We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists



186,000

200M



Our authors are among the

TOP 1% most cited scientists





WEB OF SCIENCE

Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us? Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected. For more information visit www.intechopen.com



The Use of Photomedicine in Musculoskeletal Pain

Abdullah M. Al-Shenqiti

Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/65748

Abstract

Musculoskeletal pain is a major cause of disability. Myofascial trigger points (MTrPs) in particular are a common source of pain in a substantial number of patients presenting at a pain clinic. Many different invasive and non-invasive forms have been advocated to the treatment of MTrPs. However, favourable outcome rates are inconsistent and some of these treatment forms described are often painful and have potentially dangerous side effects. Photomedicine including the coherent light sources (lasers) and more recently, non-coherent light sources have been reported to be beneficial in soft tissue lesions including MTrPs. Their beneficial therapeutic effects can be obtained without undesired effects. The main intentions of this chapter are to bring the attention of the doctors and physical therapists to the scientific approach of photomedicine, in particular laser therapy for the relief of pain arising from MTrPs, and to demonstrate how this type of therapy can be utilized in a rational manner for the relief of musculoskeletal pain. In addition, it has been found necessary to include or to start with an overview of the recently recognized diagnostic and therapeutic importance of MTrPs. Attention will therefore first be drawn mainly to incidence, types, aetiology, clinical diagnostic criteria and conventional forms of MTrPs.

Keywords: photomedicine, myofascial pain, trigger points, laser therapy, phototherapy

1. Introduction

Skeletal muscle contractile tissues are subject to constant wear and tear, which makes them prone to development of myofascial trigger points (MTrPs) that result in referred pain and motor dysfunction [1]. MTrPs are extremely common and a major source of musculoskeletal pain and dysfunction that can affect anyone at one time or another [1–3].

MTrPs are one of the profound reasons of pain in clinical practice [4, 5]. They are the source of pain in 30% of patients seeking treatment or medical advices for pain in primary care and the greatly noticeable cause of pain in 85% of patients presenting at a pain centre [6, 7].





Figure 1. Simplified schematic of taut band and myofascial trigger point (MTrP).

Myofascial pain syndromes are common conditions that, by definition, result from trigger points (TrPs). Unfortunately, practitioners often do not recognise the myofascial pain syndrome [7, 8]. Unrecognised myofascial headaches, low back pain and shoulder pain have been considered to be one of the major causes of chronic pain, disability and industrial time lost, which plays a factor in most worker's compensation claims [1, 9].

Musculoskeletal pain originating from muscle has concerned the medical community for more than a century [10]. The subject has been appointed by multiple terms that emphasize various signs and symptoms representing basically the same phenomenon [11].

History has introduced terms such as fibrositis, myalgia, rheumatic myalgia and non-articular rheumatism. However, it should be noted that all of these terms and many others that are used for myofascial trigger points (primary cause of muscle pain) and fibromyalgia (central cause of muscle pain) are now no longer utilized [1, 10, 12].

The entity of MTrP has now been widely acknowledged on the basis of clinical observation and basic scientific research [13, 14]. A myofascial trigger point (MTrP) has been defined as a highly localised, sensitive, hyperirritable spot in a palpable taut band of skeletal muscle fibres [1, 15] (**Figure 1**). MTrPs are frequently found at or near to a muscle's origin and insertion, as well as along the belly of a muscle particularly at the motor points [2, 12].

2. Types of MTrPs

There are two main types of MTrPs active and latent. An active MTrP is one associated with spontaneous pain or occurring in response to movement [16–18]. It has also been defined as one whose nociceptors have undergone sufficient activation and sensitisation to cause pain to be referred to a site some distance from it (the zone of pain referral) [12].

A latent MTrP is a sensitive spot with pain or discomfort, which occurs in response to compression only [16, 17]. It may also be defined as one in which its nociceptors have undergone a limited amount of trauma-induced activation and sensitisation, but not sufficient to cause the development of pain [12].

Active TrPs might cause agonising incapacitating pain particularly when associated with active satellite TrPs in another muscles [1]. By contrast, despite the fact that, latent TrPs do not produce spontaneous pain, they can cause some increased muscle tension, limitation of passive range motion and may also cause some muscle shortening [1, 17]. Both active and latent TrPs can therefore cause motor dysfunction [1].

MTrPs may become activated either through a primary or through a secondary event [12, 18]. Primary activation of the TrPs usually takes place as a result of direct trauma to a muscle, sudden strain, or when there is excessive or unusual exercise. The activation might also be the result of cumulative effects due to long-standing repetitive minor trauma or overloading [1, 12, 17, 18].

Secondary activation of TrPs usually takes place in synergistic and antagonistic muscles. This may be due to compensatory actions or by counteracting tension in the primary muscles. Referral from visceral sources such as in a myocardial infarction or connective tissue disorder such as osteoarthritis, rheumatoid arthritis may also contribute to this secondary phenomenon [1, 19].

3. Clinical diagnostic criteria of MTrPs

There are certain clinical characteristics that should be looked for during the examination in order to confirm the presence of MTrPs. These include:

3.1. Taut band

Muscles sometimes contain taut cord-like bands. Palpable taut band is considered to be a basic diagnostic criterion of an MTrP [20].

3.2. Local twitch response

The local twitch response (LTR) is a transient contraction of the palpable taut band of muscles comprising MTrPs. It can be visualised, or palpated through the skin of the patient, or seen by ultrasound imaging [1, 15, 18].

The LTR is elicited mechanically, usually by a vigorous snapping palpation of the TrP in a direction opposite to the muscle fibres, or by needle penetration of the TrP [1, 18].

3.3. Spot tenderness or jump sign

Spot tenderness is an essential diagnostic criterion in the MTrP examination. However, spot tenderness alone has a limited value because it might be due to other reasons such as fibromyalgia.

The jump sign is a characteristic behavioural response to pressure on a MTrP. Patients often withdraw and sometimes complain of pain particularly with active MTrPs [1].

3.4. Pain recognition

Digital pressure on an MTrP can be used to elicit referred pain patterns characteristic of that muscle and the patient symptoms. This is considered as one of the most important diagnostic criteria particularly when accompanied by other signs [1, 20, 21].

3.5. Limited range of motion

Restricted range of motion is more severe in more active MTrPs and is a fundamental characteristic of MTrPs [1]. A muscle containing an MTrP restricts range of motion due to pain [1, 12, 22]. When MTrPs are treated, range of motion increases and often returns to normal [1].

3.6. Referred pain

Pain is often felt a considerable distance from the MTrPs and as a consequence, most patients are unaware of the presence of the TrP despite its exquisite tenderness. The referred pain either occurs spontaneously, particularly when the MTrP is very active [1, 2] or through palpation [15].

Referred pain by itself is not considered a diagnostic criterion of an MTrP unless accompanied by other findings [1, 20, 23]. However, the referred pain patterns play an important role in the initial examination as they direct the examiner to the muscle that harbours the MTrPs. In addition, knowledge of referral patterns minimises the chances of missing some TrPs [12] (**Figure 2**).



Figure 2. The pattern of pain referral from myofascial trigger points (•) in the rhomboid muscles.

4. Diagnosis of MTrPs

The diagnosis of MTrPs primarily relies on manual palpation and clinical judgment. However, manual palpation may be imprecise and not a reliable [24]. Therefore, specific training coupled with clinical experience is needed to obtain good reliability for the MTrP diagnosis. It has been showed that a combination of "spot tenderness," "taut band" and "pain recognition" are the basic clinical criteria to diagnose a MTrP, while "referred pain" and "local twitch response" are considered to be confirmatory signs [20, 21].

More recently, detection of biochemicals related to pain and inflammation in MTrP site [25], the sonographic methods of MTrPs [26] and the magnetic resonance elastography for taut band image [27] are potential objective outcome assessment tools in the MTrPs diagnosis.

5. Conventional forms of MTrPs treatment

There are several forms that are conventionally used to treat the MTrPs, which include:

5.1. Dry needling

The possibility of treating the MTrPs by dry needling techniques has been noted as early as 1952 [28], but Lewit [29] was the first investigator to employ dry needling techniques.

More recently, the effectiveness of dry needing in reducing the tenderness of MTrPs has been reported by a number of authors (e.g. [30–32]). However, dry needling is occasionally associated with adverse events such as post-needling soreness, bruising, dizziness and infection [32–34].

5.2. The injection of a local anaesthetic into an MTrP

The use of local anaesthetics such as Procaine Lidocaine has been reported to be an effective method for reducing post-injection soreness [1, 30, 32]. However, the use of them may occasionally give rise to toxic, allergic and anaphylactic reactions [15].

5.3. Botulinum toxin A injection

Botulinum toxin A injection (BTA) has been utilized in treatment for MTrPs [35–38]. However, it is rare clinically indicated, as it may be associated with possible local and systemic side effects such as muscle weakness and serious respiratory compromise [38, 39].

5.4. The injection of non-steroidal and steroidal anti-inflammatory drugs into MTrPs

The injection of non-steroidal anti-inflammatory drugs into the MTrPs has been used successfully to treat them [40, 41]. However, repeated injection of it into muscle might lead to skin necrosis [40]. The injection of steroids has also been used to treat the MTrP, and a good result has been reported [40, 42]. However, the use of steroids should be discouraged because of the risk of inducing local myopathy and the possible muscle fibre damage that is frequently associated with repeated injections [40].

5.5. Therapeutic ultrasound

The literature advocates therapeutic ultrasound as an effective modality for MTrP treatment in the clinical practice [1, 43]. In a study by Hong et al. [44] revealed that pain pressure threshold of MTrPs was increased immediately after ultrasound therapy with intensities of (1.2–1.5 W/cm²) as compared to placebo therapy. However, Lee et al. [45] could not obtain similar finding at a lower intensity of ultrasound (0.5 W/cm²).

More recently, two studies conducted by Srbely et al. [46] and Srbely and Dickey [47] revealed improvement in pressure pain threshold value (less tenderness). However, the study of Srbely and Dickey [47] was not blinded and did not address the long-term benefit.

5.6. Electric stimulation

Electrotherapy has been advocated as an effective therapeutic modality to alleviate pain emanating from MTrPs [43, 48, 49]. Graff-Radford et al. [50] showed that high frequency transcutaneous electrical nerve stimulation (TENS) could alleviate pain but they did not succeed to show any improvement in the MTrP sensitivity. More recently, Lee et al. [45] utilised electrical muscle stimulation and they concluded that pain was significantly decreased compared to the placebo group, but no significant improvement in pressure threshold or range of motion was found. The long-term influence of the electrical stimulation on MTrP was not addressed in the methodology of the above two trials.

6. Photomedicine

Photomedicine has progressed and come to be one of the most inspiring fields in the medical research in the past 50 years. The coherent light sources (lasers) and more recently, non-coherent light sources, e.g. light emitting diodes (LEDs) and superluminous diodes (SLDs) used in the musculoskeletal disorders are those with an athermic effect. Frequently used lasers include the helium-neon (HeNe gas) and infrared lasers with gallium arsenide (GaAs) or gallium aluminium arsenide (GaA1As) diodes [51–53].

Laser therapy (coherent sources) or low reactive-level laser therapy (LLLT) has been reported to be beneficial in soft tissue lesions including MTrPs [54–60]. Its beneficial therapeutic effect can be obtained without undesired effects. The below section of this chapter considers mainly the background of laser and characteristics of laser light, laser treatment parameters, treatment approaches and the possible mechanisms of action of laser in MTrPs.

7. Background and historical perspectives of laser

While laser is a relatively new form of treatment, the therapeutic benefits of light energy are not a new concept [51, 61, 62]. The sun was the first source of light that was employed in the treatment of several conditions.

Laser was not developed until 1960; however, the concept behind it was described at the beginning of the century by Albert Einstein in his 'quantum theory' [62, 63]. The development of LASER—Light Amplification by stimulated Emission of Radiation—then arose when Theodore Maiman in 1960 amplified light (using a ruby crystal as a lasing media) [51, 62].

In the 1960s, rapid development took place and variety of laser types appeared, based on different lasing media and resulting in different wavelengths. For example, Johnson in 1961 developed the neodymium YAG (Nd:YAG) laser followed by the Argon laser developed by Bennet in 1962. This was followed by the carbon dioxide (CO_2) laser 2 years later by Patel and colleagues [64]. These kinds of laser in their medical applications relied upon the photothermal and photoablative interactions with the tissues at relatively high power and energy densities [62, 63].

In contrast, other types of laser were developed by Professor Master's group in Budapest during the late 1960s and early 1970. These types of laser relied upon the non-thermal interactions of laser irradiation with tissues at low power and energy densities had a photobiostimulation effect on experimental wounds, which increased the rate of healing [62–65].

Another possible biological effect of low power laser was described in 1973 by Friedrich Plog in Canada, who presented his work on the use of HeNe laser as an alternative to metal needles for acupuncture treatments [51, 66, 67].

After these initial successful reports of Professor Mester's group at Budapest and Dr. Plog in Canada, low power laser treatment has become more frequently utilized by physicians and physical therapists for the alleviation of pain [68–70]. More recently, low power laser has got approval of Food and Drug Administration (FDA), as a pain reliever for soft tissue lesion in 2005 in the USA [71]. Furthermore, the appearance of a number of clinical research papers with very promising results particularly for MTrPs treatment have led to the popularity of laser therapy (e.g. [54–58]).

8. Principal components of a laser system

The laser device consists of three essential components.

8.1. Lasing medium

A lasing medium is a material that can absorb the energy generated by an external source. It can be gaseous, liquid, solid, crystal or a semiconductor [51, 62, 65, 72–74].

8.2. Energy source

Energy laser device must have an energy source to excite the lasing medium in order to emit laser radiation [51, 62, 65, 73, 74].



Figure 3. Simplified schematic of laser basic components.

8.3. Mechanical structure

The mechanical structure consists of the lasing medium within a central chamber located between two parallel mirrors. Reflection of photons of light back and forth between the two mirrors an across the chamber takes place, which lead to an intense photon production [51, 62, 72, 73].

The reflective extent of the two mirrors is not the same. While one of them is 100%, the other one is slightly less reflective to allow a small amount of the laser beam to pass through as irradiation output of the device [51, 62, 72, 73] (**Figure 3**). However, in the semiconductor devices, the ends of the diodes can be polished or coated with a highly reflective material to work as an alternative to the mirrors. Similarly, one end of the diode is slightly less reflective to allow a certain amount of the laser beam to pass as an output of irradiation [51, 73, 75].

9. Characteristics of laser light

Laser light differs from the ordinary light in terms of its monochromaticity, collimation and coherence. The biological and clinical significance of these characteristics is still relatively questionable and under investigations:

9.1. Monochromaticity

Monochromaticity indicates to single, defined wavelength, which consequently gives (mono) single colour [51, 73, 76–78]. Research evident showed that biological process possibly altered within a very narrow bandwidth, as distinct from the board spectrum of natural light [79].

9.2. Collimation

Collimation indicates to the minimal divergence of the laser beam. Compare to, the emitted radiation of non-laser light sources radiates in various directions [51, 72, 75].

9.3. Coherence

Coherence indicates to the inherent 'synchronicity' of the light emitted by laser devices, which means that all energy waves are in phase [51, 63, 75, 77, 78] (**Figure 4**).

Some in-vitro studies have found that it is substantially critical to use a coherent laser source to attain photobiological modulations (e.g. [80, 81]), whilst others have found that coherence is not necessary [79, 82, 83]. Therefore, some of manufacturers have presented a cheaper phototherapy units, e.g. superluminous diodes (SLDs) and light-emitting diodes (LEDs), which are non-coherent [51, 63].

Clinically, researchers have disputed over the possible loss of coherency when subjecting laser light to human tissues and recently have shown positive outcomes when using light-emitting diodes (LEDs) (non-coherent light sources) in experimental muscle injury [84, 85].

Conversely, it has been reported that the photon density of coherent laser beam ensures a greater and more efficient penetration [67, 86, 87]. Simunovic [88] has also shown that coherency possibly maintained when passing through tissue. The significance of coherency has been also stressed by Antipa [89], as one of the characteristics that are necessary to obtain a higher clinical efficacy of laser therapy [89].



Figure 4. (a) Coherent light and (b) incoherent light.

9.4. Laser therapy treatment approaches

Laser therapy usually involves two main types of treatment approaches.

9.4.1. Contact approach

In the contact approach, the probe or the treatment head is applied perpendicular to the treated tissue. This technique greatly intensifies the irradiance on the tissue surface and consequently a greater proportion of laser energy can be directed to the target tissue [51, 63, 72, 75] (**Figure 5**).

The contact approach has been reported to be the most efficient treatment approach and must be used whenever it is possible [51, 62]. The treatment is also relatively safe as it reduces the potential hazard that comes from accidental intrabeam viewing [51, 62, 63].

A contact approach is commonly utilized in treating MTrPs. Beneficial effects have been shown in a number of clinical trials adopting this approach [54, 56, 90] while others failed to reveal any successful effects [91, 92]. However, in the latter two studies, no treatment parameters details and/or poor treatment parameters were evident.

9.4.2. Non-contact approach

In the non-contact approach, the probe or the treatment head is used out of contact with the tissue (**Figure 5**). This technique attenuates the irradiance on the irradiated tissue according to the 'inverse square law', thus more reflection of the incident photons will occur [51, 62, 63].



Figure 5. (a) Non-contact approach and (b) contact approach.

10. Treatment parameters

Treatment parameters are extremely important and their detailed specification can allow replication of experimental or clinical findings.

10.1. Wavelength

Wavelength is the most important treatment parameters that determine the depth of penetration of laser irradiation [93]. Laser therapy units are usually with single wavelength sources. However, more recently, laser therapy units with a broad spectrum of wavelengths are available.

Therapeutic lasers have commonly wavelengths of approximately 602–1064 nm and give visible (red) or invisible (near infra-red) radiation (HeNe laser) [51, 65, 94].

10.2. HeNe lasers (632.8 nm)

The insufficient of penetration of this visible short wavelength laser source (HeNe laser) [51, 65, 94] might be one the justifications for its ineffectiveness. However, positive outcomes were evident with this type of laser in some MTrP clinical studies (e.g. [54, 95]).

10.3. CO₂ Lasers (10,600 nm)

The CO₂ lasers produce infrared radiation at 10.600 nm and such devices have been used successfully as pain relief modalities by some researchers (e.g. [96–98]). However, this type of laser is almost totally absorbed by water, thus reducing great proportion of light penetration in biological tissue [67].

10.4. Diode lasers (820–950 nm)

Wavelengths that are in the range of 820–950 nm (diode lasers) are known as near infrared radiation in the electromagnetic spectrum [51]. Their tissue penetration ability is quite high compared to other sources with different wavelengths [67].

Most of myofascial trigger point clinical studies utilized lasers with longer wavelength, ranging from 780 to 904, because it can transmit light energy with greater penetration and, therefore, they are the most appropriate to treated trigger points that particularly located in deep muscles [99].

10.5. Radiant power output

The radiant power output of laser systems is generally measured in watts (W), but as a result of the relatively low power output employed in LLLT systems, it is more frequently measured in milliwatts (mW = thousands of a watt) [63, 65, 100].

It has also been reported that more penetration can be gained with greater average output power, as a greater number of photons will be presented at deeper depths [72]. However,

radiant power output was greatly variable between studies with possible pain reduction when irradiating painful MTrPs with a range of radiant power outputs of 0.95–120 mW.

10.6. Irradiance

Irradiance is defined as the incident photon density of laser irradiation at the target tissue and utilized to express to the intensity of light [67]. The significance of irradiance has been stressed by a number of researchers as one of the most influential treatment parameters [101, 102]. However, unfortunately, quite few numbers of researchers reported this treatment parameter [99].

Some literature reported positive results for tissue repair and anti-inflammatory effects when the range of 5–55 mW/cm2 was employed [103–105], while higher irradiances of 300–1730 mW/cm2 was recommended for analgesic purposes [106].

The irradiance can be obtained from the following equation:

Irradiance =
$$\frac{\text{Output power (W)}}{\text{Irradiated area (cm2)}}$$
 (1)

From the above equation, it can be seen that the value of the irradiance (measured in watts) will be profoundly affected by the spot size (measured in square centimetre) of the laser beam [67].

10.7. Radiant energy

The radiant energy delivered to a region of the target tissue over a period of time is commonly expressed in joules (J) and can be calculated by the following equation:

Radiant Energy (J) = power output (W) × time (s)
$$(2)$$

From the above equation, if a certain energy is required in a treatment session, the time (in seconds) needed to obtain that energy can be obtained, by dividing the energy by the radiant power output [51, 63].

The total radiant energy is one of critical treatment parameters [107]. The energy employed in the MTrPs clinical trials ranged from 0.275 to 8 J/point [99].

10.8. Radiant exposure

Radiant exposure is a critical factor in ascertaining whether the laser light will influence photobiological modulation process [108, 109]. A recent study also revealed that the radiant exposure provided by laser therapy is one of the factors that can influence biochemicals related to pain in the treatment of MTrPs [60].

It is worth noting, energy density is not a fixed parameter as it is dependent on time and can be manipulated by the operator [110]. The radiant exposure can be obtained from the following equation:

Energy density
$$(J/cm^2) = \frac{Power (W) \times times (s)}{area (cm^2)}$$
 (3)

Treatment exposure is usually in a range between 1.44 and 12 J/cm² in the treatment of MTrPs. However, radiant exposures of up to 32 J/cm² have been employed also [75, 99].

10.9. Pulse repetition rate

Generally, the laser devices that are available in research or clinical applications deliver a continuous wave or allow some form of pulsing of their output [63, 65, 72, 73].

The pulse repetition rate in the pulsed devices is expressed in Hertz (Hz, pulses per second) and can vary from 2 to thousands of Hz [63]. However, the pulse rate is restricted in some laser devices.

It has been reported that pulsed light possibly more effective than continuous one, as it allow a potentially much higher peak power densities without causing a significant tissue heating and consequently greater treatment depth [93]. Additionally, different biological effects were obtained, when experiments were conducted to determine the effect of different pulsing frequencies (e.g. [111–115]).

10.10. Frequency of treatment and length of treatment course

Clinical practitioners and researchers advocate multiple treatment sessions for successful laser treatment [51, 65, 94] (**Figure 6**). In MTrPs studies, laser treatment were commonly



Figure 6. Clinical laser session.

utilized 2-3 times per week However, there is no consensus about the optimum frequency of treatment or length of treatment course [56, 99].

11. The possible mechanisms of action of laser in MTrPs

Three possible mechanisms of action were proposed included: stimulation of the local metabolism; modulation of neurotransmitter; anti-inflammatory effect and laser-induced neuronal suppression.

11.1. Stimulation of the local metabolism

In the mechanism of taut band formation, certain muscle fibres react to trauma or abnormal stress by excessive release of calcium ions. This would cause uncontrolled muscle fibres contracture with increase metabolic demands and consequently, MTrPs formation [1]. Laser therapy reported to have the potential to cause rotation and vibration on the membrane molecules that make up the calcium channels that may alter the function of these channels [116]. This might help in removing or minimizing the excessive amount of calcium that may causes uncontrolled shortening activity of the muscle fibres.

The characteristic shortening of muscle fibres associated with MTrPs results in deteriorated local circulation leading to loss of oxygen and nutrient supply. Laser therapy has the ability to enhance the local microcirculation and subsequently decrease the muscular tension and emanate pain in the area [88].

11.2. Modulation of neurotransmitter

Modulation of neurotransmitters has proposed as a mechanism for pain alleviation [117]. For instance, serotonin levels are relatively intensifying when the treatment of MTrPs with laser treatment are employed. A trial carried out by Walker [118] applied laser irradiation on MTrPs patients attributed the analgesic effects to changes in serotonin metabolism.

More recently, a double blind study conducted by Ceylan et al. [57] also found that laser irradiation is an effective method of treatment of MTrPs associated with the elevation of the serotonin. However, more studies may be required in this area.

11.3. Anti-inflammatory effect

The cause of pain in active MTrPs may be as a result of direct trauma to a muscle, sudden strain, or when there is excessive or unusual exercise, a study by Shah et al. [25] also documents a high concentration of nociceptive substances, e.g. protons, bradykinin, calcitonin gene-related peptide, substance p, serotonin and noradrenaline) in the active MTrPs.

As the rise of the above biochemical milieu of substances are usually associated with pain and inflammation and in accordance with the above findings, evidence from the literature reported that laser therapy inhibits peripheral nerves afferent terminals prohibits peripheral nerve sensitization and hinder further release of the nociceptive substances, thus possible mechanisms of pain relief and anti-inflammatory effect may occur [119, 120].

Research has also clearly revealed anti-inflammatory effects of laser irradiation particularly in acute injury [120]. The tissue repair is associated with the release of prostaglandins E_2 Laser irradiation showed the ability to reduce the formation of the inflammatory markers including the prostaglandins E2 [119]. Therefore, laser might be able promote resolution of the inflammatory process vital for the tissue repair.

11.4. Laser-induced neuronal suppression

Laser-induced suppression of neuronal activity is another potential mechanism for pain relieving influences of laser irradiation. In advocate of laser-induced neuronal suppression mechanisms are a number of human trials that showed that laser relatively hinders nerve conduction velocity and augments latency in median [51], radial [121] and sural nerves [122]. However, light-emitting diodes (LEDs) and superluminous diodes (SLDs) more recently failed to act as a direct suppression of neuronal activity [123].

In consistent with the above findings, a number of electrophysiological experiments were carried out, to assess neuronal mediated inhibitory effects of laser irradiation [124–126]. Laser irradiations were able more specifically to influence the nerve conduction of small diameter, thinly myelinated A and unmyelinated C fibres. Therefore, in the light of these encouraging research reports using laser as an alternative to needle in acupuncture for MTrPs treatment might not be excluded.

Author details

Abdullah M. Al-Shenqiti

Address all correspondence to: monuhama@yahoo.co.uk

Faculty of Medical Rehabilitation Sciences, Taibah University, Al-Madinah Al-Munawarah, Saudi Arabia, Centre for Rehabilitation Science, University of Manchester, Manchester, UK

References

- [1] Simons D, Travell J, Simons L. Travell & Simons's myofascial pain and dysfunction: the trigger point manual, vol. 1, second edition. William & Wilkins, Baltimore. 1999.
- [2] Baldry P. Acupuncture, Trigger Points and Musculoskeletal Pain. Second Edition. Churchill Livingstone, Longman Group UK limited, Harcourt Publishers Limited, Edinburgh 1993.

- [3] Fleckenstein J. Epidemiology. In: Irnish D (ed). Myofascial trigger points, comprehensive diagnosis and treatment. E-Book, 1st edition. Churchill Livingstone. 2013.
- [4] Baldry P. Myfascial Pain and Fibromyalgia Syndromes. A clinical Guide to Diagnosis and Management. Churchill Livingstone, Harcourt Publishers Limited, London 2001.
- [5] Cummings M, Baldry P. Regional myofascial pain: diagnosis and management. Best Pract Res Clin Rheumatol 2007; 21: 367–387.
- [6] Fishbain D, Goldberg M, Meagher B, Steele R, Rosomoff H. Male and femal chronic pain patients categorized by DSM-III psychiatric diagnostic criteria. Pain 1986; 26: 181–197.
- [7] Skootsky S, Jaeger B, Oye R. Prevalence of myofascial pain in general internal medicine practice. West J Med 1989; 151: 157–160.
- [8] Sola A, Bonica J. Myofascial pain syndromes. In: Bonica J, Loeser J, Chapman C. (eds) The management of pain. Lea & Febiger, Philadelphia. 1990. pp. 352–367.
- [9] Fricton J. Myofascial pain syndrome. Neurol Clin 1989; 7: 413–427.
- [10] Simons D. Myofascial trigger points: the critical experiment. J Musculoskelet Pain 1997; 5: 113–118.
- [11] Simons D, Stolov W. Microscpic features and transient contraction of palpable bands in canine muscle. Am J Phys Med 1976; 55: 65–88.
- [12] Baldry P. Trigger point acupuncture. In: Filshie Jand white A. (eds) Medical acupuncture, a western scientific approach. Churchill Livingstone, London. 1998. pp. 33–60.
- [13] Kuan T. Current studies on myofascial pain syndrome. Curr Pain Headache Rep 2009; 13: 365–369.
- [14] Zhuang X, Tan S, Huang Q. Understanding of myofascial trigger points. Chin Med J 2014; 127(4): 4271–4277.
- [15] Travell J, Simons D. Myofascial pain and dysfunction. The trigger point manual. Williams and Wilkins, Baltimore. 1983.
- [16] Simons D. Myofascial pain syndrome due to trigger points. In: Goodgold J (ed). Rehabilitation medicine. St Louis, Mosby. 1988. pp. 686–723.
- [17] Rachlin E. Trigger point. In: Rachlin E (ed) Myofascial pain and fibromyalgia: trigger point management. St. Louis, Mosby. 1994. pp. 145–157.
- [18] Irnish D, Gautschi R, Behrens N. Terminology. In: Irnish D (ed) Myofascial trigger points, comprehensive diagnosis and treatment. E-Book, 1st edition. Churchill Livingstone. 2013.
- [19] Renolds M. Myofascial trigger point syndromes in practice of rheumatology. Arch Phys Med Rehabil 1981; 62: 111–114.
- [20] Gerwin R, Shannon S, Hong C, Hubbard D, Gevirtz R. Interrater reliability in myofascial trigger point examination. Pain 1997; 69: 65–73.

- [21] Al-Shenqiti A, Oldham J. Test-retest reliability of myofascial trigger point detection in patients with rotator cuff tendonitis. Clin Rehabil 2005; 19: 482–487.
- [22] Macdonald A. Abnormally tender muscle regions and associated painful movements. Pain 1980; 8: 197–205.
- [23] Hong C. Pathophysiology of myofascial trigger point. J Formos Med Assoc 1996; 95: 93–104.
- [24] Myburgh C, Larsen A, Hartvigsen J. A systematic, critical review of manual palpation for identifying myofascial trigger points: evidence and clinical significance. Arch Phys Med Rehabil. 2008; 89:1169–1176.
- [25] Shah J, Phillips T, Danoff J, Gerber L. An in vivo microcanalytical technique for measuring the local biochemical milieu of human skeletal muscle. J Appl Physiol 2005; 99: 1977–1984.
- [26] Sikdar S, Shah J, Gebreab T, et al. Novel applications of ultrasound technology to visualize and characterized myofascial trigger points and surrounding soft tissues. Arch Phys Med Rehabil 2009; 90: 1829–1838.
- [27] Chen Q, Basford J, An K-N. Ability of magnetic resonance elastography to assess taut bands. Clin Biomech 2008; 23(5): 623–629.
- [28] Travell J, Rinzler S. The myofascial genesis of pain. Postgrad Med 1952; 11: 425–435.
- [29] Lewit K. The needle effect in the relief of myofascial pain. Pain 1979; 6: 83–90.
- [30] Hong C. Trigger point injection: dry needling versus lidocaine injection. Am J Phys Med Rehabil 1994; 73: 256–263.
- [31] Gunn C. The Gunn approach to the treatment of chronic pain. Intramuscular stimulation for myofascial pain of radiculopathic origin, 2nd edition. Churchill Livingstone, New York. 1996.
- [32] Irnish D, Euler D, Gleditsch J, Banzer W, Bachmann J. Acupuncture and related procedures. In: Irnish D (ed) Myofascial trigger points, comprehensive diagnosis and treatment. E-Book, 1st edition. Churchill Livingstone. 2013.
- [33] Ramps H. Adverse reactions to acupuncture. In: Filshie J, White A (eds) Medical acupuncture, a western scientific approach. Churchill Livingstone, London. 1998.
- [34] Vickers A, Zollman C. ABC of complementary medicine: acupuncture. BMJ 1999; 319: 973–976.
- [35] Ojala T, Arokoski J, Partanen J. The effect of small doses of Botulinum Toxin A on neckshoulder myofascial pain syndrome: a double-blind randomized, and controlled crossover trial. Clin J Pain 2006; 22: 90–96.
- [36] Ho K, Tan, K. Botulinum toxin A for myofascial trigger point injection: a qualitative systematic review. Eur J Pain 2007; 11: 519–527.

- [37] Jeynes L, Gauci C. Evidence for the use of Botulinum Toxin in the chronic pain setting a review of the literature. Pain Pract 2008; 8: 269–279.
- [38] Schmitt H, Irnish D. Triger point infiltration. In: Irnish D. Myofascial trigger points, comprehensive diagnosis and treatment. E-Book, 1st edition. Churchill Livingstone. 2013.
- [39] Borodic J, Joseph M, Fay L. Botulinum A toxin for the treatment of spasmodic torticollis: dyphagia and regional toxin spread. Head Neck 1990; 12: 392–398.
- [40] Drewes A, Andreason A, Poulsen L. Injection therapy for treatment of chronic myofascial pain: a double-blind study comparing corticosteroid versus diclofenac injections. J Musculoskelet Pain 1993; 1: 289–294.
- [41] Frost A. Diclofenac versus lidocaine as an injection therapy in myofascial pain. Scand J Rheumatol 1986; 15: 153–156.
- [42] Lang P, Irnish D. Systemic pharmacotherapy. In: Irnish D (ed) Myofascial trigger points, comprehensive diagnosis and treatment. E-Book, 1st edition. Churchill Livingstone. 2013.
- [43] Schmitt H, Pothmann R, Banzer W, Hübscher M, Maier M, Kosub M. Physical procedures. In: Irnish D (ed) Myofascial trigger points, comprehensive diagnosis and treatment. E-Book, 1st edition. Churchill Livingstone. 2013.
- [44] Hong, C.Z., Chen, Y.C., Pon, C.H., Yu, J. Immediate effects of various physical medicine modalities on pain threshold of an active myofascial trigger point. J Musculoskelet Pain 1993; 1: 37–53.
- [45] Lee J, Lin D, Hong C. The effectiveness of simultaneous thermotherapy with ultrasound and electrotherapy with combined AC and DC current on the immediate pain relief of myofascialtrigger point. J Musculoskelet Pain 1997; 5: 81–90.
- [46] Srbely J, Dickey J, Lowerison M, Edwards A, Nolet P, Wong L. Stimulation of myofascial trigger points with ultrasound induces asegmental antinociceptive effects: a randomized controlled study. Pain 2008; 139: 260–266.
- [47] Srbely J, Dickey J. Randomized controlled study of the antinociceptive effect of ultrasound on trigger point sensitivity: novel applications in myofascial therapy? Clin Rehabil 2007; 21: 411–417.
- [48] Khan J. Electrical modalities in the treatment of myofascial conditions. In: Rachlin E (ed) Myofascial pain and frbromyalgia. Trigger point management. St. Louis, Mosby. 1994.
- [49] Hsueh T, Cheng P, Kuan T, Hong C. The immediate effectiveness of electrical nerve stimulation and electrical muscle stimulation on myofascial trigger points. Am J Phys Med Rehabil 1997; 76: 471–476.
- [50] Graff-Radford S, Reeves J, Baker R, Chiu D. Effects oftranscutaneous electrical nerve stimulation on myofascial pain and trigger point sensitivity. Pain 1989; 37: 1–3.
- [51] Baxter GD. Therapeutic laser. Theory and practice. Churchill Livingstone, London. 1994.

- [52] Basford J. Low intensity laser therapy: still not an established clinical tool. Laser Surg Med 1995; 16: 331–342.
- [53] Borges L, Cerqueira M, dos Santos Rocha J, Conrado L, Machado M, Pereira R, Net O. Light-emitting diode phototherapy improves muscle recovery after a damaging exercise. Lasers Med Sci 2014; 29: 1139–1144.
- [54] Snyder-Mackler L, Barry A, Perkins A, Soucek M. Effects of helium-neon laser irradiation on skin resistance and pain in patients with trigger points in the neck or back. Phys Therapy 1989; 69: 336–341.
- [55] Olavi A, Pekka R, Pertti K J. Effects of the infrared laser therapy at treated and nontreated trigger points. Acupunct Electrother Res Int J 1989; 14: 9–14.
- [56] Al-Shenqiti A, Oldham J. The use of low level laser therapy (LLLT) in the treatment of trigger points that are associated with rotator cuff tendonitis. In: Longo L, Hofstetter AG, Pascu M-L, Waidelich WRA (eds) Proceedings of SPIE vol. 5287 Laser Florence 2002: a window on the Laser Medicine World, Bellingham, WA: SPIE. 2003. pp. 91–101.
- [57] Ceylan Y, Hizmetli S, Silig Y. The effects of infrared laser and medical treatments on pain and serotonin degradation products in patients with myofascial pain syndrome. A Controlled Trial Rheumatol Int 2004; 24: 260–263.
- [58] Lam L, Cheing G. Effects of 904 nm low-level laser therapy in the management of lateral epicondylitis: a randomized controlled trial. Photomed Laser Surg 2007; 25: 65–71.
- [59] Demirkol N, Sari F, Bulbul M, Demirkol M, Simsek I, Usumez A. Effectiveness of occlusal splints and low-level laser therapy on myofascial pain. Lasers Med Sci 2015; 30: 1007–1012.
- [60] Hsieh Y, Hong C, Chou L, Yang S, Yang C. Fluence-dependent effects of low-level laser therapy in myofascial trigger spots on modulation of biochemicals associated with pain in a rabbit model. Lasers Med Sci 2015; 30: 209–216.
- [61] Castel J, Abergel R, Willner R, Baumann J. low energy laser biostimulation: new prospects for medical applications. Lasers Med 1986; 712: 242–247.
- [62] Ohshiro T, Calderhead R. Low level laser therapy: a practical introduction. John Wiley and Sons, Chichester. 1988.
- [63] Baxter D. Low intensity laser therapy. In: Kitchen S, Bazin S (eds) Clayton's electrotherapy. 10th edition. W. B. Saunders Company Limited, London. 1996. pp. 197–217.
- [64] Patel C. CW high-power N₂-CO₂ laser. Applied Physics Letters 1965; 7: 15–17
- [65] Turner J, Hode L. Low level laser therapy, clinical practice and scientific background, a guide for research scientists, doctors, dentists, veterinarians, and other interested parties within the medical field. Gra¨ngesberg: Prima Books in Sweden AB. 1999.
- [66] Kitchen S, Patridge C. A review of low level laser therapy. Physiotherapy 1991; 77: 161–168.

- [67] Ohshiro T. Light and life: a review of low reactive-level laser therapy, following 13 years' experience in over 12000 patients. Laser Ther 1993; 1: 5–22.
- [68] Bischko J. Use of the laser beam in acupuncture. Acupunct Electrother Res 1980; 5: 29–40.
- [69] Waylonis G, Wilke S, O'Tool D, Waylonis D, Waylonis D. Chronic myofascial pain: management by low-output helium-neon laser therapy. Arch Phys Med Rehabil 1988; 69: 1017–1020.
- [70] Ceccherelli F, Altafini L, Lo Castro G, Avila A, Ambrosio F, Giron G. Diode laser in cervical myofascial pain: a double-blind study versus placebo. Clin J Pain 1989;5: 301–204.
- [71] FDA report, (File 510 'K' Number: K043353) 1st July 2005. Department of health & human services. Public Health Service. Food and Drug Administration, Rockville MD 20850. Low level laser therapy (LLLT) Omega XP Laser System. Class II, Performance Standards.
- [72] Kert J, Rose L. Clinical laser therapy: low level laser therapy. Scandinavian Medical Laser Technology, Copenhagen. 1989.
- [73] Pontinen P. Low level laser therapy as a medical treatment modality. A manual for physician, dentists, physiotherapists and veterinary surgeons. Tampere. 1992.
- [74] Pascu M. Laser physics. In: Simunovic Z (ed) Lasers in medicine and dentistry. Basic scientific and up-to-date clinical application of low energy-level laser therapy LLLT. European Medical Laser Association. Vitagraf d.o.o. 2000. pp. 24–74.
- [75] Low J, Reed A. Electrotherapy explained. Principals and practice, 3rd edition. Butterworth-Heinemann, Oxford. 2000.
- [76] England S. Introduction to mid laser therapy. Physiotherapy 1988; 74: 100–102.
- [77] Snyder-Mackler L, Seitz L. Therapeutic uses of light in rehabilitation. In: Michlovitz S. Thermal agents in rehabilitation, 2nd edition. F A Davis Company, Philadelphia. 1990.
- [78] Weisberg J. Ultraviolet irradiation. In: Hecox B, Mehreteab T, Weisberg J (eds). Physical agents. A comprehensive text for physical therapists. Appleton and Lange, Norwalk, Connecticut. 1994.
- [79] Karu T. Photobiological fundamentals of low-power laser therapy. IEEE J Quantum Electron 1987; 23: 1703–1717.
- [80] Boulton M, Marshall J. He-Ne laser stimulation of human fibroblast proliferation and attachment in vitro. Lasers Life Sci 1986; 1: 125–134.
- [81] Berki T, Nemeth P, Hegedus J. Biological effect of low power Helium-Neon (HeNe) laser irradiation. Lasers Med Sci 1988; 3: 35–39.
- [82] Karu T. Photochemical effects upon the cornea, skin and other tissues. Photobiology of low-power laser effects. Health Phys 1989; 56: 691–704.
- [83] Young S, Bolton P, Dyson M, Harvey W, Diamantopoulos C. Macrophage responsiveness to light therapy. Lasers Surg Med 1989; 9: 497–505.

- [84] Kelencz C, Muñoz I, Amorim C, Nicolau R. Effect of low-power gallium-aluminumarsenium noncoherent light (640 nm) on muscle activity: a clinical study. Photomed Laser Surg 2010; 28: 647–52.
- [85] Leal Junior E, de Godoi V, Mancalossi J, Rossi R, De Marchi T, Parente M, Grosselli D, Generosi R, Basso M, Frigo L, Tomazoni S, Bjordal J, Lopes-Martins R. Comparison between cold water immersion therapy (CWIT) and light emitting diode therapy (LEDT) in short-term skeletal muscle recovery after high-intensity exercise in athletes—preliminary results. Lasers Med Sci 2011; 26: 493–501.
- [86] Kubota J, Ohshiro T. The effects of diode laser low-reactive-level laser therapy (LLLT) on flab survival in a rat model. Laser Ther 1989; 127–133.
- [87] Ohshiro T. Low reactive level laser therapy. Practical applications. John Wiley and Sons, Chichester. 1991.
- [88] Simunovic Z. Lasers in medicine and dentistry. Basic science and up-to-date clinical application of low energy-level laser therapy LLLT, part one, European Medical Laser Association. Vitagraf d.o.o. 2000.
- [89] Antipa C. Contributions to LLL clinical therapy. In: Simunovic Z (ed) Lasers in medicine and dentistry. Basic science, and up-to-date clinical application of low energy-level laser therapy LLLT, part 1, European Medical Laser Association. Vitagraf d.o.d. 2000.
- [90] Simunovic Z. Low level laser therapy with trigger points technique: a clinical study on 243 patients. J Clin Laser Med Surg 1996;14:163–167.
- [91] Altan L, Bingol U, Aykac M, Yurtkuran M. Investigation of the effect of GaAs laser therapy on cervical myofascial pain syndrome. Rheumatol Int 2005; 25: 23–27.
- [92] Dundar U, Evcik D, Samli F, Pusak H, Kavuncu V. The effect of gallium arsenide aluminium laser therapy in the management of cervical myofascial pain syndrome: a double blind, placebocontrolled study. Clin Rheumatol 2007; 26: 930–934.
- [93] Hashmi J, Huang Y, Sharma S, Kurup D, MSEE L, Carroll J, Hamblin M. Effect of pulsing in low-level light therapy. Lasers Surg Med 2010; 42: 450–466.
- [94] Laakso L, Richardson C, Cramond T. Factors affecting low level laser therapy. Australian Physiotherapy 1993; 39: 94–98.
- [95] Simunovic Z, Trobonjaca T, Trobonjaca Z. Treatment of medial and lateral epicondylitis-tennis and golfer's elbow-with low level laser therapy: a multicenter double blind, placebo-controlled clinical study on 324 patients. J Clin Laser Med Surg 1998;16:145–151.
- [96] Morselli M, Soragni O, Anselmi C, Farinelli F. Very low energy-density treatments by CO₂ laser in sport medicine. Laser Surg Med 1985; 5: 150
- [97] Morselli M, Soragni O, Lupia P et al: Effects of very low energy-density treatment of joint pain by CO, laser, Laser Surg Med 1985; 5: 149
- [98] Martino G, Fava G, Galperti G. CO₂ laser therapy for women with mastalgia. Lasers Surg Med 1987; 7: 78–82.

- [99] Al-Shenqiti A, Oldham J. The use of low intensity laser therapy in the treatment of myofascial trigger points: an updated critical review. Phys Ther Rev 2009; 14: 115–123.
- [100] Diamentopoulos C. Bioenergetics and tissues optics. In: Baxter GD (ed) Therapeutic lasers. Theory and practice. Churchill Livingstone, Edinburgh. 1994.
- [101] Trelles M, Mayayo E, Miro L, Rigau J, Baudin G, Calderhead R. The action of low reactive level laser therapy (LLLT) on mast cells: a possible pain relief mechanism examined. Laser Ther 1989; 1: 27–30.
- [102] Nussbaum E, Baxter GD, Lilge L. A review of laser technology and light-tissue interactions as a background to therapeutic applications of low intensity lasers and other light sources. Phys Ther Rev 2003; 8: 31–44.
- [103] Castano A, Dai T, Yaroslavsky I et al. Low-level laser therapy for zymosan-induced arthritis in rats: Importance of illumination time. Laser Surg Med 2007; 39: 543–550.
- [104] Lanzafame R, Stadler I, Kurtz A et al. Reciprocity of exposure time and irradiance on energy density during photoradiation on wound healing in a murine pressure ulcer model. Laser Surg Med 2007; 39: 534–542.
- [105] Oron, U, Yaakobi T, Oronet A et al. Attenuation of infarct size in rats and dogs after myocardial infarction by lowenergy laser irradiation. Laser Surg Med 2001; 28: 204–211.
- [106] Chow R, Armati P, Laakso E, Bjordal J, Baxter G D. Inhibitory effects of laser irradiation on peripheral mammalian nerves and relevance to analgesic effects: a systematic review. Photomed Laser Surg 2011; 29: 365–381.
- [107] Enwemeka C. Intricacies of dose in laser phototherapy for tissue repair and pain relief. Photomed Laser Surg 2009; 27: 387–393.
- [108] Karu T, Tiphlova O, Samokhina M, Diamontopoulos C, Sarantsev V, Shveikin V. Effects of infra-red laser and superluminous diode irradiation on Escherichia coli division rate. IEEE J Quantum Electron 1990; 26; 2163–2165.
- [109] Karu T, Pyatibrat L, Kalendo G, Esenaliev R. Effects of monochromatic low-intensity light and laser irradiation on adhesion of HeLa cells in vitro. Lasers Surg Med 1996; 18: 171–177.
- [110] Nussbaum E, Van Zuylen J, Baxter D. Specifications of treatment dosage in laser therapy: Unreliable equipment and radiant power determination as confounding factors. Physiother Can 1999; 51: 159–167.
- [111] Dyson M, Young S. Effect of laser therapy on wound contraction and cellularity in mice. Lasers Med Sci 1986; 1: 125–130.
- [112] Longo I, Evangelista S, Tinnaci G, Sesti A. Effects of diodes-laser-silver-aluminium (Ga-Al-As) 904 nm on healing of experimental wounds. Lasers Surg Med 1987; 7: 444–447.
- [113] Rajaratnam S, Bolton P, Dyson M. Macrophage responsiveness to laser therapy with varying pulsing frequencies. Laser Ther 1994; 6: 107–112.

- [114] El-Sayed S, Dyson M. Effect of laser pulse repetition rate and pulse duration on mast cell number and degranulation. Lasers Surg Med 1996; 19: 433–437.
- [115] Sushko B, Lymans'kyi I, Huliar S. Action of the red and infrared electromagnetic waves of light-emitting diodes on the behavioral manifestation of somatic pain. Fiziol Zh. 2007; 53: 51–60.
- [116] Smith K. The photobiological basis of low level laser radiation. Laser Ther 1991; 3: 19–24.
- [117] Navratil L, Dyleysky I. Mechanisms of the analgesic effect of therapeutic lasers in vivo. Laser Ther 1997; 9: 33–39.
- [118] Walker J. Relief from chronic pain by low power laser irradiation. Neurosci Lett 1983; 43: 339–344.
- [119] Siddall P, Cousins M. Neural blockade in clinical anesthesia. In: Cousins M, Bridenbaugh P (eds) Introduction to pain mechanisms—implications for neural blockade. Lippincott-Raven, Philadelphia. 1998. pp. 675–713.
- [120] Bjordal J, Johnson M, Iverson V, Aimbire F, Lopes-Martins R. Photoradiation in acute pain: a systematic review of possible mechanisms of action and clinical effects in randomized placebo-controlled trials. Photomed Laser Surg 2006; 24: 158–168.
- [121] Kramer J, Sandrin M. Effect of low-power laser and white light on sensory conduction rate of the superficial radial nerve. Physiother Can 1993; 45:165–170.
- [122] Cambier D, K. Blom, E. Witvrouw E, Ollevier G, De Muynck M, Vanderstraeten G. The influence of low intensity infrared laser irradiation on conduction characteristics of peripheral nerve: a randomised, controlled, double blind study on the sural nerve. Laser Med Sci 2000; 15:195–200.
- [123] Telemeco T, Schrank E. The effect of light therapy on superficial radial nerve conduction using a clustered array of infrared superluminous diodes and red light emitting diodes. J Lasers Med Sci 2013; 4:17–24.
- [124] Mezawa S, Iwata K, Naito K, Kamogawa H. 1988. The possible analgesic effect of softlaser irradiation on heat nociceptors in the cat tongue. Archs Oral Biol 1988; 33: 693–694.
- [125] Tsuchiya K, Kawatani M, Takeshige C, Sato T, Matsumoto I. Diode laser irradiation selectively diminishes slow component of axonal volleys to dorsal roots from the saphenous nerve in the rat. Neurosci Lett. 1993; 161: 65–68.
- [126] Tsuchiya D, Kawatani M, Takeshige C. Laser irradiation abates neuronal responses to nociceptive stimulation of rat-paw skin. Brain Res Bull 1994; 34: 369–374.



IntechOpen