

Low-Level Laser Therapy for Trigeminal Neuralgia

Case reports on two patients whose unrelenting facial pain and hypersensitivity from their diagnosed trigeminal neuralgia resolved with low-level laser therapy.



In this issue, Drs. Leonard Vernon and Rafael Hasbun present two case reports of trigeminal neuralgia pain successfully treated with low-level laser therapy (LLLT). Trigeminal neuralgia can be a challenging painful condition to treat and/or manage. This article also includes a review of the etiology, involved anatomy, and typical outcomes of conventional modalities in comparison to LLLT.

— William Kneebone, RN, CRNA, DC
Department Head



by Leonard F. Vernon, DC and Rafael J. Hasbun, MD

Low-level laser therapy (LLLT) has been used extensively as modality in the treatment of a wide variety of clinical conditions. Until recently, most of this clinical use has been outside of the United States. Although this therapy method remains controversial, many authors have reported significant pain reduction in a number of conditions such as rheumatoid arthritis, osteo-arthritis, fibromyalgia, post-operative pain, and low back pain.

LLLT has been shown to effect many sub-cellular and cellular processes and, although the exact mechanism have not been well defined, it is believed that light is absorbed by mitochondrial chromophores leading to an increase in adenosine triphosphate (ATP), reactive oxygen species and/or cyclic AMP production, and consequent gene transcription via activation of transcription factors. Despite the many case reports, the use of LLLT remains controversial.

In this article, we report on two patients who were diagnosed with trigeminal neuralgia (CN V). Both patients had unrelenting facial pain, with one of the two patients experiencing a motor nerve component (CN VII), as well as a hyper-sensitivity. Both patients had tried various medications including analgesics, NSAIDs, and steroids—all of which offered no relief. Both patients were treated with a Ga-Al-As diode system that produces low-energy infrared light (808nm diodes/200mW). The laser was applied to the multiple locations within a defined region of the facial area. Both patients were treated once per day for five consecutive days, followed by a two-day interval.

This regimen continued until the total number of applications reached 20.

Etiology

Trigeminal neuralgia (TN) has been referred to in the medical literature for centuries. References to unilateral facial pain causing facial spasms can be seen in the writings of Aretaeus of Cappadocia in the 2nd century A.D.¹ and those of the Arab physician Jujani in the 11th century A.D.² John Fothergill,³ in a paper published in 1773, described the typical features of TN, including its paroxysmal nature and association with triggering factors such as eating, speaking, or touching the face. In 1756, the French surgeon Nicholas André coined the term “tic douloureux” to describe at least three patients with TN.⁴

Trigeminal neuralgia (TN), or tic douloureux (also known as prosopalgia), is a neuropathic disorder of the trigeminal nerve that causes episodes of intense pain in the eyes, lips, nose, scalp, forehead, and jaw, with the majority of cases being unilateral (>95%).⁵ This lancinating pain is typically in the distribution of the second and third divisions of the trigeminal nerve and can be triggered by facial movement, cold temperature, talking, and other common activities.⁶ Trigeminal neuralgia is considered by many to be among the most painful of conditions and is often labeled the “suicide disease” because of the significant numbers of people taking their own lives when they cannot find effective treatments. An estimated 1 in 15,000 people suffers from trigeminal neuralgia, although numbers

may be significantly higher due to frequent misdiagnosis. It usually develops after the age of 40, although there have been cases with patients being as young as 13 months of age.⁷

On physical examination, the neurological examination of other cranial nerves is usually within normal limits, as neurological signs rarely accompany TN. If a neurological abnormality is revealed, such as absent or diminished corneal reflex, it is important to consider an alternative diagnosis such as a central lesion (posterior fossa tumor) or MS.⁸ When attempting to examine the affected side, the patient will shrug away or prevent the examiner's hand from touching the face for fear of inducing pain. If the examiner can examine the area, there may be a mild light touch or pin perception loss in the central area of the face.⁹

The mechanism of pain production remains controversial. One theory suggests that peripheral injury or disease of the trigeminal nerve increases afferent firing in the nerve. Failure of central inhibitory mechanisms may be involved as well. Some authors have hypothesized that trigeminal neuralgia has a peripheral cause as well as a central pathogenesis. They speculate that chronic irritation of the trigeminal nerve apparently leads to both a failure of segmental inhibition in the trigeminal nucleus, and ectopic action potentials in the trigeminal nerve. In 90-95% of cases, no lesion is identified, and the etiology is labeled idiopathic by default.¹⁰ Some authors have speculated that, like Bell's palsy, a neurotropic virus may be the causative agent and the proximity of the two nerves may give cause to further support this theory.⁷ Ch'ien and Halsey reported two cases of concomitant trigeminal sensory neuropathy and Bell's palsy.¹¹ Although the exact pathogenesis is unknown, it is, however, generally accepted that focal demyelination in the root of the trigeminal nerve is involved.^{12,13} Of course, any injury to the trigeminal nerve may also cause the condition. Trigeminal Neuropathy or Post-Traumatic TN may develop following cranio-facial trauma (such as from a car accident), dental trauma, or sinus trauma.

The syndrome presents as classic episodes of shooting pain in areas of the face. The disorder is frequently associated with gradual worsening of the pain pattern and shortening of pain-free intervals. If left untreated, it may develop new features, including sensory disturbances and constant pain, which are uncharacteristic of trigeminal neuralgia and evolve toward another pain syndrome, generally known as atypical trigeminal neuralgia.¹⁴

Anatomy

The trigeminal nerve is the largest of all the cranial nerves. It exits laterally at the mid-pons level and has two divisions—a smaller motor root (portion minor) and a larger sensory root (portion major). The motor root supplies the temporalis, pterygoid, tensor tympani, tensor palati, mylohyoid, and anterior belly of the digastric. The motor root also contains sensory nerve fibers that particularly mediate pain sensation.

The gasserian ganglion is located in the trigeminal fossa (Meckel cave) of the petrous bone in the middle cranial fossa. It contains the first-order general somatic sensory fibers that carry pain, temperature, and touch. The peripheral processes of neurons in the ganglion form the three divisions of the trigeminal nerve.

The ophthalmic nerve (V1), which exits the cranium via the



FIGURE 1. Here the patient is treated with the laser at the point where an incision would be made if decompression surgery was being performed on trigeminal nerve (about two-inches deep).

superior orbital fissure, is a sensory nerve that controls feeling in the skin on the top of the head, the mucous membrane of the nose and sinuses, the cornea, and the conjunctiva. In trigeminal neuralgia, damage to, or pressure on, the ophthalmic nerve can cause pain along the jaw line, to the eye, and back across the forehead.

The maxillary nerve (V2), which exits via the foramen rotundum, controls sensation in the dura mater of the brain and spinal cord, the gums and teeth in the upper jaw, the upper lip, and the orbit. If this nerve is affected, pain often radiates from the jaw to the upper lip, nose, and underneath the eye.

The mandibular nerve (V3) exits via the foramen ovale, and is the largest branch of the fifth cranial nerve. This nerve controls sensation in the lower jaw (including the gums and teeth), the tongue, the lower lip, and the skin of the cheek. It also controls the muscles that are used for mastication. In patients who have TN, damage to, or pressure on, the mandibular nerve can cause pain that radiates from the jaw and chin to the lower lip and tongue.^{15,16}

Treatment Modalities

Standard first line treatment is pharmacological, usually with carbamazepine.^{17,18} Other drugs including lamotrigine, phenytoin, gabapentin, oxcarbazepine, topiramate, baclofen, and clonazepam have also been reported to have some effect, although studies are more limited.¹⁸ Because many patients fail to have a sustained response to pharmacological intervention, some “non-drug” options for TN patients have evolved.

Interventions include microvascular decompression, which treats the compressive etiology of TN surgically by separating the trigeminal nerve from adjacent blood vessels, and a variety of methods of producing a partial trigeminal nerve lesion including neurectomy, radiofrequency thermal ablation, balloon compression, glycerol injections, and radiosurgery.

The evidence for these treatments for trigeminal neuralgia does not come from randomized trials.¹⁷ Given the severity of the pain, it is not surprising that no major randomized studies have been conducted of the natural history of untreated patients with trigeminal neuralgia.

Some specialist centers report that microvascular decompres-

sion renders over two thirds of patients pain free at 10 years with 1% experiencing facial numbness.¹⁹ Other studies are less optimistic and highlight complications, which include injury to the cerebellar and eighth cranial nerve²⁰ and death rates of 0.2-1%.²¹ Microvascular decompression offers a treatment that is not designed to damage the trigeminal nerve and has good results in expert hands. However, it carries a small but definite risk of major, including fatal, complications and, like all surgical procedures, is operator-dependent.

More recently, there has been an increased use of stereotactic gamma knife radiosurgery, which entails the delivery of a focused beam of radiotherapy to the proximal trigeminal nerve. First used in 1951, it has been more widely used since the mid-1990s and reports of 70-80% of patients describing freedom from pain in the short term have been reported,²²⁻²⁴ although up to 50% may relapse.²⁵ Side effects include facial dysaesthesia (up to 12%),²³ corneal irritation, vascular damage, hearing loss, and facial weakness, varying with the dose schedule and target area. Follow up is short compared with the 10 years cited for other treatment modalities, and uncertainty persists about possible late complications of radiotherapy—for example, cerebral edema or neoplastic transformation.

Therapeutic Effects of Low-Level Laser Therapy

There have been numerous claims in the literature for the therapeutic effects of LLLT treatment such as acceleration of wound healing,²⁶ pain attenuation,^{27,28} restoration of normal neural function following injury,^{29,32} enhanced remodeling and repair of bone, normalization of abnormal hormonal function, stimulation of endorphin release and modulation of the immune system.³³ Published data on efficacy exist for some, but not all, of these applications.

Low level laser (LLLT) therapy has also been used for the treatment of nerve injuries. Clinical studies of the effects of LLLT on injured nerves have revealed an increase in nerve function and improved capacity for myelin production.²⁹ LLLT has been shown to be effective for promoting axonal growth in injured nerves in animal model.³³⁻³⁵

LLLT acts on the prostaglandin (PG) synthesis, increasing the change of PGG2 and PGH2 into PG12 (also called prostacyclin, or epoprostenol) and PG12. The latter is the main product of the arachidonic acid into the endothelial cells and into the smooth muscular cells of vessel walls and has a vasodilating and anti-inflammatory action.³⁶ In a case report of arthralgia of the temporomandibular joint treated with LLLT, the analgesic and anti-inflammatory effects of LLLT were confirmed by infrared thermography.³⁷ The main benefit of LLLT is that it is relatively noninvasive and may have the ability to treat nerve injuries without surgical intervention thus making it a desirable treatment option.³⁸

An online search of PubMed using the words “trigeminal neuralgia” and “low level laser” yielded only one published report since 1998 that specifically dealt with the use of LLLT in the treatment of TN.³⁹

Case Report 1

Patient is a 47-year-old female who presented a history of insidious onset of left-sided facial weakness that began about one month prior to her initial examination. The examination was unremarkable with the exception of moderate facial weakness

on the left side resulting in difficulty in raising of the eyebrow on that side. There was also difficulty in closing the eyelid on the same side. The deep tendon reflexes were symmetric, the gait was normal, and there were no cerebellar signs.

An MRI of the brain with and without gadolinium was ordered as was an electromyography. The results of this examination revealed a rather severe lesion of the 7th cranial nerve on the left, a diagnosis of Bell’s palsy (axonal type) was made. The brain MRI was reported as unremarkable. Approximately two months following the onset of Bell’s palsy, the patient began to develop pain in the right side of her face, which became increasingly severe over the next few days. Trigeminal sensory neuropathy is often associated with facial idiopathic nerve paralysis (Bell’s palsy).⁴⁰ Hanner et al,⁴¹ have suggested that there may be coordinating functions between the facial and trigeminal neuronal systems that are influenced by facial nerve dysfunction. Nonetheless, in most instances the etiology remains unclear.

The patient was started on Neurontin(r) (gabapentin) 300 mg b.i.d along with Valtrex(r) (valacyclovir) 500mg b.i.d. Both were initially prescribed for 10 days. A Medrol-Dose Pack® (methylprednisolone) was also prescribed. Only minimal relief was

“Trigeminal sensory neuropathy is often associated with facial idiopathic nerve paralysis (Bell’s palsy).”⁴⁰

obtained and this relief lasted only about one week. Over the next eight weeks the patient’s facial pain increased in intensity and her current medication program offered no relief. The patient was referred for LLLT for her condition.

LLLT was begun utilizing a Ga-Al-As diode system that produces low-energy infrared red light (808nm diodes/200mw) wavelength, (6J/pm) once per day for five consecutive days, followed by a two-day interval. The number of applications was 20 with the treatment model adapted from Claus et al.⁴²

The patient was irradiated over the foramina where the sensory portions of the nerve exits: the superior orbital fissure, the foramen rotundum, and the foramen ovale, as well as over the striated muscles adjacent to relevant sensory branches of the nerve (see Figure 1). Each area was irradiated for a three-minute period for a total of 15-18 minutes. Although treatment of the 7th cranial nerve was not begun at the time of the writing of this paper, placement of the laser head would be over the stylomastoid foramen.

On the 5th treatment day, the patient reported greatly diminished pain in the region, there was no change noted of the facial palsy. Treatment of the 5th cranial nerve was continued using the protocol as noted above for a total of 20 treatments, even though the patient was completely symptom-free from pain by the 12th treatment session.

Case Report 2

Patient is a 61-year-old female who was diagnosed by her family physician (RH) to be suffering from typical TN. The physical examination was unremarkable. The deep tendon reflexes were

symmetric, the gait was normal, and there were no cerebellar signs. The patient exhibited an unwillingness to have her face examined on the left side. She indicated that the pain began insidiously about four weeks ago and has become increasingly severe. The patient's pain was unresponsive to pharmacological management that included: Neurontin® (gabapentin) 300 mg b.i.d and Valtrex® (valacyclovir) 500mg b.i.d. Both were initially prescribed for 10 days along with a Medrol-Dose Pack® (methylprednisolone). Following relief with these pharmacological agents, the patient was referred for LLLT.

Treatment protocol was the same as in the previous patient and consisted of a Ga-Al-As diode system that produces low-energy infrared light (808nm diodes/200mW) wavelength. Application was performed once per day for five consecutive days, followed by a two-day interval. The total number of applications was 20. Anatomical areas and treatment times for this patient were the same as in the previous patient.

Response to treatment was again seen by the 5th treatment and by the 9th treatment the patient reported only minimal discomfort. By treatment session 12, the patient was essentially pain free.

“LLLT appears to be a highly effective modality in treating intractable facial pain as seen with trigeminal neuralgia.”

Treatment of the 5th cranial nerve was continued using the protocol as noted above for a total of 20 treatments, even though the patient was totally symptom-free from pain by the 12th treatment session. Although there are, as yet, no established treatment protocol for various conditions treated with LLLT, we chose 20 treatments based on standard protocol used in wound care.

Follow-up of this patient was not possible as she returned to her home country the week following her 20th session.

Discussion

Our report indicates that LLLT appears to be effective in the treatment of TN. We were unable to treat the patient in Case Report 1 for her Bell's palsy, although the literature does indicate that in cases where nerve regeneration or stimulation is desirable (e.g., Bell's palsy or facial paralysis), LLLT may be beneficial.⁴³

Another major issue when utilizing LLLT is the rationale behind the selection of application technique and treatment parameters, such as power density, size of exposure, timing or treatment frequency, all of which currently remain unclear.⁴⁴ The reason for this is that low-level laser therapy (LLLT) has received only specific US Food and Drug Administration clearance, for the treatment of carpal tunnel syndrome, thus manufacturers are prohibited from suggesting protocols for other conditions. This leaves the practitioner with only anecdotal reports to rely on. Additional studies and FDA clearance of the LLL for other conditions would greatly enhance the utilization of this instrument.

Conclusion

LLLT appears to be a highly effective modality in treating intractable facial pain as seen with trigeminal neuralgia. The beneficial effect from LLLT on the motor segment of the nerve was not evaluated. Relief was obtained by both patients by the seventh treatment session; however, both patients continued treatment and completed all 20 sessions. Neither patient has returned for additional care. Although the pain and hypersensitivity resolved in both patients, the one patient with a motor deficit (CN VII) continued to experience this problem. ■

Dr. Kneebone studied nursing at Cook County Hospital in Chicago graduating as an RN in 1972. He completed an anesthesia program at St. Francis Hospital in La Crosse, Wisconsin in 1974. Dr. Kneebone practiced anesthesia until he graduated from Palmer College of Chiropractic in 1978. He has been in a complimentary medicine practice in the San Francisco Bay area since 1978. He has post graduate certification in nutrition and homeopathy. He is also a Fellow of the International Academy of Medical Acupuncture and a Diplomate of the International Academy of Clinical Thermology. Dr. Kneebone has been using therapeutic lasers in his practice for over 7 years and has been teaching laser seminars for the past four years. He teaches Cutting Edge Laser Seminars™ all around the US. He can be contacted at drknee@pacbell.net

References

- Rose FC. Trigeminal neuralgia. Arch Neurol. 1999. 56: 1163-1164.
- Ameli NO. Avicenna and trigeminal neuralgia. J Neurol Sci. 1965. 2: 105-107.
- Fothergill J. Of a painful affection of the face. Medical observations and inquires by a society of physicians. Med Observ Inquir. 1773. 5: 129-142.
- Brown JA, Coursaget C, Preul MC, et al: Mercury water and cauterizing stones: Nicolas André and tic douloureux. J Neurosurg. 1999. 90: 977-981.
- Bayer DB and Stenger TG. "Trigeminal neuralgia: an overview". Oral Surg. Oral Med. Oral Pathol. 1979. 48 (5): 393-399.
- Headache Classification Committee of the International Headache Society, The international classification of headache disorders. Cephalalgia 24. 2004. pp 1-160.
- Mason WE, Kollros P, and Jannetta PJ. Trigeminal neuralgia and its treatment in a 13-month-old child: A review and case report. J Craniomandib Disord Facial Oral Pain. 1991. 5: 213-216.
- Jackson EM, Bussard GM, Hoard MA, and Edlich RF. Trigeminal neuralgia: a diagnostic challenge Am J Emerg Med. Oct 1999. 17(6): 597-600.
- ibid. ref 8; Jackson et al.
- Fromm GH, Terrence CF, and Maroon JC. Trigeminal neuralgia. Current concepts regarding etiology and pathogenesis. Arch Neurol. Nov 1984. 41(11): 1204-1207.
- Ch'ien LT and Halsey JH: Trigeminal sensory neuropathy and Bell's palsy. N Engl J Med. 1970. 282: 224-225.
- Burchiel KJ. Trigeminal neuropathic pain. Acta Neurochir Suppl (Wien). 1993. 58: 145-149.
- Burchiel KJ. Abnormal impulse generation in focally demyelinated trigeminal roots. J Neurosurg 53. 1980. pp 674-683.
- International Association for the Study of Pain, Subcommittee on Taxonomy: Classification of chronic pain. 1986. Pain Suppl 3: S1-S226.
- Hickey JV. The clinical practice of neurological and neurosurgical nursing (5th ed.). Philadelphia: Lippincott Williams & Wilkins. 2002.
- Jannetta PJ. The pathophysiology of trigeminal neuralgia. In M. Samii & P.J. Jannetta (Eds.), The cranial nerves. Berlin: Springer-Verlag. 1981. pp 312-320.
- Wiffen P, Collins S, McQuay H, Carroll D, Jadad A, and Moore A. Anticonvulsant drugs for acute and chronic pain. Cochrane Database Syst Rev. 2000. 3: CD001133.
- Zakrzewska JM. Trigeminal neuralgia. Clin Evid. Jun 2002. (7): 1221-1231.
- Barker FG, Jannetta PJ, Bissonette DJ, Larkins MV, and Jho HD. The long-term outcome of microvascular decompression for trigeminal neuralgia. N Engl J Med. 1996. 334: 1077-1083.
- McLaughlin MR, Jannetta PJ, Clyde BL, Subach BR, Comey CH, and Resnick

- DK. Microvascular decompression of cranial nerves: lessons learned after 4400 operations. *J Neurosurg.* 1999. 90: 1-8.
21. Sweet WH and Poletti CE. Complications of percutaneous rhizotomy and microvascular decompression operations for facial pain. In: Schmidek HH, Sweet WH, eds. *Operative neurosurgical techniques: indications, methods and results, II.* 2nd ed. Grune and Stratton. Orlando, FL. 1988. 1139-1143.
22. Young RF, Vermeulen SS, Grimm DO, Blasko J, and Posewitz A. Gamma knife radiosurgery for treatment of trigeminal neuralgia. Idiopathic and tumor related. *Neurology.* 1997. 48: 608-613.
23. Pollock BE, Phuong LK, Gorman DA, Foote RL, and Stafford SL. Stereotactic radiosurgery for idiopathic trigeminal neuralgia. *J Neurosurgery.* 2002. 97: 347-353.
24. Brisman R. Gamma knife radiosurgery for primary management for trigeminal neuralgia. *Journal of Neurosurg.* 2000. 93:(suppl 3): S159-61.
25. Kondziolka D, Lunsford LD, and Flickinger JC. Stereotactic radiosurgery for the treatment of trigeminal neuralgia. *Clin J Pain.* 2002. 18: 42-47.
26. Abergel RP, Lyons RF, Castel JC, Dwyer RM, and Uitto J. Biostimulation of wound healing by lasers: experimental approaches in animal models and in fibroblast cultures. *J Dermatol Surg Oncol.* 1987. 13: 127-133.
27. Walker J. Relief from chronic pain by low power laser irradiation. *Neurosci Lett.* 1983. 43: 339-344.
28. Naeser MA, Hahn KA, Lieberman BE, and Branco KF. Carpal tunnel syndrome pain treated with low-level laser and microamperes transcutaneous electric nerve stimulation: A controlled study. *Arch Phys Med Rehabil.* 2002. 83: 978-988.
29. Miloro M and Repasky M. Low-level laser effect on neurosensory recovery after sagittal ramus osteotomy. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2000. 89:12-18.
30. Tay AB and Go WS. Effect of exposed inferior alveolar neurovascular bundle during surgical removal of impacted lower third molars. *J Oral Maxillofac Surg.* 2004. 62: 592-600.
31. Haug RH, Perrott DH, Gonzalez ML, and Talwar RM. The American Association of Oral and Maxillofacial Surgeons Age-Related Third Molar Study. *J Oral Maxillofac Surg.* 2005. 63: 1106-1114.
32. Poole TE, Holland I, and Peterson LJ. Clinical efficacy of low level laser treatment of oro-facial neurosensory deficits. *J Oral Maxillofac Surg.* 1993. p 182.
33. Walsh LJ. The current status of low level laser therapy in dentistry. Part 1. Soft tissue applications. *Aust Dent J.* 1997. 42: 247-254.
34. Mester AF, Snow JB Jr, and Shaman P. Photochemical effects of laser irradiation on neuritic outgrowth of olfactory neuroepithelial explants. *Otolaryngol Head Neck Surg.* 1991. 105: 449-456.
35. Solomon A, Lavie V, Ben-Bassat S, Belkin M, and Schwartz M. New surgical approach to overcome the inability of injured mammalian axons to grow within their environment. *J Neural Transplant Plast.* 1991. 2: 243-248.
36. Tam G. Low power laser therapy and analgesic action. *J Clin Laser Med Surg.* Feb 1999. 17(1): 29-33.
37. Fikáková H, Dostálová T, Vosická R, Peterová V, Navrátil L, and Lesák J. Arthralgia of the temporomandibular joint and low-level laser therapy. *Photomed Laser Surg.* Aug 2006. 24(4): 522-527.
38. Robinson PP. Observations on the recovery of sensation following inferior alveolar nerve injuries. *Br J Oral Maxillofac Surg.* 1988. 26: 177-189.
39. Pinheiro AL, Cavalcanti ET, Pinheiro TI, Alves MJ, Miranda ER, De Quevedo AS, Manzi CT, Vieira AL, and Rolim AB. Low-level laser therapy is an important tool to treat disorders of the maxillofacial region. *J Clin Laser Med Surg.* Aug 1998. 16(4): 223-226.
40. Adour KK, Byl FM, Hilsinger RL, Kahn ZM, and Sheldon MI. The true nature of Bell's palsy: analysis of 1,000 consecutive patients. *Laryngoscope.* 1978. 88: 787-801.
41. Hanner P, Badr G, Rosenhall V and Edstrom S. Trigeminal dysfunction in patients with Bell's palsy. *Acta Otolaryngol (Stock L)* 1986. 101: 224-230.
42. Claus EE, Fusco CF, Ingram T, Ingersoll CD, Edwards JE, and Melham TJ. Comparison of the effects of selected dressings on the healing of standardized abrasions. *J Athl Train.* 1998. 33:145-149.
43. Vargas JT. Low-Level Laser Acupuncture. *Medical Acupuncture.* 2005. 16 (2): 38-41.
44. Bjordal JM, Couppe C, and Ljunggren A. Low level laser therapy for tendinopathy: evidence of a dose-response pattern. *Physical Therapy Reviews.* 2001. 6(2): 91-99.