Review Article

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Laser Induced Photobiomodulation for Acceleration of Orthodontic Tooth Movement

Sameer Ahmed[1] Manjula Rathod[2] Venkata Naidu[3*] Sanagamesh B[4]

[1]Reader, Department of Orthodontics, AME Dental College, Raichur, Karnataka, India.
[2]PG Student, Department of Orthodontics, AME Dental College, Raichur, Karnataka, India.
[3]Senior Lecturer, Department of Orthodontics, AME Dental College, Raichur, Karnataka, India.
[4]Professor and Head, Department of Orthodontics, AME Dental College, Raichur, Karnataka, India.

ABSTRACT

Background: There are various invasive and non invasive methods for accelerating the orthodontic tooth movement. The semiconductor diode laser being the recent and non invasive approach for accelerating the orthodontic tooth movement. 1953 John von Neumann described the concept of semiconductor laser. The application of low-level lasers in medicine was introduced in the 1970s and 1980s. LASER is an acronym for light amplification by stimulated emission of radiation.Laser therapy causes the increase of receptor activator of nuclear factor kappa-B ligand in periodontal ligament thereby increasing the rate of tooth movement during orthodontic treatment. Low level laser therapy can increase macrophage-colony stimulating factor on the compressed side and may also increase osteoclastogenesis leading to tooth movement. There are various types of laser which are helpful for acceleration of orthodontic tooth movement**.** Which are, GaAlAs: 860nm, 940nm, 830nm, 940nm,760 nm, 785 nm, 808 nm and 980 nm; He-Ne laser of less than 1mW, Argon lasers and Indium-gallium-aluminum-phosphide laser. The Ga-Al-As semiconductor diode laser of different wavelength ranging from 808nm to 980nm; and with power output of 50 to 100 mW can be used for acceleration of orthodontic tooth movement. The initiation for acceleration of orthodontic tooth movement varies from 7 days to 3 months. The acceleration in tooth movement is in a range between 0.5mm to 4.45mm. This can reduce the treatment duration range form 18-24 months to 6-8 months.

Keywords: Laser, Photobiomodulation, Orthodontic.

INTRODUCTION

Light Amplification by Stimulated Emission of Radiation **(**LASER) was built in 1960 by Theodore H. Maiman at Hughes Research Laboratories, based on theoretical work by Charles Hard Townes and Arthur Leonard Schawlow. The use of lasers on the gingiva was first approved by the US Food and Drug Administration in the early 1990s, and use on hard tissue like teeth or the bone of the mandible gained approval in 1996. Several variants of dental lasers are in use with different wavelengths suited for different applications.[1]

Lasers are classified as follows:

A] Based on the type of active state of material (LASER medium):

1. Gas lasers
2. Liquid lasers
3. Solid-state lasers
4. Semiconductor diode lasers.

B] Based on element and intensity:

1. Low-level (Soft) Lasers : < 500 mW

i) Helium Neon (He-Ne) of 632.8 nm,
ii) Gallium Aluminum Arsenide (Ga-Al-As) of 940 nm and
iii) Semiconductor diode laser (630-980 nm).

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***Correspondence Dr. Venkata Naidu.**
Department of Orthodontics, Ame Dental College, Raichur, Karnataka, India.
Email: ameortho2003@gmail.com

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2. High-intensity (Hard) Lasers: > above 500 mW.

i) Yag lasers - (Nd:Yag), Erbium Yag lasers (Er:Yag),
ii) CO2 lasers,
iii) Argon lasers.[10 11 12 13]

C] General usage based on average power required:

Power	**Use**
1–5 mW	Laser pointers
5 mW	CD-ROM drive
5–10 mW	DVD player or DVD-ROM drive
100 mW	High-speed CD-RW burner
250 mW	Consumer 16×DVD-R burner
400 mW	Burning through a jewel case including disc within 4 seconds
	DVD 24× dual-layer recording
1 W	Green laser in Holographic Versatile Disc prototype development
1–20 W	Output of the majority of commercially available solid-state lasers used for micro machining
30–100 W	Typical sealed CO_2 surgical lasers[83]
100–3000 W	Typical sealed CO_2 lasers used in industrial laser cutting

D] Based on the clinical method of application:

i) Contact (Point contact or woodpecker technique)
ii) Contact-free techniques

E] Based on safety class number, which identifies how dangerous the laser is:

i) Class 1 is inherently safe, usually because the light is contained in an enclosure.
ii) Class 2 is safe during normal use; the blink reflex of the eye will prevent damage. Usually up to 1 mW power.
iii) Class 3R (formerly IIIa) lasers are usually up to 5 mW and involve a small risk of eye damage within the time of the blink reflex. Staring into such a beam for several seconds is likely to cause damage to a spot on the retina.
iv) Class 3B can cause immediate eye damage upon exposure.
v) Class 4 lasers can burn skin, and in some cases, even scattered light can cause eye and/or skin damage.

Uses of Lasers in medicine and dentistry:

Medicine	**Dentistry**
Wound healing	Cutting of hard and soft tissues
Reduce inflammation	Coagulation
Surgical operations	Disinfection of root canals
Skin treatment	Caries removal
Removing kidney stones	Periodontal therapy
Removing the tumors	Pain management
Repairing the retina of eye	Orthodontic tooth movement

Low Level Laser Therapy (bio-stimulation) is based on stimulation of cellular activity as a result of absorbing laser radiation. It exerts influence on tissues without increasing their temperature above 36.5°C, or exceeding the normal body temperature.[2] On the other hand, the energy absorbed by a cell is used in biophysical and biochemical processes.[3] The activity of laser light is accompanied by several physical phenomena, i.e. transmission, reflection, scattering and absorption. The depth of transmission (light penetration into the tissue) depends on the length of a light wave, as well as on the power of radiation, and the properties of the tissue undergoing treatment (its density, thickness, hydration, presence of pigments). Biostimulation lasers may emit waves within the range of visible (380-700 nm) and invisible (700-1000 nm) light. The radiation of visible waves and near infrared radiation penetrates the osseous tissue to a considerable depth.[4]

Mechanism of LLLT on tissue:

The precise biochemical mechanism underlying the therapeutic effects of LLLT are not yet well-established. From observations, it appears that LLLT has a wide range of effects at the molecular, cellular, and tissular levels. In addition, its specific modes of action may vary among different applications. Within the cell, there is strong evidence to suggest that LLLT acts on the mitochondria[5] to increase adenosine triphosphate (ATP) production,[6] modulation of reactive oxygen species (ROS), and the induction of transcription factors. Several transcription factors are regulated by changes in cellular redox state. Among them are: redox factor:- dependent activator protein (AP-1), nuclear factor kappa B (NF-κB), p53, activating transcription factor/cAMP-response element–binding protein (ATF/CREB), hypoxia-inducible factor (HIF)-1, and HIF-like factor.[7] These transcription factors then cause protein synthesis that triggers further effects down-stream, such as increased cell proliferation and migration, modulation in the levels of cytokines, growth factors and inflammatory mediators, and increased tissue oxygenation.[8] Immune cells, in particular, appear to be strongly affected by LLLT. Mast cells, which play a crucial role in the movement of leukocytes, are of considerable importance in inflammation. Specific wavelengths of light are able to trigger mast cell degranulation,[9] resulting in the release of the pro-inflammatory cytokine TNF-α from the cells. This leads to increased infiltration of the tissues by leukocytes. LLLT also enhances the proliferation, maturation, and motility of fibroblasts, and increases the production of basic fibroblast growth factor.[10,11] Lymphocytes become activated and proliferate more rapidly, and epithelial cells become more motile, allowing wound sites to close more quickly. The ability of macrophages to act as phagocytes is also enhanced under the application of LLLT.

Radiation of tissue with light causes an increase in mitochondrial products such as ATP, NADH, protein, and RNA,[12] as well as a reciprocal augmentation in oxygen consumption. Various *in vitro* experiments have confirmed that cellular respiration is up regulated when mitochondria are exposed to an HeNe laser or other forms of illumination. The relevant chromophore can be identified by matching the action spectra for the biological response to light in the NIR range to the absorption spectra of the four membrane-bound complexes identified in mitochondria.[13] This procedure indicates that complex IV, also known as cytochrome *c* oxidase (CCO), is the crucial chromophore in the cellular response to LLLT.[14] CCO is a large transmembrane protein complex, consisting of two copper centers and two heme–iron centers, which is a component of the respiratory electron transport chain.[15] The electron transport chain passes high-energy electrons from electron carriers through a series of transmembrane complexes (including CCO) to the final electron acceptor, generating a proton gradient that is used to produce ATP. Thus, the application of light directly influences ATP production by affecting one of the trans-membrane complexes in the chain: in particular, LLLT results in increased ATP production and electrontransport.[16,17]

The precise manner in which light affects CCO is not yet known. The observation that nitric oxide(NO) is released from cells during LLLT has led to speculation that CCO and NO release are linked by two possible pathways. It is possible that LLLT may cause photo dissociation of NO from CCO.[18] Cellular respiration[19,20] is down regulated by the production of NO by mitochondrial nitric oxide synthase (mtNOS-NOS isoform specific to mitochondria), that binds to CCO and inhibits the process. The NO displaces oxygen from CCO, inhibiting cellular respiration and thus decreases the production of ATP.[21] By dissociating NO from CCO, LLLT prevents this process from taking place and results in increased ATP production. An alternative or parallel mechanism to explain the biological activity of NIR light to release NO from cells or tissue is the following.[22] A new explanation has been recently proposed for how light increases NO bio availability. CCO can act as a nitrite reductase enzyme (a one electron reduction of nitrite gives NO) particularly when the oxygen partial pressure is in low. concentrations or at hypoxia condition.

The influence of LLLT on the electron transport chain extends far beyond simply increasing the levels of ATP produced by a cell. Oxygen acts as the final electron acceptor in the electron transport chain and is, in the process, converted to water. Part of the oxygen that is metabolized produces reactive oxygen species (ROS) as a natural by-product. Chemically active molecules that play an important

role in cell signaling, regulation of cell cycle progression, enzyme activation, and nucleic acid and protein synthesis. Because LLLT promotes the metabolism of oxygen, it also acts to increase ROS production. In turn, ROS activates transcription factors, which leads to the upregulation of various stimulatory and protective genes. These genes are most likely related to cellular proliferation, migration, and the production of cytokines and growth factors, which have all been shown to be stimulated by low-level light. The processes described above are almost certainly only part of the story needed to explain all the effects of LLLT. Among its many effects, LLLT has been shown to cause vasodilation by triggering the relaxation of smooth muscle associated with endothelium, which is highly relevant to the treatment of joint inflammation. This vasodilation increases the availability of oxygen to treated cells, and also allows for greater traffic of immune cells into tissue. These two effects contribute to accelerated healing. NO is a potent vasodilator via its effect on cyclic guanine monophosphate production, and it has been hypothesized that LLLT may cause photo dissociation of NO, not only from CCO, but from intracellular stores such as nitrosylated forms of both hemoglobin and myoglobin, leading to vasodilation.

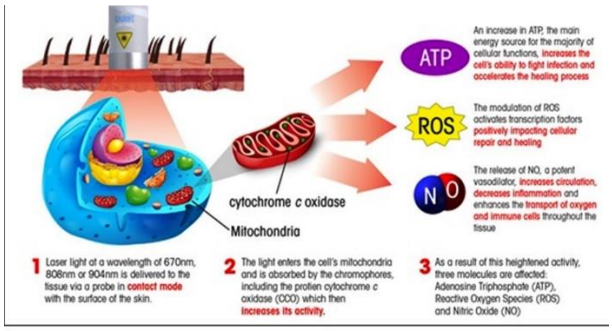

Fig 1: The mechanism of LLLT in Tissue

Discussion of Review of literature:

Delma R. Cruz, et al in 2004[23] studied the effects of Low-Intensity Laser Therapy on the Orthodontic movement velocity of human teeth. Iirradiated with a diode laser emitting light at780 nm, during 10 seconds at 20 mW, 5 J/cm2, on 4 days of each month. Data of the bio metrical progress of both groups were statistically compared. And concluded that LILT does accelerate human teeth movement and could therefore considerably shorten the whole treatment duration.

Fujita et al in 2008[24] studied the effect of low level laser therapy stimulates tooth movement velocity by expression of RANK and RANKL. The wavelength used is 810nm, energy density of 27.99J/cm^2. They concluded that irradiation of laser accelerate orthodontic tooth movement by induction of RANK and RANKL.

Nayer S. AboElsaad et al in 2009[25] studied **e**ffect of soft laser and bioactive glass on bone regeneration in the treatment of infra-bony defect slow-power 830 nm Gallium–Aluminium–Arsenide (GaAlAs)laser [continuous wave (CW) 40 mW and fluence 4 J/cm2,with total energy density of 16 J/cm2] on the healing of human infra-bony defects treated with bioactive glass graft Material in 20 patients with chronic periodontitis. Results have confirmed the positive effect of soft laser in accelerating periodontal wound healing.

AndreTortamano et al in 2009[26] studied Low-level laser therapy for pain caused by placement of the first orthodontic arch wire. A split mouth design of 60 patients was conducted. Laser irradiation was done on experimental group immediately after placement of first arch wire for 10 seconds. All patients received a questionnaire to be filled out at home describing their pain during the next 7 days. The result was low level laser therapy group had lower mean scores for oral pain and intensity of pain on the most painful day. Also, their pain ended sooner. Low level laser therapy did not affect the start of pain perception or alter the most painful day. There was no significant difference in pain symptomatology in the maxillary or mandibular arches in an evaluated parameter. They concluded that Low level laser therapy efficiently controls pain caused by the first arch wire.

Yamaguchi et al in 2010[27] studied the effect of Ga-Al-As laser of wavelength 810nm, energy density of 54.0J for orthodontic tooth movement. Concluded that Ga-Al-As helps in tooth movement.

Gauri Dosi Mehta et al in 2012[28] studied on the efficacy of low intensity laser therapy in reducing treatment time and orthodontic pain which was a split mouth design on twenty patients with premolar extraction. The experimental side was

irradiated with a Ga Al As diode laser. They concluded that Low level Laser therapy is a good option to reduce a treatment duration and pain.

Eliziane Cossetina et al 2013[29] studied the influence of low-level laser on bone remodeling during induced tooth movement in rats. They selected the 40-70-day old Wistar rats. They concluded that low level laser therapy significantly increased the osteoclastic but not the osteoblastic activity during the initial phases of tooth movement.

Won Tae Kim et al 2012[30] **s**tudied low-level laser therapy on perception of pain after separator placement and compare it with perceptions of control and placebo groups using a frequent irradiation protocol. They concluded, on experimental side that low level laser therapy is an effective method of reducing orthodontic pain.

Arezoo Jahanbin et al 2014[31] studied effectiveness of Er:YAG laser-aided fiberotomy and low-level laser therapy in alleviating relapse of rotated incisors. They concluded that Er-YAG laser-aided circumferential supracrestal fiberotomy proved to be an effective alternative to conventional circumferential supracrestal fiberotomy in reducing rotational relapse. Low level laser therapy with excessively high energy density was also as effective as the circumferential supracrestal fiberotomy procedures in alleviating relapse, at least in the short term.

Nikolaos Gkantidis at el 2014[32] studied effectiveness of non-conventional methods for accelerated orthodontic tooth movement. They concluded that there was some evidence that low laser therapy and corticotomy are effective.

Kyung-A Kim et al 2015[33] studied effect of low-level laser therapy on orthodontic tooth movement into bone-grafted alveolar defects. They concluded that low level laser therapy significantly decreased the rate of orthodontic tooth movement into the bone-grafted surgical defects by accelerating defect healing and maturation, particularly when the start of postoperative orthodontic tooth movement was delayed.

Irfan Qamruddin et al 2016[34] studied the effect of a single dose low-level laser therapy on spontaneous and chewing pain caused by elastomeric separators. They stated that a single dose of low-level laser therapy can be an efficient modality to reduce the postoperative pain associated with the placement of elastomeric separators.

Varella A M., et al 2018[35] studied the effect of low-level laser therapy, its correlation with interleukin-1b in gingival crevicular fluid and the rate of orthodontic tooth movement. They concluded that in combination with light orthodontic force, application of low-level laser therapy increased the levels of interlukin-1b in gingival crevicular fluid and accelerated orthodontic tooth movement.

According to different authors, the optimum range of laser energy doses that have the biostimulating effect, ranges from 0.1-12 J/cm2 to 1-20 J/cm2. Also the parameters have been systematically tabulated below.

Table 1: Various types of laser used in acceleration of orthodontic tooth movement.

Materials	Wavelengths	Power out put	Energy density	Tooth movement	Duration
Ga-Al-As	810nm[1]	100mW	54.0J	0.6mm	7 days
Ga-Al-As	800nm[2]	0.7mW	8J	5.49mean	4months
Ga-Al-As	808nm	20mW	0.7J/cm2	287mean	14days
Ga-Al-As	780nm[4]	70mW	4.5J/cm2	1.59 mm	6 weeks
Ga-Al-As	980nm[5]	100mw	5.6J/cm2	1.57mean	4months

Ga-Al-As	830nm[6]	150mW	2J/cm2	0.2mm	4months
Ga-Al-As	940nm[7]	100mW	8J/cm2	4.45mm	2months
Ga-Al-As	808nm[3]	100mW	54J	2.5mm	35 days
He-Ne	32nm	1mW		Hair growth in mice	
Ruby laser	6943nm	-	0.5-10J	wound healing	14days

Advantages:

a) Time saving
b) Less pain in some instances
c) Reduces the need of anaesthesia
d) Reduces anxiety in patient
e) Minimizes the bleeding and swelling
f) Reduces bacterial infection
g) Preserves more healthy tooth.

CONCLUSION

The Ga-Al-As semiconductor diode laser of different wavelength ranging from 808nm to 980nm; and with power output of 50 to 100 mW can be used for acceleration of orthodontic tooth movement. The initiation for acceleration of orthodontic tooth movement varies from 7 days to 3 months. The acceleration in tooth movement is in a range between 0.5mm to 4.45mm. This can reduce the treatment duration range form 18-24 months to 6-8 months.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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