# Lasers and the Treatment of Chronic Periodontitis

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**KEYWORDS**

• Lasers • Periodontitis • Bacteria • Probing depth
• Clinical attachment level • Inflammation

For many intraoral soft-tissue surgical procedures the laser has become a desirable and dependable alternative to traditional scalpel surgery. The dental literature contains many case reports and uncontrolled case studies that report on the use of various laser wavelengths, predominantly diode, $CO_2$, Nd:YAG, Er:YAG, and Er, Cr:YSGG, for various intraoral soft-tissue procedures, such as frenectomy, gingivectomy and gingivoplasty, de-epithelization of reflected periodontal flaps, second stage exposure of dental implants, lesion ablation, incisional and excisional biopsies, irradiation of aphthous ulcers, removal of gingival pigmentation, and soft-tissue crown lengthening.[1–12] Lasers easily ablate and reshape oral soft tissues. In addition, lasers increase hemostasis through heat-induced coagulation and occlusion of arterioles, venules, and capillaries. The resulting hemostasis allows for a clear and fully visible surgical field. Because of the intense heat, lasers also have the advantage of a bactericidal effect at the target site. A few studies have reported that laser surgery, compared with traditional scalpel surgery, is less painful, features less swelling, and heals faster with less wound contraction.[13,14] However, there are conflicting opinions on pain and speed of wound healing. Several papers comparing lasers with traditional scalpel wounding have reported either an equivalent effect or that laser surgery is accompanied by more pain and slower healing.[15–19] The issues of pain and wound

This work was not funded by any agency or commercial enterprise and none of the authors has a conflict of interest that would compromise or affect on the manuscript content.
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Dent Clin N Am 54 (2010) 35–53
doi:10.1016/j.cden.2009.08.007 **dental.theclinics.com**
0011-8532/09/$ – see front matter 

healing, and wound contraction seem dependent on the judicious choice of parameters such as power, hertz, pulse duration, and time of exposure.[13]

Given the apparent usefulness of the laser, why, after almost 2 decades, does the use of dental lasers in periodontal therapy remain controversial? Is it because lasers challenge the traditional modalities of treating periodontitis or because of a lack of hard evidence on which to make an informed decision? One may argue in favor of one or both of these reasons. It is well known that many in private practice are using various types of lasers for the treatment of periodontal disease and most have expressed satisfaction with the results of therapy. However, several recent systematic reviews of the literature have suggested there is little evidence in support of the purported benefits of lasers in the treatment of periodontal disease compared with traditional periodontal therapy.[13,14,20–22] The obvious question then becomes, is the current use of dental laser for the treatment of periodontitis based on peer-reviewed published evidence obtained under controlled conditions or word-of-mouth, unconfirmed evidence?

A letter to the editor in a recent issue of the *Journal of Dental Education*[23] asked the following question: ''Why is it that dentists are among the very few health professionals who can ignore critical evaluation of the scientific literature and treat patients with personal experience as its equal?'' The authors suggest that many dentists may be providing treatment without critically evaluating whether such treatment is consistent with the best evidence.

The authors also present several possible reasons for ignoring the best available evidence, such as expediency, difficulty finding reliable evidence-based references, easy access to questionable information, and a desire for quick profits. Other reasons may include the introduction of new products without rigorous clinical trials. Regulatory agencies such as the US Food and Drug Administration (FDA) do not necessarily require clinical research before product marketing. As an example, in the case of dental lasers, the 510K FDA premarket notification process requires only that the applicant provide evidence that its device is substantially equivalent to 1 or more similar devices currently marketed in the US marketplace. A 510K premarket notification does not imply therapeutic equivalency or superiority. Indeed, the 510K process does not even require a clinical trial.[24]

Given the current conflicting opinions, this article presents the current peer-reviewed evidence on the use of dental lasers for the treatment of chronic periodontitis.

## WAVELENGTH

Wavelength can be related to collateral tissue damage. In general, the shorter wavelength lasers (eg, 809 nm to 980 nm diodes and 1064 nm Nd:YAG) are more likely to penetrate deeper into soft tissues.[4,5] The extent of tissue penetration by shorter wavelengths is related to their affinity for pigmented tissues and a low absorption coefficient in water. The potential for undesired tissue penetration can be controlled with proper selection of parameters, such as power level, pulse repetition rate, pulse width, and energy density. In contrast, the longer wavelengths (2940 nm Er:YAG, 2780 nm Er, Cr:YSGG, and 10,600 nm $CO_2$) show comparatively more shallow tissue penetration because of their high absorption coefficients in water.[4,5] To avoid unintended consequences, the least amount of power required to achieve the desired clinical result should be chosen. Hard tissues and periosteum subjacent to thin oral mucosa (thin biotype), the gingival margin, or gingiva overlying prominent roots, are particularly vulnerable to thermal insult (**Fig. 1**A–D). The potential for damage to dental hard

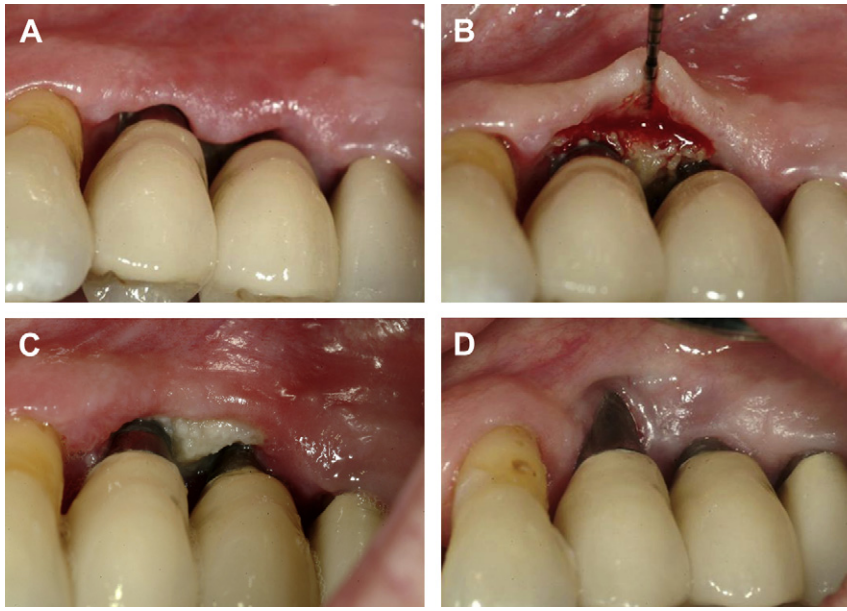


**Fig. 1.** Interproximal soft-tissue cratering with underlying bone necrosis around 2 dental implants following laser treatment of periimplantitis using inappropriate energy density and duration of exposure. Photos in sequence: (*A*, *B*) 1 month post laser treatment; (*C*) sequestration of necrotic interproximal and facial bone at 2 months post treatment; and (*D*) healing at 3 months post treatment. (Patient referred to and photos taken by Keigm Crook, DDS, Albuquerque, NM.)

tissues results from uncontrolled penetration during soft-tissue procedures that, in turn, results from improper choice of parameters while using a short wavelength laser or the conductive heat effects arising from superheating of char produced by longer wavelengths.[5] Moreover, only the erbium family of lasers have a specific indication for use on dental hard tissue, and other wavelengths should be avoided. The clinician is well advised to assess tissue thickness before initiating any laser procedure involving the gingival sulcus or the periodontal pocket area.

## LASERS AND PERIODONTAL THERAPY

As a basic premise, it should be understood that the gold standard for successful treatment of chronic periodontitis is gain in clinical attachment level.[13,25] However, other clinical goals are often considered as creditable end-points and should be considered, such as maintenance of esthetics, complete debridement of root surface accretions, regeneration of bone, periodontal ligament and cementum, and patient preference.

Basic knowledge and understanding of the pathogenesis of plaque-induced periodontal disease continues to evolve.[26–29] The current model for plaque-induced periodontal diseases includes the initial microbial challenge, a subsequent host inflammatory response, and various risk factors that contribute to host susceptibility and progression of the disease.[26–29] Considering the microbial component, it seems logical that laser irradiation with its bactericidal effect would have significant potential as an alternative or adjunct to traditional nonsurgical therapy (ie, scaling and root

planing). All dental lasers have a thermal effect. In general, most nonsporulating bacteria, including periodontopathic anaerobes, are readily deactivated at temperatures of 50°C.[30] Coagulation of the inflamed soft-tissue wall of a periodontal pocket and hemostasis are both achieved at a temperature of 60°C.[31]

Over the last decade, various dental laser wavelengths have been used by clinicians in the treatment of periodontitis, most commonly the diode lasers (809–980 nm), Nd:YAG (1064 nm), Er:YAG and Er,Cr:YSGG (2940 nm and 2780 nm, respectively), and the $CO_2$ (10,600 nm). In addition, photodynamic therapy protocols use diode lasers with wavelengths in the range of 635 nm to 690 nm combined with a photosensitizer to eradicate subgingival microbes.

#### ND:YAG LASER

Laser-mediated periodontal therapy is based on the purported benefits derived from subgingival soft-tissue curettage and significant decreases in subgingival bacterial loads. **Table 1** seems to indicate that Nd:YAG lasers are used primarily for these specific reasons, that is, laser-assisted subgingival soft-tissue curettage and reduction of subgingival microbial populations.[32–41] The exception is the laser-assisted new attachment procedure (LANAP)[39] which purports to promote regeneration of lost periodontal support structures (eg, cementum, periodontal ligament, and supporting alveolar bone).

Despite the increasing use of the Nd:YAG laser for treating periodontitis, well-designed and adequately powered studies (eg, that include enough subjects to see a difference if one exists) are severely limited and the overall quality of the body of evidence is insufficient to support evidence-based decision making. For various reasons performing a meta-analysis using data from existing clinical trials that have used the Nd:YAG laser as the test therapy is not possible. In fact, one can legitimately argue that deriving evidence-based conclusions from the published literature is overtly speculative as the body of evidence is weak and often confusing. For example, in the outcomes measurement of periodontal probing depth (PPD) in the clinical trials listed in **Table 1**, 2 studies did not measure PPD as an end-point[32,35]; 1 study did not provide data for PPD measurements[41]; 3 studies reported little or no difference in PPD reduction when comparing laser-treated sites with control sites[36–38]; 1 study reported a greater mean decrease in PPD in the control group (scaling and root planing, ie, SRP) than in the laser-treated group[33]; 1 study reported the laser improved PPD compared with untreated controls[34]; 1 study reported a decrease in PPD when the laser was used in combination with locally delivered minocycline and compared with a sham procedure[40]; and 1 study reported the laser improved PPD compared with historic controls (ie, data reported in other studies used for comparison).[39] This latter study[39] reported large standard deviations for mean PPD reductions in laser-treated pockets, indicating either a significant variation in technique or a significant degree of unpredictability in the procedure.

Other examples of inconsistencies in reporting of treatment outcomes are also seen in **Table 1**. Only 3[34,38,40] of the 10 studies measured gains in clinical attachment levels (CAL), despite the fact that CAL is considered the gold standard for demonstrating effect of periodontal therapy. Six of the 10 studies reported bleeding on probing (BOP) as an outcome although, similar to probing depth (PD), there was considerable variability in results. Five of the 10 studies did not measure reductions in subgingival microbial populations and of the 5 studies that did report on this parameter, 3 showed no significant difference,[32,36,41] 1 favored the SRP control group,[33] and 1 favored the laser-treated group[34] although no data were presented.

Although calculating the average of a series of means is risky at best and not a reliable statistical method, it does allow trends to be viewed. Given this caveat, **Table 1** shows a difference in PD reduction between laser treatment groups and controls of 0.09 mm, a gain in CAL of 0.33 mm (favoring the Nd:YAG laser) and essentially no difference in reductions in BOP or subgingival microbial loads.

Probably as a result of the nonsurgical character attributed to laser periodontal therapy, most clinical studies comparing the laser to standard therapy in the treatment of periodontitis use SRP as the control rather than conventional surgical procedures. The concept of regeneration of the periodontal attachment apparatus as an ultimate goal is, in many cases, more of an ideal than a pragmatic achievement. The debate will continue over regenerated connective tissue attachment versus long-junctional epithelial attachment, the latter often the result of many surgical and nonsurgical periodontal procedures.[42,43] However, for purposes of comparison, it would seem that laser procedures may have a positive adjunctive effect on periodontal regeneration by decreasing bacteria, producing an etching effect on root surfaces, removing granulation tissue, and de-epithelialization of the pocket soft-tissue wall. When laser therapies are compared with conventional open-flap procedures, with or without the addition of biologic mediators such as enamel matrix protein derivatives, the conclusions are consistent in that no statistical or clinically significant differences have been reported when comparing traditional surgery with laser-mediated periodontal surgery.[44,45]

A recent human histologic study using the Nd:YAG laser in a specific protocol, the LANAP, reports new cementum and new connective tissue attachment on previously diseased root surfaces, and bone regeneration.[46] By contrast, the SRP controls exhibited repair via a long-junctional epithelium with no evidence of bone regeneration. Moreover, there were no adverse changes associated with the laser group.

Several issues concerning this study are worthy of consideration. First, the study was not blinded. Second, the study is basically a proof of principle study as the number of specimens is quite small (ie, 6 pairs of single-rooted teeth). The limited number of specimens severely restricts extrapolation of results to the general population. Third, the study used pretreatment notches in the teeth as histologic reference points. Such notches are difficult to place subgingivally and, therefore, it is hard to determine the site of placement, and difficult to detect on histologic specimens that have been demineralized for sectioning. Fourth, the study did not use stents to aid clinical measurement. Consequently, given that manual probing is susceptible to variation, the results achieved fall within the range of acceptable measurement error for PPD and CAL of $\pm$1 mm reported by other clinical trials devoted to measuring such parameters.[47,48] Thus, it can justifiably be argued that the reported gain in CAL and reduction in PD may only equal that achieved by SRP. Fifth, the materials and methods are vague: ''Sections were cut in 200 micron increments until the notch was found, then serially sectioned at 200 micron increments (7 micron thick sections) until the notch was no longer visible. The 3 most central sections were then sent … for evaluation.'' Depending on interpretation, the ''3 most central sections'' can represent either 3.2% or 30% of the total possible number of 7 $\mu$m sections. Regardless of the interpretation, the resulting number of sections represents a limited histologic assessment and says nothing about consistency of effect across the depth and width of the treated defect. Lastly, all treated teeth were single-rooted. Thus, no conclusions can be made regarding the effect of treatment on multirooted teeth presenting with furcation involvement. The ultimate test of the regenerative powers of the LANAP protocol would be regeneration of horizontal bone loss, a study yet to be done.

**Table 1**
**Summary of clinical trials for Nd:YAG laser treatment of periodontitis (4–6 mm PDs)**

| Reference | No. of Subjects, Length of Study | Reduction in PPD (mm): Laser vs Control | Gain in CAL (mm): Laser vs Control | Reduction in BOP (%): Laser vs Control | Reduction in Microbes | Comment |
|---|---|---|---|---|---|---|
| Ben Hatit et al, 1996[32] | 14, 70 d | n.a. | n.a. | n.a. | No significant difference | Treatment groups: laser used at different energy densities vs SRP (control) |
| Radvar et al, 1996[33] | 11, 42 d | 0.50 vs 1.70 | n.a. | 10 vs 45 | SRP | Treatment groups: laser vs SRP (control) |
| Neill & Mellonig, 1997[34] | 10, 180d | 1.30 vs 0.40 | 1.10 vs 1.00 | Yes | Laser | Treatment groups: laser vs SRP vs untreated control |
| Liu et al, 1999[35] | 8, 84 d | n.a. | n.a. | n.a. | n.a. | Treatment groups: laser vs SRP vs laser + SRP vs SRP + laser. Outcome measure was level of IL-1β in GCF |
| Gutknecht et al, 2002[36] | 20, 175d | 0.85 vs 0.80 | n.a. | 85 vs 75 | No significant difference | Treatment groups: SRP+laser once/wk for 3 weeks vs SRP vs untreated control |
| Sjostrom et al,[a] 2002[37] | 27, 120d | 1.40 vs 1.40 | n.a. | 27 vs 29 | n.a. | Treatment groups: laser+SRP+laser vs SRP (control) |
| Miyazaki et al, 2003[38] | 18, 84 d | 1.43 vs 1.36 | 0.50 vs 0.57 | 43 vs 34 | n.a. | Treatment groups: Nd:YAG laser vs $CO_2$ laser vs ultrasonic scaling (control) |

| | | | | | | |
|---|---|---|---|---|---|---|
| Harris et al, 2004[39] | 75,[b] 180d | 1.55 vs 1.29 | n.a. | n.a. | n.a. | Treatment groups: LANAP protocol vs historic controls |
| Noguchi et al, 2005[40] | 16, 90 d | 1.57 vs n.a. | 1.52 vs n.a. | 63 vs n.a. | n.a. | Treatment groups: laser vs laser + local minocycline vs laser + povidone-iodine irrigation vs sham laser control |
| Verhagen et al, 2006[41] | 15, 90 d | No significant difference (no data given) | n.a. | 16 vs 17 | No significant difference | Treatment groups: SRP + laser with and without systemic antibiotics vs SRP with and without systemic antibiotics. Use of antibiotics had no impact on parameter outcomes |
| Average Difference: laser vs control | | 1.23 vs 1.14<br>0.09 | 1.04 vs 0.71<br>0.33 | 41 vs 40<br>1 | 1 favored laser<br>1 favored SRP<br>3 no significant difference<br>5 studies n.a. | |

All numbers for reductions in PD and BOP and gains in CAL represent means.

*Abbreviation:* n.a., did not measure parameter.

[a] Nd:YCG (1061 nm).

[b] Includes 10 patients from Neill and Mellonig's study.[34]

**Table 2**
**Summary of clinical trials for Er:YAG/Er,Cr:YSGG laser treatment of periodontitis (4–6 mm PDs)**

| Reference | No. of Subjects, Length of Study | Reduction in PPD (mm): Laser vs Control | Gain in CAL (mm): Laser vs Control | Reduction in BOP (%): Laser vs Control | Reduction in Microbes | Comment |
|---|---|---|---|---|---|---|
| Schwarz et al, 2001[59] | 20, 180 d | 2.00 vs 1.60 | 1.90 vs 1.00 | 77 vs 56 | No significant difference | Treatment groups: laser vs SRP (control) |
| Schwarz et al, 2003[60] | 20, 1 y | 2.00 vs n.a. | 1.60 vs n.a. | 16 vs n.a. | No significant difference | Treatment groups: laser+SRP vs laser (control) |
| Schwarz et al,[a] 2003[61] | 20, 2 y | 1.60 vs 1.30 | 1.40 vs 0.70 | 64 vs 46 | No significant difference | Article reports long-term results of the Schwarz, et al, 2001 study[59] |
| Schwarz et al, 2003[44] | 22, 180 d | 4.00 vs 4.10 | 3.20 vs 3.30 | 35 vs 26 | n.a. | Treatment groups: access flap surgery + laser debridement + enamel matrix protein derivative (test) vs access flap surgery + SRP + enamel matrix protein derivative (control) |
| Sculean et al, 2004[45] | 23, 180 d | 1.52 vs 1.57 | 1.11 vs 1.11 | 23 vs 31 | n.a. | Treatment groups: laser vs flap surgery and debridement of root and defect (control) |
| Sculean et al, 2004[62] | 20, 180 d | 3.70 vs 3.20 | 2.60 vs 1.50 | 63 vs 59 | n.a. | Treatment groups: laser vs ultrasonic scaling (control) |
| Tomasi et al, 2006[63] | 20, 120 d | 1.10 vs 1.00 | 0.60 vs 0.40 | 40 vs 40 | No significant difference | Treatment groups: laser vs ultrasonic scaler (control) |

| | | | | | | |
|---|---|---|---|---|---|---|
| Crespi et al,[b] 2007[64] | 25, 2 y | 2.88 vs 1.00 | 2.92 vs 1.32 | n.a. | n.a. | Treatment groups: laser vs ultrasonic scaler (control). Treated 5–6 mm $\geq$ 7 mm PDs |
| Gaspirc & Skaleric, 2007[65] | 25, 5 y | 2.79 vs 2.87 | 1.72 vs 1.76 | 39 vs 23 | n.a. | Treatment groups: laser + surgery vs modified Widman flap (control) |
| Kelbauskiene et al, 2007[66] | 10, 84 d | 2.00 vs 0.97 | n.a. | 68 vs 60 | n.a. | Treatment groups: laser vs SRP clinical trial using Er,Cr:YSGG laser |
| Lopes et al,[c] 2008[67] | 21, 30 d | 1.60 vs 1.67 (L+SRP vs SRP) | 0.21 vs 0.48 (L+SRP vs SRP) | n.a. | n.a. | Treatment groups: laser + SRP vs laser vs SRP vs untreated control |
| Average of the mean | | 2.29 vs 1.93 | 1.73 vs 1.23 | 47 vs 43 | 4 no significant difference | |
| Difference: laser vs control | | 0.36 | 0.45 | 4 | 7 studies n.a. | |

All numbers for reductions in PD and BOP and gains in CAL represent means.

*Abbreviation:* n.a., did not measure parameter.

[a] Paper reports the long-term results of the Schwarz et al, 2001 study.[59]

[b] Treated 5–6 mm and 7 mm PD.

[c] Treated 5–9 mm PD. Mean PD for all Tx groups ranged from 6.28 to 6.87 mm.

**Table 3**
**Summary of clinical trials for diode laser treatment of periodontitis (4–6 mm PDs)**

| References | No. of Subjects, Length of Study | Reduction in PPD (mm) Laser vs Control | Gain in CAL (mm) Laser vs Control | Reduction in BOP (%) Laser vs Control | Reduction in Microbes | Comment |
|---|---|---|---|---|---|---|
| Moritz et al, 1998[68] | 50, 180 d | 1.30 vs 0.40 | n.a. | 97 vs 67[a] | No significant difference | Treatment groups: scaling + laser at 1, 8, and 16 weeks vs scaling + $H_2O_2$ (control) rinsing at 1, 8, and 16 weeks |
| Borrajo et al, 2004[69] | 30, 42 d | n.a. | 0.81 vs 0.85 | 72 vs 53 | n.a. | Treatment groups: SRP + laser vs SRP (control) |
| Qadri et al, 2005[70] | 17, 42 d | 0.90 vs 0.20 | n.a. | n.a. | No significant difference | Treatment groups: laser vs laser sham (control). Measured GCF levels of IL-1β and MMP-8 and reported no significant difference between groups |
| Kreisler et al, 2005[71] | 22, 90 d | 1.8 vs 1.6 | | 38 vs 34 | n.a. | Treatment groups: all patients received SRP. Subsequently, 2 quadrants in each patient were treated with the laser |
| Kamma et al, 2006[72] | 30, 84 d | Laser 2.00<br>SRP 2.34<br>L+SRP 2.80<br>Control 0.13 | Laser 1.94<br>SRP 1.87<br>L+SRP 2.14<br>Control 0.27 | Laser 65<br>SRP 57<br>L+SRP 63<br>Control 61 | Laser | Treatment groups: laser vs SRP vs laser + SRP vs untreated control. Aggressive periodontitis with clinical attachment loss of $\geq$ 5 mm |
| Average of the means | | 1.7 vs 1.14 | 1.52 vs 1.34 | 68 vs 53 | 1 favored laser | |
| L+SRP vs SRP | | | | | 2 no significant difference | |
| Difference: L+SRP vs SRP | | 0.56 | 0.18 | 15 | 2 studies n.a. | |

All numbers for reductions in PD and BOP and gains in CAL represent means.
*Abbreviation:* n.a., did not measure parameter.
[a] Percent of sites showing improvement.

With respect to laser-mediated periodontal regeneration, in a study by Schwartz and colleagues[49] beagle dogs with naturally occurring periodontitis were treated with an Er:YAG laser; ultrasonic scaling was used as a control. Both treatment groups exhibited new cementum formation with embedded collagen fibers. The investigators concluded that both therapies supported the formation of new connective tissue attachment.

The Nd:YAG laser is absorbed selectively by certain pigments, including melanin and hemoglobin. Given this selective absorption in darker pigments, proponents of this wavelength have promoted the laser as being effective against the pigmented bacteria frequently associated with periodontal diseases, eg, *Porphyromonas* spp, *Prevotella* spp, *Tannerella* spp. However, the common periodontal diseases exhibit a subgingival biofilm comprised of a diverse population of bacteria, most of which are not pigment producers.[50]

#### ER:YAG AND ER,CR:YSGG LASERS

Two different wavelengths of erbium lasers are currently available for clinical use: the Er:YAG (2940 nm) and the Er,Cr:YSGG (2780 nm). Each system ablates soft and hard tissues with minimal heat-related side effects. It has been suggested that the erbium wavelengths present the broadest range of application for clinical dentistry and are likely the most suitable lasers for periodontal therapy.[13,21,51,52]

The erbium lasers are effective in removing calculus and reducing PPD. Several studies have demonstrated safe and effective root substance removal without negative thermal effects, comparable with conventional instrumentation.[53–55] Not surprisingly, these lasers are bactericidal against in vitro cultures of *Porphyromonas gingivalis* and *Aggregatibacter* (formally *Actinobacillus*) *actinomycetemcomitans*,[56] and effective in removing absorbed root surface endotoxins.[57,58]

In **Table 2**, a collective average for the 11 clinical trials shows equivalent or slightly greater reductions in PPD (2.29 mm vs 1.93 mm), gains in CAL (1.73 vs 1.26 mm), and decreased BOP (47% vs 43%) when comparing laser therapy with the control treatments.[44,45,59–67] The one paradoxic exception is that of the 4 studies reporting the effect of treatment on subgingival microbial levels; none showed a significant difference between treatment groups.[59–61,63]

Three of the clinical trials deviated from the usual design in that they either combined the Er:YAG laser with flap surgery, with and without adjunctive use of enamel matrix protein,[44] compared the laser with traditional access flap surgery,[45] or compared the laser with the modified Widman flap.[65] For PPD reduction and gains in CAL the results were essentially equivalent.

#### DIODE LASER

The most widely used lasers in the diode family are the gallium-aluminum-arsenide (GaAlAs) laser (810 nm) and the indium-gallium-arsenide (InGaAs) laser (980 nm). A low initial investment cost and ease of use by dental hygienists are undoubtedly major factors for this popularity. Thus, given the apparent widespread use of the diode for treatment of slight to moderate periodontitis, it is surprising to realize that currently there are only 5 published clinical trials (**Table 3**). As with the Nd:YAG laser, the purported benefits of diode laser periodontal therapy are based on the premise that subgingival curettage is an effective treatment and that significant reduction in subgingival microbial populations are predictably achieved.

The 5 studies[68–72] presented in **Table 3** used various control groups with which to compare diode laser therapy. In 4 studies,[68,69,71,72] the diode laser was used

**Table 4**
**Summary of clinical trials for photodynamic therapy (PDT) using a diode laser plus a photosensitizer for the treatment of periodontitis pockets with 4–6 mm probing depths**

| Reference | Laser Type and Photosensitizer | No. of Subjects, Length of Study | Reduction in PPD (mm): PDT vs SRP | Gain in CAL (mm): PDT vs SRP | Reduction in BOP (%): PDT vs SRP | Reduction in Microbes | Comment |
|---|---|---|---|---|---|---|---|
| Yilmaz et al, 2002[80] | Diode (685 nm); Methylene Blue | 1032, d | PDT + SRP 0.66<br>PDT 0.23<br>SRP 0.49<br>OHI 0.19 | n.a. | PDT + SRP 60<br>PDT 17<br>SRP 50<br>OHI 20 | No significant difference | Treatment groups: laser + SRP vs laser vs SRP vs OHI (control). Photosensitizer used in both laser groups |
| Andersen et al, 2007[81] | Diode (670 nm); Methylene Blue | 33, 84 d | PDT+ SRP 1.11<br>PDT 0.67<br>SRP 0.74 | PDT+ SRP 0.62<br>PDT 0.14<br>SRP 0.36 | PDT+SRP 73<br>SRP 56 | n.a. | Treatment groups: PDT + SRP vs PDT vs SRP |
| de Oliveira et al, 2007[82] | Diode (690 nm); Phenothiazine | 10, 90 d | PDT+SRP 1.43<br>SRP 0.94 | PDT+ SRP 1.19<br>SRP 1.52 (relative clinical attachment level) | PDT+ SRP 38<br>SRP 39 | n.a. | Treatment groups: PDT vs SRP (control). Mean PD was 4.92 mm in aggressive periodontitis |
| Braun et al, 2008[83] | Diode (660 nm); Phenothiazine | 20, 90 d | PDT+SRP 0.8<br>SRP 0.7 | n.a. | PDT+SRP 56<br>SRP 51 | n.a. | Treatment groups: PDT + SRP vs SRP (control) |
| de Oliveira et al, 2009[84] | Diode (660 nm); Phenothiazine | 10, 90 d | n.a. | n.a. | n.a. | n.a. | Treatment groups: PDT + SRP vs SRP (control). No significant difference in GCF levels of TNF-$\alpha$ or RANKL |
| Polansky et al 2009[85] | Diode (680 nm); HELBO blue | 58, 90 d | PDT+SRP 1.24<br>SRP 1.03 | n.a. | PDT+SRP 53<br>SRP 41 | No significant difference | Treatment groups: PDT+SRP vs SRP (control) |
| Average of the means | | | PDT+SRP 1.05 | PDT+SRP 0.91 | PDT+SRP 56 | No significant difference | |
| PDT+SRP vs SRP | | | SRP 0.78 | SRP 0.94 | SRP 47 | | |
| Difference: PDT+SRP vs SRP | | | 0.27 | 0.03 | 9 | | |

All numbers for reductions in PD and BOP and gains in CAL represent means.
*Abbreviations:* n.a., did not measure parameter; OHI, oral hygiene index.

**Table 5**
**Comparative summary of results from clinical trials using Nd:YAG, Er:YAG, or diode lasers for treatment of periodontal (4–6 mm PDs)**

| LaserType (Number of Clinical Trials) | Reduction in PPD (mm) | Gain in CAL (mm) | Reduction in BOP (%) | Reduction in Microbes |
|---|---|---|---|---|
| Nd:YAG (n = 10) | 1.23 | 1.04 | 41 | 2/10[a] |
| Er:YAG/Er,Cr:YSGG (n = 11) | 2.30 | 1.68 | 47 | 0/11 |
| Diode (n = 5) | 1.70 | 1.52 | 68 | 1/5[b] |
| Photodynamic therapy (n =5) | 1.05 | 0.91 | 56 | 0/5 |

[a] 1 study favored the laser and 1 study favored SRP.
[b] 1 study favored the laser.

adjunctively with SRP. Control groups consisted of SRP in 2 studies,[69,71] a sham laser treatment in 1 study,[70] and an initial scaling followed by periodic hydrogen peroxide or saline oral rinses in the remaining 2 studies.[68,71] None of the studies measured all 4 of the usual clinical parameters, that is, reductions in PPD, BOP, and subgingival microbes, or gains in CAL. Consequently, given the limited number of studies and the diversity in experimental design, it is not possible to combine the 5 studies for the purpose of a meta-analysis.

Despite these limitations it is possible to discern trends. For example, as noted in **Table 3**, when comparing the laser treatment groups with their specific controls, the laser groups showed greater reductions in PPD (1.70 mm vs 1.14 mm) and BOP (68% vs 53%) but a nearly equivalent gain in CAL (1.52 mm vs 1.34 mm). Three of the 5 studies measured reductions in microbes but only 1[72] reported a significant difference that favored the laser. The remaining 2 studies reported no significant differences between treatment groups.[68,70] Despite the equivalency between laser-treated sites versus controls, uncontrolled case studies continue to report successful periodontal therapy when using the diode and Nd:YAG lasers as adjuncts to SRP.

Diode lasers are effective for soft-tissue applications, offering excellent incision, hemostasis, and coagulation.[73] However, diode wavelengths when combined with the appropriate choice of parameters can result in penetration of soft tissues ranging from about 0.5 mm to 3 mm.[14] Thus, 1 must select parameters with caution to avoid undesired collateral damage. In this regard, the diode and Nd:YAG lasers are contraindicated for calculus removal. They both exhibit poor energy absorption in mineralized tissues and thus offer the possibility of excessive generation of heat caused by their interaction with darkly colored deposits. However, given the current recommended parameters, the possibility of inducing root surface damage is virtually impossible.[74,75]

## PHOTODYNAMIC THERAPY

Photodynamic therapy (PDT) involves the combination of visible light, usually using a low wavelength diode laser (635 nm to 690 nm) and a photosensitizer. The photosensitizer is generally an organic dye or similar compound capable of absorbing light of a specific wavelength, after which it is transformed from a ground singlet state to a longer-lived excited triplet state.[76] The longer lifetime of the triplet state enables the interaction of the excited photosensitizer with the surrounding tissue molecules. It is generally accepted that the generation of the cytotoxic species produced during

PDT occurs while in the triplet state.[77,78] The cytotoxic product, generally $O_2$, cannot migrate more than 0.02 μm after this formation, thus making it ideal for the local application of PDT without endangering distant biomolecules, cells, or organs.[79]

As with the diode laser, there are a relatively small number of published clinical trials.[80–84] Given the apparent potential for PDT, it is discouraging that the collective differences reported for measurable clinical parameters are not particularly noteworthy (**Table 4**). The aggregate of clinical trials shows a reduction in PPD for PDT versus SRP of 1.0 mm versus 0.72 mm, respectively. Reduction in BOP was somewhat better: 57% for PDT versus 49% for SRP. Gains in CAL were nearly equal, 0.91 mm for PDT versus 0.94 mm for SRP. The major reason for using PDT is to effect reductions in subgingival microbes. Thus, it is surprising that only 1 of the 5 published clinical trials measured this parameter[80] and that study reported no significant difference between PDT and SRP treatment groups. In addition, de Oliveira and colleagues[84] compared treatment of aggressive periodontitis with PDT versus SRP and reported no significant differences between treatment groups for measures of gingival crevicular fluid (GCF) levels of tumor necrosis factor-alpha (TNF-α) or nuclear factor-kappa B ligand (RANKL), both factors being involved in bone resorption.

### $CO_2$ LASER

The carbon dioxide wavelength is effective in removing soft tissue and inflamed pocket tissues while achieving good hemostasis. However, there are only 2 published clinical trials showing the effect of this wavelength on PD.[38] One study compared the Nd:YAG with ultrasonic scaling and the $CO_2$ laser and reported no significant difference between the 3 treatments. Choi and colleagues[86] measured changes in GCF levels of interleukin-1beta (IL-1β) at 6 weeks and PPD, BOP, and CAL at 6 months following treatment of periodontitis by traditional flap surgery versus flap surgery plus $CO_2$ laser irradiation of the exposed root surfaces using 2 different energy densities. The investigators reported no significant differences between treatment groups for reduced PPD (flap + laser groups, 2.7 mm vs flap surgery alone, 2.2 mm) and reduction in BOP (flap + laser groups, 61% vs flap surgery alone, 69%). Paradoxically, CAL gains were statistically significantly better for 1 of the $CO_2$ treatment groups, as were reductions in IL-1β. The primary caution when using the $CO_2$ laser for subgingival periodontal therapy relates to the wavelength's high absorption by hydroxyapatite and water. The clinician is well advised to carefully direct the energy beam and use low powers and low energy densities to avoid damage to healthy hard tissues. The recently introduced super-pulse mode reduces the potential for adverse effects caused by excessive heat generation during interaction with hard tissues.

### SUMMARY

Many questions remain to be answered on use of lasers as a singular modality or as an adjunct for the treatment of periodontitis. Although the adjunctive use of lasers with traditional treatment modalities is less controversial, published clinical trials indicate only a slightly greater benefit should be expected with respect to gains in CAL and reductions in PPD, BOP, and subgingival microbial loads (**Table 5**). A recent publication noted that a meta-analysis of clinical trials was impossible because of the lack of homogeneity between studies.[21] Although the collective evidence was considered weak, the investigators did note that Er:YAG laser monotherapy resulted in similar clinical outcomes compared with SRP for up to 24 months post treatment.[21] Clearly, additional well-designed randomized, blinded, controlled longitudinal studies are necessary to provide clear and meaningful evidence to validate the use of this

technology in periodontal therapy. Because of the current lack of published well-designed clinical trials, clinicians using lasers for the treatment of periodontitis must be cognizant of safety issues and should expect limited clinical improvement in periodontal status.

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