Chapter 19

Low-Level Laser Therapy

The light spectrum encompasses ultraviolet, visible, and infrared energy. Although some thermal effects may be obtained from these modalities, the primary benefits are derived from photochemical effects.

Electromagnetic energy is the most abundant form of energy in the universe. The energy found in the electromagnetic spectrum, including radio waves, and x-rays, is categorized by the frequency and length of its wave (Appendix B). Light, a form of electromagnetic energy, has three general classifications: ultraviolet, visible, and infrared (Fig. 19-1). Energy having a wavelength greater than 780 **nanometers** (nm) (the upper end of visible light) is infrared energy. The ultraviolet (UV) spectrum is located in the area below the range of visible light (380 nm).

Many therapeutic modalities presented in this text use energy within the light range of the electromagnetic spectrum, although the light energy may not be visible to humans. UV light is used for the treatment of certain skin conditions. Medical lasers produce beams of energy that can cause either tissue destruction or therapeutic effects within the tissues.

Therapeutic Lasers

Lasers produce highly refined, **monochromatic** light in the ultraviolet, visible, or infrared range. Lasers, an acronym for ***L***ight ***A***mplification by ***S***timulated ***E***mission of ***R***adiation, consist of highly organized light (**photons**) that elicits physiological events in the tissues. **Low-level laser therapy (LLLT)** does not normally cause tissue destruction.[198] The U.S. Food and Drug Administration (FDA) has approved LLLT for the treatment of **carpal tunnel syndrome** and musculoskeletal shoulder and neck pain; however, clinically it is used for a wide range of conditions.

The energy produced by therapeutic lasers can have a wavelength between 650 and 1200 nanometers (nm).[199] This range includes UV, visible, and infrared light on the electromagnetic spectrum. The frequency (wavelength) determines the color of the laser light. Frequency and wavelength, often

Nanometer: One-billionth (10^{-9}) of a meter.

Monochromatic: Light that consists of only one color.

Carpal tunnel syndrome: Compression of the median nerve that produces pain, numbness, and weakness in the palm and ring and index fingers.

Photon: A unit of light energy that has zero mass, no electrical charge, and an indefinite life span.

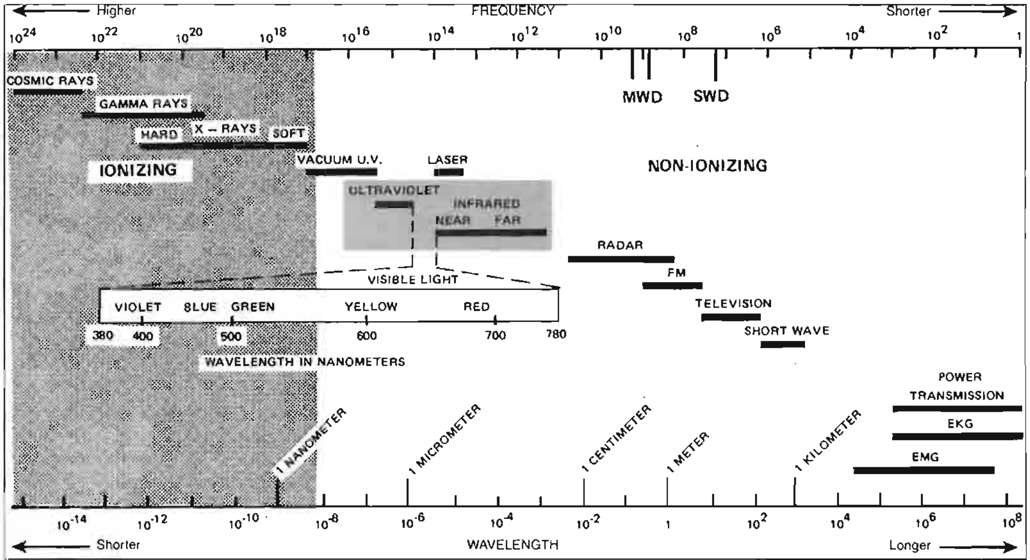


Figure 19-1. **The Electromagnetic Spectrum.** The visible portion of the light spectrum consists of energy having a wavelength of approximately 380 to 780 nm. Infrared light has a wavelength between 780 and 12,500 nm; energy with a wavelength between 180 and 400 nm is in the ultraviolet range. There is a slight amount of overlap between the visible light range and that of ultraviolet and infrared light.

used interchangeably, are inversely related to each other: as frequency increases, wavelength decreases (and vice versa). The photons emitted during LLLT activate certain skin receptors that stimulate or inhibit physiological events. These effects are caused by activation of **chromophores,** parts of a molecule (generally **melanin** • and hemoglobin) that absorb light having a specific color (wavelength). Because of the specificity of chromophores in absorbing light energy, wavelength is thought to determine which skin receptors are affected.[200]

Class 4 lasers ("hot lasers") produce thermal changes in the tissues, causing the tissues to be destroyed, evaporated, or dehydrated or causing protein coagulation (see Table 19-1).[201] Hot lasers are used for surgery, capsular shrinkage, ocular surgery, and wrinkle and tattoo removal. Because of their destructive potential, high-power lasers are not found in the rehabilitation setting.

Other forms of light therapy include ultraviolet lamps, superluminous diodes (SLDs), and light-emitting diodes (LEDs). These types of light modalities are reported to evoke some of the effects of phototherapy, but their biophysical effects are different. A laser applicator may include LEDs and SLDs to produce visible light to assist in targeting the laser, especially when the laser output is not in the visible light range. Some manufacturers may include LEDs and SLDs to promote an additive effect to the laser treatment.

Laser Characteristics

Lasers produce a refined, homogeneous beam of light characterized by three features: the light is (1) monochromatic, (2) coherent, and (3) collimated. **Monochromatic** means all of the light energy has the same wavelength, thereby producing the same color. Sunlight passing through a prism creates a rainbow of colors because it has light of different wavelengths. A laser light passed through a prism simply bends and the color leaving the prism the same as that entering it.

Light photons travel in waves having different lengths. This wavelength determines the color of the light. When all of the light waves are in phase, they are said to be **coherent** (Fig 19-2). Light from a light bulb spreads—diverges—as it travels. Laser light is **collimated** because the beam does not tend to diverge as it travels through space.

Lasers are classified by the FDA's Center for Devices and Radiological Health (CDRH) based on the **Accessible Emission Limit** (AEL). The AEL is the maximum permissible power level for each class, ranging from 1 (minimum risk of causing harm) to 4 (extreme risk) (Table 19-1).

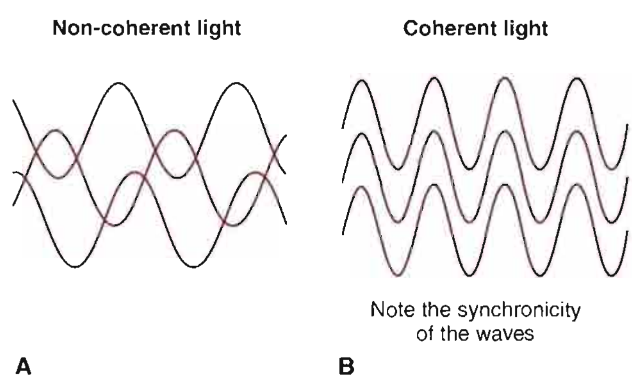


Figure 19-2. **Light Waves In and Out of Phase.** Light having the same wavelength, but the photons are out of phase. With coherent light the photons are in phase.

Melanin: Pigmentation of the hair, skin, and eye produced by melanocytes.

TABLE 19-1 U.S. Food and Drug Administration's Center for Devices and Radiological Health Laser Classification System

Classification	Description
1	These lasers are exempt from most control measures. The laser output is either safe to the human eye or contained within the device in a manner that keeps the laser from escaping. No special labeling is required.
2	Low-power lasers—visible light may be emitted. Output does not exceed 1 mW. The normal eye blink reflex (approximately 0.25 s) will protect the eye from direct contact with the laser output. Must be labeled with **"CAUTION—Laser Radiation: Do not stare into beam."**
2a	Visible laser is produced (e.g., a bar code scanner). Eye damage can occur if the laser enters the eye for more than 1000 seconds. The labeling is the same as for type 2 lasers.
3a	Produces an output up to 5 mW. Direct contact with the eye for short periods is not hazardous. Viewing the laser through magnifying optics such as eyeglasses can present a hazard. Must be labeled with **"CAUTION—Laser Radiation: Do not stare into beam or view directly with optical instruments."** Class 3a lasers do not require that the patient and clinician wear goggles during treatment.
3b	Medium power lasers producing an output of 5 mW to 500 mW. Direct contact of the laser output with the eyes can result in damage. Must be labeled with **"DANGER—Visible and/or invisible laser radiation—avoid direct exposure to beam."** The patient and the clinician (and anyone in the immediate area) must don goggles during treatment.
4	High power lasers having an output of greater than 500 mW. Direct or indirect contact with the skin and eyes can be hazardous. Toxic airborne contaminants may be produced. The output creates a fire hazard. Must be labeled with **"DANGER—Visible and/or invisible laser radiation—avoid eye or skin exposure to direct or scattered beam."**

Laser Output Parameters

In the United States therapeutic lasers have a wavelength of 650 nm to 1200 nm. The low and medium power output associated with LLLT does not cause significant thermal changes in the tissues, so therapeutic benefits are thought to be related to photochemical events.[199,202] The magnitude of the tissue's reaction to laser light is based on the physical characteristics of the output frequency/wavelength (absorption, reflection, and transmission), power density (irradiance), the duration of the treatment, and the vascularity of the target tissues.[199,203] The depth of penetration is also affected by the skin's melanin and blood hemoglobin content, both of which absorb photons.[200]

Effects that occur from the absorption of photons are termed the **direct effect.** An **indirect effect** is produced by chemical events caused by the interaction of the photons emitted from the laser and the tissues. The indirect effect may produce changes that occur deeper in the tissues than those caused by the direct effect.

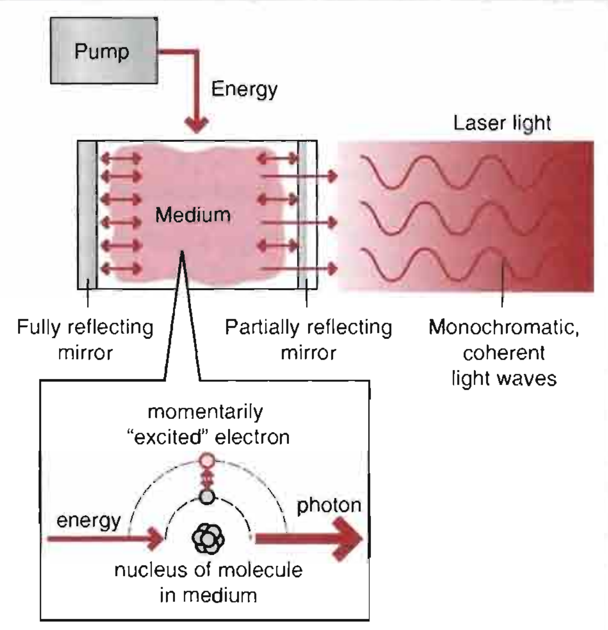
In some cases therapeutic lasers require a base unit that houses the laser generator (Box 19-1). The output is delivered to the body through a fiber optic cable and a hand-held applicator. The laser medium (e.g., gallium aluminum arsenide, argon, helium-neon) determines the wavelength—and therefore the effects of—the laser output.

Wavelength and Frequency

Wavelength and frequency are inversely related. Long wavelengths have a lower frequency than shorter wavelengths (see Appendix B). LLLT has a frequency of up to 5000 Hz, a duration of 1 to 500 milliseconds, and an interpulse interval of 1 to 500 milliseconds, although there is variability depending on the make and model of the unit.[204]

The laser's depth of penetration, the point where photons are absorbed by the tissues, depends on the wavelength of the light. Longer wavelengths (800 to 1000 nm) penetrate deeper into the tissues than lasers

Box 19-1. PRODUCTION OF LASER



Laser production requires four essential components: (1) an active (amplifying) medium, (2) a mechanism for exciting the medium (a pump), (3) a reflective mirror, and (4) a partially reflective mirror that allows for some transmission of light and reflects the rest.

Lasers are referred to by the type of active medium (the atoms that are stimulated to produce the light) they use, HeNe (helium-neon) or GaAs (gallium-arsenic), for example. The active medium is a solid, liquid, or gas that contains atoms, molecules, or ions that are capable of storing energy that, when stimulated, release their energy as light. Any subsequent increase in light energy through the lasing mechanism is known as **gain.**

Through a process known as **pumping,** energy is introduced into the active medium. For a solid medium, the pumping is obtained by irradiating the medium with a bright light; gaseous mediums are energized by passing an electrical current through the medium.

When a photon of light is absorbed by an atom, molecule, or ion, an outer electron moves from its normal orbit, its **ground state,** to a higher orbit termed the **excited state.** By being moved to a higher orbit, the electron, and therefore the atom, reaches a higher energy state. After a brief stay (less than a millionth of a second) in the higher orbit, the electron spontaneously returns to its ground state and in the process releases another photon. The released photon will have the same wavelength (and therefore frequency) as the photon that was absorbed.

Stimulated response occurs when a photon strikes an atom that is already in the excited state and causes the electron that is in the higher orbit to move to a lower one. In this case, instead of a single photon being released, two photons are released. Each of these photons has the same wavelength and is in phase. When more atoms are in an excited state than in the ground state, a **population inversion,** more photons are emitted than absorbed, forming the basis for the emission of laser.

A laser generator consists of an **oscillator,** the active medium located between two mirrors. One mirror reflects 100% of the photons that strike it and the other mirror is only partially reflective. Photons that reflect off the mirrors are reflected back into the medium for further amplification. Those photons that transmit through the partially reflective mirror form the laser output.

having shorter wavelengths (600 to 700 nm). There is an "optical window" where the penetration of light energy through the tissues is maximized. This window is contingent on the type and consistency of the tissues.[199] The amount of power—discussed in the next section—also affects the depth of penetration.

* Practical Evidence

Helium-neon (HeNe) lasers have been demonstrated to be the most effective for stimulating tissue healing; gallium aluminum arsenide (GaAlAs) lasers appear to be more effective in reducing pain.[205,206]

The laser's wavelength depends on the medium used (Table 19-2). New evaluation methods have indicated that some low-power lasers can affect tissues up to 2 cm deep secondary to indirect responses, especially when applied over bony prominences such as the cervical spine and skull.[207]

Helium-neon (HeNe) lasers stimulate a mixture of helium and neon gases, producing light with a wavelength of 632.8 nm, within the visible red light range. The maximum output of HeNe lasers is usually 1 mW or less (although some models may produce output between 0.5 to 35 mW), and the energy can penetrate up to 0.8 to 15 mm deep.[208] The indirect effect may produce tissue changes deeper than 15 mm. HeNe lasers typically have an output in the range of 14 to 29 mJ (millijoule, 10^{-3} joule). The clinical availability of HeNe laser is limited because of their relative expense.

Gallium arsenide (GaAs) laser is produced by a semiconductor diode chip and delivers a light wave between 904 to 910 nm. This wavelength places the GaAs laser within the infrared spectrum (invisible to the human eye). The energy can penetrate the tissues up to 2 cm. GaAs lasers may produce up to 2 mW output that is often pulsed, delivering a significantly lower average power than HeNe lasers (see Power and Treatment Dosage). GaAs lasers have a visible light pointing system that illuminates when the laser output is being emitted to target the treatment effects.

Gallium aluminum arsenide (GaAlAs) laser multiple diodes with each diode producing up to 30 mW of power at a wavelength of 830 nm. The outputs of the diodes combine to produce a total treatment output of 90 mW, theoretically producing a deeper depth of penetration.[209] Although multidiode systems produce an increased output, their dosage specifications are unclear.[202]

Power

Dosage, the amount of energy applied to the tissues, is similar to that used for therapeutic ultrasound. The power density of the treatment is expressed in milliwatts per square centimeter (mW/cm²) and is based on the laser output (expressed in mW) and the surface area (circumference) of the emitted energy. This calculation is based on the formula:

$$\text{Power density (mW/cm}^2\text{)} = \text{Watts (mW)/Target area (cm}^2\text{)}$$

Laser output is described in joules, and is the most meaningful output measure when pulsed laser output is used. This calculation factors in the actual amount of time that the energy produced and is expressed in terms of joules per square centimeter (J/cm²):

$$\text{Energy density (J/cm}^2\text{)} = \text{Watts (W)} \times \text{Time (Sec)/Target area (cm}^2\text{)}$$

Treatment Dosage

Most LLLT treatments are based on dose-oriented treatments per unit of area. The treatment duration is based on the energy density (joules per square centimeter [J/cm²]), the average power of the output, and the area of the output beam. Most generators perform the following calculation to determine the treatment duration:

$$\text{Treatment duration} = (\text{J/cm}^2\text{/Average power}) \times \text{Target area (cm}^2\text{)}$$

* Practical Evidence

Similar to therapeutic ultrasound, many LLLT treatments are delivered at a dosage that is well below that expected to evoke biological responses.[210] Proper dosing is required to produce the desired physiological effects.

For example, assume a treatment dosage of 5.0 J/cm² with an average output of 1 mW over an area of 1 square centimeter. The total treatment time would be:

$$\text{Treatment duration} = (5\ \text{J/cm}^2/0.001\ \text{W}) \times 0.01\ \text{cm}^2$$
$$\text{Treatment duration} = 5000 \times 0.01\ \text{cm}^2$$
$$\text{Treatment duration} = 50\ \text{seconds}$$

Common clinical dosage of LLLT ranges from 0.5 to 10.0 J/cm². As with most therapeutic modalities, the Arndth-Schultz principle dictates appropriate dosing (see Appendix A).[211] Insufficient dosages will not stimulate the desired response; doses that are too intense will cause tissue damage or hinder the healing process.

Acute conditions are treated with an output of less than 0.5 J/cm². The dosage for chronic conditions is normally less than 3.0 J/cm² (Table 19-3). Individual

TABLE 19-2 Types of Therapeutic Lasers

Name	Abbreviation	Wavelength (nm)	Light Band
Argon	Ar	488	Blue
Gallium-arsenide	GaAs	904	Infrared
Gallium aluminum arsenide*	GaAlAs	830	Infrared
Helium-neon	HeNe	632.8	Red
Indium gallium aluminum phosphate	$InGaAlPO_4$	670	Red

** Gallium aluminum arsenide can be manipulated to create different wavelengths.*

TABLE 19-3 Treatment Dosing Based on the Pathology

	Inflammatory State	
CONDITION	ACUTE	CHRONIC
Tendinopathy	24 to 30 J	35 to 40 J
Sprains	25 to 30 J	35 to 45 J
Strains	25 to 35 J	35 to 45 J

The applied treatment dosage should be based on the density of the energy (J/cm^2).

This table is provided as a sample. Refer to the manufacturer's recommendation and the current literature for the recommended dosage.

Adapted from McLeod IA. Low-level laser therapy in athletic training. Athl Ther Today. 9:17, 2004.

laser manufacturers publish recommended treatment intensity and durations, although the evidence base for making this decision is currently lacking. On units that combine laser and LED and/or SLD emitters, the dosage and application should be determined only by the number of laser diodes.

EFFECTS ON The Injury Response Process

Laser energy can stimulate tissues at depths up to 2 cm below the surface of the skin.[212,213] Although several studies have demonstrated positive effects of LLLT, the exact mechanism of action has yet to be identified. Current evidence suggests that the biophysical benefits are related to photomechanical or photochemical, rather than photothermal effects.[204] This response is based on the **first law of photobiology,** which states that for light to affect tissue, it must be absorbed by specific receptors (Fig. 19-3).[199]

In human tissues the primary photoreceptors (chromophores) include hemoglobin, COX, **myoglobin** •, and **flavoproteins** •. The energy from the absorbed photons affects the mitochondria, which stimulates the production of adenosine triphosphate (ATP).[205] The energy produced by ATP alters molecular-level activity, including short-term stimulation of the electron transport chain, stimulation of

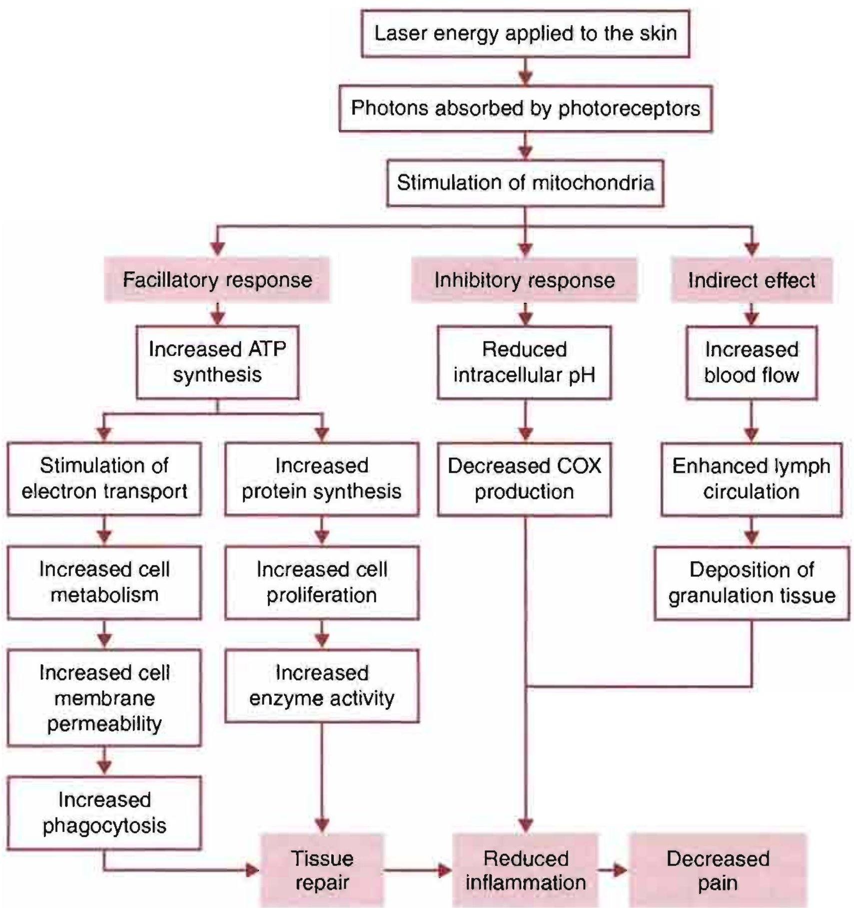


Figure 19-3. **Proposed Mechanism of Low-Level Laser Therapy.**

Myoglobin: A blood-based protein that stores oxygen in the tissues.
Flavoprotein: A protein involved in oxidation within a cell.

the mitochondrial respiratory chain, increased synthesis of adenosine triphosphate (ATP), and a reduction in intracellular pH and COX production.[199,202]

These actions are theorized to affect pain-producing tissue, such as areas of muscle spasm, by restoring the normal properties of muscle tissue via the increased formation of ATP and increased enzyme activity.[213,214] The effects of laser energy are most pronounced when the cells are traumatized, possibly explaining the lack of physiological changes noted in subjects using healthy subjects.[204,209,215]

Inflammation and Tissue Repair

Although the exact mechanism of action is yet to be substantiated, LLLT can produce anti-inflammatory or proinflammatory responses that affects healing. The biophysical effects depend on the type of laser applied and the power applied (Fig. 19-4).[216] There are overlapping areas where multiple effects occur. In the range of approximately 2.5 to 5 J, an anti-inflammatory effect and fibroblast stimulation occurs. Between the range of approximately 7 to 14 J anti-inflammatory effects continue, but fibroblast production is inhibited.[210,216] Anti-inflammatory effects appear to be more pronounced in laser in the high red or infrared bands (e.g., HeNe, GaAs, and GaAlAs).[210]

GaAs lasers promote muscle healing in the active inflammatory phase.[217] HeNe laser applied 2 days post injury and applied at 48-hour intervals is more effective in promoting healing than placebo for the first 21 days.[205]

* Practical Evidence

When applied at a sufficient and appropriate dosage, LLLT yields pain relief and produces anti-inflammatory markers that are significantly better than markers observed using a placebo.[210]

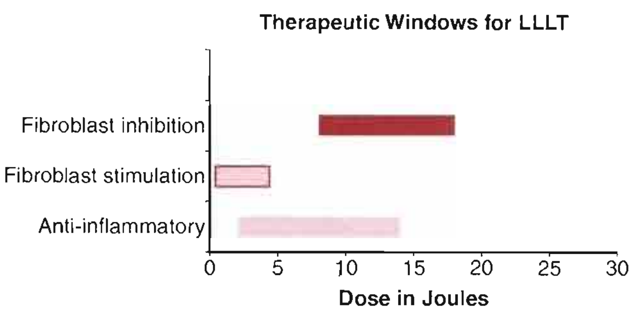


Figure 19-4. **Dosage in Joules Required to Affect Tissue Repair.** Adapted from Lopes-Martin RAB, Penna SC, Joensen J, Iversen VV, et al: Low level laser therapy [LLLT] in inflammatory and rheumatic diseases: A review of therapeutic mechanisms. *Curr Rheumatol Rev.* 3:147, 2007.

Wound Healing

Lasers are used to assist in the healing of superficial wounds including skin ulcerations, surgical incisions, and burns. Because of their relatively low power output, HeNe lasers are less effective in destroying bacteria than indium gallium aluminum phosphate ($InGaAlPO_4$) lasers.[218]

The healing process is enhanced by the accelerated phagocytic activity and the selective destruction of bacteria.[218,219] The absorption of photons is believed to cause increased ATP synthesis that accelerates cell metabolism and encourages the release of free radicals.[218,220] Cell membrane permeability is altered and there is an increase in fibroblast, lymphocyte, and macrophage activity.[221] Blood and lymph circulation is improved in the area surrounding the treatment area, promoting the growth of granulation tissue[222] and the growth of new capillaries.[210] Increased fibroblast proliferation has been associated with LLLT; the response appears to be tissue and wavelength-specific.[204] Laser therapy is also thought to increase collagen content and increase tensile strength of healing wounds.[223]

The bactericidal effects of low-power lasers are enhanced by the preapplication of photosensitizers. These substances, not to be confused with photosensitizing medications such as tetracycline, absorb light having a specific frequency, increasing the intensity of the laser output. The increased laser uptake increases cell membrane permeability and enhances the production of free radicals and **singlet oxygen** •, and selective cell death occurs.[218]

Pain Reduction

LLLT has been used to decrease acute and chronic pain. Proposed mechanisms of pain reduction include altering nerve conduction velocity or decreasing muscle spasm.[224] These pain control approaches have been augmented with the use of sympathetic blocks and antidepressant medication.[225] Low-intensity laser can reduce the rate and velocity of sensory nerve impulses in inflamed nerves, but changes are seldom observed in healthy nerves.[210] Decreased prostaglandin synthesis may also account for decreased pain.[215,216]

Theories on how laser disrupts sensory nerve condition include effects similar to that seen during cold application but without the thermal changes.[208] Low-intensity GaAlAs laser having a wavelength of 830 nm and applied at 9.6 J/cm^2 [227] and HeNe having a wavelength of 632.8 nm applied at 19 mJ/cm^2 [208] reduced the rate of nerve conduction at the site of the treatment and distally. These effects had a significant latency period following the treatment.

Decreased skin resistance indicates the presence of local hypersensitive areas such as trigger points and acupuncture points. HeNe laser applied to cervical trigger points[228] and acupuncture points corresponding to pain caused by

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Singlet oxygen: An uncharged form of oxygen that can selectively destroy cells.

fibromyalgia[229] resulted in increased skin resistance and decreased pain. GaAlAs laser applied at 50 mW (total dose = 2 J) for 1 minute (total dose = 2 J) at each tender joint demonstrated significant decrease in pain and improved clinical function following 10 days of treatment (5 days per week for 2 weeks).[215]

Laser therapy has demonstrated the ability to reduce the pain associated with post-herpetic neuralgia[225] and to resolve neurapraxia.[222] Another possible explanation for pain reduction following laser treatment is the release of endogenous opiates.[210,230]

* Practical Evidence

Laser having a wavelength of 904 nm (GaAs) has demonstrated effectiveness in the short-term relief of pain associated with lateral epicondalygia.[231]

Fracture Healing

Many of the same biophysical effects that assist soft tissue healing are theorized to enhance fracture healing and bone remodeling via an indirect effect. These effects, including increased capillary formation, calcium deposition, increased callus formation, and reduction of hematoma, may be associated with the direct or indirect effect. Photons striking the tissues may create micropressure waves that affect the healing bone in a manner similar to ultrasonic bone growth stimulators.[221,232]

Using an animal model, both GaAs and GaAlAs lasers applied daily at 4.0 J/cm^2 increased the density of the healing callus relative to a control group. However, the longer wavelength GaAs laser group had significantly denser callus than the GaAlAs group, but there was no improvement in the bones' tensile strength.[232,233] Laboratory studies have also demonstrated that GaAlAs improves bony fixation to titanium implants.[234] A higher-power carbon dioxide (CO_2) laser increased the rate of hematoma absorption and the removal of necrotic tissue, leading to enhanced fracture healing in laboratory animals.[221]

The preponderance of studies comparing the effects of laser-induced fracture healing relative to other bone growth stimulation techniques involves animal studies. At this point these studies are limited and inconclusive. One study suggests that there is no difference in healing between laser and pulsed ultrasound (US),[235] while other studies suggest improved healing relative to pulsed US.[236,237] These limited results prevent them from being applied to human bone growth stimulation.

Contraindications and Precautions

Laser produces nonionizing radiation, greatly reducing the possibility of causing permanent damage to cellular structures or damaging DNA. The retina is sensitive to low-power laser exposure; even brief (1 second) contact can result in permanent damage to the retina. Infrared energy is invisible, negating the eye's protective blink reflex. Depending on the type of laser being used, appropriate safety goggles should be worn by both the patient and the clinician (refer to the manufacturer's instructions regarding eye protection). Because of the risk of increasing the rate of cancerous cell growth, low-power laser therapy must not be applied to tumors or cancerous lesions.

Some medications such as tetracycline, antihistamines, oral contraceptives, and antidepressants increase the skin's sensitivity to sunlight. In these cases begin the patient with a below normal LLLT dosage and note any adverse reaction. If a question exists about the potential contraindication to laser therapy, contact the patient's physician or a pharmacist.

Some tattoo inks function as a photosensitizer, potentially predisposing the patient to adverse treatment effects. When possible, avoid the application of laser directly over tattoos. If direct application over tattoos is unavoidable, decrease the intensity of the initial treatment and monitor the patient for signs of "burning" (similar to a sunburn) or increased inflammation. These signs may take up to 48 hours to be seen.

Overview of the Evidence

LLLT is an evolving treatment approach that must be considered in the context of unclear treatment protocol, different forms of laser energy applied, conflicting results, and unknown biophysical effects. Although the evidence is building to support the use of LLLT, it is difficult to make an informed decision because of methodological issues associated with several studies. The application of laser energy to the tissues with a power well below that needed to evoke physiological changes is perhaps the single largest confounder in substantiating the efficacy of laser therapy.[210] Another confounding factor is that healthy tissue appears not to react to laser energy, negating the findings of studies performed on healthy subjects.[204,210,215]

Although therapeutic laser has been used to control pain and otherwise alter nerve conduction velocity, research studies using various types of lasers and a range of output parameters have not significantly substantiated this effect.[202,209,212,239] The anti-inflammatory benefits of GaAs laser was substantiated by a well-designed, controlled study.[226] However, several prior studies have concluded that laser treatment was not effective in treating musculoskeletal pain[210] including myofascial pain,[240] lateral epicondylagia,[239] traumatic orthopedic pain,[241] rheumatoid arthritis,[230] and tooth extraction.[242] Two early meta-analyses of the effect of laser therapy in treating orthopedic and skin conditions strongly suggest that this is not an effective modality in the treatment of these conditions.[243,244]

A study investigating the effects of HeNe laser, GaAs laser, and standard treatment protocol on pain and range of motion associated with **tendinopathies** demonstrated that all three treatment groups improved over a 2-week period, but the laser treatment groups had no significant benefits relative to the control group.[245] Other studies, however, do demonstrate improved function and reduced pain following LLLT.[215,231,246]

Several studies investigating the effect of laser on wound healing have questioned the efficacy of this technique.[204] Using human subjects, no significant difference in the healing rates of chronic venous leg ulcers was found between a group receiving standard treatment protocol augmented by HeNe laser (applied at 6 mW) and a group receiving standard treatment and sham laser.[247] A laboratory study investigating the healing characteristics of straight-line incisions in rat skin concluded that HeNe laser application demonstrated slight increases in tensile strength and other healing measures early, but these differences were only statistically significant, not clinically significant. There was no long-term difference in healing characteristics between irradiated and nonirradiated groups.[248]

Although cellular level effects and increased callus formation in fractures treated with GaAs and GaAlAs,[232] and CO_2 [221] lasers have been identified, these effects were not replicated using the more common HeNe lasers applied at 2 or 4 J.[211] The evidence that laser improves fracture healing is lacking.[232]

The lack of a substantiated biological mechanism of action in response to LLLT, identification of the specific wavelength for use with certain conditions, and evidence of adequate treatment dosages must be addressed for LLLT to be accepted by mainstream medicine.[210]

Clinical Application of Therapeutic Lasers

Setup and Application

The setup, application, and dosage are device-specific. Refer to the user's manual for the exact application procedures. The following is provided as a general overview and should not replace formal training on the device being used. Most units have preprogrammed protocol based on the clinical problem being treated (e.g., pain, muscle spasm, wound healing).

Laser therapy should be administered in a space that prevents unintended exposure to laser output by others in the facility. Class II and III lasers should be administered only by those who have been trained to use these devices.[238]

Instrumentation

Although there are a limited number of laser units marketed in the United States, the available functions and types of laser output produced (e.g., HeNe, GaAs) create diversity in the instrumentation. When units produced outside of the United States are considered, the difference in instrumentation becomes even greater.

Timer: Selects the duration of the treatment. On some units, the timer function may be overridden by selecting the **MANUAL** switch.

Frequency: For pulsed output, adjusts the frequency or duration, or both, of the laser pulses. Do not confuse this parameter with the output frequency of the laser. Wavelength is the best description of the energy being administered.

Source: Selects the type of laser, typically GaAlAs, GaAs, or HeNe.

Power: Adjusts the output in watts. The total amount of energy is equal to the output wattage and the treatment duration (joules = power × duration). The total output per unit of area is measured as joules per square centimeter (J/cm^2).

Preparation of the Generator

1. If applicable, clean the laser lens(es) with an approved cleaner and/or polish.
2. Determine the treatment dosage and technique to be administered during the treatment.
3. Select the appropriate size laser applicator head.
4. If laser is being applied to open wounds, cover the applicator face with a clear plastic wrap to prevent transmission of contaminants. This technique results in the loss of approximately 8% of the total laser energy.[249]
5. Select the desired power output display. Joules per square centimeter (J/cm^2) is the recommended output measure.

Preparation of the Patient

1. Assure that the patient is free on any contraindications to the application of LLLT.
2. If applicable to the type of laser being used, the patient and clinician should wear goggles.
3. Clean the area to be treated with soap and water or alcohol swabs. Allow the area to dry thoroughly before initiating the treatment.
4. Cryotherapy may be administered prior to LLLT. The decreased blood flow and decreased tissue profusion are believed to increase the depth of laser penetration and decrease the inflammatory effects of the treatment. Heating the area prior to the application increases blood flow, thereby decreasing the depth of penetration and increasing the inflammatory effects.[200]
5. Determine the application technique to be used:

   Point technique: Laser is applied to predetermined points for a duration sufficient to deliver the appropriate amount of energy

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Tendinopathy: Any disease or trauma involving a muscle's tendon, tissues surrounding the tendon, or the tendon's insertion into the bone.

At a Glance: Therapeutic "Cold" Lasers

Description

Laser is a highly organized form of ultraviolet, visible, or infrared light. Photons that are absorbed by the cells produce direct changes in their function. Indirect effects occur secondary to photochemical events.

Indications

- Wound healing
- Fracture healing
- Musculoskeletal pain
- Myofascial pain/fibromyalgia[215]
- Trigger points
- Inflammatory conditions
- Osteoarthritis
- Rheumatoid arthritis
- Arthritis
- Carpal tunnel syndrome

Primary Effects

- Altered nerve conduction velocity
- Vasodilation of microvessels
- Increased ATP production
- Increased collagen production
- Increased macrophage activity

Contraindications

- Application to the eyes
- Application over the thyroid gland
- High-intensity application over areas of hemorrhage
- Over areas of active deep vein thrombosis or thrombophlebitis
- Over cancerous areas
- Do not apply to the low back or abdomen during pregnancy
- Do not apply to the testicles

Treatment Duration

The treatment duration depends on the type of laser being used (e.g., helium-neon), the pathology being treated, and the power of the output.

Precautions

- LLLT should not be applied within 6 months of radiation therapy.
- Because of unknown effects, lasers should not be applied over unfused epiphyseal plates, or be administered to small children.
- The patient may experience dizziness during the treatment. If this occurs, discontinue the treatment. If the episode recurs, laser therapy should not be applied to the patient.
- Caution should be used with patients who are taking medications that increase sensitivity to light including certain antihistamines, oral contraceptives, NSAIDs, tetracyclines, and antidepressants.
- Some tattoo inks may increase the absorption of laser energy.

Grid technique: Used for larger areas, an imaginary grid with points spaced 1 cm apart is placed over the treatment area. The laser is then applied to each point.[200]

Scanning technique: This method of laser application resembles traditional US application. The laser probe is slowly moved over the target tissues until the desired treatment dosage is reached. This method of application can decrease energy transmission.

6. When treating a joint, position it in the open-packed position (usually flexion) within patient comfort.

Initiation of the Treatment

1. Determine the treatment **DOSAGE** in J/cm^2.
2. Select the treatment **DURATION** (seconds). If the generator uses a dose-oriented treatment, the output (J/cm^2) will change in response to changes in the treatment duration.
3. Select the output mode. If the pulsed mode is selected, use the **FREQUENCY** control to select the number of pulses. Low pulse frequencies (1 to 20 pps) are used to promote tissue healing; pain is treated with a frequency greater than 20 pps.
4. View the dosage based on the treatment duration, frequency, and duty cycle used. If the dosage is out of range of the desired treatment parameters make adjustments as needed.
5. Hold the applicator so that the laser energy strikes the skin at a 90-degree angle.
6. Press the **START** button. If the laser head requires charging a countdown timer will indicate the time until the treatment actually begins.
7. Unless otherwise indicated, the applicator should remain in contact with the patient's skin throughout the treatment.

Maintenance

Following Each Treatment

1. Clean the head using a manufacturer-approved cleanser.

At Regular Intervals

1. Check all cords and cables for kinks, frays, and cuts.
2. Check lens for dirt, grime, and/or oil buildup.

Annually

1. Have the unit inspected and calibrated by an authorized technician. LED lasers may need to be recalibrated every 6 months.[238]

End of Section

Case Study: Bertha

Bertha is a 62-year-old female who sustained a right wrist fracture 6 weeks ago, which was medically managed with immobilization of a fiberglass cast. The cast was removed 3 days ago by the physician, and today you are the treating therapist for this patient. Her primary limitations are pain 4/10 rest, 10/10 activity; greater than 50% limited active and passive wrist range of motion; and poor strength due to pain and edema. Her edema measures 3 cm greater midpalm right versus left. She is medically healthy, except for a history of ovarian cancer 20 years ago that was treated successfully.

1. What are some edema management options to consider in this situation?
2. What are the physiological effects on the injury response cycle from the application of this thermal agent?
3. What are the clinical symptoms that you hope to address with this intervention?
4. What other interventions may be appropriate over the following 2 weeks for this diagnosis? Why?

Case Study: Sam

Sam is a 40-year-old man who is diagnosed with a herniated lumbar disk with left leg radiculopathy. He has mild osteoarthritis. His evaluation shows limited trunk active range of motion, moderate spasm in his lumbosacral paraspinals, and pain that radiates into the left buttock. The physician would like him to receive a course of treatment of lumbar traction.

1. What are the indications for traction?
2. What are the physiological effects on the injury response cycle from the application of this agent?
3. What are the clinical symptoms that you hope to address with this intervention?
4. What other thermal modalities may be appropriate over the following 2 weeks for this diagnosis? Why?

Case Study: Mr. Smith

Mr. Smith is a 77-year-old man with 10 years of progressive degenerative joint disease at the knee. He is currently 2 days out of surgery for a total knee arthroplasty. A continuous passive machine is requested by the physician.

1. What are three contraindications to the use of a continuous passive machine?
2. What are the physiological effects on the injury response cycle from the application of this thermal agent?
3. What are the clinical symptoms that you hope to address with this intervention?

Case Study: Continuation of Case Study From Section 1

(The following discussion relates to Case Study 2 in Section 1.)

Two of the modalities presented in this section, cervical traction and massage, would be appropriate for our patient's cervical trauma.

Massage

Soft tissue massage using effleurage and pétrissage strokes can promote relaxation of the involved muscles. Depending on the clinician's preference, the patient could be placed in the supine, prone, or seated position, with the head resting on a table to promote relaxation. The massage strokes should run parallel to the muscle fibers to help lengthen them. The patient would further benefit from massage by increased local blood flow and decreased neuromuscular excitability. Deep, localized friction massage can be used to help break up trigger points.

Cervical Traction

Cervical traction would be used only in the later stages of this patient's treatment protocol. Recall that the patient has been diagnosed as having a cervical strain and sprain. If traction were used too soon after the injury, the force may cause further damage to the cervical ligaments. Likewise, the patient does not show signs of radiating pain, decreasing the likelihood of cervical nerve root impingement.

Intermittent cervical traction, applied in two 5-minute intervals with a maximum of 25 pounds of tension, will assist in decreasing muscle spasm and pain, especially if the treatment is preceded by the application of moist heat packs. Placing the patient in the supine position lowers the amount of tension needed to elongate the muscles by decreasing motor activity in the cervical musculature.

Section 5 Quiz

1. All of the following effects have been attributed to continuous passive motion (CPM) except:
   A. Increased nutrition to the meniscus
   B. Increased nutrition to the articular cartilage
   C. Increased tensile strength of tendons and allografts
   D. Increased nutrition to the anterior cruciate ligament

2. Intermittent cervical traction can be useful in relieving the pain associated with intervertebral disk herniations. This reduction of pain occurs by reducing the bulge of the __________ through the __________.
   A. Fibrous pulposus • nucleus pulposus
   B. Annulus fibrosus • nucleus pulposus
   C. Nucleus pulposus • annulus fibrosus
   D. Nucleus pulposus • fibrous pulposus

3. When applying intermittent compression to an extremity, the pressure in the appliance should not exceed:
   A. The diastolic blood pressure
   B. The systolic blood pressure
   C. The difference between the diastolic and systolic blood pressure
   D. The resting heart rate

4. Electromyographic biofeedback measures:
   A. The amount of tension produced by a muscle group
   B. The amount of electrical activity within a muscle
   C. The amount of myelin activity within a muscle
   D. All of the above

5. Which of the following techniques produces the greatest amount of femoral blood flow?
   A. Pneumatic sleeve
   B. Manual calf compression
   C. Straight-leg raises
   D. Anatomic CPM

6. All of the following are indications for the use of intermittent compression except:
   A. Postsurgical edema
   B. Gangrene
   C. Lymphedema
   D. Venous stasis ulcers

7. Which of the following types of continuous passive motion designs provides for the most joint stability?
   A. Free linkage
   B. Anatomical
   C. Nonanatomical

8. Light having a wavelength of 780 to 12,500 nm would be classified as:
   A. Ultraviolet
   B. Blue
   C. Red
   D. Infrared

9. Therapeutic laser is being applied at a total of 5 watts for 10 seconds over an area of 10 square centimeters. What is the energy density (J/cm²)?
   A. 5 J/cm²
   B. 25 J/cm²
   C. 0.5 J/cm²
   D. 50 J/cm²

10. The depth that laser energy penetrates into the body is related to:
    A. Total watts
    B. Duty cycle
    C. Wavelength
    D. Total joules

11. The body's fascia can be elongated using a ____________ force.
    A. Quick, high-intensity
    B. Slow, high-intensity
    C. Quick, moderate-intensity
    D. Slow, moderate-intensity

12. In addition to the amount of force applied, what other parameters influence the effect of cervical traction?
    A.
    B.
    C.
    D.

13. List two reasons why separation of the vertebral column occurs at a lower percentage of the patient's body weight in the reclining position than in the sitting position.
    A.
    B.

14. Match the following massage strokes to method of delivery:
    A. Pétrissage ________ Pounding of the skin
    B. Tapotement ________ Kneading of the skin
    C. Effleurage ________ Stroking of the skin

References

1. Capps SG: Cryotherapy and intermittent pneumatic compression for soft tissue trauma. *Athl Ther Today.* 14:2, 2009.
2. Partsch H: Intermittent pneumatic compression in immobile patients. *Int Wound J.* 5:389, 2008.
3. Pierce C, McLeod KJ: Feasibility of treatment of lower limb edema with calf muscle pump stimulation in chronic heart failure. *Eur J Cardiovasc Nurs.* 8:345, 2009.
4. Goddard AA, Pierce CS, McLeod KJ: Reversal of lower limb edema by calf muscle pump stimulation. *J Cardiopulm Rehabil Prev.* 28:174, 2008.
5. Muhe E: Intermittent sequential high-pressure compression of the leg: A new method of preventing deep vein thrombosis. *Am J Surg.* 147:781, 1984.
6. Olavi A, et al: Edema and lower leg perfusion in patients with posttraumatic dysfunction. *Acupunct Electrother Res.* 16:7, 1991.
7. Miranda F, et al: Effect of sequential intermittent pneumatic compression on both leg lymphedema volume and on lymph transport as semi-quantitatively evaluated by lymphoscintigraphy. *Lymphology.* 34:135, 2001.
8. Rucinski TJ, et al: The effects of intermittent compression on edema in postacute ankle sprains. *J Orthop Sports Phys Ther.* 14:65, 1991.
9. Iwama H, Obara S, Ohmizo H: Changes in femoral blood flow velocity by intermittent pneumatic compression: calf compression device versus plantar-calf sequential compression device. *J Anesth.* 18:232, 2004.
10. Stöckle U, et al: Fastest reduction of posttraumatic edema: Continuous cryotherapy or intermittent impulse compression? *Foot Ankle Int.* 18:432, 1997.
11. Hopkins JT, et al: Cryotherapy and transcutaneous electric neuromuscular stimulation decrease arthrogenic muscle inhibition of the vastus medialis after knee joint effusion. *J Athl Train.* 37:25, 2001.
12. Hopkins JT, Ingersoll CD: Arthrogenic muscle inhibition: A limiting factor in joint rehabilitation. *J Sports Rehabil.* 9:135, 2000.
13. Hopkins JT, et al: The effects of cryotherapy and TENS on arthrogenic muscle inhibition of the quadriceps. *J Athl Train.* 36:S49, 2001.
14. Fanelli G, Zasa M, Baciarello M, et al: Systemic hemodynamic effects of sequential pneumatic compression of the lower limbs: A prospective study in healthy volunteers. *J Clin Anesth.* 20:388, 2008.
15. Boris M, et al: The risk of genital edema after external pump compression for lower limb lymphedema. *Lymphology.* 31:15, 1998.
16. Gilbart MK, et al: Anterior tibial compartment pressures during intermittent sequential pneumatic compression therapy. *Am J Sports Med.* 23:769, 1995.
17. Wright RC, Yacoubian SV: Sequential compression device may cause peroneal nerve palsy. *Orthopedics.* 9:444, 2010.
18. Mayrovitz HN, Macdonald J, Davey S, et al: Measurement decisions for clinical assessment of limb volume changes in patients with bilateral and unilateral limb edema. *Phys Ther.* 87:1362, 2007.
19. Segers P, Belgrado JP, Leduc A, et al: Excessive pressure in multichambered cuffs used for sequential compression therapy. *Phys Ther.* 82:1000, 2002.
20. Salter RB: The biologic concept of continuous passive motion of synovial joints: The first 18 years of basic research. *Clin Orthop.* 12, May, 1989.
21. McCarthy MR, et al: The clinical use of continuous passive motion in physical therapy. *J Orthop Sports Phys Ther.* 15:132, 1992.
22. Saringer J: Engineering aspects of the design and construction of continuous passive motion devices for humans. In Salter RB (ed): Continuous Passive Motion (CPM): A Biological Concept for the Healing and Regeneration of Articular Cartilage, Ligaments, and Tendons—From Origination to Research to Clinical Applications. Williams & Wilkins, Baltimore, 1993, pp 403–410.
23. Diehm SL: The power of CPM: Healing through motion. *Patient Care* 8:34, 1989.
24. O'Donoghue PC, et al: Clinical use of continuous passive motion in athletic training. *J Athl Train.* 26:200, 1991.
25. Jordan LR, et al: Early flexion routine: An alternative method of continuous passive motion. Clin Orthop. 231, June, 1995.

26. Ferretti M, Srinivasan A, Deschner J, et al: Anti-inflammatory effects of continuous passive motion on meniscal fibrocartilage. *J Orthop Res*. 23:1165, 2005.
27. Williams JM, et al: Continuous passive motion stimulates repair of rabbit knee cartilage after matrix proteoglycan loss. *Clin Orthop*. 252, July, 1994.
28. Plessis MD, Eksteen E, Jenneker A, et al: The effectiveness of continuous passive motion on range of motion, pain and muscle strength following rotator cuff repair: A systematic review. *Clin Rehabil*. 25:291, 2011.
29. McInnes J, et al: A controlled evaluation of continuous passive motion in patients undergoing total knee arthroplasty. *JAMA*. 268:1423, 1992.
30. O'Driscoll SW, Nicholas JG: Continuous passive motion (CPM): Theory and principles of clinical application. *J Rehabil Res Dev*. 37:178, 2000.
31. Ring D, et al: Continuous passive motion following metacarpophalangeal joint arthroplasty. *J Hand Surg A]*. 23:505, 1998.
32. LaStayo PC, et al: Continuous passive motion after repair of the rotator cuff. A prospective outcome study. *J Bone Joint Surg Am*. 80:1002, 1998.
33. Trumble T, Vedder NB, Seiler JG, et al: Zone-IT flexor tendon repair: A randomized prospective trial of active place-and-hold-therapy compared with passive motion therapy. *J Bone Joint Surg*. 92A:1381, 2010.
34. Gates HS, et al: Anterior capsulotomy and continuous passive motion in the treatment of posttraumatic flexion contracture of the elbow: A prospective study. *J Bone Joint Surg Am*. 74:1229, 1992.
35. Nadler SF, et al: Continuous passive motion in the rehabilitation setting: A retrospective study. *J Phys Med Rehabil*. 72:162, 1993.
36. Montgomery F, Eliasson M: Continuous passive motion compared to active physical therapy after knee arthroplasty: Similar hospitalization times in a randomized study of 68 patients. *Acta Orthop Scand*. 67:7, 1996.
37. Chiarello CM, et al: The effect of continuous passive motion duration and increment on range of motion in total knee arthroplasty patients. *J Orthop Sports Phys Ther*. 25:119, 1997.
38. McNair PJ, et al: Stretching at the ankle joint: Viscoelastic responses to holds and continuous passive motion. *Med Sci Sports Exerc*. 33:354, 2001.
39. London N, et al: Continuous passive motion: Evaluation of a new portable low cost machine. *Physiother*. 85:610, 1999.
40. Chin B, et al: Continuous passive motion after total knee arthroplasty. *Am J Phys Med Rehabil*. 79:421, 2000.
41. Ververeli PA, et al: Continuous passive motion after total knee arthroplasty: Analysis of costs and benefits. *Clin Orthop*. 208, December, 1995.
42. Lachiewicz PF: The role of continuous passive motion after total knee arthroplasty. *Clin Orthop*. 380:144, 2000.
43. Harvey LA, Brosseau L, Herbert RD: Continuous passive motion following total knee arthoplasty in people with arthritis. *Cochrane Database Sys Rev*. 2:CD004260, 2003.
44. Leach W, Reid J, Murphy F: Continuous passive motion following total knee replacement: A prospective randomized trial with follow-up to 1 year. *Knee Surg Sports Traumatol Arthrosc*. 14:922, 2006.
45. Lenssen TAF, van Steyn MJA, Crijins YHF, et al: Effectiveness of prolonged use of continuous passive motion (CPM), as an adjunct to physiotherapy, after total knee arthroplasty. *BMC Musculoskelet Disord*. 9:60, 2008.
46. Bruun-Olsen V, Heiberg KE, Mengshoel AM: Continuous passive motion as an adjunct to active exercises in early rehabilitation following total knee arthroplasty—a randomized controlled trial. 31:277, 2009.
47. Flowers KR, LaStayo, P: Effect of total end range time on improving passive range of motion. *J Hand Ther*. 7:150, 1994.
48. Wright A, et al: An investigation of the effect of continuous passive motion and lower limb passive movement on heart rate in normal volunteers. *Physiother Theory Pract*. 9:13, 1993.
49. Gershuni DH, et al: Regional nutrition and cellularity of the meniscus. Implications for tear and repair. *Sports Med*. 5:322, 1988.
50. Kim HKL, et al: The potential for regeneration of articular cartilage in defects created by chondral shaving and subchondral abrasion: An experimental investigation in rabbits. *J Bone Joint Surg Am*. 73:1301, 1991.
51. Kim HKW, et al: Effects of continuous passive motion and immobilization on synovitis and cartilage degeneration in antigen induced arthritis. *J Rheumatol*. 22:1714, 1995.
52. Alfredson H, Lorentzon R: Superior results with continuous passive motion compared to active motion after periosteal transplantation. A retrospective study of human patella cartilage defect treatment. *Knee Surge Sports Traumatol*. 7:232, 1999.
53. Mussa R, et al: Condylar cartilage response to continuous passive motion in adult guinea pigs: A pilot study. *Am J Orthod Dentofacial Orthop*. 115:360, 1999.
54. Moran ME, Salter RB: Biological resurfacing of full-thickness defects in patellar articular cartilage of the rabbit. Investigation of autogenous periosteal grafts subjected to continuous passive motion. *J Bone Joint Surg Br*. 74:659, 1992.
55. Namba RS, et al: Continuous passive motion versus immobilization: The effect on posttraumatic joint stiffness. *Clin Orthop*. 218, June, 1991.
56. Von Schroeder HP, et al: The changes in intramuscular pressure and femoral vein flow with continuous passive motion, pneumatic compression stockings, and leg manipulations. *Clin Orthop*. 218, May, 1991.
57. Grumbine NA, et al: Continuous passive motion following partial ankle joint arthroplasty. *J Foot Surg*. 29:557, 1990.
58. Mullaji AB, Shahane MN: Continuous passive motion for prevention and rehabilitation of knee stiffness: A clinical evaluation. *J Postgrad Med*. 35:204, 1989.
59. Giudice ML: Effects of continuous passive motion and elevation on hand edema. *Am J Occup Ther*. 44:914, 1990.
60. Dirette D, Hinojosa J: Effects of continuous passive motion to the edematous hands of two persons with flaccid hemiplegia. *Am J Occup Ther*. 48:403, 1994.
61. Macdonald SJ, et al: Prospective randomized clinical trial of continuous passive motion after total knee arthroplasty. *Clin Orthop*. 380:30, 2000.
62. Barthelet Y, et al: Effects of perioperative analgesic technique on the surgical outcome and duration of rehabilitation after major knee surgery. *Anesthesiology*. 91:8, 1999.
63. Kannus P: Immobilization or early mobilization after an acute soft-tissue injury? *Phys Sports Med*. 28:55, 2000.
64. Skyhar MJ, et al: Nutrition of the anterior cruciate ligament: Effects of continuous passive motion. *Am J Sports Med*. 13:415, 1985.
65. Smith TO, Davies L: The efficacy of continuous passive motion after anterior cruciate ligament construction: A systematic review. *Phys Ther Sport*. 8:141, 2007.
66. Wright RW, et al: A systematic review of anterior cruciate ligament reconstruction rehabilitation. Part I: Continuous passive motion, early weight bearing, postoperative

bracing, and home-based rehabilitation. *J Knee Surg*. 21:217, 2008.
67. Wright RW, Preston E, Flemming BC, et al: A systematic review of anterior cruciate ligament reconstruction rehabilitation. Part I: Continuous passive motion, early weight bearing, postoperative bracing, and home-based rehabilitation. *J Knee Surg*. 21:217, 2008.
68. Engstrom B, et al: Continuous passive motion in rehabilitation after anterior cruciate ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc*. 3:18, 1995.
69. McCarthy MR, et al: The effect of immediate continuous passive motion on pain during the inflammatory phase of soft tissue healing following anterior cruciate ligament reconstruction. *J Orthop Sports Phys Ther*. 17:96, 1993.
70. Witherow GE, et al: The use of continuous passive motion after arthroscopically assisted anterior cruciate ligament reconstruction: Help or hindrance? *Knee Surg Sports Traumatol Arthrosc*. 1:68, 1993.
71. Zarnett R, et al: The effect of continuous passive motion on knee ligament reconstruction with carbon fiber. *J Bone Joint Surg Br*. 73:47, 1991.
72. Drez D, et al: In vivo measurement of anterior tibial translation using continuous passive motion devices. *Am J Sports Med*. 19:381, 1991.
73. Rosen MA, et al: The efficacy of continuous passive motion in the rehabilitation of anterior cruciate ligament reconstructions. *Am J Sports Med*. 20:122, 1992.
74. McCarthy MR, et al: Effects of continuous passive motion on anterior laxity following ACL reconstruction with autogenous patellar tendon grafts. *J Sports Rehabil*. 2:171, 1993.
75. Wasilewski SA, et al: Value of continuous passive motion in total knee arthroplasty. *Orthopedics*. 13:291, 1990.
76. Yashar AA, et al: Continuous passive motion with accelerated flexion after total knee arthroplasty. *Clin Orthop*. 345:38, 1998.
77. Chen B, et al: Continuous passive motion after total knee arthroplasty: A prospective study. *Am J Phys Med Rehabil*. 79:421, 2000.
78. Sperber A, Wredmark T: Continuous passive motion in rehabilitation after anterior cruciate ligament reconstruction. *Knee Surg Sports Traumatol*. 3:18, 1995.
79. Richmond JC, et al: Continuous passive motion after arthroscopically assisted anterior cruciate ligament reconstruction: Comparison of short- versus long-term use. *Arthroscopy*. 7:39, 1991.
80. Takai S, et al: The effects of frequency and duration of controlled passive mobilization on tendon healing. *J Orthop Res*. 9:705, 1991.
81. Graham G, Loomer RL: Anterior compartment syndrome in a patient with fracture of the tibial plateau treated by continuous passive motion and anticoagulants: Report of a case. *Clin Orthop*. 197, May, 1985.
82. Bible JE, Simpson AK, Biswas D, et al: Actual knee motion during continuous passive motion protocols is less than expected. *Clin Orthop Relat Res*. 467:2656, 2009.
83. LaBan MM, et al: Intermittent cervical traction: A progenitor of lumbar radicular pain. *Arch Phys Med Rehabil*. 73:295, 1992.
84. Saunders H: The use of spinal traction in the treatment of neck and back conditions. *Clin Orthop Rel Res*. 179:31, 1983.
85. Roberts S, Evans H, Trivedi J, et al: Histology and pathology of the human intervertebral disc. *J Bone Joint Surg Am*. 88:S10, 2006.
86. Lundon K, Bolton K: Structure and function of the lumbar intervertebral disk in health, aging, and pathologic conditions. *J Orthop Sports Phys Ther*. 31:291, 2001.
87. Murakami H, Yoon TS, Attallah-Wasif ES, et al: Quantitative differences in intervertebral disc-matrix composition with age-related degeneration. *Med Biol Eng Comput*. 48:469, 2010.
88. Sari H, Akarirmak U, Karacan I, et al: Computed tomographic evaluation of lumbar spinal structures during traction. *Physiother Theory Pract*. 21:3, 2005.
89. Ozturk B, Gunduz OH, Ozoran K, et al: Effect of continuous lumbar traction on the size of herniated disc material in lumbar disc herniation. *Rheumatol Int*. 26:622, 2006.
90. Tekeoglu I, et al: Distraction of lumbar vertebrae in gravitational traction. *Spine*. 23:1061, 1998.
91. Moeti P, Marchetti G: Clinical outcome from mechanical intermittent cervical traction for the treatment of cervical radiculopathy: A case series. *Phys Ther*. 31:207, 2001.
92. Meszaros TF, et al: Effect of 10%, 30%, and 60% body weight traction on the straight leg raise test of symptomatic patients with low back pain. *J Orthop Sports Phys Ther*. 30:595, 2000.
93. Yang KH, King AI: Mechanism of facet load transmission as a hypothesis for low-back pain. *Spine*. 9:557, 1984.
94. Judovich B, Nobel GR: Traction therapy: A study of resistance forces. *Am J Surg*. 93:108, 1957.
95. Werners R, et al: Randomized trial comparing interferential therapy with motorized lumbar traction and massage in the management of low back pain in a primary care setting. *Spine*. 24:1579, 1999.
96. Wong AM, et al: Clinical trial of a cervical traction modality with electromyographic biofeedback. *Am J Phys Med Rehabil*. 76:19, 1997.
97. Walker GL: Goodley polyaxial cervical traction: A new approach to a traditional treatment. *Phys Ther*. 66:1255, 1986.
98. Demir T, Canakci V, Eltas A, et al: Effectiveness of mouthguards on tooth pain and mobility in cervical traction treatment. *J Back Musculoskelet Rehabil*. 21:91, 2008.
99. Fater DCW, Kernozek TW: Comparison of cervical vertebral separation in the supine and seated positions using home traction units. *Physiother Theory Pract*. 24:430, 2008.
100. Wong AM, et al: The traction angle and cervical intervertebral separation. *Spine*. 17:136, 1992.
101. Chung CT, Tsai SW, Chen CJ, et al: Comparison of the intervertebral disc spaces between axial and anterior lean cervical traction. *Eur Spine J*. 18:1669, 2009.
102. Deets D, et al: Cervical traction: A comparison of sitting and supine positions. *Phys Ther*. 57:225, 1977.
103. Fater DC, Kernozek TW: Comparison of cervical vertebral separation in the supine and seated position using home traction units. *Physiother Theory Pract*. 24:430, 2008.
104. Liu J, Ebraheim NA, Sanford CG, et al: Quantitative changes in the cervical neural foramen resulting from axial traction: In vivo imaging study. *Spine J*. 8:619, 2008.
105. Raney NH, Peterson EJ, Smith TA, et al: Development of a clinical prediction rule to identify patients with neck pain likely to benefit from cervical traction and exercise. *Eur Spine J*. 18:382, 2009.
106. Young IA, Michener LA, Cleland JA, et al: Manual therapy, exercise, and traction for patients with cervical radiculopathy: A randomized clinical trial. *Phys Ther*. 89:632, 2009.
107. Jette DU, et al: Effect of intermittent, supine cervical traction on the myoelectric activity of the upper trapezius muscle in subjects with neck pain. *Phys Ther*. 65:1173, 1985.
108. Murphy, MJ: Effects of cervical traction on muscle activity. *J Orthop Sports Phys Ther*. 13:220, 1991.

109. Bradnam L, et al: Manual cervical traction reduces alpha-motoneuron excitability in normal subjects. *Electromyogr Clin Neurophysiol.* 40:259, 2000.
110. DeLacerda FG: Effect of angle of traction pull on upper trapezius muscle activity. *J Orthop Sports Phys Ther.* 1:205, 1980.
111. Borman P, Keskin D, Ekici B, et al: The efficacy of intermittent cervical traction in patients with chronic neck pain. *Clin Rheumatol.* 27:1249, 2008.
112. Cleland JA, Fritz JM, Whitman JM, et al: Predictors of short-term outcome in people with a clinical diagnosis of cervical radiculopathy. *Phys Ther.* 87:1619, 2007.
113. Latimer EA, et al: Tear of the cervical esophagus following hyperextension from manual traction: Case report. *J Trauma.* 31:1448, 1991.
114. Simmers TA, et al: Internal jugular vein thrombosis after cervical traction. *J Int Med Res.* 241:333, 1997.
115. Bridger RS, et al: Effect of lumbar traction on stature. *Spine.* 15:522, 1990.
116. Letchuman R, Deusinger RH: Comparison of sacrospinalis myoelectric activity and pain levels in patients undergoing static and intermittent lumbar traction. *Spine.* 18:1361, 1993.
117. Harrison DE, Cailliet R, Harrison DD, et al: A review of biomechanics of the central nervous system—Part III: Spinal cord stresses from postural loads and their neurologic effects. *J Manipulative Physiol Ther.* 22:399, 1999.
118. Saunders HD: Unilateral lumbar traction. *Phys Ther.* 61:221, 1981.
119. Cai C, Pua YH, Lim KC: A clinical prediction rule for classifying patients with low back pain who demonstrate short-term improvement with mechanical lumbar traction. *Eur Spine J.* 18:554, 2009.
120. Cholewicki J, Lee AS, Reeves NP, et al: Trunk muscle response to various protocols of lumbar traction. *Man Ther.* 14:562, 2009.
121. Ramos G, Martin W: Effects of vertebral axial decompression on intradiscal pressure. *J Neurosurg.* 81:350, 1994.
122. Beattie PF, Nelson RM, Michener LA, et al: Outcomes after a prone lumbar traction protocol for patients with activity-limiting low back pain: A prospective case series study. *Arch Phys Med Rehabil.* 89:269, 2008.
123. Beurskens AJ, et al: Efficacy of traction for nonspecific low back pain. 12-week and 6-month results of a randomized clinical trial. *Spine.* 22:2756, 1997.
124. Beurskens AJ, et al: Efficacy of traction for non-specific low back pain. A randomized clinical trial. *Lancet.* 346:1596, 1995.
125. Onel D, et al: Computer tomographic investigation of the effect of traction on lumbar disc herniations. *Spine.* 14:82, 1989.
126. Ljunggren AE, et al: Manual traction versus isometric exercises in patients with herniated intervertebral lumbar discs. *Physiother Theory Pract.* 8:207, 1992.
127. Schillinger A, et al: Effect of manual lymph drainage on the course of serum levels of muscle enzymes after treadmill exercise. *Am J Phys Med Rehabil.* 85:516, 2006.
128. Huggenberger R, Siddiqui SS, Brander D, et al: An important role in lymphatic vessel activation in limiting acute inflammation. *Blood.* 117:4667, 2011.
129. Cyriax, JH: Clinical applications of massage. In Rogoff JB (ed): Manipulation, Traction, and Massage, ed 2. Williams & Wilkins, Baltimore, 1980.
130. Brasseau, L, et al: Deep transverse friction massage for treating tendinitis. *Cochrane Database Syst Rev.* 1:CD003528, 2002.
131. Sefton J: Myofascial release for athletic trainers, part 3: Specific techniques. *Athl Ther Today.* 9(3):40, 2004.
132. Simons D, Travell J, Simons L: Upper Half of the Body. Travell & Simons' Myofascial Pain and Dysfunction: The Trigger Point Manual, ed. 2 vol. 1. Williams & Wilkins, 1999.
133. Sefton J: Myofascial release for athletic trainers, part 2: Guidelines and techniques. *Athl Ther Today.* 9(2):52, 2004.
134. Hou C, Tsai L, Cheng K, et al: Immediate effects of various physical therapeutic modalities on cervical myofascial pain and trigger-point sensitivity. *Arch Phys Med Rehabil.* 83:1406, 2002.
135. Hains G, Descarreaux M, Lamy A, et al: A randomized controlled (intervention) trial of ischemic compression therapy for chronic carpal tunnel syndrome. *J Can Chiropr Assoc.* 54: 155, 2010.
136. Goldenberg DL: Fibromyalgia, chronic fatigue syndrome, and myofascial pain syndrome. *Curr Opin Rheumatol.* 3:247, 1991.
137. Wolfe F, et al: The fibromyalgia and myofascial pain syndromes: A preliminary study of tender points and trigger points in persons with fibromyalgia, myofascial pain syndrome and no disease. *J Rheumatol.* 19:944, 1992.
138. King JC, Goddard MJ: Pain rehabilitation: II. Chronic pain syndrome and myofascial pain. *Arch Phys Med Rehabil.* 75:S9, 1994.
139. Sefton J: Myofascial release for athletic trainers, part 1: Theory and session guidelines. *Athl Ther Today.* 9(1):48, 2004.
140. Remvig L, Ellis RM, Patijn J: Myofascial release: An evidence-based treatment approach? *Int Musculoskelet Med.* 30:29, 2008.
141. Curran PF, Fiore RD, Crisco JJ: A comparison of the pressure exerted on soft tissue by 2 myofascial rollers. *J Sport Rehabil.* 17:432, 2008.
142. Stasinopoulos D, Johnson MI: Cyriax physiotherapy for tennis elbow/lateral epicondylitis. *Br J Sports Med.* 38:675, 2004.
143. Hammer WI, Pfefer MT: Treatment of a case of subacute lumbar compartment syndrome using the Graston technique. *J Manipulative Physiol Ther.* 28:199, 2005.
144. Gehlsen GM, Ganion LR, Helfst RH: Fibroblast responses to variation in soft tissue mobilization pressure. *Med Sci Sports Exerc.* 31:531, 1999.
145. Hammer WI: The effect of mechanical load on degenerated soft tissue. *Journal of Bodywork and Movement Therapies.* 12:246, 2008.
146. Burke J, Buchberger DJ, Carey-Logmani T, et al: A pilot study comparing two manual therapy interventions for carpal tunnel syndrome. *J Manipulative Physiol Ther.* 30:50, 2007.
147. Ferrell-Torry AT, Glick OJ: The use of therapeutic massage as a nursing intervention to modify anxiety and the perception of cancer pain. *Cancer Nurs.* 16:93, 1993.
148. Felhendler D, Lisander B: Effects of non-invasive stimulation of acupoints on the cardiovascular system. *Complement Ther Med.* 7:231, 1999.
149. Delaney JP, et al: The short-term effects of myofascial trigger point massage therapy on cardiac autonomic tone in healthy subjects. *J Adv Nurs.* 37:364, 2002.
150. Morelli M, et al: Changes in H-reflex amplitude during massage of triceps surae in healthy subjects. *J Orthop Sports Phys Ther.* 12:55, 1990.
151. Sullivan SJ, et al: Effects of massage on alpha motoneuron excitability. *Phys Ther.* 71:555, 1991.
152. Morelli M, et al: H-reflex modulation during manual muscle massage of human triceps surae. *Arch Phys Med Rehabil.* 72:915, 1991.

153. Crosman LJ, et al: The effects of massage to the hamstring muscle group on range of motion. *J Orthop Sports Phys Ther*. 6:168, 1984.
154. Smith LL, et al: The effects of athletic massage on delayed onset muscle soreness, creatine kinase, and neutrophil count: A preliminary report. *J Orthop Sports Phys Ther*. 19:93, 1994.
155. Tiidus PM, Shoemaker JK: Effleurage massage, muscle blood flow and long-term post exercise strength recovery. *Int J Sports Med*. 16:478, 1995.
156. Nalilbolff BD, Tachiki KH: Autonomic and skeletal muscle response to nonelectrical cutaneous stimulation. *Percept Motor Skills*. 72:575, 1991.
157. Knygsand-Roenhoej K, Maribo T: A randomized clinical controlled study comparing the effect of modified manual edema mobilization treatment with traditional edema technique in patients with a fracture of the distal radius. *J Hand Ther*. 24:184, 2011.
158. Kriederman B, et al: Limb volume reduction after physical treatment by compression and/or massage in a rodent model of peripheral lymphedema. *Lymphology*. 35:23, 2002.
159. Howard SB, Krishnagiri S: The use of manual edema mobilization for the reduction of persistent edema in the upper limb. *J Hand Ther*. 14:291, 2000.
160. Nixon M, et al: Expanding the nursing repertoire: The effect of massage on post-operative pain. *Australian J Adv Nurs*. 14:21, 1997.
161. Malkin K: Use of massage in clinical practice. *Br J Nurs*. 3:292, 1994.
162. Furlan AD, et al: Massage for low back pain. *Cochrane Database Syst Rev*. 2:CD001929, 2002.
163. Goldberg J, et al: The effect of two intensities of massage on H-reflex amplitude. *Phys Ther*. 72:449, 1992.
164. Goldberg J, et al: The effect of therapeutic massage on H-reflex amplitude in persons with a spinal cord injury. *Phys Ther*. 74:728, 1994.
165. Day JA, et al: Effect of massage on serum level of a-endorphin and a-lipotropin in healthy adults. *Phys Ther*. 67:926, 1987.
166. Goats GC, Keir KA: Connective tissue massage. *Br J Sports Med*. 25:131, 1991.
167. Ching M: The use of touch in nursing practice. *Australian J Adv Nurs*. 10:4, 1993.
168. Field T, et al: Massage therapy reduces anxiety and enhances EEG pattern of alertness and math computations. *Int J Neurosci*. 86:197, 1996.
169. Weinrich SP, Weinrich MC: The effect of massage on pain in cancer patients. *Appl Nurs Res*. 3:140, 1990.
170. Ferrell BA, et al: A randomized trial of walking versus physical methods for chronic pain management. *Aging* (Milano). 9:99, 1997.
171. Shoemaker JK, et al: Failure of manual massage to alter limb blood flow: Measures by Doppler ultrasound. *Med Sci Sports Exer*. 29:610, 1997.
172. Boone T, et al: A physiologic evaluation of the sports massage. *J Athl Train*. 26:51, 1991.
173. Harmer PA: The effect of pre-performance massage on stride frequency in sprinters. *J Athl Train*. 26:55, 1991.
174. Cafarelli E, et al: Vibratory massage and short-term recovery from muscular fatigue. *Int J Sports Med*. 11:474, 1990.
175. Tiidus PM: Manual massage and recovery of muscle function following exercise. A literature review. *J Orthop Sports Phys Ther*. 25:107, 1997.
176. Martin NA, Zoeller RF, Robertson RJ, et al: The comparative effects of sports massage, active recovery, and rest in promoting blood lactate clearance after supramaximal leg exercise. *J Athl Train*. 33:30, 1998.
177. Striggle JM, et al: Effects of vibrational massage on delayed onset muscle soreness and H-reflex amplitude. *J Athl Train*. 37(S):S105, 2002.
178. Lehmann JF, et al: Effect of therapeutic temperatures on tendon extensibility. *Arch Phys Med Rehabil*. 51:481, 1970.
179. Madeleine P, Vedsted P, Blangsted K, et al: Effects of electromyographic and mechanomyograpic biofeedback on upper trapezius muscle activity during standardized computer work. *Ergonomics*. 49:921, 2006.
180. Croce RV: The effects of EMG biofeedback on strength acquisition. *Biofeedback Self Regul*. 11:299, 1986.
181. Peek CJ: A primer of biofeedback instrumentation. In Schwartz MS (ed): Biofeedback: A Practitioner's Guide. Guilford Press, New York, 1987.
182. Araujo RC, et al: On the inter- and intra-subject variability of the electromyographic signal in isometric contractions. *Electromyogr Clin Neurophysiol*. 40:225, 2000.
183. Intiso D, et al: Rehabilitation of walking with electromyographic biofeedback in drop-foot after stroke. *Stroke*. 25:1189, 1994.
184. Utz SW: The effect of instructions on cognitive strategies and performance in biofeedback. *J Behav Med*. 17:291, 1994.
185. Segreto J: The role of EMG awareness in EMG biofeedback learning. *Biofeedback Self Regul*. 20:155, 1995.
186. Vander Linden DW, et al: The effect of frequency of kinetic feedback on learning an isometric force production task in nondisabled subjects. *Phys Ther*. 73:79, 1993.
187. Draper V: Electromyographic biofeedback and recovery of quadriceps femoris muscle function following anterior cruciate ligament reconstruction. *Phys Ther*. 70:25, 1990.
188. Wolf SL: Neurophysiological factors in electromyographic feedback for neuromotor disturbances. In Basmajian JV (ed): Biofeedback: Principles and Practice for Clinicians. Williams & Wilkins, Baltimore, 1983.
189. Ingersoll CD, Knight KL: Patellar location changes following EMG biofeedback or progressive resistive exercises. *Med Sci Sports Exerc*. 23:1122, 1991.
190. Coleborne GR, et al: Feedback of ankle joint angle and soleus electromyography in the rehabilitation of hemiplegic gait. *Arch Phys Med Rehabil*. 74:1100, 1993.
191. Yip SLM, Ng GYF: Biofeedback supplementation to physiotherapy exercise programme for rehabilitation of patellofemoral pain syndrome: A randomized controlled pilot study. *Clin Rehabil*. 20:1050, 2006.
192. Wang S, Li S, Xu X, et al: Effect of slow abdominal breathing combined with biofeedback on blood pressure and heart rate variability in prehypertension. *J Altern Complement Med*. 16:1039, 2010.
193. Cummings MS, et al: Flexibility development in sprinters using EMG biofeedback and relaxation training. *Biofeedback Self Regul*. 9:395, 1984.
194. Flor H, Birbaumer N: Comparison of the efficacy of electromyographic biofeedback, cognitive-behavioral therapy, and conservative medical interventions in the treatment of chronic musculoskeletal pain. *J Consult Clin Psychol*. 61:653, 1993.
195. Newton-John TR, et al: Cognitive-behavioural therapy versus EMG biofeedback in the treatment of chronic low back pain. *Behav Res Ther*. 33:691, 1995.
196. Valeyen J, et al: Behavioural rehabilitation of chronic low back pain: Comparison of an operant treatment, an operant-cognitive treatment and an operant-respondent treatment. *Br J Clin Psychol*. 34:95, 1995.

197. Silkman C, McKeon J: The effectiveness of electromyographic biofeedback supplementation duration knee rehabilitation after injury. *J Sport Rehabil.* 19:343, 2010.
198. Bischko JJ: Use of the laser beam in acupuncture. *Acupunct Electrother Res.* 5:29, 1980.
199. Huang YY, Chen AC, Carroll JD, et al: Biphasic dose response in low level light therapy. *Dose Response.* 7:358, 2009.
200. McLeod IA. Low-level laser therapy in athletic training. *Athl Ther Today.* 9:17, 2004.
201. Perkins SA, Massie JE: Patient satisfaction after thermal shrinkage of the glenohumeral-joint capsule. *J Sports Rehabil.* 10:157, 2001.
202. Bartlett WP, et al: Effect of Gallium-aluminum-arsenide triple-diode laser irradiation on evoked motor and sensory action potentials of the median nerve. *J Sports Rehabil.* 11:12, 2002.
203. Lehmann JF, De Lateur BJ: Laser as a physical treatment modality. In Lehmann JF (ed): Therapeutic Heat and Cold, ed 4. Williams & Wilkins, Baltimore, 1990, p. 582.
204. Posten W, Wrone DA, Dover JS, et al: Low-level laser therapy for wound healing: Mechanism and efficacy. *Dermatol Surg.* 31:3, 2005.
205. McBrier N, Olczak JA: Low level laser therapy for stimulating muscle regeneration following injury. *Athl Ther Today.* 14:20, 2009.
206. Enewemeka C, Parker J, Dowdy D, et al: The efficacy of low-power lasers in tissue repair and pain control: A meta-analysis study. *Photomed Laser Surg.* 22:323, 2004.
207. Ohshiro T, et al: Penetration depths of 830 nm diode laser irradiation of the head and neck assessed using a radiographic phantom model and wavelength-specific imaging film. *Laser Ther.* 8:197, 1996.
208. Snyder-Mackler L, Bork CE: Effect of helium-neon laser irradiation on peripheral sensory nerve latency. *Phys Ther.* 68:223, 1988.
209. Bartlett WP, et al: Effect of gallium aluminum arsenide triple-diode laser on median nerve latency in human subjects. *J Sports Rehabil.* 8:99, 1999.
210. Bjordal JM, Johnson MI, Iversen V, et al: Low-level laser therapy in acute pain: A systematic review of possible mechanisms of action and clinical effects in randomized placebo-controlled trials. *Photomed Laser Surg.* 24:158, 2006.
211. David R, et al: Effect of low-power He-Ne laser on fracture healing in rats. *Lasers Surg Med.* 19:458, 1996.
212. Greathouse DG, et al: Effects of clinical infrared laser on superficial radial nerve conduction. *Phys Ther.* 65:1184, 1985.
213. King CE, et al: Effect of helium-neon laser auriculotherapy on experimental pain threshold. *Phys Ther.* 70:24, 1990.
214. Pires Oliveria DAA, De Oliveria RF, Magini M, et al: Assessment of cytoskeletan and endoplasmic reticulum of fibroblast cells subjected to low-level laser therapy and low-intensity pulsed ultrasound. *Photomed Laser Surg.* 27:461, 2009.
215. Armagan O, Tascioglu F, Ekim A, et al: Long-term efficacy of low level laser therapy in women with fibromyalgia: A placebo-controlled study. *J Back Musculoskelet Rehabil.* 19:135, 2006.
216. Lopes-Martin RAB, Penna SC, Joensen J, et al: Low level laser therapy [LLLT] in inflammatory and rheumatic diseases: A review of therapeutic mechanisms. *Curr Rheumatol Rev.* 3:147, 2007.
217. Rizzi CF, et al: Effects of low-level laser therapy (LLLT) on the nuclear factor (NF)-kB signaling pathway in traumatized muscle. *Lasers Surg Med.* 38:704, 2006.
218. DeSimone NA, et al: Bactericidal effect of 0.95-mW helium-neon and 5-mW indium-gallium-aluminum-phosphate laser irradiation at exposure times of 30, 60, and 120 seconds on photosensitized *Staphylococcus aureus* and *Pseudomonas aeruginosa* in vitro. *Phys Ther.* 79:839, 1999.
219. Hawkins D, Houreld N, Abrahamse H: Low level laser therapy (LLLT) as an effective modality for delayed would healing. *Ann NY Acad Sci.*1056:486, 2005.
220. Ricevuti G, et al: In vivo and in vitro HeNe laser effects on phagocyte functions. *Inflammation.* 13:507, 1989.
221. Tang XM, Chai BP: Effect of CO_2 laser irradiation on experimental fracture healing: A transmission electron microscope study. *Lasers Surg Med.* 6:346, 1986.
222. Hamilton GF, et al: The effects of helium-neon laser upon regeneration of the crushed peroneal nerve. *J Orthop Sports Phys Ther.* 15:209, 1992.
223. Wood VT, Pinfildi CE, Neves MAI, et al: Collagen changes and realignment induced by low-level laser therapy and low-intensity ultrasound in the calcaneal tendon. *Lasers Surg Med.* 42:559, 2010.
224. Ilbuldu E, Cakmak A, Disci R, et al: Comparison of laser, dry needling, and placebo laser treatments in myofascial pain syndrome. *Photomed Laser Surg.* 22:306, 2004.
225. Numazawa R, et al: The role of laser therapy in intensive pain management of postherpetic neuralgia. *Laser Ther.* 8:143, 1996.
226. Bjordal JM, Lopes-Martins RAB, Iversen VV: A randomised, placebo controlled trial of low level laser therapy for activated Achilles tendinitis with microdialysis measurement of peritendinous prostaglandin E_2 concentrations. *Br J Sports Med.* 40:76, 2006.
227. Baxter GD, et al: Effects of low intensity infrared laser irradiation upon conduction in the human median nerve in vivo. *Exp Physiol.* 79:227, 1994.
228. Snyder-Mackler L, et al: Effects of helium-neon laser irradiation on skin resistance and pain in patients with trigger points in the neck or back. *Phys Ther.* 69:336, 1989.
229. Sprott H, Mueller W: Efficiency of acupuncture in patients with fibromyalgia. *Reumatologia* (Warsaw). 32:414, 1994.
230. Heussler JK, et al: A double blind randomized trial of low power laser treatment in rheumatoid arthritis. *Ann Rheum Dis.* 52:703, 1993.
231. Bjordal J, Lopes-Martins RAB, Joensen J, et al: A systematic review with procedural assessments and meta-analysis of low level laser therapy in lateral elbow tendinopathy (tennis elbow). *BMC Musculoskelet Disord.* 9:75, 2008.
232. Glinkowski W, Rowinski, J: Effect of low incident levels of infrared laser energy on the healing of experimental bone fractures. *Laser Ther.* 7:67, 1995.
233. Shakouri SK, Soleimanpour J, Salekzamani Y, et al: Effect of low-level laser therapy on the fracture healing process. *Lasers Med Sci.* 25:73, 2010.
234. Khadra M, Lyngstadaas SP, Haanaes HR, et al: Effect of laser therapy on attachment, proliferation and differentation of human osteoblast-like cells cultured on titanium implant material. *Biomaterials.* 26:3503, 2005.
235. Oliveria P, Sperandio E, Fernandes KR, et al: Comparison of the effects of low-level laser therapy and low-intensity pulsed ultrasound on the process of bone repair in the rat tibia. *Rev Bas Fisioter.* 15:200, 2011.
236. Fávaro-Pipi E, Feitosa SM, Riberio DA, et al: Comparative study of the effects of low-intensity pulsed ultrasound and low-level laser therapy on bone defects in tibias of rats. *Lasers Med Sci.* 25:727, 2010.

237. Lirani-Galvão AP, Jorgetti V, da Silva OL: Comparative study of how low-level laser therapy and low-intensity pulsed ultrasound affect bone repair in rats. *Photomed Laser Surg*. 24:735, 2006.
238. Houghton PE, Nussbaum EL, Hoens AM: Low-level laser therapy (LLLT)/non-coherent light. *Physiother Can*. 62:39, 2010.
239. Lundeberg T, et al: Effect of laser versus placebo in tennis elbow. *Scand J Rehabil Med*. 19:135, 1987.
240. Thorsen H, et al: Low level laser therapy for myofascial pain in the neck and shoulder girdle. A double-blind cross-over study. *Scand J Rheumatol*. 21:139, 1992.
241. Mulcahy D, et al: Low level laser therapy: a prospective double blind trial of its use in an orthopaedic population. *Injury*. 26:315, 1995.
242. Fernando S, et al: A randomized double blind comparative study of low level laser therapy following surgical extraction of lower third molar teeth. *Br J Oral Maxillofac Surg*. 31:170, 1993.
243. Gam AN, et al: The effect of low-level laser therapy on musculoskeletal pain: A meta-analysis. *Pain*. 52:63, 1993.
244. Beckerman H, et al: The efficacy of laser therapy for musculoskeletal and skin disorders: A criteria-based meta-analysis of randomized clinical trials. *Phys Ther*. 72:483, 1992.
245. Siebert W, et al: What is the efficacy of soft and mid lasers in therapy of tendinopathies? A double-blind study. *Arch Orthop Trauma Surg*. 106:358, 1987.
246. Ay S, Dogan SK, Evcik D: Is low-level laser therapy effective in acute or chronic low back pain? *Clin Rheumatol*. 29:905, 2010.
247. Lundeberg T, Malm M: Low-power HeNe laser treatment of venous leg ulcers. *Ann Plastic Surg*. 27:537, 1991.
248. Surinchak JS, et al: Effects of low level energy lasers on the healing of full thickness skin defects. *Lasers Surg Med*. 2:267, 1983.
249. Chen C, Diven DG, Lockart S, et al: Laser transmission through transparent membranes used in cutaneous laser treatment. *J Am Acad Dermatol*. 45:919, 2001.