

Effect of the Clinical Application of the GaAlAs Laser in the Treatment of Dentine Hypersensitivity

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ABSTRACT

Objective: The aim of this study was to evaluate the effectiveness of the clinical use of the gallium-aluminum-arsenium (GaAlAs) laser at the maximum and minimum energies recommended by the manufacturer for the treatment of dentine hypersensitivity. **Background Data:** Dentine hypersensitivity (DH) is a response to a stimulus that would not usually cause pain in a healthy tooth. It is characterized by sharp pain of short duration from the denuded dentin. Its etiology is unknown. The dentin only begins to show sensitivity when exposed to the buccal environment. This exposure can result after removal of the enamel and/or dental cement, or after root denudation. Different treatments are proposed for this disorder. **Materials and Methods:** In this study, 25 patients, with a total number of 106 cases of DH, were treated with GaAlAs low-level laser therapy (LLLT). 65% of the teeth were premolars; 14% were incisors and molars; 6.6% were canines. The teeth were irradiated with 3 and 5 J/cm² for up to six sessions, with an interval of 72 h between each application, and they were evaluated initially, after each application, and at 15 and 60 days follow-up post-treatment. **Results:** The treatment was effective in 86.53% and 88.88% of the irradiated teeth, respectively, with the minimum and maximum energy recommended by the manufacturer. There was a statistically significant difference between DH and after a follow-up of 60 days for both groups. The difference among the energy maximum and minimum was not significant. **Conclusion:** The GaAlAs low-level laser was effective in reducing initial DH. A significant difference was found between initial values of hypersensitivity and after 60 days follow-up post-treatment. No significant difference was found between minimum (3 J/cm²) and maximum (5 J/cm²) applied energy.

INTRODUCTION

DENTINE HYPERSENSITIVITY (DH) is described clinically as an exaggerated response to a non-noxious sensory stimulus that would not usually cause a response in a healthy tooth.^{1–4} Thermal, tactile, evaporative, osmotic, and some chemical stimuli applied to the exposed dentin cause a sharp pain with short duration, but do not cause pathological alterations to the dentin–pulp complex in this disorder.^{5–7}

DH affects one in seven adult patients.⁸ The most affected age group is 20–30 years old, with no significant difference in prevalence between the sexes.^{1,8} Premolars have a larger incidence, and the cervical area of the buccal surface has a larger frequency of this disorder than other teeth.^{8,9}

Dentin, in normal conditions, is covered by enamel and cementum and is not sensitive to external stimuli. Dentin only begins to show sensitivity when exposed to the buccal atmosphere, due to the removal of the enamel layer and/or dental cementum by processes that include attrition, abrasion, and erosion, or by radicular denudation as a result of gingival recession or periodontal disease.¹⁰

Once exposed, the fine cementum layer (20–50 µm) is removed easily by periodontal scaling, abrasive toothpastes, acid and abrasive foods, and toothbrushing.¹¹ In addition, in about 10% of individuals, the enamel and the cementum do not meet, exposing an area of dentin.^{6,11}

It is known that innervation does not exist in the whole extension of the dentin and that the nerve terminations present in

the dentinal tubules are close to the pulp.¹² This characteristic supports the theory that a mechanism of indirect stimulation of the nerve terminations exists, causing the pain.

Transmission of stimuli is the theory of the movement of the dentinal fluid (or the hydrodynamic mechanism) that is most widely accepted.¹³ According to this theory, the pain results from stimulus of the nerve terminations close to the odontoblastic layer, due to the movement of the dentinal fluid in the direction of the pulp or opposite to it, depending on the nature of the stimulus.¹⁴

Several materials and different methods are used for the treatment of DH.^{15,16} For safe and effective treatment, the desensitizing agent should not be irritating to the pulp, should present easy application, should be well tolerated by the patient, should leave no dental staining, should have reasonable cost, and should promote an immediate and durable effect.¹⁷

Low-level laser therapy (LLLT) is a densitizing method that shows promise. This treatment induces alterations within the net of nerve transmission of the dental pulp, instead of altering the exposed dentinal surface, as in most other types of treatment.¹⁸ LLLT has been used for DH since the 1980s. Studies using the GaAlAs laser showed DH reduction in the range of 60–98%.^{19–25}

This study had the objective of evaluating the effectiveness of the clinical application of the gallium-aluminum-arsenium laser (GaAlAs) as well as the maximum and minimum energies, recommended by the manufacturer for the treatment of DH.

MATERIALS AND METHODS

For the accomplishment of this clinical study, patients presenting with DH were selected, and the research project was submitted to the evaluation of the Committee of Ethics of the FOSJC–UNESP.

The patients were selected independently of the presence or absence of cervical lesions such as erosions, abrasions, and abfractions, as well as gingival recessions. Patients of both sexes were admitted, most of adult age, with absence of dental mobility or presenting mobility lower than first degree.²⁴ Patients with one of the following characteristics were excluded: active periodontal disease; periodontal surgery in the last 6 months; carious lesions; extensive restorations; pulpitis; dental fractures; or chronic or weakening disease with constant episodes of pain and ingestion of daily doses of medications (e.g., analgesics, anticonvulsants, antihistamines, sedatives, tranquilizers, antidepressants, or anti-inflammatories).^{4,24,26–28}

The number of selected patients was 25 (14 females and 11 males), with ages between 14 and 58 years, presenting 106 cases of DH. These patients divided into two homogeneous groups: Group A consisted of 13 patients with mean age of 34.70 years, with 52 cases of DH. Group B had 12 patients with mean age of 34.75 years, with 54 cases of DH.

DH was evaluated by means of evaporative stimulus. Air emission from a standard air-water syringe was applied on the buccal cervical area, approximately 3 mm away from

the tooth surface, perpendicular to the cement enamel junction, for 5 sec and with constant pressure of 80 pounds. During the evaluation, the operative field was maintained without rubber dam isolation, and the adjacent teeth were protected with the fingers of the operator.^{4,7} This procedure was used to establish a baseline value at the end of each laser session and at 15 and 60 days post-treatment. Evaluation of the pain was based on the patient's subjective answer, using the Visual Analog Scale.^{4,24,25,27–31} Ordinal values from 0 to 10 are located at the opposite ends of this scale and represent "pain absence" (value 0) and "intolerable pain" (value 10). The patients were asked to indicate a value from 0 to 10 that best represented their pain level due to the air blast application. Only patients with minimum initial evaluation, of score 3, were selected.

To determine the effects of laser on DH pain, the following approach was used:

- (a) Excellent—DH reached value 0, meaning pain absence.
- (b) Good—DH reached value 1, 2, or 3, that is to say, tolerable light pain.
- (c) Unsatisfactory—DH reached value 4, 5, or 6 (moderate pain) and 7, 8, or 9 (strong pain); even so, the pain was tolerated.
- (d) Bad—Final DH was higher than the initial and the pain was not tolerated.

Treatment was considered effective in those cases where DH reached excellent or good values.

The low-level laser used as desensitizing agent for clinical application was the GaAlAs laser VR K-611,* visible in red, with density of adjustable energy of 1–15 joules/cm², continuous potential of 15-mW diode laser and 670-nm wavelength. Operator and patients used appropriate protective glasses during the laser application.^{28,30,32–35}

During irradiation, the teeth were isolated through the use of cotton rolls, and the laser was applied at the buccal cervical area, approximately 3 mm away in a perpendicular direction to the cemento–enamel junction of the committed teeth.

Each tooth of group A received 3 J/cm² for 1 min and 54 sec, while the teeth of group B were irradiated with 5 J/cm² for 3 min and 10 sec, periods pre-determined by the manufacturer. Only the maximal number of six applications was reached when needed, with an interval of 72 h between each irradiation.

The DH value was verified and registered at the beginning of the treatment (baseline values) and after each application. The initial DH values for both groups (baseline A and B) had been admitted as a control value, and further used for comparison of therapy effectiveness on DH reduction, as a subjective evaluation, control maintenance would be subjected to a lower variation of pain interpretation. DH was logged at the end of six applications, and two follow-ups were accomplished after 15 and 60 days following termination of laser therapy.

One of two different events determined treatment ending: (a) when/if the patient reached the zero value in the Visual Analog Scale, independent of the number of applications to which the patient had been submitted; or (b) after the sixth application, the maximum number recommended by

TABLE 1. MEAN VALUES AND STANDARD DEVIATION OF THE VALUES OF DENTINAL HYPERSENSITIVITY TO THE AIR BLAST CALCULATED ON THE VISUAL ANALOG SCALE WITH RELATIONSHIP TO THE DIFFERENT EVALUATION PERIODS OF THE MINIMUM (3 J/cm²) AND MAXIMUM (5 J/cm²) LASER ENERGY APPLIED

<i>Period of evaluation</i>	<i>Group A, 3 J/cm²</i>		<i>Group B, 5 J/cm²</i>	
	<i>M</i>	<i>(SD)</i>	<i>M</i>	<i>(SD)</i>
Baseline	7.57	(2.51)	7.22	(2.51)
1 st session	5.34	(3.10)	5.40	(3.12)
2 nd session	3.98	(2.86)	4.05	(2.87)
3 rd session	3.01	(2.81)	3.40	(2.85)
4 th session	2.30	(2.49)	3.01	(2.91)
5 th session	1.78	(2.26)	2.18	(2.76)
6 th session	1.34	(2.12)	1.72	(2.36)
15 days follow-up	1.30	(1.96)	1.20	(1.83)
60 days follow-up	1.44	(2.20)	1.18	(1.97)

the manufacturer, even though DH had not reached the zero value.

The laser's effectiveness was verified with Wilcoxon test for paired samples (at 5% significance), comparing initial DH and DH at 60 days post-treatment, for both groups. Statistical analysis was performed using the Mann-Whitney test to verify the comparison among the minimum and maximum energy recommended by the manufacturer, (at 5% significance).

RESULTS

A total of 25 patients were analyzed in this study, with 106 hypersensitive teeth, which resulted in an average of 4.24 teeth per patient. The most frequently affected teeth were the premolars (65.09%), followed by incisors and molars (14.15%) and canines (6.60%).

After laser irradiation of the teeth in group A (3 J/cm²), there was a total pain regression to the zero value in 57.69% and regression to light pain in 28.84% of cases. In group A, we verified DH regression up to 86.53%. DH was invariable in 13.45%, because there was a regression to moderate pain in 9.61% and to strong pain in 3.84% of cases; the sensitivity experienced by these patients was tolerable.

After laser irradiation of the teeth in group B (5 J/cm²), we verified that the treatment was effective in 88.88% of the cases. There was total pain regression to the zero value in 57.40% and regression to tolerable light pain in 31.48% of cases.

In this group, DH remained invariable in 11.11% of cases, because there was regression to moderate pain in 7.40% and to tolerable strong pain in 3.70% of the cases; the sensitivity experienced by these patients was once again tolerable.

Among the 52 teeth treated in group A, there was pain recurrence in five cases (9.61%), and among the 54 teeth treatments in group B, there was pain recurrence in only two

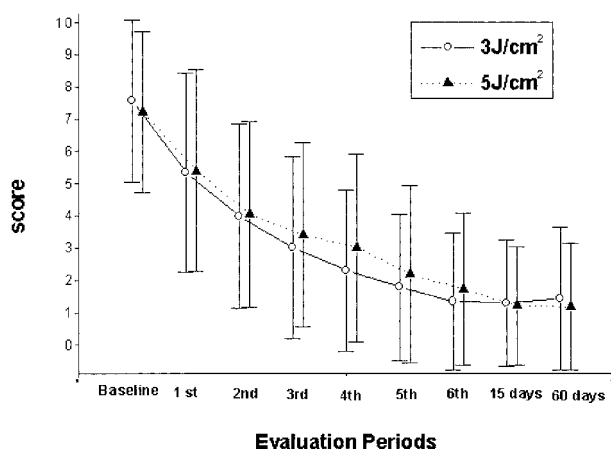


FIG. 1. Mean values and standard deviation of the values of dentinal hypersensitivity calculated on the Visual Analog Scale with relationship to the different evaluation periods of the minimum (3 J/cm²) and maximum (5 J/cm²) laser energy applied.

cases (3.70%); both occurred 60 days after the end of the treatment.

The averages and deviation patterns determined among the DH values obtained in response to air blast of the groups A and B are presented in Table 1 and illustrated in Figure 1.

Figure 2 shows the comparison among averages of initial DH and DH at 60 days after treatment conclusion for groups A and B. We obtained a statistically significant difference between these two periods, which confirmed the effectiveness of the applied laser.

The results of the statistical analysis on the difference between the minimum and maximum energy used in the treatment are presented in Figure 3; no significant difference between both groups was found.

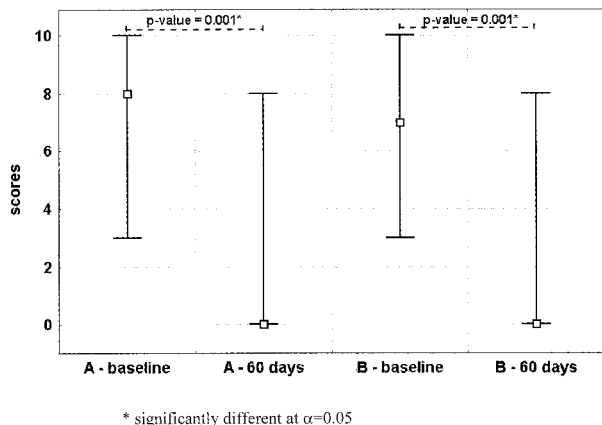


FIG. 2. Wilcoxon test ($\alpha = 0.05$) for paired samples for verification of the effectiveness of the minimum (A) and maximum (B) laser energy between the baseline values of the dentinal hypersensitivity and values at 60 days follow-up.

DISCUSSION

In our study, the mean age of subjects was 34.7 years, similar to the age range of the study by Flynn et al.,¹ whose patients presented a mean age of 34 years. Among our 25 evaluated patients, an average of 4.24 teeth was diagnosed per patient, also observed by Orchardson and Collins,⁹ who found in their study an average of four DH teeth per patient. The premolars were affected by DH with larger frequency as per Graf and Galasse⁸ and Orchardson and Collins,⁹ who also observed a larger incidence of these teeth in their studies. In our study, all teeth presented DH in the coronary buccal cervical surface, as was found in works of Flynn et al.,¹ Graf and Galasse,⁸ and Orchardson and Collins.⁹

In the literature discussed, two main forms of treatment—tubular occlusion and reduction of sensorial nervous activity—were found.^{14,15,36–40}

Several mechanisms are proposed to explain the effects of LLLT on DH; however, in spite of the vast literature, information on the neurophysiologic mechanism was not conclusive.^{41,42}

Our results are in agreement with those observed by Wakabayashi and Matsumoto,¹⁹ who verified effectiveness in the DH treatment with GaAlAs laser in 98% of the cases, confirming that LLLT was an effective desensitizing agent. Furuoka et al.²⁰ demonstrated the effectiveness of DH treatment with GaAlAs laser in 92% of cases, with few recurrent signs. Kumazaki et al.,²¹ using a semiconductor GaAlAs laser, observed an effectiveness of 69.2% in the laser-treated group in comparison to the placebo group, with a statistically significant difference among groups. Yamaguchi et al.²² verified effectiveness of the low-level GaAlAs laser in 60% in the control group and 22.2% in the placebo group, which indicates that the laser can be effective in DH pain reduction.

The decrease in DH observed in our study agrees with the results obtained by Groth,²³ who showed DH reduction with the use of a low-level GaAlAs laser.

Gerschman et al.²⁴ observed that thermal and tactile sensitivity was reduced, respectively in 67% and 65%, when the

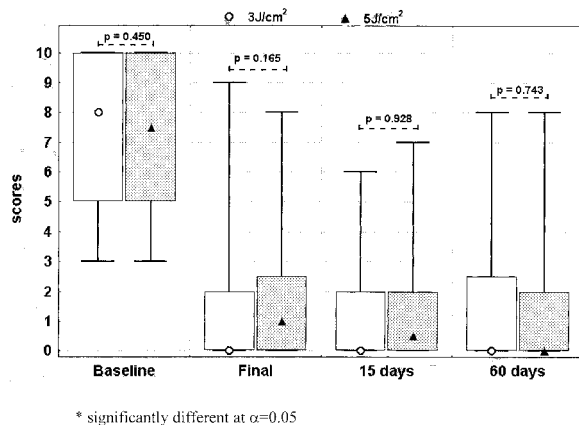


FIG. 3. Mann-Whitney test ($\alpha = 0.05$) for verification of the difference between the minimum (3 J/cm²) and maximum (5 J/cm²) laser energy used in the treatment according to evaluation periods.

GaAlAs laser was applied to patients with hypersensitive teeth, demonstrating that the laser was an effective treatment method for this condition. The authors affirmed that, while the mechanism of the low-level laser on DH remained uncertain, the results follow the line of most recent research in the area, suggesting modification in neuronal activity.

Liu and Lan²⁵ also found that treatment of HD with the GaAlAs semiconductor diode laser was effective. These authors verified reduction of sensitivity to air blast and a mechanical stimulus, respectively, in 70% and 72% of their cases.

Brugnera Júnior et al.⁴⁴ showed that younger patients, with better dentinal and pulp conditions, tend to recover more rapidly during laser treatment.

Gomi et al.⁴⁵ verified that the increase in effectiveness of the treatment is directly proportional to the increase in the number of applications. Similar results were also found in our study, because the averages of the DH values of groups A and B reduced gradually as the number of applications increased.

As in our study, Senda et al.⁴⁶ did not observe increase of pain or collateral effects after laser treatment in any of the treated cases.

Similar to the results of our study, Wakabayashi and Matsumoto¹⁹ showed a lower effectiveness of the GaAlAs laser when the teeth presented strong and intolerable pain in response to applied stimulus, as well as a larger recurrence of hypersensitivity.

Analyzing the cases where there was recurrence, we noticed in most of our cases that initial pain was at the highest value of the Visual Analog Scale, which was also found in other studies.^{19,33} The comparison among the different applied energies was not statistically significant. We verified, in this study, that painful sensitivity in response to an applied stimulus exhibited great variation, not only among patients, but also within a patient. Each tooth reacted to the treatment in a unique way, making it impossible to establish a pattern. It is important to note good patient acceptance of the laser treatment in this and in other studies found in the literature.^{25,47,48}

We observed, in our literature review, no data on the long-term effects of LLLT for DH; therefore, more studies evaluating this new treatment modality are necessary.

CONCLUSION

The initial DH was reduced after treatment with the low-level GaAlAs laser. The difference between initial DH and DH at 60 days post-treatment was statistically significant. The difference among the minimum (3 J/cm²) and maximum (5 J/cm²) energies recommended by the manufacturer was not statistically significant.

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REFERENCES

1. Flynn, J., Galloway, R., and Orchardson, R. (1985). The incidence of "hypersensitive" teeth in the West of Scotland. *J. Dent.* 13, 230–236.
2. Bissada, N.F. (1994). Symptomatology and clinical features of hypersensitive teeth. *Arch. Oral Biol.* 39, suppl., 31S–32S.
3. Curro, F.A. (1990). Tooth hypersensitivity in the spectrum of pain. *Dent. Clin. North Am.* 34, 429–437.
4. Plagmann, H.C., Konig, J., Bernimoulin, J.P., et al. (1997). A clinical study comparing two high-fluoride dentifrices for the treatment of dentinal hypersensitivity. *Quintessence Int.* 28, 403–408.
5. Abel, I. (1958). Study of hypersensitivity teeth and a new therapeutic aid. *Oral Surg. Oral Med. Oral Pathol.* 11, 491–495.
6. Trowbridge, H.O., and Silver, D.R. (1990). A review of current approaches to in-office management of tooth hypersensitivity. *Dent. Clin. North Am.* 34, 561–581.
7. Kleinberg, I., Kaufman, H.W., and Wolff, M. (1994). Measurement of tooth hypersensitivity and oral factors involved in its development. *Arch. Oral Biol.* 39, 63S–71S.
8. Graf, H., and Galasse, R. (1977). Morbidity, prevalence and intra-oral distribution of hypersensitive teeth. *J. Dent. Res.* 56, 230–236 (abst).
9. Orchardson, R., and Collins, W.J.N. (1987). Clinical features of hypersensitive teeth. *Br. Dent. J.* 162, 253–256.
10. Dowell, P., Addy, M., and Dummer, P. (1985). Dentine hypersensitivity: aetiology, differential diagnosis and management. *Br. Dent. J.* 158, 92–96.
11. Wichgers, T.G., and Emert, R.L. (1996). Dentine hypersensitivity. *Gen. Dent.* 44, 225–230.
12. Brännström, M. (1963). Dentin sensitivity and aspiration of odontoblasts. *J. Am. Dent. Assoc.* 66, 366–370.
13. Brännström, M., and Åström, A. (1964). A study on the mechanisms of pain elicited from dentin. *J. Dent. Res.* 43, 618–625.
14. Pashley, D.H. (1986). Dentin permeability, dentin sensitivity and treatment through tubule occlusion. *J. Endodont.* 12, 465–474.
15. Zappa U. (1994). Self-applied treatments in the management of dentine hypersensitivity. *Arch. Oral Biol.* 39, 107S–112S.
16. Gangarosa, L.P. (1994). Current strategies for dentist-applied treatment in the management of hypersensitive dentine. *Arch. Oral Biol.* 39, 101S–106S.
17. Grossman, L.I. (1935). A systematic method for the treatment of hypersensitivity dentin. *J. Am. Dent. Assoc.* 22, 592–602.
18. Karu, T. (1989). Photobiology of low-power laser effects. *Health Phys.* 56, 691–704.
19. Wakabayashi, H., and Matsumoto, K. (1988). Treatment of dentine hypersensitivity by GaAlAs soft laser irradiation. *J. Dent. Res.* 67, 182(abst).
20. Furuoka, M., Yokoi, T., Fukuda, S., et al. (1988). Effects of GaAlAs laser diode in treatment of hypersensitive dentine. *J. Fukuoka Dent. Coll.* 15, 42–48.
21. Kumazaki M., Zennyu, K., Inoue, M., et al. (1990). Clinical evaluation of GaAlAs—semiconductor laser in the treatment of hypersensitive dentin. *Jpn. J. Conserv. Dent.* 33, 911–918.
22. Yamaguchi, M., Ito, M., Miwata, T., et al. (1990). Clinical study on the treatment of hypersensitive dentin by GaAlAs laser diode using the double-blind test. *Aichi Gakuin Daigaku Shigakkai Shi.* 28, 703–707.
23. Groth, E.B. (1993). Contribuição para o estudo da aplicação do laser de baixa potência de GaAlAs no tratamento da hipersensibilidade dentinária [Master's thesis]. Faculdade de Odontologia, Universidade de São Paulo.
24. Gerschman, J.A., Ruben, J., Gebart-Eaglemon, J. (1994). Low-level laser therapy for dentinal tooth hypersensitivity. *Aust. Dent. J.* 39, 353–357.
25. Liu, H.C., Lan, W.H. (1994). The combined effectiveness of the semiconductor laser with Duraphat in the treatment of dentin hypersensitivity. *J. Clin. Laser Med. Surg.* 12, 315–319.
26. Collins, J.F., Perkins, L. (1984). Clinical evaluation of the effectiveness of three dentifrices in relieving dentin sensitivity. *J. Periodontol.* 55, 720–725.
27. Clark, G.E., Troullos, E.S. (1990). Designing hypersensitivity clinical studies. *Dent. Clin. North Am.* 34, 531–544.
28. Gelskey, S.C., White, J.M., Pruthi, V.K. (1993). The effectiveness of the Nd:YAG laser in treatment of dental hypersensitivity. *J. Can. Dent. Assoc.* 59, 377–386.
29. McGrath, P.A. (1986). The measurement of human pain. *Endodont. Dent. Traumatol.* 2, 124–129.
30. Holland, G.R., Narhi, M.N., Addy, M., et al. (1997). Guidelines for the design and conduct of clinical trials on dentine hypersensitivity. *J. Clin. Periodontol.* 24, 808–813.
31. West, N.X., Addy, M., Jackson, R.J., et al. (1997). Dentine hypersensitivity and the placebo response. A comparison of the effect of strontium acetate, potassium nitrate and fluoride toothpastes. *J. Clin. Periodontol.* 24, 209–215.
32. Wilder-Smith, P. (1988). The soft laser: therapeutic tool or popular placebo? *Oral Surg. Oral Med. Oral Pathol.* 66, 654–658.
33. Iwase, T., Nara, Y., Morioka, T. (1990). Relieving pain in patients with hypersensitive dentine by He-Ne laser. *Surg. Med. Lasers* 3, 193–195.
34. Miller, M., Truhe, T. (1993). Lasers in dentistry: an overview. *J. Am. Dent. Assoc.* 124, 33–35.
35. Walsh, L. J. (1997). The current status of low-level laser therapy in dentistry. Part 2. Hard tissue applications. *Aust. Dent. J.* 42, 302–306.
36. Kim, S. (1986). Hypersensitivity teeth: desensitization of pulpal sensory nerves. *J. Endodont.* 12, 482–485.
37. Kleinberg, I. (1986). Dentinal hypersensitivity. Part I: The biologic basis of the condition. *Compend. Cont. Educ. Dent.* 7, 182–187.
38. Rosenthal, M.W. (1990). Historical review of the management of tooth hypersensitivity. *Dent. Clin. North Am.* 34, 403–427.
39. Addy, M., West, N. (1994). Etiology, mechanisms and management of dentine hypersensitivity. *Curr. Opin. Periodontol.* 71–77.
40. Siqueira Junior, J.F. (1994). Hipersensibilidade dentinária. Visão atual dos mecanismos envolvidos e medidas terapêuticas. *Rev. Bras. Odontol.* 51, 55–58.
41. Ribeiro, M.S. (1999). Lasers de baixa intensidade. Presented at USP/FDCTO, IPEN/CNEN, Workshop—Utilização Clínica do Laser, São Paulo.

42. Wakabayashi, H., Hamba, M., Matsumoto, K., et al. (1993). Effect of irradiation by semiconductor laser on responses evoked in trigeminal caudal neurons by tooth pulp stimulation. *Lasers Surg. Med.* 13, 605–610.
43. Pashley, D.H. (1990). Mechanisms of dentin sensitivity. *Dent. Clin. North Am.* 34, 449–473.
44. Brugnera Junior, A., Cruz, F.M., Zanin, F. (1998). Dentinary hypersensibility treatment with low-level laser therapy. Presented at the International Congress on Lasers in Dentistry, Maui.
45. Gomi, A., Kameya, K., Yamashita, H., et al. (1986). A clinical study on “Soft Laser 632,” a He-Ne low energy medical laser. 2: The effects in relieving the pain of hypersensitive dentin and the pain during seating inlay. *Aichi Gakuin Daigaku Shigakkai.* 24, 390–399.
46. Senda, A., Gomi, A., Tomi, T., et al. (1985). A clinical study on “Soft Laser 632,” a He-Ne low energy medical laser. 1: The effects in relieving pain just after irradiation. *Aichi Gakuin Daigaku Shigakkai Shi.* 23, 773–780.
47. Smith, T.A., Thompson, J.A., Lee, W.E. (1991). Patient response to dental laser treatment; a preliminary report. *CDA J.* 19, 37–41.
48. Pick, R.M. (1993). Using lasers in clinical dental practice. *J. Am. Dent. Assoc.* 124, 37–47.

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