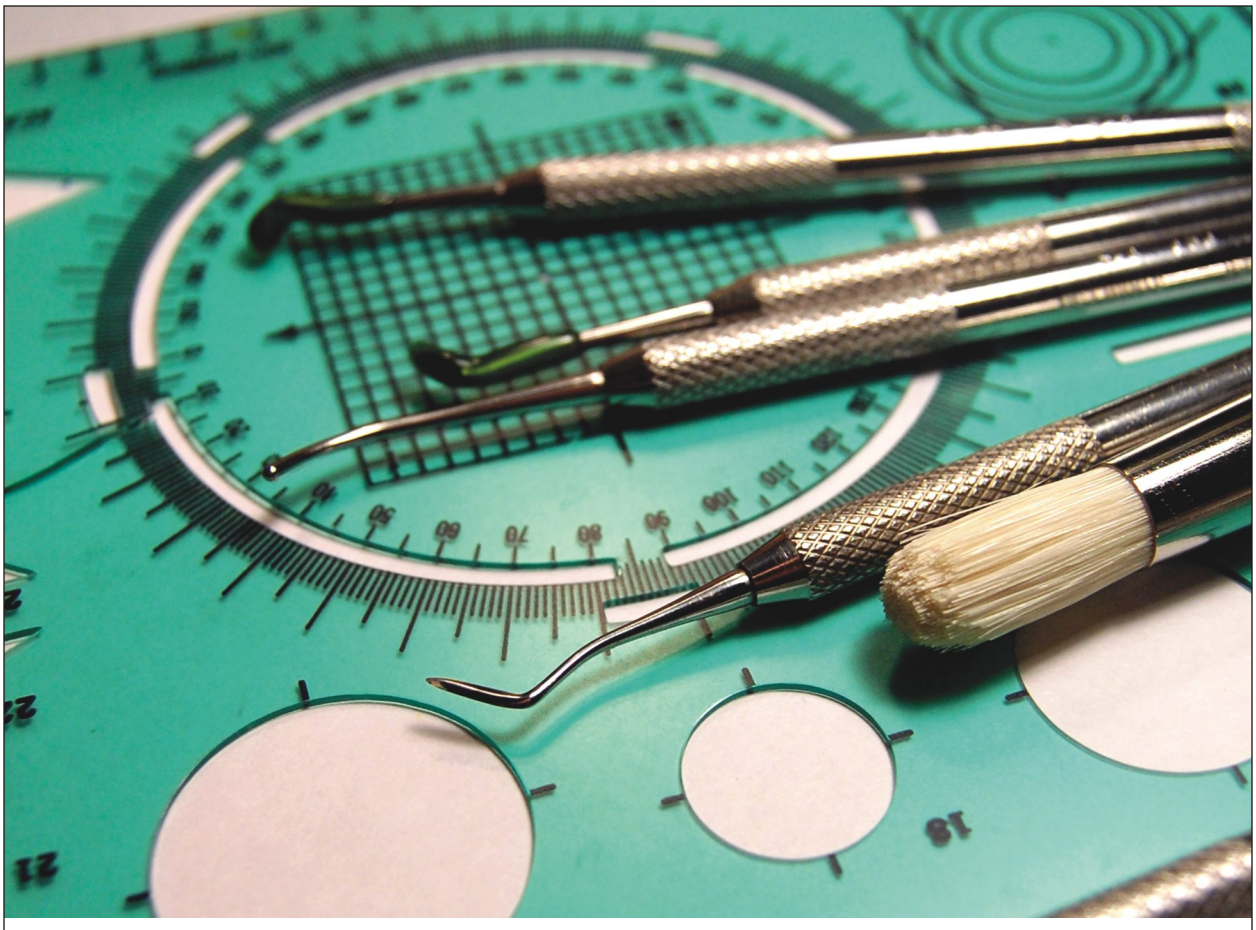

European Journal of Dentistry and Medicine

ISSN 1996-3378

Academic Journals Inc.

www.academicjournals.com

OPEN ACCESS European Journal of Dentistry and Medicine

ISSN 1996-3378
DOI: 10.3923/ejdm.2016.1.5

Review Article
Pain Pathways and Effect of Low Level Laser Therapy on Pain During Fixed Orthodontic Treatment-an Overview

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Abstract
Pain is a subjective phenomenon extremely difficult to generalise presenting itself in various forms, which motivate an individual more than any other life experience. It is a typical symptom seen during the initial stage of orthodontic treatment which leads to decreased acceptance and non compliance over the next few stages and ultimately may result in treatment interruption in a few. It is dependent upon a number of factors, such as gender, age, stress and the magnitude of orthodontic force applied. The mechanism of pain perception can be described by a number of theories as the specificity, intensity and the most importantly the gate control theory of pain. Many non pharmacological and pharmacological methods are being explored to determine ways to reduce the perception of pain. Low level laser therapy as a non pharmacological method came to view after the discovery of its biostimulatory effects in 1971. Low level laser therapy appears to produce photobiomodulation in the body and can reduce pain, quicken wound healing and exercise a positive effect on the inflammatory process, which is the core reason for pain during the initial phases of orthodontic treatment. Hence, low level laser therapy has a lot of scope in the field of orthodontics as one of the methods to improve patient compliance. The following study is literature review of the pain pathways and how low level laser therapy acts on them to inhibit the pain.

Key words: Pain, separators, inflammation, low level laser therapy

Received: May 08, 2016 Accepted: May 31, 2016 Published: June 15, 2016

Citation: T. Tripathi, G. Ganesh and P. Rai, 2016. Pain pathways and effect of low level laser therapy on pain during fixed orthodontic treatment-an overview. Eur. J. Dent. Med., 8: 1-5.

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Competing Interest: The authors have declared that no competing interest exists.

Data Availability: All relevant data are within the paper and its supporting information files.

INTRODUCTION

The International Association for the study of pain describes the term ''Pain'' as an ''Unpleasant sensory and emotional experience associated with actual or potential tissue damage[1].''

The frequency, duration and magnitude varies from person to person as pain is subjective in nature, thus becoming one of the most frequent complications of initial orthodontic treatment causing patients to discontinue treatment[2].

Pain during the initial stages of orthodontic treatment is mainly inflammatory in nature thus, a lot of orthodontists have been prescribing analgesics or Non Steroidal Anti Inflammatory Drugs (NSAIDs) in adult patients to relieve the pain[3]. However, it has been suggested that tooth movement required during treatment may be affected in patients who use NSAIDs[3,4].

Low Level Laser Therapy (LLLT) per se came into existence soon after the invention of the ruby laser in 1960 and the helium-neon (HeNe) laser in 1961[5]. However, the biostimulatory effects were only seen by 1971[6].

Low level laser therapy produces biostimulation without producing any direct thermal effects in the area of application[7].

There is evidence that low level laser therapy can reduce pain, quicken wound healing and exercise a positive effect on the inflammatory process.

The analgesic efficacy of LLLT has been investigated and several mechanisms for pain relief have been proposed like the modulation of endorphin production, the anti-inflammatory effect and the direct inhibition of neural activity[8].

The following study is literature review of the pain pathways and how low level laser therapy acts on them to inhibit the pain.

The various presentations of pain: Pain can present itself in numerous forms, such as acute or chronic, superficial or deep, somatic or visceral to name a few.

Acute v/s chronic pain: Acute and chronic pain are different clinical entities. Acute pain is the pain, which is self limiting in nature, provoked by a specific disease or injury but serves a useful biologic purpose and is associated with skeletal muscle spasm and sympathetic nervous system activation. On the other hand, chronic pain is a disease state where pain outlasts the normal time of healing if associated with a disease or injury. Chronic pain serves no biologic purpose and has no recognizable end-point[8].

Primary v/s secondary pain: The site where the pain is felt may or may not identify the location of the source of pain. If the pain does emanates from the structures that hurt it is primary pain while if the source of pain is located elsewhere, it represents secondary pain[9].

Superficial v/s deep pain: Pain emanating from the cutaneous and mucogingival tissues represent superficial pain which can be precisely located and related to timing, location and intensity. In contrast, pain from stimulation of deeper musculoskeletal and visceral structures is more diffusely felt and responds less to provocation[9].

Theories of pain: A number of theories have been postulated to describe mechanisms underlying pain perception. These theories date back several centuries but the three most accepted theories of pain perception include the specificity, intensity and gate control theories of pain.

Receptors for pain: Normally, pain perception is evoked only at extreme pressures and temperatures enough to potentially injure tissues using toxic molecules and inflammatory mediators. Nociceptors, the pain receptors, detect high threshold physical and noxious chemical stimuli[10].

Nociceptors essentially are A delta and C nerve fibres which are electrically silent and transmit all or-none action potentials when stimulated. However, nociceptor activity does not lead to the perception of pain per se. This requires peripheral information to reach higher centres which depends on the frequency of action potentials in primary afferents, temporal summation of pre and postsynaptic signals and central influences[10].

Pain pathways: Pain is perceived at the site of the injury after the signals are relayed to the higher centres by means of a pathway that involves 2 tracts: The neospinothalamic and paleospinothalmic tracts (Fig. 1).

Neospinothalamic tract[11] carries the signals from the A delta pain fibres, which transmit acute thermal and mechanical pain.

Glutamate is the neurotransmitter substance secreted in the spinal cord at the A delta nerve fibre endings.

Paleospinothalamic pathway[11] transmits pain mainly from the peripheral slow-chronic type C pain fibres.

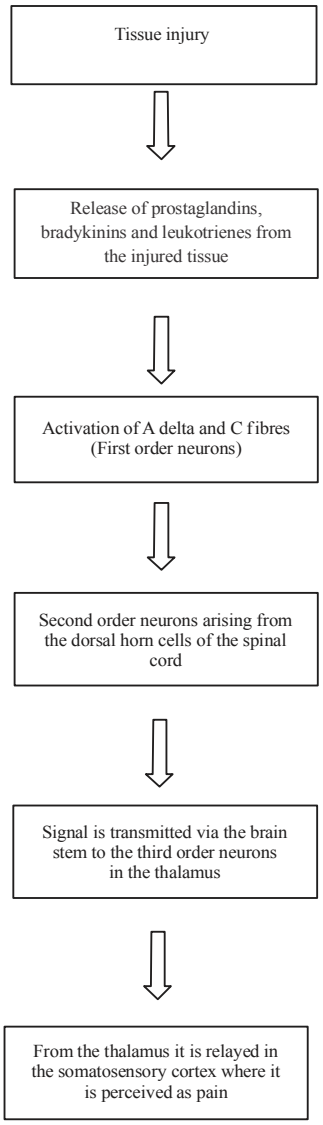

Fig. 1: This flowchart traces the pathway for transmission of pain from the point of tissue injury till the cerebral cortex where the pain is perceived

Substance P is the slow-chronic neurotransmitter of type C nerve endings.

Pain during orthodontic treatment: Pain during orthodontic treatment is an inflammatory type of pain, not an infection-related pain it is localized and of short duration[7].

According to the literature, pain starts 2 h after the application of orthodontic fixed appliance, rises over the next 24-36 h, starts decreasing on day 3 and disappears within 6-7 days[12,13].

It has been reported that the initial tooth displacement caused by using dental separators causes pain and immediate release of mediating biochemical compounds into the gingival fluid. This is attributed to the increase in the levels of prostaglandin E2 tin the initial stage and the increase in interleukin (IL)-1 one day later[14].

Low level laser therapy: The experience of pain during the initial orthodontic treatment represents an important area of concern which may affect the patient's cooperation and lead to early treatment interruption[15].

The biostimulatory effects of low level laser therapy were introduced in 1971 following, which it developed into a massive therapeutic procedure utilised not only for pain but also other purposes like tissue repair and healing.

Low level laser therapy is exposing cells or tissue to low levels of red and near infrared light (NIR) and is called "Low level" as it uses light at energy densities which are low as compared to other forms of laser therapies which are used for cutting, ablation and thermally coagulating tissue. This therapy is also known as "Cold laser" therapy as the power density used is lower than that needed to produce heating of tissue. Originally believed to be using coherent laser light, currently as a cheaper alternative and Light Emitting Diodes (LEDs) are being considered[5].

The effects of low level lasers on cells are related to various parameters such as wavelength, power density, frequency and time[7]. According, to the Arndt-Schulz law, if the light stimulus is delivered below the recommended dose it will be insufficient to trigger the target functions and may even cause inactivation of these functions if a higher dose is given[16].

Karu[17] said that lasers that provide this type of effect are within the wavelength range of 600-1000 nm. Chung *et al.*[5] report that low level laser therapy used wavelength of light between 600-1070 nm. The tissue penetration is maximum in this range as the principal tissue chromatophores (haemoglobin and melanin) have high absorption bands at wavelengths less than 600 nm. Wavelengths in the range 600-700 nm are used to treat superficial tissue and longer wavelengths between 780-950 nm are used to treat deeper-seated tissues. Wavelengths from 700-770 nm have limited biochemical activity and are hence not used[5].

Mechanism of action of low level laser therapy: Several studies have been done in the past which demonstrate the effectiveness of low level laser therapy in reduction of pain by describing various mechanisms.

Low level laser therapy basically acts by inducing a photochemical reaction in the cell, a process referred to as biostimulation or photobiomodulation. It has a wide range of effects at the molecular, cellular and tissue levels[5].

Immune cells responsible for the inflammatory process are strongly affected by low level laser therapy. Mast cells playing a major role in inflammation undergo degranulation at specific wavelengths which results in the release of the pro-inflammatory cytokine TNF-a from the cells[18,19]. Low level laser therapy enhances the proliferation, maturation and motility of fibroblasts and increases the production of basic fibroblast growth factor[20,21]. Lymphocytes proliferate more rapidly and epithelial cells exhibit increased motility thus allowing wound sites to close more quickly. The ability of macrophages to act as phagocytes is also enhanced under the application of LLLT[5].

Honmura *et al*.[22] have shown that low level laser therapy can modulate the inflammatory process and thus reduce pain by preventing the release of prostaglandins, leukotrienes and bradykinins from the injured tissue[22]. After tissue injury, arachidonic acid released from the damaged cell membrane acts as a precursor for the release of inflammatory mediators such as prostaglandins and leukotrienes. Along with that, mast cell degranulation also releases certain chemical mediators responsible for inflammation.

Low level laser therapy basically prevents the release of prostaglandins and other inflammatory mediators thus, reducing pain perception.

A second view is that low level laser therapy alters nerve conduction and excitation in peripheral nerves by its action on the sodium potassium pump[23]. As it alters the conduction of nerve impulses it decreases the perception of pain.

The third view suggests that low level laser therapy may stimulate and activate the production of endogenous endorphins which act on the mu receptors and prevent the action of substance P on these receptors thus having morphine like action[24]. Endorphins are naturally produced compounds that act on the pain receptors and compete with substance P, the neurotransmitter for pain. Recent studies suggest that cells of the immune system are also capable of B endorphin synthesis as they possess mRNA transcripts for T, B lymphocytes, monocytes and macrophages.

However, a large number of factors, such as the wavelength, power, pulse structure and timing of the applied light are chosen and are specific for each effect of the treatment. A less than optimal choice can result in reduced effectiveness of the treatment or even a negative therapeutic outcome. Hence, many of the published results of low level lasers include negative results simply because of an inappropriate choice of light source and dosage[5].

CONCLUSION

Pain is one of the most common symptoms for which the patient seeks treatment. Managing pain and relieving suffering should be at core of the heath professional community.

The field of orthodontics involves long span treatments hence it is extremely essential to put the patient in comfort at the beginning itself which improves the patients compliance and thus the treatment outcome.

Low level laser therapy targets orthodontic pain which is inflammatory in nature and seen during the initial stages of treatment. It acts by producing photobiomodulation in the body and reduces pain, quickens wound healing and exercises a positive effect on the inflammatory process.

There are considerable evidences stating the effectiveness of low level laser therapy on reduction of orthodontic pain. However, the parameters like wavelength, power and frequency of the low level laser need to be standardized .

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