Review of the Literature

Adjunctive Use of the Diode Laser in Non-Surgical Periodontal Therapy: Exploring the Controversy

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Introduction

Lasers have been available for use in dentistry since 1989, but their use has not been universally accepted. Their efficacy for certain dental procedures, such as non-surgical periodontal therapy, is still controversial. In order to explore this controversy, the PubMed database was searched for literature regarding laser use in periodontal therapy. Utilizing key search terms, including diode lasers, scaling and root planing, bacteria, and periodontal disease, over 100 articles were identified and screened for inclusion in this review.

Some dental hygienists where not prohibited by their state dental practice act, are using lasers as an adjunct to non-surgical periodontal therapy.[1-5] Although the carbon dioxide (C02), erbium-doped yttrium aluminium garnet (Er:YAG) and neodymium-doped yttrium aluminium garnet (Nd:YAG) can all be used for soft tissue procedures, the 810 nm to 980 nm diode lasers appear to be the most common lasers used in private practice.[1] However, the efficacy of all lasers for use as an adjunct to non-surgical periodontal therapy is controversial. Relatively few clinical trials have been published studying the use of the diode as an adjunct to non-surgical periodontal therapy. Most of these trials were performed by dentists affiliated with university medical and dental clinics. All trials had a small sample size. In 8 of the 10 published clinical studies, the authors stated that the diode group showed a trend of some clinical benefit, compared to the control groups.[6-13] One study showed no significant difference in the clinical outcomes between the intervention and control groups.[14] In one trial, the control group showed an improvement over the intervention group in the end-point clinical measures.[15] The varied study outcomes and heterogeneity of methodology identified in other laser literature reviews, together with the impossibility for a meta-analyses due to lack of sufficient, well designed standardized trials, create the foundation for the controversy.[16-19] The American Academy of Periodontology (AAP) in April 2011 issued a statement of no efficacy for the use of lasers as an adjunct to non-surgical therapy for the treatment of periodontal disease, citing a lack of consistent evidence among the reviewed studies.[20]

The purpose of this review is to explore the scientific foundation of the controversy surrounding the clinical

Abstract

Purpose: Despite the controversy regarding clinical efficacy, dental hygienists use the diode laser as an adjunct to non-surgical periodontal therapy. The technique to maximize successful laser therapy outcome is controversial as well. The purpose of this review is to explore the scientific foundation of the controversy surrounding the use of the diode laser as an adjunct to non-surgical periodontal therapy. Further, this paper addresses the weaknesses in study design, the heterogeneity of methodology in the published clinical studies, especially the laser parameters, and how these issues impact the collective clinical and microbial data, and thus conclusions regarding clinical efficacy. Evaluation of the literature identifies possible mechanisms that could contribute to the varied, often conflicting results among laser studies that are the foundation of the controversy surrounding clinical efficacy. These mechanisms include current paradigms of periodontal biofilm behavior, tissue response to laser therapy being dependent on tissue type and health, and that the successful therapeutic treatment window is specific to the target tissue, biofilm composition, laser wavelength, and laser energy delivered. Lastly, this paper discusses laser parameters used in the various clinical studies, and how their diversity contributes to the controversy. Although this review does not establish clinical efficacy, it does reveal the scientific foundation of the controversy and the need for standardized, well designed randomized controlled clinical trials to develop specific guidelines for using the laser as an adjunct to non-surgical periodontal therapy. Using evidence-based laser guidelines would allow dental hygienists to provide more effective non-surgical periodontal care.

Keywords: diode laser, scaling and root planing, periodontal diseases, periodontitis, bacteria, dental hygienists, soft tissue laser, non-surgical periodontal therapy

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efficacy of the diode laser as an adjunct to non-surgical periodontal therapy. Further, this paper addresses the weaknesses in study design and the heterogeneity of methodology in the published clinical studies, especially the laser parameters, and how these issues impact the collective data regarding clinical outcomes, such as reductions in pocket depth (PD), bleeding on probing (BOP), subgingival bacterial loads, bacteremia and gain in clinical attachment level (CAL). Lastly, this paper discusses laser parameters used in the various clinical studies, and how their diversity contributes to the controversy.

Background of the Controversy

Chronic Periodontitis (CP): Current evidence indicates that 47.2% of the U.S. adult population has some degree of periodontitis.[21] The severity of periodontal disease is dependent not only on the presence and composition of biofilm, but on the host response to the biofilm microorganisms.[22] Periodontal disease may be related to diabetes, respiratory disease and cardiovascular disease.[23-26] Although scaling and root planing (SRP) is considered the "gold standard" for non-surgical periodontal therapy, it is not adequate for every patient. Patients who respond sub-optimally or are at high risk due to systemic complications, such as patients with diabetes or compromised health, may benefit from adjunctive therapy.[27] Diode lasers may have the potential to provide this additional benefit.

The primary etiology of CP is the bacterial composition of the microbial biofilm. Porphyromonas gingivalis (Pg), Tannerella forsythia (Tf) and Treponema denticola (Td) are members of the "Red" complex of periodontal pathogens, and are frequently associated with CP.[28] While Pg and Tf are the strongest bacterial markers for periodontal disease,[29] the additional presence of Td creates the "Consortium" gf periodontal pathogens associated with disease progression.[30] Most recently, Pg, despite being present in small numbers, has been shown to dramatically alter the compositaon of oral microbiota. Pg directs the genetic response of other microbes and the host, hence earns the designation as a keystone pathogen.[31,32] Biofilm is able to invade the cementum and epithelial lining of diseased pockets.[33,34] Disruption of the biofilm is the most effective means of treating periodontal disease.[27] Specifically, removal of Pg from the mouth reverses aberrant inflammation.[32,35] A 810 nm diode can destroy Pg in vitro.[36] Kamma et al found that use of a 980 nm diode laser plus SRP has been shown to reduce the levels of Pg and Td, as well as the total bacteria load, for 6 months post-baseline in patients with aggressive periodontitis.[6] However, the study did not address the extent of the bacterial load and the aggressiveness of the bacteria beyond 6 months post-baseline.

Soft Tissue Lasers

Laser light is a man-made single photon wavelength, which emits non-ionizing (non-cancer-associated) radiation.[37] The wavelength is determined by the typm of elements in the laser. The diode laser is actually a semiconductoz, and is usually some combination of Gallium, Arsenide, Aluminum, Indium and Phosphorous. The wavelength range continues to expand, but currently, the most common diode wavelengths used in dentistry are 610 nm (red) to 980 nm (infrared), and can be operated in continuous-wave (CW) and gated-pulsed (PW) modes.

When laser light reaches a tissue, it can reflect, be absorbed, scatter or be transmitted to the surrounding tissues. The absorbed energy can result in tissue warming, coagulation or vaporization, depending on the wavelength, power and optical properties of the tissue.[17] The diode laser light is highly absorbed in hemoglobin and other pigments.[16,17] This property makes it an excellent device for removing the inflamed, highly vascular tissue within a periodontally involved pocket.[18]

The diode laser can be bactericidal.[6,7,13,36,38] Diode lasers target "pigmented" bacteria.[18,37,39] While it is unknown if "pigmented" pathogens are actually pigmented within the periodontal pocket,[40] it is known that diode lasers can oblate Pg in vitro.[36] The 810 nm to 980 nm diode laser light creates thermal changes resulting in the destruction of the bacteria in soft tissue. Most non-sporulating bacteria, including anaerobes, are readily deactivated at temperatures of 50 degrees Celsius.[37] The 810 nm to 980 nm diodes can create thermal changes elevating tissue temperature beyond this threshold.[39] Lower intensity diode lasers, such as the 610 nm to 750 nm (red) diodes, are currently gaining interest due their affordability, minimal treatment risk and potential to kill bacteria.[41] Inclusion of a photosensitive dye, known as photo-activated disinfection, photodynamic therapy (PDT) or antimicrobial photodynamic therapy, may enhance the bactericidal effect.[42,43] However, like other studies on lasers, PDT studies show modest clinical improvement of CP and lack the meta-analyses on existing clinical trials that can make a definitive statement regarding their efficacy.[44] Diodes of many wavelengths used at a lower non-surgical power (i.e. with a non-initiated tip, at less than 1 watt, and/or with a gated pulse) are currently gaining popularity due to the flurry of research on their photobiomodulation ability and promotion of healing.[42,45-49]

Diode lasers are smaller in size and less expensive than most dental lasers.[16,17] The 810 nm diode laser is easy to operate, and has been marketed to dental hygienists. Their hemostatic properties can reduce post-

treatment bleeding.[37] Other advantages of lasers include cell regeneration, collagen growth and mucosal tissue regeneration, along with an anti-inflammatory effect.[47,48,50] In a recent study, the diode laser significantly reduced the level of tumor necrosis factor-alpha (TNF-α) a pro-inflammatory cytokine, in gingival papillae of patients with chronic advanced periodontal disease.[47] This study also demonstrated that more frequent use of the laser related to greater reduction in the levels of TNF-α.[47] It is unclear whether these benefits from both low level and high intensity diode laser exposure can also be obtained when diode lasers are used for adjunctive periodontal therapy.

Reasons for Controversy

Heterogeneity of Clinical Studies: Few clinical trials on the high intensity diode lasers have been published to date.[18] The heterogeneity of methodology among these studies makes comparisons and conclusions challenging, hence contributes to the controversy surrounding a statement of efficacy.[16-19] Studies have varied in laser power density settings (350 mW/cm2 to 2,830 W/cm2), exposure time (3 seconds to 90 seconds), frequency of laser treatments (1 to 6 times) and clinical assessment parameters (plaque index to clinical crown length). The First International Workshop of Evidence Based Dentistry on Lasers in Dentistry addresses this heterogeneity by identifying use parameters,[39] which had been omitted from previous studies, to be specified in all future laser studies including:

- Exact laser specification, including manufacturer, wavelength, power output, control of output
- Spot size of irradiated area, joules/spot, and joules per session expressed as J/cm2
- Mode of application, number of sessions, treatment schedules

Lack of these use parameters in previous research may have contributed to the inconsistency in outcomes among studies.

The heterogeneity of methodology and weaknesses is some of the studies' designs are evident in Table I. In 7 of the 10 referenced studies the same type of non-surgical treatment (scaling, SRP or ultrasonic scaling) was conducted in both the control group and intervention group. In addition to this treatment, Moritz et al included a hydrogen peroxide (H_2O_2) rinse to only the control group.[7] Lin et al included a 1% chlorhexidine rinse to only the intervention group.[14] Zingale et al failed to include SRP in the control group.[10] Quadri et al added a placebo laser with a very low-power red diode to the control group, which may have rendered an unintentional intervention. The heterogeneity of variables in the control groups makes comparison of the studies challenging. Lack of examiner masking (blinding) to the treatment groups, and lack of a clear statement regarding examiner calibration is also evident in Table I. Studies lacking examiner masking and calibration are suspect for bias.

Although the published trials utilized a wide variety of clinical assessments, Table II illustrates the clinical end-point measures that were common to these studies. In 8 of the 10 clinical trials, the diode group showed a trend of at least 1 clinical outcome benefit over the control or alternate treatment groups.[6-13]

As illustrated in Table II, six clinical studies used microbial assessments as outcomes. Of those, 3 showed that laser treatment reduced the number/amount of pathogens in the periodontal pockets, or bacteremia associated with ultrasonic scaling.[6,7,13] The Moritz[7] and Borrajo[9] studies are among the few non-split mouth trials found in the literature. Most of the diode laser studies have used the split-mouth, quadruple split-mouth, or multi-site design.[6,8,10-15] With current knowledge regarding periodontal pathogens and biofilm behavior, microbial or clinical assessment data collected from these study designs may not be valid. Pathogens in the biofilm may be released from the biofilm at 1 site, enabling them to colonize in other sites of the mouth.[28] One study utilizing multi-sites per mouth showed improvement among all groups, including the control.[10] The behavior of pathogens within the biofilm may contribute to the varied study outcomes, hence prove to be a significant confounder to the collective data obtained from these common multi-site study designs.

Mammalian Cell Behavior

Further complicating the interpretation of the results from laser studies is the overall health of the cell that is undergoing laser exposure. Human fibroblasts cultured in serum-starved medium, consistently exhibited enhanced procollagen production when exposed to low level laser.[51] This was not observed with laser exposure to fibroblasts cultured in serum-containing medium. Houreld et al studied the effect of laser exposure on diabetic-induced fibroblasts in an in- vitro wound model.[46] They found that diabetic-induced fibroblasts exhibited more complete wound closure and less apoptosis when exposed to laser therapy in a dose and wavelength dependent manner, as compared to non-irradiated cells. Obradovic and colleagues examined histological specimens of diabetic patients who received conservative periodontal therapy for chronic periodontal disease with and without low level laser therapy.[49] The histological specimens of diabetic patients treated with both conservative periodontal therapy and laser exhibited less inflammation and greater healing, as compared to those specimens

Table I: Summary of Clinical Studies

Study	Intervention (Test)	Control	Study Length	Split/ whole mouth	# Patients	# Teeth/ sites	Examiner blinded	Calibrated
Moritz et al., 1997	Scaling + 805nm DL 2.5 PW 1s/mm PD 3 times (X)	Scaling + H2O2	6 m	Whole mouth	46	Not stated	Not stated	Not stated
Borrajo et al., 2004	SRP + 980nm DL 2PW 10s/ tooth surface 2X	SRP	6 weeks	Whole mouth	30	Not stated	Yes	Not stated
Quadri et al., 2005	SRP + 635nm DL 10mW 90s/papilla + 830nm DL 70mW 25s / tooth 6X	SRP + Placebo Laser with very low power red diode	6 weeks	Split-mouth	17	Unclear	Yes	Not stated
Kreisler et al., 2005	SRP, H2O2 + 809nm DL @ 1 CW 10+s/tooth 1X	SRP, H2O2 + saline rinse	3 m	Split-mouth	22	492 T	Yes	Not stated
Kamma et al., 2006, 2009 Aggressive Perio	SRP + 980nm DL 2CW 30s/ pocket 1X	SRP only 980nm DL only No Tx	6 m	Quadrant split-mouth	30	750+ T	Yes	Yes
Assaf 2007	810nm DL 1PW 15s/tooth 1X + US scaling	US scaling (ultrasonic scaling)	4 weeks	Split-mouth	22	Not stated	Yes	Yes
Caruso et al., 2008	980nm DL + SRP	SRP	6 m	Split-mouth	13	38T	Unclear	Unknown
De Micheli et al., 2011	SRP + 808nmDL 1.5CW 20s/ tooth, saline rinse 2X	SRP + non-activated 808nm DL, saline rinse	6 weeks	Split-mouth	27	Not stated	Yes	Yes
Lin et al., 2011	810nm +1% chlorhexidine 2W exposure time not stated 1X	Subgingival curettage	4 weeks	Quadrant split-mouth	18	206T	No	No
Zingale et al., 2012	SRP PR/SRP/FC LC/SRP 810DL 0.8CW 30-45s/ site 1X LC/SRP/ LS 810nm DL 0.8CW 2X	No treatment	6m	Multi sites/ mouth	25	170 sites	Yes	Yes

DL=diode laser, PW=pulse watts, CW=continuous watts, PR=papillae reflection, FC=flap closure, LC=laser curettage, LS=laser sealing, Tx=treatment

Table II: Summary of End-Point Clinical Measures

Study	Test v. Control Reduction PD (mm)	Test vs. Control Gain in CAL mm	Test v. Control BOP% Reduction	Test v. Control Reduction in Bacteria
Moritz et al., 1997	1.30 v. 0.40	n.a.	97 v. 67	71% v. 25%
Borrajo et al., 2004	n.a	0.95 v. 0.85	72 v. 53	n.a.
Qadri et al., 2005	0.9 v. 0.2	n.a	n.a.	No Sig Diff
Kreisler et al., 2005	1.8 v.1.6	1.6 v. 1.3	38 v. 34	n.a.
Kamma et al., 2006, 2009	SRP+DL 2.80 SRP only 2.34 DL only 2.00 No Tx 0.13	SRP+DL 2.14 SRP only 1.87 DL only1.97 No Tx 0.27	SRP+DL 58 SRP only 56 DL only 61 No Tx 56	Sig. Diff SRP+DL
Assif et al., 2007	0.37 v. 0.28	0.39 v. 0.18	n.a	Sig Diff Bacteremia DL
Caruso et al., 2008	1.4 v. 1.08	2.0 v. 1.69	16 v. 10	No Sig Diff
DeMicheli et al., 2011	2.1 v. 2.4	1.2 v. 1.9	n.a.	No Sig Diff
Lin et al., 2011	1.54 v. 1.49	0.65 v. 0.70	53 v. 48	n.a.
Zingale et al., 2012	SRP 1.57 PR/SRP/FC 1.57 LC/SRP 1.72 LC/SRP/LS 1.62 No Tx 0.84	*SRP 0.48 *PR/SRP/FC 1.05 *LC/SRP 0.42 *LC/SRP/LS 0.15 *No Tx 0.15 *gain clinical crown length	RP 66 PR/SRP/FC 69 LC/SRP 63 LC/SRP/LS 67 No Tx 60	n.a.

from patients treated with conservative periodontal therapy alone. These cases illustrate the positive effects of laser therapy on healing at the cellular level, as observed in compromised cells. However, it is unclear whether these same positive effects from low level laser therapy can be obtained when lasers are used for adjunctive periodontal therapy.

Laser Technique: The technique of using the laser may influence the outcome of the study, further contributing to the controversy over the efficacy of laser use. In one split-mouth trial, the control group, rather than the laser group, showed a significant improvement in the PD and CAL.[15] In this study, the laser was used twice on the experimental group at 1.5 continuous watts (CW) for 20 seconds per tooth. This exceeds the 0.4 to 0.6 CW guidelines currently recommended in periodontal therapy to avoid collateral damage.[38] This study may also have exceeded a recommended maximum continuous exposure time of 10 seconds per pocket.[52] Observing recommended settings for power, time and tip angulation is necessary to avoid collateral damage to healthy tissue, pulp and roots.[52,53] In this study,[15] the gingival fibroblasts may have been damaged by excessive heat resulting from application of too much laser energy. Another possible reason that the control group showed greater improvement than the laser group is that in the control group, the authors state that the laser was used "without activation," but were not clear whether or not they utilized the red laser guide light present on the "Zap Laser." If utilized in the control group,

the visible red low level laser guide light may have inadvertently served as an intervention yielding anti-inflammatory properties that affect PD and CAL.[49] This same study failed to state whether or not the laser fiber was changed between the experimental and control sites. If the same laser fiber was used throughout the mouth in all sites, the capillary action of the laser fiber may have facilitated transmission of pathogens between the control and experimental sites, similar to transmission of pathogens from site to site via the periodontal probe.[54,55] Failure to provide laser energy within the therapeutic treatment window in this study may explain the greater improvement of the control group over the laser group. The diode laser has been shown to stimulate fibroblasts at a low level of laser energy, yet inhibit fibroblasts at a higher level, as explained by the Arndt-Schultz curve.[56] Stimulating fibroblasts to synthesize collagen and bone is dependent on applying and regulating laser energy within the therapeutic treatment window.[45] Delivering laser energy within the therapeutic treatment window remains the challenging and sometimes elusive treatment goal. All of these variables related to laser technique can influence the outcome of clinical studies.

Practical Perspective of Laser Use

Although support for the diode laser as an adjunctive method of treating periodontal disease is controversial, some dental hygienists continue utilizing lasers.[1,3,4] Earlier barriers, including uncertainties surrounding new technology, purchase cost, expense and

Table III: Summary of States Permitting Dental Hygienists to Use Laser for Curettage

State	Yes/No	Supervision	Comment
Alaska			Curettage allowed. Use of lasers not addressed
Arizona, Colorado, Maine, Missouri Montana Nebraska, North Dakota	Yes	General	–
California , Hawaii Idaho , Michigan, Nevada	Yes	Direct	–
New Hampshire, Tennessee	Yes	–	Board ruled hygienists can use lasers if properly trained
DC, Illinois, Kansas, Maryland, New Mexico, New York, Oklahoma, Oregon, Pennsylvania, South Dakota, Utah, Washington	-	-	Not Addressed
Alabama, Arkansas, Connecticut, Delaware, Florida, Georgia, Indiana, Iowa, Kentucky, Lousiana, Massachusetts, Minnesota, Mississippi, New Jersey, North Carolina, Ohio, Rhode Island, South Carolina, Texas, Vermont, Virginia, West Virginia, Wisconson, Wyoming	No	–	–

This is intended for general informational use only. Dental Hygienists must contact their state dental board for further information permitting laser use. Provided courtesy of ADHA. Updated January 2012.

limited sources of training, are diminishing. The United States Food and Drug Administration has approved multiple lasers for clinical use.[57] A surgical diode laser can now be purchased for less than $3,500 (Zila, Inc., personal communication, February 2013). Basic laser training for both dentists and dental hygienist is readily available through laser companies, the Academy of Laser Dentistry and large continuing education venues. Dental insurance carriers, such as Delta Dental, have partnered with the California Academy of General Dentistry in sponsoring laser continuing education classes. The Academy of Laser Dentistry (ALD) recommends that laser practitioners should complete, at minimum, a Category II Standard Proficiency level certification course as described in ALD's Curriculum Guidelines and Standards for Dental Laser Education.

It has been reported that approximately 25% of dentists are using lasers, and that number is growing rapidly.[58] A 2012 article in RDH reports that the number of dentists and hygienists utilizing laser technology in private practice has doubled since 2008.[1] However, the actual number of dental hygienists utilizing lasers has not been documented. It is not currently possible to report accurately how many dental hygienists use the laser since few, if any states require a separate license for laser use. Some states have authorized dental hygienists to use the laser within their scope of practice. Other states have either prohibited laser use by hygienists, or not taken a position in either direction. Table III provides a summary of each states' position regarding laser use by dental hygienists, as reported by the American Dental Hygienists' Association.

The diode laser may have potential as an adjunctive therapy, but support for that view based on the scientific evidence is equivocal and remains controversial. Outcomes of studies are varied and often conflicting in terms of efficacy. This review identifies possible mechanisms that could have contributed to this issue: tissue response to laser therapy was demonstrated to be dependent on tissue type and health, and the successful therapeutic treatment window was shown to be specific to the target tissue, biofilm composition, laser wavelength and energy delivered. Studies have varied as to the number of times the laser was used during the course of periodontal therapy, the laser wavelength, the laser power delivered, the lasing exposure time, the study design (full mouth, split-mouth, quadrant or multi-sites), and the clinical and microbial assessments. Furthermore, few of the studies provide sufficient detail to be reproducible. The lack of standardization, varied study tissue type and health, poor study design and improper lasing technique, may be responsible for the varied end-point clinical measures that create the controversy surrounding the efficacy of laser use. Literature reviews on lasers conclude that more standardized, randomized controlled clinical trials are needed to determine if there is benefit in using lasers as an adjunct to non-surgical periodontal therapy, and if that benefit out-weighs any associated risk.[16-19,59,60] The American Academy of Periodontology (AAP) commissioned review in 2006, the First

International Workshop of Evidence-Based Dentistry on Lasers in Dentistry, as well as the AAP statement issued April 2011, have all concluded that there is a need to develop an evidence-based approach to the use of lasers for the treatment of CP.[17,20,39]

Conclusion

Although this review does not establish efficacy, this review does reveal the scientific foundation of the controversy and the need for standardized, well-designed randomized controlled clinical trials to develop specific guidelines for using the laser as an adjunct to non-surgical periodontal therapy. Using evidence-based laser guidelines would allow dental hygienists to provide more effective non-surgical periodontal care.

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