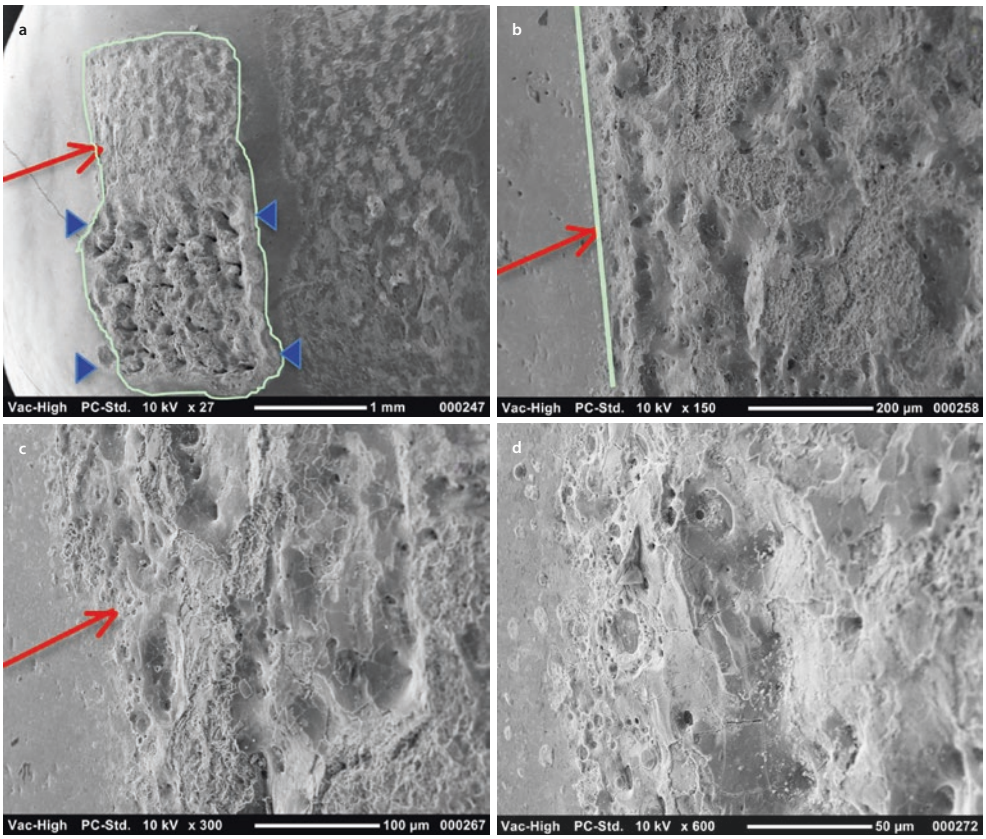

Fig. 17.7 SEM pictures of enamel after 6 and 7 µs pulse duration irradiation; rough surface morphology with slight ablation of the enamel occurs at 6 µs pulses; 7 µs pulses (in *1*, between *triangles*) result in noticeable ablation of the enamel (*lines* are drawn between irradiated and non-irradiated surfaces; *arrows* indicate area showed at the next higher magnification) (Adapted from Refs. [19, 22])

remineralization in the pH cycling model revealed significantly reduced mineral loss in the laser treatment group; thus, one could conclude that enamel melting is not necessary in driving out carbonate as reported above and achievement of enhanced caries resistance [19].

It had been established that ablation of enamel occurs at temperatures above 1200 °C [17]. In the laboratory study presented here [19], when 9.3 µm CO_2 laser short-pulsed laser energies were applied, causing ablation of enamel for cutting teeth (Fig. 17.7), caries resistance of the remaining enamel was also enhanced. A 65% reduction in mineral loss in comparison to the non-irradiated surfaces was shown (Fig. 17.5a, b). This effect is an advantage when cavities are drilled with a 9.3 µm CO_2 short-pulsed laser, and a restoration then is placed. The restoration margins will be better protected against recurrent caries. A failure of the restoration will be more unlikely.

17.2 Pulpal Safety Study

Before proving that both short-pulsed CO_2 9.6 and 9.3 µm laser irradiation clinically render enamel more caries resistant, a pulpal safety study had to be performed. The intention of such a study was to show that the laser treatment would not harm the dental pulp. A clinical study using third molars was completed performing pulp histology after laser treatment and sham dental procedures, respectively. The conclusion was that the 9.6 µm CO_2 laser, pulsed with 5–8 µs pulse width, can be safely used to alter enamel surfaces to render them more resistant to caries without permanently damaging the dental pulp. Histological examination of all teeth disclosed no indication of an inflammatory response in the pulp tissue at any time point. All histological sections appeared normal with no changes seen in the normal pulpal morphology. Permanently or serious damaging of the dental pulp was not observed [23]. Lately a second pulpal safety study performed with a 9.3 µm, 15 µs pulsed CO_2 laser confirmed that this laser can ablate enamel safely without harming the pulp [24].

17.3 Inhibition of Caries in Vital Teeth by CO_2 Laser Treatment: First In Vivo Study Using the Orthodontic Bracket Model

To test the caries preventive, specific CO_2 laser irradiation for the first time in vivo, an orthodontic model was used [25]. Typically, orthodontic treatment is associated with rapidly enhanced demineralization of the enamel due to the increased plaque accumulation around the brackets [26]. This also includes a change to a more cariogenic bacterial milieu [27, 28]. After bracket bonding in orthodontic patients, demineralization takes place at the gingival and middle thirds of the facial surfaces [29]. Demineralization switches from typically the interproximal areas to the facial as well as from posterior to anterior areas of the mouth [30, 31]. This well-established caries pattern was used as a model system [25, 32] to determine whether the laser treatment prevents demineralization

and/or even enhances remineralization in vital human teeth at those more caries prone regions.

Twenty-four orthodontic patients with need for extraction of bicuspids within the planed orthodontic treatment gave their consent and were enrolled into the study. Brackets were bonded with a conventional light-cured composite resin (Transbond XT, 3M ESPE, St. Paul, MN) [25] onto the buccal surface of the bicuspid scheduled for extraction. An enamel area directly next to the bracket at the cervical area of the tooth (Fig. 17.8) was treated according to the laser treatment protocol. The two pictures in Fig. 17.9 demonstrate the clinical aspect of the laser-treated surfaces; due to the biofilm removal, the irradiated enamel is very shiny. Nevertheless, after 1 week of homecare typically the shiny aspect faded off.

The laser employed was a CO_2 laser, Pulse Systems, Inc. (PSI) (Model #LPS-500, Los Alamos, NM), which had been designed for dermatology indications (9.6 μm wavelength, 20 μs pulse duration, 20 Hz pulse repetition rate, 1100 μm beam diameter). To achieve caries preventive changes, each irradiated spot had to receive 20 laser pulses; the laser fluence per pulse averaged is 4.1 ± 0.3 J/cm^2 (for more details, see [33]). An area cervical to the bracket (Fig. 17.8) on one side of the bracket was irradiated, while the opposite site on the same tooth served as the control side later on.

The patients were instructed to brush for 1 timed minute twice daily with a provided over-the-counter (OTC) toothpaste (1100 ppm fluoride as NaF).

The study teeth were extracted 4 or 12 weeks after irradiation, respectively. In the laboratory they were cut into halves through the bracket so that the laser-treated area was on one half and the non-laser control area on the other half. Afterward they were embedded in acrylic and prepared for the cross-sectional microhardness testing. Figure 17.10 shows a microscopic picture of a typical cross section with enamel, dentin, and the orthodontic bracket with the composite and the micro-indents. During the cross-sectional microhardness testing procedure, a pyramid-shaped diamond tip is pushed into the enamel with a defined weight. The softer the enamel (due to the occurred demineralization), the wider the indent will be. The width of the indent is measured and the actual mineral loss can be calculated.

In Fig. 17.11 the mineral loss of enamel (volume percentage) is plotted versus the depth into the tooth presenting a mineral loss profile for the 4-week study arm teeth. The green line with the triangle symbols represents the average mean volume % mineral at each depth for the laser-treated teeth and

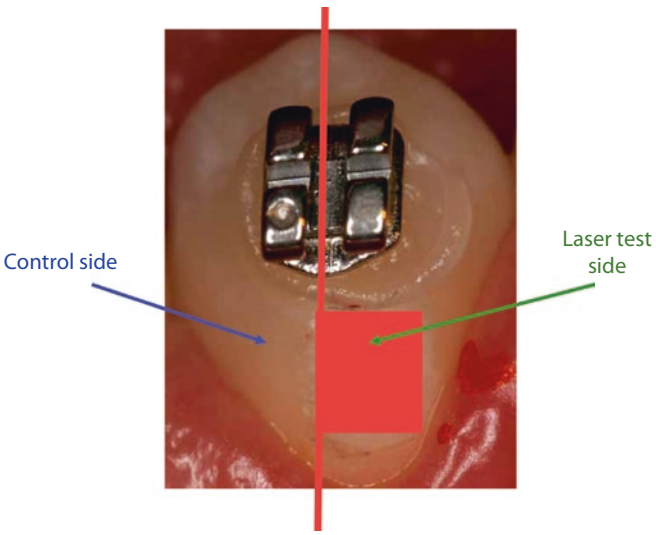

Fig. 17.8 Orthodontic bracket placed on a study bicuspid using an abundant amount of composite to create a microbial plaque trap; the irradiated area cervical to the bracket is marked *red* in this picture; the other side served as control

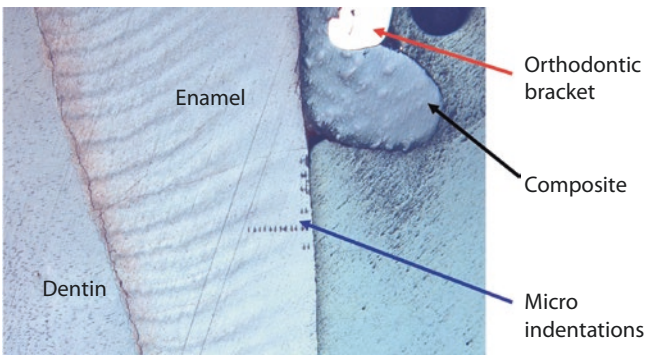

Fig. 17.10 Microscopical picture demonstrating the cross-sectional microhardness testing; the bicuspid histological cross section shows the dentin, enamel, and the composite, used to bond the orthodontic bracket onto the enamel; the micro-indentations were placed right below the enamel surface following an elaborated distribution pattern; the indents are located right below the area where the orthodontic bracket was bonded to the enamel; this area represents the microbial plaque challenge and consequently demineralization occurs here (Adapted from Ref. [33])

Fig. 17.9 Orthodontic bracket placed on the lower right (*left picture*) and on the upper left (*right picture*) study bicuspid showing an irradiated area beyond the bracket. Obviously the biofilm is removed by the laser irradiation, and the area appears white and shiny, but after a period of a week of homecare, the colors matched again

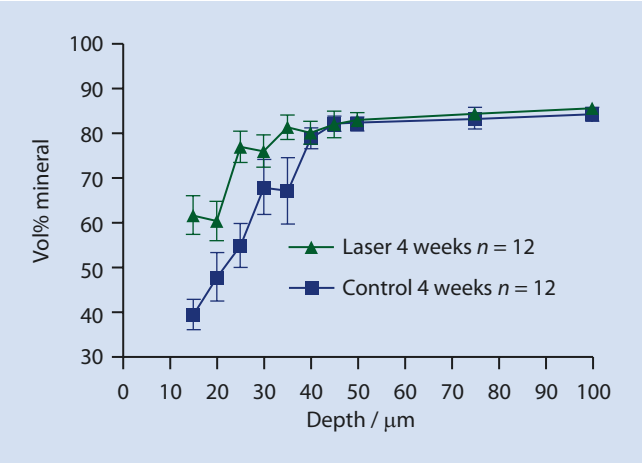

Fig. 17.11 A depth profile of volume % mineral loss for the laser-irradiated areas (*green line* with *triangle* symbols) and for the controls (*blue line* with *square* symbols) for the bicuspids at 4 weeks' time point (mean ± standard error) (Adapted from Ref. [33])

the blue line with the square symbols for the non-laser-treated controls. At 15 µm depth the control teeth (square symbols) show a very high loss of mineral with a remaining only 40% average volume % mineral, increasing to an average of 82% at a depth of 45 µm. In contrast, the CO_2 9.6 µm laser-irradiated enamel (triangle symbols) shows 62% volume % mineral at the 15 µm depth. Further into the tooth, the volume % mineral increases at 45 µm depth to 85%, which represents the typical volume % mineral content of sound enamel.

To compare the «mineral loss» between groups, the mean relative mineral loss, ΔZ (vol% × µm) can be calculated. After the 4-week observation, the mean relative mineral loss ΔZ for the laser-treated enamel was 402 ± 85 (mean ± standard error [SE]), while the controls showed an almost doubled mineral loss ΔZ of 737 ± 131. The laser treatment resulted in a significant 46% reduction of demineralization around the orthodontic brackets in comparison to the controls (Fig. 17.12) (significance level $P = 0.04$).

The reduction in mineral loss was even more impressive after 12 weeks (Fig. 17.12). The control areas showed a fairly high relative mineral loss ΔZ of 1067 ± 254. In contrast, for the CO_2 laser-treated area, the mean relative mineral loss was much lower (ΔZ 135 ± 98) and even lower than for the 4-week mineral loss. The difference in mineral loss between laser and control for the 12-week observation was significant (significance level $P = 0.002$). For the 12-week group, the laser treatment produced a noticeable 87% inhibition of enamel demineralization.

Previous studies had demonstrated that the orthodontic bracket model employed in this study here presents a high caries demineralization challenge to the enamel. In addition, this demineralization challenge cannot simply be overcome by using an OTC 1100 ppm fluoride toothpaste [25]. Gorton et al. in 2003 reported using the orthodontic bracket model that the mean mineral loss value ΔZ in their control group was 805 ± 78 (Mean ± SE) vol% × µm. This establishes a considerable, measurable, demineralization in only 1 month even when fluoride toothpaste is used. In this CO_2 laser orthodontic bracket model study, the participants in the control regions around the brackets had a high mineral loss of ΔZ of 737 ± 131 vol% × µm at the 4-week and even higher one of 1067 ± 254 vol% × µm at the 12-week arm, similar to the mineral loss Gorton et al. stated [25].

However, the application of the CO_2 9.6 µm short-pulsed laser irradiation significantly reduced the mineral loss in the 4-week and in the 12-week arm of the study by 46% and 87%, respectively. While the mineral loss for the 12-week group controls was higher than for the 4-week group controls, the additional reduced mineral loss for the treatment group after 12 weeks in comparison to the 4 weeks might be explained by enhanced remineralization over a this longer observation time period.

This study showed that caries inhibition demonstrated in numerous models and experiments in the laboratory [34–37]

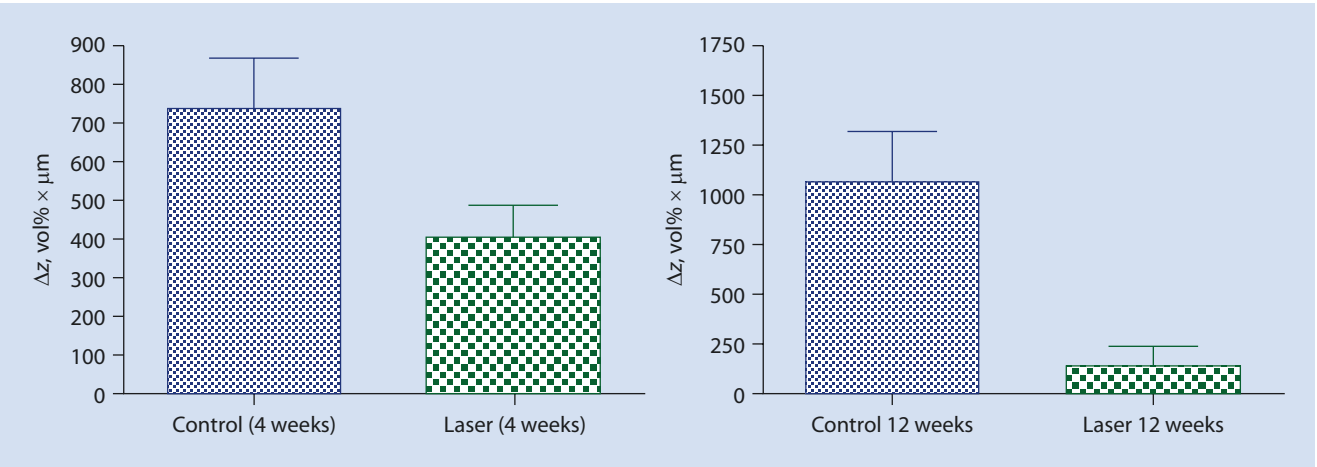

Fig. 17.12 Relative mineral loss ΔZ (vol% × µm) for the laser-treated enamel and for the non-laser-treated controls ($n = 12$ for both groups, mean ± SE) 4 weeks (*left* and 12 weeks *right* graphs) after treatment. The differences in relative mineral loss between laser-treated and control groups are statistically significant with a significance level of $P = 0.04$ for the 4-week and $P = 0.002$ for the 12-week observations (Adapted from Ref. [33])

can also be achieved in humans in vital teeth using short-pulsed 9.6 μm CO_2 laser irradiation [33]. Moreover this study showed that the orthodontic bracket model could be considered to explore any caries inhibition agents or tools in living teeth in humans.

17.4 In Vivo Occlusal Caries Prevention by Pulsed CO_2 Laser and Fluoride Varnish Treatment

While in the orthodontic bracket model study, the test teeth were extracted to perform the cross-sectional microhardness testing, a subsequent, in vivo study was designed with a goal to determine the caries prevention effects of a short-pulsed CO_2 laser without the need to extract the study teeth for analytical assessments [38].

In this study for the first time, in vivo occlusal pits and fissures of second molars in the oral cavity were irradiated; and the change in demineralization and remineralization was assessed by three visual methods: (1) the International Caries Detection and Assessment System (ICDAS), (2) SOPROLIFE in daylight and in blue fluorescence mode, and (3) the DIAGNOdent fluorescence tool. The study's intention was to show caries inhibition in fissures of molars [38]. The significant challenge was to reach normal as well as deep pits and fissures with the laser irradiation. In order to facilitate adequate laser interaction with the walls of deep fissures, we designed and used a custom contra-angle laser handpiece made for this study.

Twenty subjects were recruited and consented for the study. Their age ranged between 10 and 15 years; they were at high caries risk according to CAMBRA rules [39–41] and presented at least two fully erupted second molars in the same arch (contralateral) with untreated, non-carious (non-cavitated) occlusal surfaces. One molar was randomly selected for the laser irradiation; the tooth on the opposite site in the same jaw functioned as control.

At baseline, right after laser treatment and at the 6- and 12-month recall visit, the occlusal surfaces of the study molars were visually judged for decalcification applying the ICDAS II criteria (International Caries Detection and Assessment System) and the SOPROLIFE light-induced fluorescence evaluator system (SOPRO, ACTEON Group, La Ciotat, France).

17.4.1 Caries Assessment Methods Applied in In Vivo Occlusal Caries Prevention by Pulsed CO_2 Laser Study

The *International Caries Detection and Assessment System* provides a standardized method of lesion detection and assessment, leading to a caries diagnosis [42, 43]. ICDAS scores range from code 0 to 6, with code 0 as no mineral loss, code 1 and 2 as precavitated lesions, and code 3 and above showing the first physical enamel break down to more than 50% of the tooth surface is cavitated. ICDAS criteria are built on the visual enamel properties of translucency and microporosity. After increasing number of demineralization attacks, the microporosity of the subsurface of enamel intensifies. Consequently a change in translucency and light refraction of the enamel surface represents the first sign of a carious alteration. If the demineralization process continues, the enamel microporosity increases, which results in an even further decrease in the refractive index of enamel [44].

Ekstrand et al. [45–47] validated ICDAS by demonstrating an association between the lesions' histological depth and the severity of caries lesions (as described by ICDAS codes). Other authors confirmed this close relationship between ICDAS scoring and the histological depth of the caries lesion, especially in precavitated (ICDAS codes 1 and 2) but also in slightly cavitated stages (ICDAS code 3 and above [48, 49]).

Fluorescence is a property of specific materials to absorb energy at shorter wavelengths and emit light at longer wavelengths. Several caries detection methods use fluorescence. The *SOPROLIFE system* basically combines the benefits of a visual inspection method (high specificity) by employing a high-magnification oral camera with a laser fluorescence instrument (high reproducibility and discrimination [50]). The system uses four white LEDs in the daylight mode and four blue LEDs in the blue fluorescence mode. Bacteria and their by-products trigger the fluorescence signal and expression. The blue light transmits through healthy enamel and evokes a green fluorescence of the dentin core. The green fluorescence light coming back from the dentin core then leads to a red fluorescence from bacteria and bacterial by-products like porphyrins [50–52]. Figure 17.13 shows a graphic representation of the SOPROLIFE system, and Fig. 17.14 depicts both the visible light and SOPROLIFE images of an ICDAS code 3 occlusal lesion.

The laser was the same instrument that was used in the inhibition of caries in vital teeth by CO_2 laser treatment orthodontic bracket model study (CO_2 laser, Pulse Systems, Inc. (PSI) Los Alamos, NM), with a 9.6 μm wavelength, 20 μs

Fig. 17.13 The SOPROLIFE light-induced fluorescence evaluator system uses in the blue fluorescence mode four blue LEDs emitting light at 450 nm. The light is transmitted through the enamel and then induces *green* fluorescence from the dentin body. If *green light* on the way back hits porphyrins in caries, the emitted fluorescence is *red*

Fig. 17.14 The left picture of the occlusal surface of a molar is taken with SOPROLIFE in daylight mode (white LEDs illumination) and the right taken in blue fluorescence mode (blue LEDs illumination). The tooth shows an ICDAS 3 code, already exhibiting physical enamel breakdown in the central grove. The enamel breakdown became clearly visible

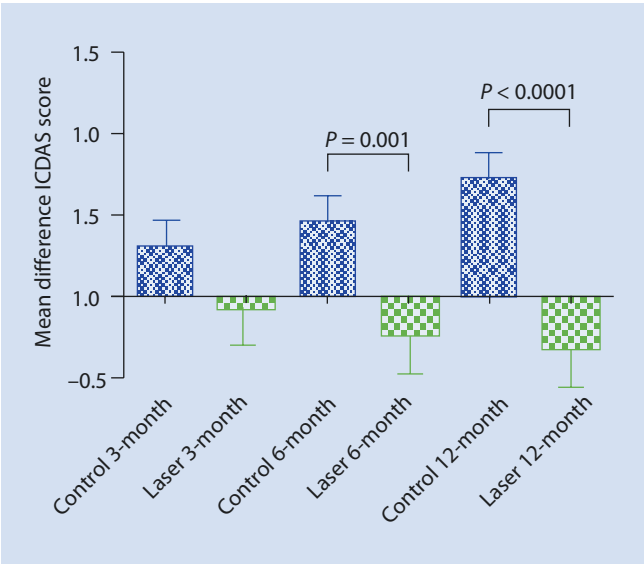

Fig. 17.15 Average change of ICDAS scores between baseline and 3-month, baseline and 6-month, and baseline and 12-month recall (mean ± SE) for laser-treated and control teeth; statistically significant differences between laser and control at 6- and 12-month recalls ($P = 0.001$ and $P < 0.0001$, respectively)

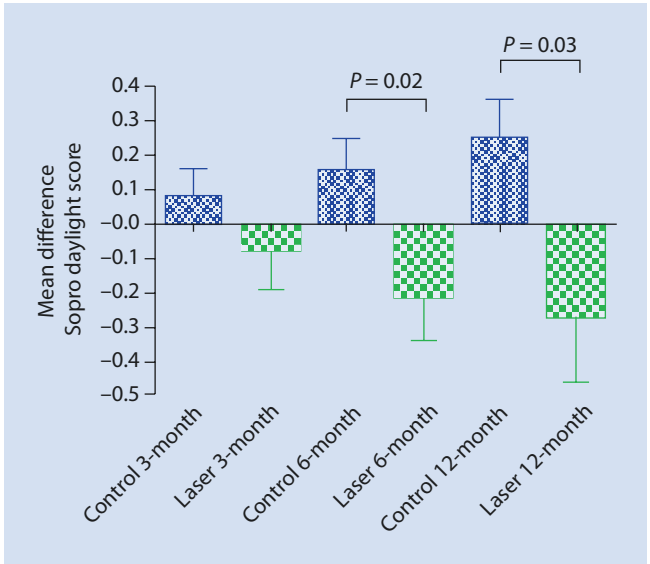

Fig. 17.16 Average changes of SOPROLIFE daylight scores for laser-treated and control teeth between baseline and the 3-, 6-, and 12-month recall (mean ± SE) showing statistically significant differences at the 6- and 12-month interval (significance level $P = 0.02$ and $P = 0.03$, respectively) (Graph adapted from Ref. [38])

17

pulse duration, 20 Hz pulse repetition rate, and 800 µm beam diameter delivered through a custom-made contra-angle handpiece. Each irradiated spot received 20 laser pulses; the laser fluence per pulse averaged 4.5 ± 0.5 J/cm^2.

All subjects received fluoride varnish applications to all teeth (Omni varnish fluoride varnish, Omni Preventive Care, West Palm Beach, FL) at baseline and 6-month recall (for more details, see [38]).

The ICDAS scores ranged at baseline from code 0 to code 2. Figure 17.15 shows the average change of ICDAS scores between baseline and 3-month, baseline and 6-month, and baseline and 12-month recall (mean ± SE) for laser-treated and control teeth. While for the controls the average ICDAS increased over time, a decrease in ICDAS could be observed for the laser-treated fissures. The differences in average change in ICDAS scores over time were statistically significant between laser and control at 6-month and again between laser and control at the 12-month recalls (significance level $P = 0.001$ and $P < 0.0001$, respectively).

In addition to the ICDAS scoring, the study teeth were also evaluated with the SOPROLIFE system. They were scored with a recently presented scoring system, which was developed for the SOPROLIFE light-induced fluorescence evaluator for daylight and for the blue fluorescence mode [51, 52]. For the control as well as the laser-treated teeth, the SOPROLIFE scores ranged between 0 and 3 at baseline with no statistically significant differences between study and control group.

Calculating the changes in SOPROLIFE daylight scores between baseline and each recall time point (Fig. 17.16) revealed an increasing daylight score for the controls and a decreasing score for the laser-treated fissures similar to the ICDAS scoring. The differences between the average changes were again like the ICDAS scores statistically significant between baseline and 6-month and baseline and 12-month recall.

Figures 17.17 and 17.18 show the occlusal surface of a control and a laser-treated tooth, respectively, with the pictures taken with the SOPROLIFE camera in daylight mode

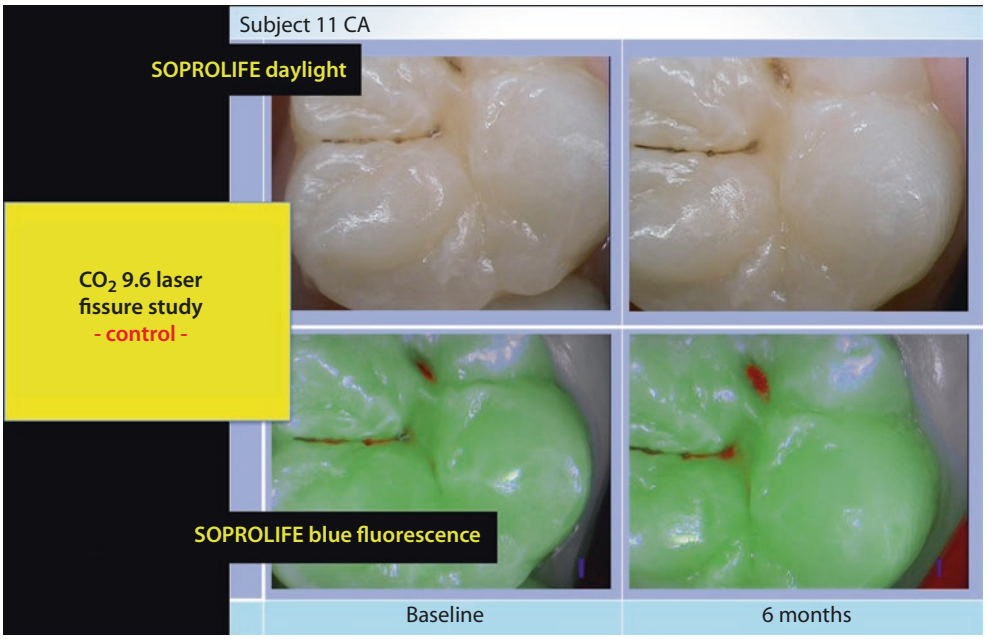

Fig. 17.17 Control tooth of a subject at baseline and at the 6-month recall, pictures taken in daylight and fluorescence mode, respectively. The area of demineralization appears wider in daylight mode and in fluorescence mode after 6 months (*upper row* daylight, *lower row* blue fluorescence mode; baseline left, 6-month recall right) (Photos from Ref. [38])

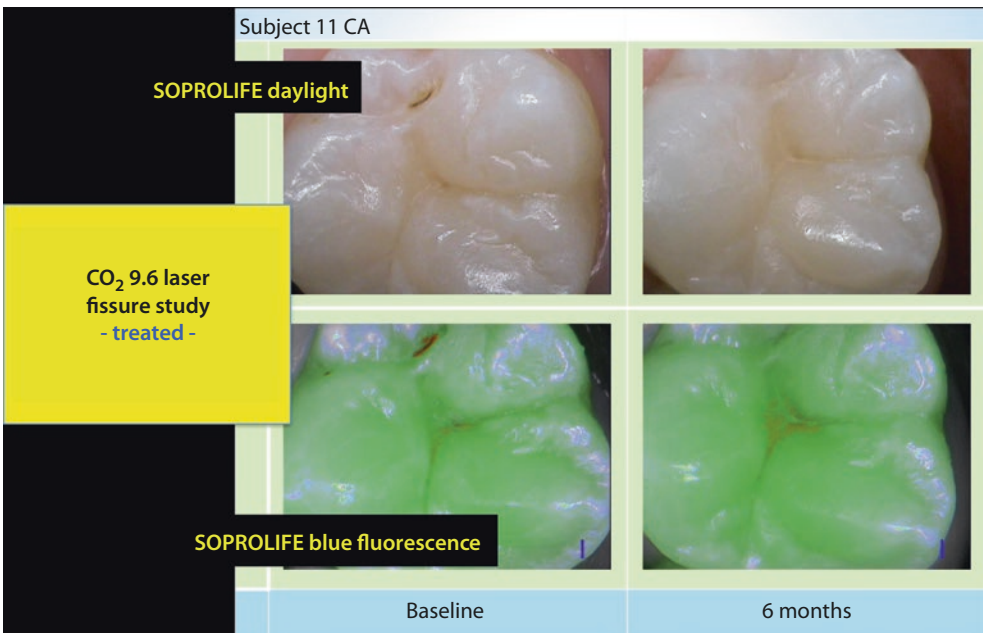

Fig. 17.18 Laser-irradiated tooth of a subject at baseline and at the 6-month recall, pictures taken in daylight and fluorescence mode, respectively. The area of demineralization in the distal fossa of the laser-treated tooth in daylight mode and fluorescence mode, respectively, is not visible anymore (*upper row* daylight, *lower row* blue fluorescence mode; baseline left, 6-month recall right) (Photos from Ref. [38])

and in the blue fluorescence mode. The pictures demonstrate the obvious differences over time between the baseline and the 6-month recall. Both molars show noticeable changes in the fissure system, being very distinct in the distal groove. While the area of demineralization in the control tooth appears to become more extended in both daylight mode and fluorescence mode, after 6 months the demineralization zone and the red fluorescence width and intensity, respectively, disappeared on the laser-irradiated tooth.

Similar to the SOPROLIFE daylight scores, the SOPROLIFE blue fluorescence average scores for baseline and the 3-, 6-, and 12-month recall demonstrated increased scores for the controls, and decreased scores for the laser-treated fissure were observed (except for the 12-month recall for the laser group, which did not change). Figure 17.19 demonstrates the average changes for SOPROLIFE blue fluorescence scores between baseline and 3-month, baseline and 6-month, and baseline and 12-month recall for the laser-treated and control teeth, respectively. The differences are statistically significant between the laser and the control group at 6- and 12-month recalls.

In summary, this single-blind, controlled, randomized clinical pilot trial showed that using a microsecond-pulsed 9.6 μm CO_2 laser with additional fluoride varnish

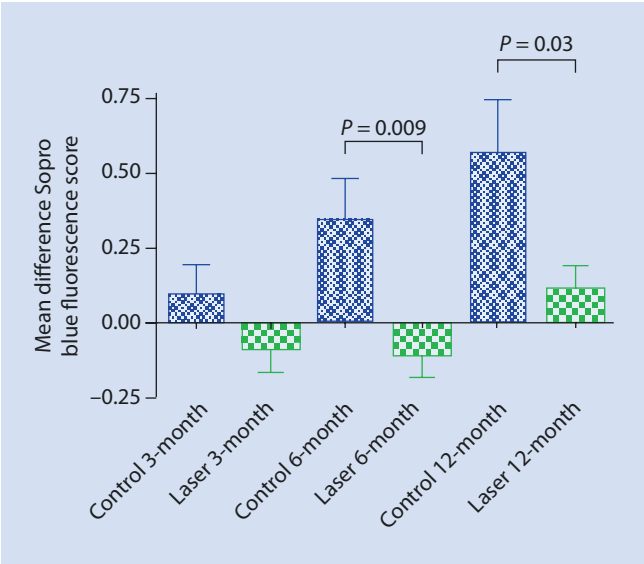

Fig. 17.19 Average change of SOPROLIFE blue fluorescence scores between baseline and 3-month, baseline and 6-month, and baseline and 12-month recall (mean ± SE) for laser-treated and control teeth; statistically significant differences between laser and control at 6- and 12-month recalls ($P = 0.009$ and $P = 0.03$, respectively) (Adapted from Ref. [38])

applications significantly inhibits the formation of carious lesions in fissures of molars in vivo in comparison to a non-irradiated control tooth in the same arch over a 1-year observation interval. With regard to the ICDAS score change over time, the control teeth scores constantly increased describing more severe mineral loss, while the control teeth showed constantly decreasing ICDAS scores, demonstrating a certain mineral gain.

From both the occlusal caries prevention study and the orthodontic bracket study, it can be reasonably concluded that the CO_2 short-pulsed laser irradiation drives out the carbonated phase from the enamel crystal and decreases the demineralization of the modified hydroxyapatite in an acid environment. Specifically when fluoride is present, the transformed hydroxyapatite appears to be prone to higher remineralization. The phenomenon of increased remineralization proven by ICDAS and SOPROLIFE daylight and fluorescence assessments was observed in this study over a period of 12 months.

17.5 Cavity Preparation and Soft Tissue Cutting with the CO_2 9.3 µm Short-Pulsed Laser

The previously mentioned 9.3 µm wavelength short-pulsed carbon dioxide laser (Solea, Convergent Dental, Inc., Natick, MA) has been available on the US market for soft and hard tissue procedures. The instrument offers a wide range of beam diameters; the basic beam diameter is actually 0.25 mm, but by using computer-controlled galvo mirrors, the beam can cover up to a diameter of 1.25 mm by using a spiral movement of the focus point. The focus distance is relatively long and is set between 10 and 19 mm. The emitted laser energy is controlled by the pulse width, varying between 10 and 130 µs, controlled by the foot pedal.

In addition to preventive procedures, this laser can perform carious lesion removal, tooth preparation, and osseous surgery. Figure 17.20 shows the first patient receiving a Solea laser cavity preparation in 2013. The green pilot laser helps in guiding the laser beam; the computerized laser control facilitates easy cavity preparation. Clinical case examples will be found in Chap. 8.

Figure 17.21 shows that, for the first time, true minimal invasive treatment becomes a reality. This fissure on a molar is prepared with a 0.25 mm laser beam. The preparation borders are completely smooth. The scanning electron microscopic picture (Fig. 17.22) shows a similar smooth cut with a diameter of roughly 0.75 mm, done by the author, in 1997 with an experimental carbon dioxide laser used to produce a carbon-13 (^{13}C) isotope. Thus 20 years ago, the search for a second-generation hard tissue cutting laser had begun.

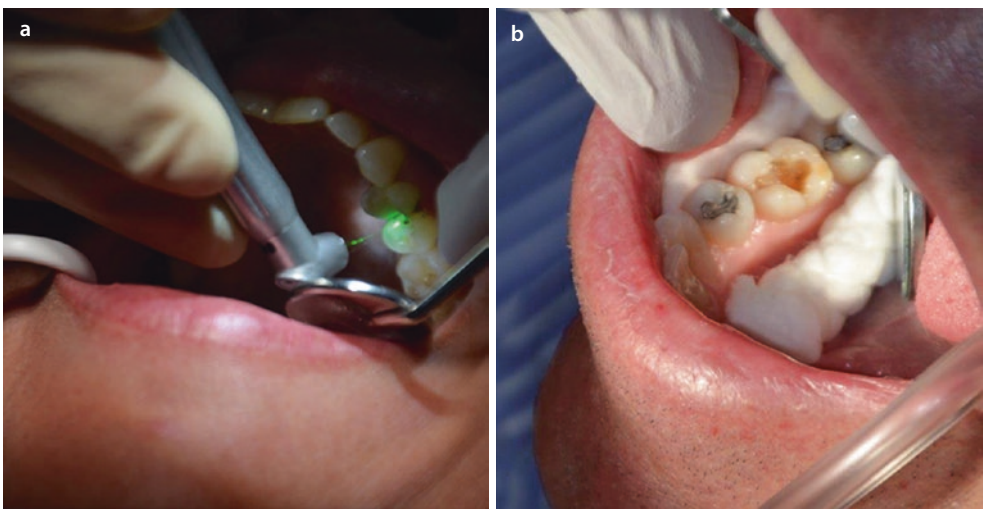

Fig. 17.20 First Solea cavity preparations: **a** upper left jaw with the *green* pilot laser visible; **b** lower right jaw after the clean laser preparation was finished (Clinical photos courtesy of Dr. Mark Mizner)

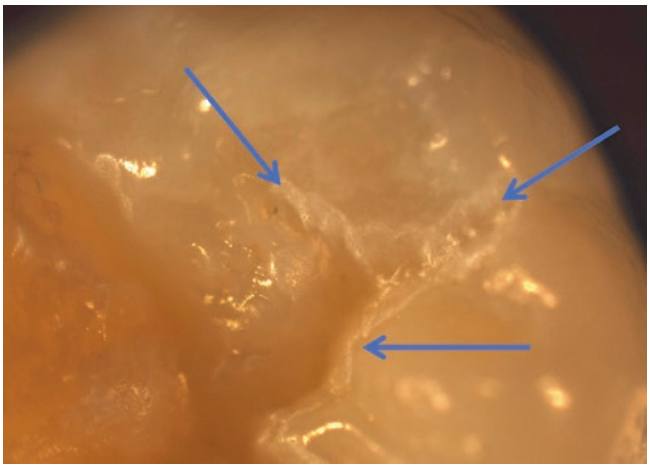

Fig. 17.21 The Solea was used for a minimal invasive preparation of an occlusal fissure. Note the very smooth preparation in enamel

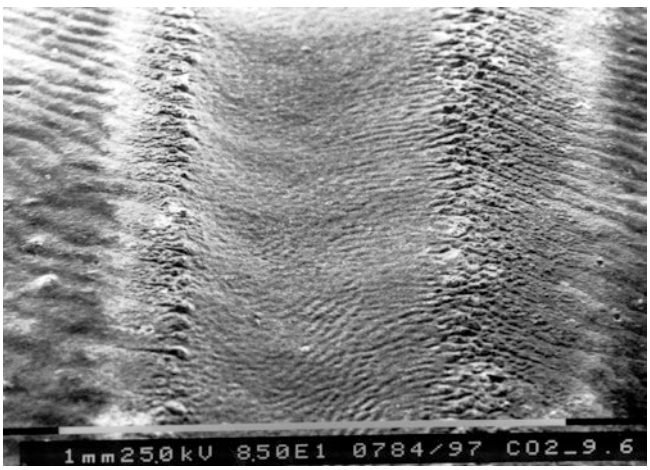

Fig. 17.22 Scanning electron microscope picture showing a similar smooth surface cut in enamel as in Fig. 17.21. Here an experimental 9.6 µm short-pulsed CO_2 laser (described above) was used

Figure 17.23 depicts a distal occlusal cavity preparation with the Solea laser. In addition, the overgrown gingival tissue was quickly removed with the laser without any bleeding (Clinical case courtesy of Dr. Joshua Weintraub).

The CO_2 9.3 µm short-pulsed laser offers when it is already too late for caries prevention a wide potential for cutting hard tissues as well as soft tissue.

17.6 Shear-Bond Strength Testing to Human Enamel

A series of bond strength testing was conducted to answer the question whether enamel, already rendered caries resistant with the CO_2 9.3 µm short-pulsed laser, would still allow sufficient bonding of composites. The additional question how bond strength to dentin might be influenced by the laser irradiation was also of interest. Consequently, enamel and dentin samples were irradiated with a wide range of laser parameters. Different total-etch and self-etch bonding systems of the fourth- and fifth-generation bonding materials were tested using Clearfil AP-X, a micro-hybrid composite (Kuraray America, New York, NY) as testing composite. The adhesive bonding strength was determined by performing a single-plane shear-bond test with the UltraTester testing device (Ultradent Products, Inc., South Jordan, UT).

Figure 17.24 shows as an example the shear-bond strength values using a self-etch bonding system Clearfil SE bond (Kuraray America, New York, NY) after the enamel was irradiated with laser parameters known to render enamel more caries resistant. Irradiation with the two different 3 µs caries preventive patterns (3 µs pulse width engaging the straight 0.25 mm beam and the 1 mm spiral patterns) resulted in higher bond strength values compared to the non-laser-treated controls. The 16.9% increase in bond strength observed for the 3 µs pulse duration with the 1 mm spiral irradiation pattern was even statistically significant (* $P \leq 0.05$).

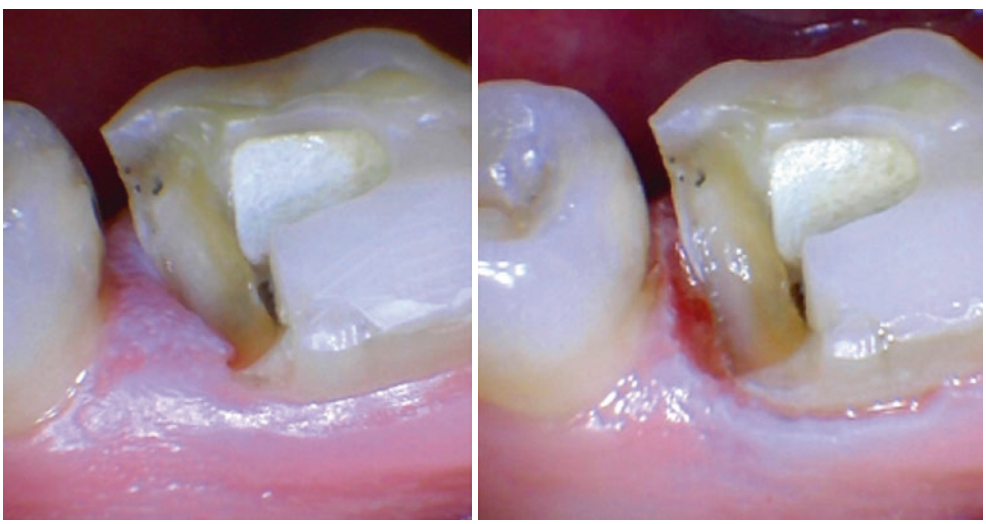

Fig. 17.23 Solea cavity preparation – *left* picture after laser preparation with overgrown gingiva; *right* picture after gingivectomy was performed without anesthesia; no bleeding occurs due to the high hemostatic effect when cutting soft tissue with a CO_2 laser (Clinical photos courtesy of Dr. Joshua Weintraub)

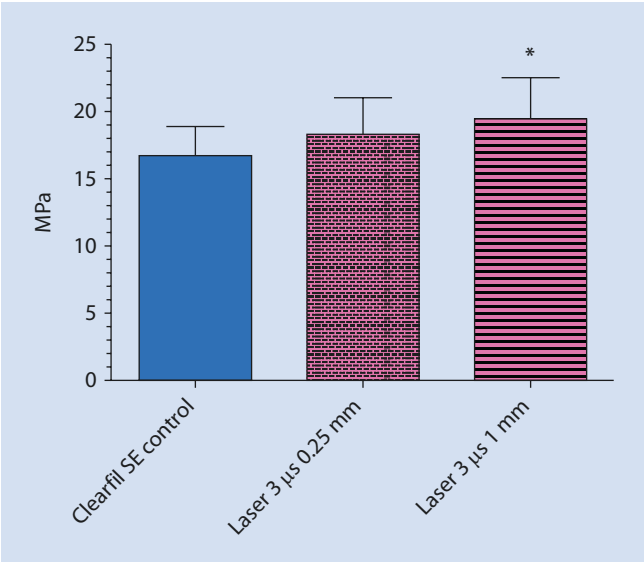

Fig. 17.24 Human enamel, uncut – Clearfil SE bond self-etch shear-bond strength values (mean ± SD), caries preventive laser irradiation increased the bond strength to enamel; the 1 mm spiral pattern showed even statistically significant differences to the control marked with *, significance level $P \leq 0.05$

The results for all laser settings tested for shear-bond strength in enamel and dentin as well as for the different self- and total-etch bonding systems were promising and will soon be reported.

17.7 Future Dreams

Research continues to both improve existing instruments and to discover new technologies, wavelengths, and clinical applications. The possibilities are certainly numerous within any area of study.

As mentioned in Chap. 3, production of very high-power densities coupled with extremely short-pulsed durations can result in photoablation, plasma-induced ablation, and molecular photodisruption. Indeed, over 30 years ago, investigators were experimenting with pulse durations of 30 picoseconds (10^{-12} s) for removing initial carious lesions in tooth enamel [53]. A recently published study described using a 400 femtosecond (10^{-15} s) pulse duration and its effects on ablation efficiency of dentin and enamel [54]. It would seem that a trend is emerging toward ultrashort-pulsed irradiance, which could provide a new horizon in plasma physics and target molecular cleavage. Chapter 7 discusses using completely opposite parameters: very low power density and very long and/or continuous pulse emissions to achieve photochemical cellular responses. All of these tissue interactions will be novel concepts compared with our current photonic energy effects on dental tissue.

The use of a laser in hard and soft tissue for diagnostics, described in Chap. 6, will certainly be enhanced by developments of scanning and reflective photonic energy. Many chapters describe current delivery systems for the laser beam, and their further refinement will allow access to the complex anatomy of dental structures. The ability to select several wavelengths from a tunable instrument could aid in the quest for having the ideal wavelength available for any procedure. As a natural result of research, well-designed clinical trials are essential. Ultimately, the goal of all the studies is to provide credible evidence for improved patient care.

Conclusion

9.3 and 9.6 µm CO_2 µs short-pulsed lasers are the most highly absorbed laser in dental hard tissues. They can safely and efficiently be used to render enamel caries resistant by transforming carbonated apatite into the much less soluble hydroxyapatite. Adding fluoride after the laser treatment additionally reduces the acid solubility of enamel and creates the desired least acid-soluble fluorapatite. These 9.3 and 9.6 µm CO_2 µs-short-pulsed lasers are very efficient in cutting dental hard and soft tissue. Results of shear-bond strength testing with multiple bonding agents to such laser cuts are promising.

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