



LASERS IN DENTISTRY

Guide for Clinical Practice

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[Figure 15.1 Different stages and prognosis of caries lesions: \(a\) cavitated caries lesion – more prone to cause pulpal damages and to progress \(b\) initial active caries lesions: sign of dental caries present – need intervention to be arrested \(c\) initial inactive caries lesions: past sign of dental caries – do not need intervention to be arrested.](#)

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[Figure 15.6 Innovation in the use of the LFpen: examination of approximal surfaces.](#)

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Chapter 16

[Figure 16.1 Scanning electron micrograph of the enamel surface after laser–fluoride treatment. Note the calcium fluoride-like deposits.](#)

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Chapter 17

[Figure 17.1 Light transmission through increasing concentrations of methylene blue in a 96-well plate \(300 µL per well\). At higher concentration \(150 µM\), light is absorbed at the surface and does not penetrate the PS volume.](#)

[Figure 17.2 Light distribution during irradiation performed inside an extracted human molar \(a\) with the laser tip at the pulp chamber, \(b\) with the optical fiber inside the root canal. Note that with the use of an optical fiber the amount of light, especially in the apex region, is higher than when no fiber is used.](#)

[Figure 17.3 \(a\) Conventional endodontic treatment, \(b\) after chemomechanical treatment, the root canal was filled with 1 M \(3% or 10 vol.\) hydrogen peroxide for 1 minute, \(c\) root canal filled with 60 µM methylene blue. Note the PS concentration \(color\) used for endodontic aPDT, \(d\) irradiation with a 660-nm diode laser coupled with an optical fiber.](#)

[Figure 17.4 \(a\) Clinical and radiographic image of the lesion, \(b\) a full mucoperiosteal flap, allowing access to the lesion, \(c\) after the conventional procedure, the cavity was filled with a sterile aqueous solution of methylene blue for 3 minutes, \(d\) irradiation with the laser tip covering all cavity surfaces, \(e\) the retrograde cavity was also filled with the PS solution for 1 minute, \(f\) to irradiate the cavity, the laser was coupled to an optical fiber, \(g\) radiographic images immediately after the surgery \(left\) and 6 months later \(right\). Note the healing of the lesion compared to the initial radiographic exam \(courtesy of Debora Parra Sellera\).](#)

Chapter 18

Figure 18.1 (a) Initial clinical image. Note the presence of the fistula on the mucosa of the apical region of tooth 11. (b) Initial radiograph showed a large periapical lesion. (c) After the incision (handpiece 2062, 50/10 fiber of the KAVO KEY II) and removal of the flap, the erbium laser defined the area of the cortical bone defect to be removed. (d) The Er:YAG laser (handpiece 2060, 350 mJ/4 Hz, KAVO KEY) was used to perform the apicoectomy. (e) Nd:YAG laser irradiation (1.5 W, 100 mJ, 15 Hz; 124 J/cm²) on the cut dentin surface after apicoectomy promoted sealing of the dentinal tubules. (f) Disinfection of the periapical region with Nd:YAG laser irradiation (1.5 W, 100 mJ, 15 Hz; 124J/cm²). (g) Radiographic imaging 1 month after apicoectomy of tooth 11, (h) 6 months after apicoectomy shows a decrease in the periapical radiolucent area, indicating bone repair, and (i) 3 years after apicoectomy shows a significant decrease in the periapical radiolucent area. (j) Radiographic imaging 5 years after apicoectomy showed bone healing, albeit with the presence of scar tissue.

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Chapter 19

Figure 19.1 (a) Presence of a large restoration cavity on the distal aspect of the tooth. (b) Presence of a fistula at the buccal mucosa. (c) Radiograph image showing a large, irregular radiolucency around the tooth apex of the upper right lateral incisor. (d) Root canal opening was worn progressively using a #3 ultrasonic tip on the pulp chamber walls. (e) Abundant exudate aspirated throughout root canal negotiation. (f) Root canal was dried with sterile paper points and filled. (g) Follow-up radiograph at 90 days.

Figure 19.2 (a) Initial periapical radiograph. (b) Computed tomographic images depicting details of the apical inflammation^{16,17} (Distel et al. 2002. Reproduced with permission of Elsevier). (c) Clinical image depicting details of the esthetics of the incisors. (d) Transillumination image depicting details of the crown microfractures. (e) Postoperative radiograph. After cleaning and shaping, the root canal was irradiated with a high power diode laser set to operate in continuous mode at 2 W with a 400- μ m optical fiber applied to the root canal surface in four applications of 5 seconds each, with a 20-second interval between applications^{18,19} (Garbuz et al. 2008. Reproduced with permission of Elsevier). (e) Abundant irrigation of the canal was performed with 2.5% NaOCl during the intervals, and the irrigant was maintained in the canal during the subsequent applications. Finally, the canal was dried with sterile paper points and filled. (f) Radiograph at 90-day follow-up (g) Radiograph at 180-day follow-up.

Figure 19.3 (a) Initial periapical radiograph. (b) Emptying of the pulp

chamber, and start of the cleaning and shaping phase. (c) Root canal preparation with 1% NaOCl and PTC gel. (d) Instruments and chemical substances. (e) DMC Thera Lase Surgery. (f) 400- μ m optical fiber and #30K-file.

Chapter 20

Figure 20.1 (a) Alteration of the contour of the marginal gingiva caused by orthodontic movement; (b) surgical gingivectomy performed with a CO₂ laser; (c) seven days and (d) 30 days after the operation.

Figure 20.2 (a) Increased gingival volume covering the end of the coronal preparation; (b) surgery for the enlarged clinical crown using the Er:YAG laser (2940 nm; 400 mJ; 10 Hz); (c) immediate clinical appearance and (d) seven days after the operation.

Figure 20.3 (a) Fibroid located in the interdental papilla; (b) excisional biopsy performed with the Er:YAG laser (400 mJ/10 Hz); (c) immediate clinical appearance and (d) seven days after the operation.

Figure 20.4 Electron photomicrography of the root surface irradiated with an Er:YAG laser set at 100 mJ and 10 Hz (original magnification 2000 \times).

Figure 20.5 Electron photomicrography of the root surface irradiated with an Er,Cr:YSGG at a power setting of 1.5 W

Figure 20.6 Fiber of the diode laser positioned in the periodontal pocket during decontamination of the periodontal pocket.

Figure 20.7 Fiber of the Er:YAG laser positioned in the periodontal pocket during root surface scaling.

Figure 20.8 Root surface irradiated with an Er,Cr:YSGG laser at a power setting of 1.5 W (arrows) (original magnification, 5 \times)

Chapter 21

Figure 21.1 Photomicrographs illustrating the areas of bone loss in the furcation regions of the mandibular first molars in induced periodontal disease in ovariectomized rats: (a) No aPDT treatment at day 7 and (b) day 15 post scaling and root planing (SRP) (c) aPDT treatment at day 7 and (d) day 15 post treatment (SRP plus aPDT) (original magnification 100 \times). The sections were stained with hematoxylin and eosin (H&E).

Figure 21.2 Photomicrograph illustrating the areas of bone loss in the furcation regions of the mandibular first molars in induced periodontal disease in rats: (a) SRP treatment at day 15 post treatment in a non-diabetic animal (b) SRP treatment at day 15 post treatment in a diabetic animal (c) aPDT treatment at day 7 and (d) day 30 post treatment in a diabetic animal (original magnification 100 \times). The sections were stained with hematoxylin and eosin (H&E).

Figure 21.3 (a–d) Initial clinical images and radiographs of a case of localized aggressive periodontitis in a 27-year-old female patient who

displayed a periodontal pocket with angled bone loss in the region of teeth 16 and 26. (e) Irrigation of the periodontal pocket with methylene blue solution; (f) irradiation with a low power laser for 133 seconds (660 nm, 57.14 J/cm²).

Figure 21.4 Application of aPDT during surgical periodontal therapy with the performance of a periodontal flap in areas of chronic periodontal disease. (a) Initial radiograph demonstrating bone loss in the molar region; (b) irrigation with methylene blue solution after scaling and root planing in an open field; (c) irradiation with a low power laser for 133 seconds (660 nm, 57.14 J/cm²); (d) immediately post operation (courtesy of JM Almeida).

Figure 21.5 Application of aPDT during surgical regenerative periodontal therapy. (a) Chronic periodontal disease in the region of the left upper molars; (b) initial periapical radiograph demonstrating angled bone loss; (c) the periodontal flap and a view of an affected furcation area; (d) irrigation with methylene blue solution after scaling and root planing (courtesy of Almeida JM).

Figure 21.6 Application of aPDT during surgical regenerative periodontal therapy. (a) Irradiation of the photosensitized area with a low power laser for 133 seconds (660 nm, 57.14 J/cm²); (b) a collagen membrane in place; (c) the repositioned and sutured flap; (d) clinical image 180 days post surgery; (e) initial radiograph compared with; (f) the radiograph 180 days post surgery (courtesy of Almeida JM).

Chapter 22

Figure 22.1 The Er:YAG laser equipment (Kavo Key Laser II; Kavo, Germany) with handpiece 2056 and chisel tip with a rectangular end of 1.65 × 0.5 mm and transmission factor of 64%.

Figure 22.2 (a) Preoperative appearance of moderate gingival melanin pigmentation. (b) Irradiation of the maxillary gingiva. Note the chisel tip in oblique contact mode. The laser beam is applied using the “brush technique” as described by Tal et al.¹⁶ (c) Immediately after irradiation of the maxillary gingiva. Note the precise and effective ablation of gingival pigmentation without any marked coagulation or carbonization on the treated surface. The papillary edges and free gingival margins were not irradiated. (d) Three months after treatment. Note the significant decrease in gingival melanin pigmentation of the maxillary and mandibular gingiva without any recurrence, or gingival recession or deformity.

Figure 22.3 (a) Preoperative appearance of moderate gingival melanin pigmentation. (b) Immediately after irradiation of maxillary gingiva without any marked coagulation or carbonization on the treated surface. (c) Twenty-four hours after treatment of the gingiva. (d) Forty-eight hours after treatment of the gingiva. (e) Two weeks after treatment, the gingiva shows almost complete healing. (f) One month after treatment, there is significant reduction of gingival melanin pigmentation of the

maxillary and mandibular gingiva without recurrence or gingival recession or deformity.

Chapter 23

Figure 23.1 (a) Abnormal inferior labial frenum that is causing retraction of the gingival margin. (b) Clinical image immediately after the irradiation and (c) at 3 weeks follow-up.

Figure 23.2 (a) Abnormal inferior labial frenum that is causing retraction of the gingival frenum. (b) Clinical image immediately after irradiation.

Figure 23.3 (a) Patient who presented with a 6-month history of retention of tooth 23. (b) Clinical picture immediately after irradiation.

Chapter 24

Figure 24.1 (a) Implant placement (b) transducer fitted intraorally (c) point distribution of infrared laser irradiation per implant .

Figure 24.2 Postoperative infrared laser irradiation of dental implants.

Figure 24.3 Second-stage surgery using a CO₂ laser for (a) implant uncovering and (b) placement of a sulcus former. (c) Excellent wound healing 2 weeks after surgery.

Figure 24.4 Proposed flap design. Occlusal view of the severe vestibular bone loss often responsible for unsatisfactory results following prosthetic rehabilitation and thus this case is a candidate for mucogingival surgery to restore lost volume .

Figure 24.5 Comparison of postoperative healing after second-stage implant surgery with that after use of the cold scalpel, based on the conventional technique versus the Er,Cr:YSGG laser. Upper panel: 1 week after surgery; lower panel: 2 weeks after surgery .

Figure 24.6 General surgical technique. (a,b) Design and raising of the trapezoidal flap, exposing the cover screw of the implant (c) substitution with the healing abutment: the flap rests on the latter to allow healing by secondary intention (d) 1 week after surgery; (e,f) appearance of the newly formed papilla on removal of the healing abutment: vestibular and occlusal views (g) rehabilitation using an esthetic zirconium post (h) final result .

Figure 24.7 Rehabilitation of a right superior molar. (a) Trapezoidal incision with the Er,Cr:YSGG laser (b) flap and the cover screw, (c) insertion of the healing abutment (d) use of a rolling flap to gain volume in the affected area. Note that after preparation and before placement of the flap folded onto itself, the external surface was vaporized with the laser to secure de-epithelialization; (e,f) healing 1 week after surgery .

Figure 24.8 Rehabilitation of a left superior lateral incisor. (a) Preoperative appearance, showing important vestibular bone loss secondary to a chronic infection; (b,c) use of a rolling flap to gain volume in the affected area. Note that after preparation and before placement of

the flap folded onto itself, the external surface was vaporized with the Er,Cr:YSGG laser to secure de-epithelialization (d) healing 1 week after surgery; (e,f) occlusal and vestibular view of the peri-implant soft tissue contour achieved (g) final result .

Figure 24.9 (a) Computerized tomograph showing thin lateral wall and low bone height of right maxillary sinus (b) window osteotomy with the Er,Cr:YSGG laser (c) implant placement and simultaneous sinus grafting (d) a bony window was repositioned over the bone graft as a barrier membrane to prevent ingrowth of soft tissue into the sinus cavity (e) allograft and implants placed; – (f) postoperative panoramic radiograph; – (g,h) placement of the final restorations 7 months after surgery and periapical X-ray control . Figure 24.9.(continued)

Figure 24.10 Advanced bone loss due to peri-implantitis, with a circumferential crater defect.

Figure 24.11 Cleaning of the abutments using a special ultrasonic device with a plastic tip.

Figure 24.12 (a) Peri-implant bone loss at element 34 (b) peri-implant infrabony defect (c) decontamination of the implant surface using a CO₂ laser (d) augmentation with mineral bovine bone (e) radiological fill 4 years after treatment.

Figure 24.13 (a) Clinical and (b) radiographic view of a patient with peri-implantitis at elements 13, 11, 21, and 23 (c) exposition of the implants due to bone loss. (d) After mechanical debridement of the implant surfaces, (e) methylene blue dye was applied for 5 minutes, and then (f) LPL irradiation was performed for aPDT.

Figure 24.14 Implant irradiation using a Nd:YAG (2W pulsed) laser achieves significant melting of the implant surface.

Figure 24.15 No modification of the implant surface after the use of a 4-W, continuous wave CO₂ laser (right) in comparison with the non-irradiated surface (left).

Figure 24.16 No changes to the implant surface after irradiation with a continuous wave diode laser (980 nm, 10 W).

Chapter 25

Figure 25.1 (a) The decorticated receptor site. (b) Before the fixation of the graft, the receptor bed is irradiated with infrared light (visible light is shown for the purpose of illustration). (c,d) The grafted site immediately after surgery. Following fixation, the blocks are irradiated with infrared light for 15 days. (e,f) The grafts at 9 weeks; already incorporated and ready to receive the implants. (g,h) Histology of the newly formed bone around the threads of the implants at 30 days after insertion. Note that the bone is mature and in close association with the implants.

Figure 25.2 (a) Clinical image of the implants inserted in the jaw. (b) Before suturing, the implanted area is irradiated with infrared laser light

at points along the length of the implant and at the occlusal surface. (c) The soft tissues are also irradiated with visible red light to improve wound healing. After surgery, irradiation with infrared light is carried out for 15 days.

Chapter 26

Figure 26.1 Lymphatic system comprised of lymph, vessels, and lymphatic organs (tonsils, thymus, lymph nodes, and spleen).

Figure 26.2 Some palpable lymph node chains used in LLLT for lymphatic drainage.

Figure 26.3 Surface parotid lymph nodes and the areas they drain, as well as the direction of this drainage.

Figure 26.4 (a,b) Application of the lymphatic drainage technique with LLLT on the surface parotid lymph nodes.

Figure 26.5 Little observed facial lymph nodes: nasolabial, zygomatic, buccinator, and mandibular. The submandibular and surface cervical lymph nodes are also shown.

Figure 26.6 (a,b) Application of the lymphatic drainage technique with LLLT on the facial lymph nodes.

Figure 26.7 Lingual and deep lower cervical lymph nodes.

Figure 26.8 (a,b) Application of the lymphatic drainage technique with LLLT on the lingual lymph nodes.

Figure 26.9 Submental and submandibular lymph nodes.

Figure 26.10 (a,b) Application of the lymphatic drainage technique with LLLT on the submental lymph nodes.

Figure 26.11 Surface and preauricular, submental, submandibular, and cervical lymph nodes.

Figure 26.12 (a,b) Application of the lymphatic drainage technique with LLLT on the submandibular lymph nodes.

Figure 26.13 Deep parotid and surface cervical lymph nodes.

Figure 26.14 (a,b) Application of the lymphatic drainage technique with LLLT on the surface cervical lymph nodes.

Figure 26.15 Panoramic view of the main head and neck surface lymph nodes, including the surface cervical lymph nodes.

Figure 26.16 (a,b) Application of the lymphatic drainage technique with LLLT on the surface cervical lymph nodes.

Figure 26.17 Detection of altered lymph nodes: Digital palpation.

Chapter 27

Figure 27.1 Points of laser application for the TMJ: (a) above, (b); anterior, and (c) posterior to the condyloid process; (d) an intra-auricular point towards the joint.

[Figure 27.2 Possible points of laser application for the masseter and temporalis muscles.](#)

[Figure 27.3 Case study: irradiated points.](#)

Chapter 28

[Figure 28.1 Orthodontic movement: –, pressure side – osteoclast activation; +, tension side – osteoblast activation.](#)

[Figure 28.2 Points of laser application. The same points were made on the lingual side, except for in the study of Lim et al.¹² in which the laser was only applied at buccal points. \(a\) Cruz et al.,²⁰ Sousa et al.,¹⁹ Angelieri et al.,⁷¹ Tortamano et al.,¹⁵, Genc et al.,⁹³ and Doshi-Mehta and Bhad-Patil²²; \(b\) Youssef et al.¹⁴; \(c\) Camacho and Cujar²¹ and Turhani et al.¹³; \(d\) Lim et al.¹²; \(e\) Artés-Ribas et al.¹⁷; \(f\) Bicakci et al.¹⁶ and Esper et al.⁷²; \(g\) Limpanichkul et al.¹⁰](#)

[Figure 28.3 Points of laser application. \(a\) Cepera et al.⁸⁷ for rapid maxillary expansion \(RME\); \(b\) Abreu et al.⁹⁰ for surgically assisted rapid maxillary expansion \(SARME\).](#)

Chapter 29

[Figure 29.1 \(a\) Visible red laser application all over the injured area. \(b\) Laser \(infrared\) acupuncture points. \(c\) Treatment evolution over 10 days.](#)

[Figure 29.2 Roots of the third molar in relation to the inferior alveolar nerve.](#)

[Figure 29.3 Proposed sites of laser application.](#)

[Figure 29.4 Paresthesia in implantology.](#)

[Figure 29.5 Acupuncture points.](#)

Chapter 30

[Figure 30.2 \(a\) Typical aspects of an IFH, located in the anterior buccal sulcus. \(b\) Clinical postoperative aspect 7 days following laser surgery. \(c\) Complete healing observed 15 days following surgery. No scarring was noted.](#)

[Figure 30.1 \(a\) Palatal squamous papilloma, with a typical warty surface. \(b\) Clinical aspect immediately following laser surgery. \(c\) Complete healing observed 07 days following surgery. No scarring was noted.](#)

Chapter 31

[Figure 31.1 \(a\) Vascular lesion on the right of the lower lip. \(b\) Clinical image immediately after photocoagulation. \(c\) After 4 weeks of follow-up.](#)

[Figure 31.2 \(a\) Lymphangioma on the labial mucosa on the left of the upper lip. \(b\) Clinical image immediately after vaporization. \(c\) After 4 weeks of follow-up.](#)

Chapter 32

Figure 32.1 (a) Extension of the inflammatory fibrous hyperplasia. (b,c) Immediately post surgery. The excisions were done with CO₂ laser irradiation on both sides. (d,e) The specimens (from the left and right sides) were sent for histopathological study. (f,g) Seven days after both surgical procedures, (h,i) 30 days after both surgical procedures, and (j) 1 month after the second surgical procedure. (k,l) Adapted full denture. Figure 32.1.(continued)

Figure 32.2 (a) Drug-induced gingival hyperplasia. (b) Immediately post surgery. The excisions were done with a Nd:YAG laser. (c) Three months later the mucosa had epithelialized.

Figure 32.3 (a) Inflammatory papillary hyperplasia of the palate. (b) Immediately after vaporization with a CO₂ laser. (c) After 4 weeks of follow-up.

Figure 32.4 Pyogenic granuloma in a pregnant patient. (b) Immediately after excision with a high power diode laser. (c) After 7 days of follow-up.

Figure 32.5 (a) Clinical intraoral image of the growth; peripheral ossifying fibroma. (b) After 24 months of follow-up.

Chapter 33

Figure 33.1 (a) Nodular lesion in the lower lip mucosa, with a translucent aspect, compatible with diagnosis of an oral mucocele. (b) Clinical image of oral mucocele excision using a high power CO₂ laser. (c) Thirty days post oral mucocele excision. There is complete wound healing and absence of scar. (d,e) Microscopic analysis of the biopsied lesion, confirming the diagnosis of a mucus extravasation phenomenon. (d) Connective tissue showing mucus extravasation surrounded by granulation tissue and thermal damage in the margins (*). (e) Minor salivary gland parenchyma exhibiting thermal damage restricted to the biopsy margins (*). (HE, original magnification ×40 and ×100).

Chapter 34

Figure 34.1 (a) Clinical presentation of oral leukoplakia at the lateral border of the tongue. (b,c) Laser surgery technique for removal of oral leukoplakia lesion: (b) the extension of area to be excised was performed by outlining the lesion through vaporization, (c) followed by undercutting the connective tissue within the limited area. (d) Clinical image immediately after laser surgery. (e) Specimen sent for histopathological examination. (f) Clinical appearance 2 weeks and (g) 5 months after the surgical procedure.

Figure 34.2 (a) Initial clinical presentation of oral leukoplakia at the buccal mucosa. (b) Immediate postoperative clinical appearance after oral leukoplakia removal with a CO₂ laser. (c) A 2-week postoperative clinical examination showed an uneventful healing process; (d) after 4 weeks, the healing was almost complete without any scar.

Figure 34.3 (a) Clinical image of actinic cheilitis. (b) Clinical image of lower lip immediately after surgery and (c) 5 years postoperatively.

Figure 34.4 Oral lichen planus on (a) the left buccal mucosa and (b) the right gingival edge. (a) Immediate postoperative clinical appearance of the left buccal mucosa and (d) of the right gingival edge. (e,f) Postoperative clinical appearance after 1 week. (g) Clinical image of the buccal mucosa 3 months after oral lichen planus vaporization with a CO₂ laser. (h) Clinical image of the left buccal mucosa and (i) of the gingival edge 1 year after laser vaporization, showing recurrence of oral lichen planus.

Figure 34.5 (a) Erythroplakia affecting the soft palate and oral pharyngeal mucosa. (b) Clinical appearance immediately after erythroplakia excision with a CO₂ laser (6 W), (c) after 2 weeks, and (c) after 6 months of follow-up, when the patient was free of the disorder. (d) Recurrence of oral erythroplakia 1 year after surgical treatment.

Chapter 35

Figure 35.1 Different stages of HSV-1 infection: (a) vesicle, (b) crust (B), (c) prodrome or latent phase.

Figure 35.2 (a,b) Severe herpes labialis lesions due to the development of drug resistance.

Figure 35.3 (a) Recurrent herpes labialis in the vesicle phase. (b) Perforation of the vesicles with a sterile needle. (c) Application of methylene blue dye solution. (d) After 5 minutes, irradiation with an LPL. (e) Clinical appearance 24 hours post treatment. (f) Complete healing of the lesion after 1 week.

Figure 35.4 (a) Initial lesion. (b) Vesicle perforation and dye application. (c) Lesion appearance immediately post irradiation. (d) 6 hours post irradiation the crust was formed and there was significant reduction of edema. (e) Advanced healing observed 24 hours post irradiation; slight edema. (f) Complete healing 1 week post treatment.

Figure 35.5 (a) Initial lesions on the upper and lower lips. (b) Application of methylene blue dye. (c,d) After 5 minutes, PDT was performed at three points on each lesion. (e) Lesions 48 hours post treatment. (f) Complete healing was achieved after 1 week.

Figure 35.6 (a) Initial lesion on the upper lip. (b) Drainage of vesicles with an Er,Cr:YSGG laser. (c) Vesicles after drainage. (d) LLLT after 24 hours. (e) LLLT at four points on the lesion area. (f) One week after HPL irradiation.

Figure 35.7 (a) Initial lesion after drainage of the vesicles with an Er:YAG laser. (b) A crust was noted after 24 hours; no pain was reported, but edema was present. (c) No edema after 72 hours. (d) Advanced healing after 1 week. (e) LLLT was performed until resolution of the case. (f) Complete healing after 10 days.

Figure 35.8 (a) Recurrent herpes labialis on the left side of the upper lip. (b) Drainage of vesicles with a diode laser. (c) A crust was present after 24 hours, without edema or pain. (d) LLLT in the crust phase. (e) Advanced healing 48 hours post treatment. (f) Complete healing after 1 week.

Figure 35.9 (a) Recurrent herpes labialis (RHL) with vesicles on the upper lip. (b) Irradiation with a Nd:YAG laser for drainage of vesicles. (c) A crust was formed after 24 hours and then LLLT was performed (d). (e) Lesion after 72 hours. (f) After 1 week, signs and symptoms related to RHL were no longer present.

Figure 35.10 (a) LLLT performed at only one point on the lesion in a single session. Result after 1 hour (b) and 2 hours (c). Note the shrinkage of the lesion. (d) Complete healing after 1 week.

Figure 35.11 (a,b) Herpes zoster affecting the left side of the face, including the eye and head. (c) Before vesicle rupture, the region was isolated. (d) After vesicles drainage, methylene blue dye was applied, and after 5 minutes PDT was performed (e). (f) Vesicles still present. (g) PDT was repeated. (h) Crusts were formed and no vesicles were observed 24 hours after the second PDT session (i), and then LLLT was performed. (j) Fast resolution of the case, with a satisfactory result after 1 week. (k) Last session of LLLT. (l) Clinical appearance 8 days after the first treatment.

Chapter 36

Figure 36.1 Clinical presentation of recurrent aphthous ulcers (RAU): (a) Minor RAU, (b) major RAU, (c) herpetiform aphthous ulcer.

Figure 36.2 LLLT treatment of a patient presenting with RAU with traumatic factor: (a) Initial lesion (b) schematic of LLLT irradiation points.

Figure 36.3 Patient with a major RAU in the area of the upper right premolars: (a) Initial lesion (b) appearance of the lesion after 3 days of LLLT (c) complete healing 7 days after the first LLLT session.

Figure 36.4 LLLT of an RAU lesion: (a) Initial appearance of the aphthous lesion (b) red laser irradiation at the center of the ulcer, showing light scattering around the lesion (observe that the laser handpiece is not in contact with the tissue) (c) schematic of the LLLT irradiation points: one point in the center and four points around the ulcer (d) remission of the lesion after 4 days.

Chapter 37

Figure 37.1 Points of irradiation: (a) On the tip of the tongue (b) on the lower lip (c) on the lower alveolar ridge region.

Chapter 38

Figure 38.1 Molecular and cellular mechanisms of LLLT. Light is absorbed by cytochrome c oxidase in the mitochondrial respiratory chain of the neurons or their axons. Cell signaling and messenger molecules are

up-regulated as a result of stimulated mitochondrial activity, including reactive oxygen species (ROS), nitric oxide (NO), and adenosine triphosphate (ATP). These signaling molecules activate transcription factors including NF-κB and AP-1 that enter the nucleus and cause transcription of a range of new gene products.

Figure 38.2 Micrograph of sciatic nerve treated with LLLT (three times a week for 4 weeks) and control evaluated at 28 days after the lesion. The immunomarker is S-100, which stains the Schwann cells. There is greater staining of the laser-treated (c,d) Schwann cells than in the control (sham-treated Schwann cells, a,b) in two magnifications.

Figure 38.3 (a) Initial presentation; (b) 11 days after the start of the laser treatment (infrared laser 830 nm, energy density 120 J/cm², power density 120 mW/cm², five times per week); (c) after 55 days of treatment; (d,e) outcome of the treatment after 4 months; (f) points of irradiation used to treat the patient, three points on each branch of the facial nerve (courtesy of Prof Luiz Ferreira Monteiro Neto).

Chapter 40

Figure 40.1 Oral mucositis grades according the WHO: (a) grade 1; (b) grade 2; (c) grade 3; (d,e) grade 4.

Figure 40.2 Both lasers (A and B) have a spot area of 0.04 cm². However, the software for equipment B uses the “treated surface area” in the formula Energy density or dose = Power × Time/Area. Observe the alterations to the laser parameters according to the area used by the laser software.

Figure 40.3 (a) Oral mucositis lesions on the tongue border, induced by chemotherapy. Lesions on the fourth (b) and ninth (c) day of laser treatment.

Chapter 41

Figure 41.1 Variables resulting from the degree (I, II, and III/IV) of oral mucositis (OM) used for evaluation of cost-effectiveness of dental management associated with laser therapy in patients undergoing high doses of chemotherapy for hematopoietic stem cell transplant (HSCT). The term “with dentist” refers to the group of patients who were under dental management associated with laser therapy, and the term “without dentist” refers to the group of patients without this treatment (based on Bezinelli4).

Chapter 42

Figure 42.1 Salivary glands from hamsters in the normal control group (Normal), 5-fluouracil (5-FU) chemotherapy group (Chemotherapy), and 5-FU plus LLLT group (Chemotherapy + Laser Irradiation). Animals who received 5-FU (Chemotherapy) showed acinar atrophy with an increase in the intercellular spaces and loss of boundary cells when compared to the Normal and Chemotherapy + Laser Irradiation groups. SD, secretory ducts; arrow, secretory endpieces (original magnifications 40.×).

Figure 42.2 (a) Clinical image of a patient with hyposalivation: dehydrated skin, dry mouth, angular cheilitis, and fissured tongue. (b) Laser irradiation on all parotid, submandibular, and sublingual glands. (c) Saliva on the commissura labiorum after LLLT.

Chapter 43

Figure 43.1 aPDT on the periodontium. (a) Clinical image before aPDT. Note the severe gingival inflammation and enlargement. (b) Oral hygiene orientation and tooth brushing under supervision. (c) MB dye applied to the periodontal pockets for 5 minutes; (d) phototherapy with a low power laser in contact mode; (e) clinical image 1 week after the single aPDT treatment.³

Figure 43.2 (a) HSV infection of the palatal mucosa in a head and neck cancer patient treated with radiotherapy. The ulcers were covered with MB for 5 minutes prior to laser irradiation. (b) The day after the first aPDT treatment there was considerable improvement and the lesion was painless (c) clinical image 5 days after aPDT(d) clinical image 13 days after aPDT.⁵

Figure 43.3 (a) Clinical image of an infected oral lesion before aPDT. (b) MB (0.01%) applied for 5 minutes before laser irradiation. (c) Irradiation with a red low power laser in contact mode. (d) Clinical image 7 days after treatment; a second aPDT session was then performed (same parameters); (e) clinical image 10 and (f) 15 days after the first aPDT session.

Figure 43.4 (a) Gum before treatment. (b) Dye carefully applied to the periodontal pockets after biofilm removal. (c) Laser irradiation with no contact. (d) Clinical image 15 days after oral treatment.

Figure 43.5 (a) Panoramic radiograph showing severe bone loss; (b) clinical image before dental extractions; (c) alveolus after curettage; (d) image after teeth removal; (e) dye applied for 5 minutes; (f) laser irradiation; (g) mass suture; (h) clinical image 2 months after extractions, showing the epithelialized area; (i) clinical image 3 months after the extractions, showing total tissue repair.

Chapter 44

Figure 44.1 Schematic of the basic principle of PDT. (a) Superficial tumor surrounded by normal tissue; (b) some time after its administration, the photosensitizer (PS) preferentially accumulates in the target tumor tissue; (c) the target site is irradiated by laser light at a specific wavelength, exciting the PS. The penetration of this light is limited and depends on its wavelength; (d) activated PS induces photochemical reactions with the local oxygen, generating mainly singlet oxygen (1O_2), other cytotoxic oxygen species, and free radicals (SH-R); (e) tissue damage (apoptosis and/or necrosis) is restricted to the tumor; (f) normal tissue after destruction of the tumor by PDT.

Figure 44.2 (a) Initial lesion: squamous cell carcinoma of the tongue; (b)

5 days after PDT; (c) 1 month after PDT

Figure 44.3 (a) Initial lesion: squamous cell carcinoma of the lip; (b) 1 month after PDT; (c) 1 year after treatment

Figure 44.4 (a) Initial lesion: squamous cells carcinoma of the cheek; (b) immediately after PDT; (c) 5 months after treatment

Lasers in Dentistry

Guide for Clinical Practice

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To God, without whom my life would have no direction and my projects would not be successful

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