Research article

Neurophysiological effect of low level laser therapy on Ulnar Nerve

Prathap Suganthirababu¹, Lavanya Prathap^{2*}, Jagatheesan Alagesan¹, Kumaresan A.¹, Vignesh Srinivasan³, Deepthi Ganesan⁴¹Professor, ³Assistant Professor, Saveetha College of Physiotherapy, Saveetha Institute of Medical and Technical Sciences, Chennai.²Associate Professor, Saveetha Dental College and Hospital, Saveetha Institute of Medical and Technical Sciences, Chennai.⁴Lecturer, School of Allied Health Sciences, AMIST, Malaysia.

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Corresponding author: **Lavanya Prathap**. Email: lavanya.anatomist@gmail.com

ABSTRACT

Introduction and Aim: Therapeutic low-level laser therapy aids in managing pain and inflammation in musculoskeletal ailments. Its effect on peripheral nervous system and its function are not yet clearly studied. The study aims to analyze the neurophysiological effect of therapeutic laser irradiation on ulnar nerve.

Materials and Methods: Sixty healthy subjects with age groups between 20-30 years of both genders were recruited in this experimental study. The procedure was clearly explained to the subjects and informed consent was obtained. Random allocation was done to participants to either as laser group or sham laser group. All the subjects were positioned in supine lying with the irradiated dominant hand kept at 135 degrees of flexion. The electrode for recording was placed on the hypothenar muscle and the stimulating electrode was placed 4 cm away distally from the medial epicondyle of the humerus and was secured with tape. The subject's skin (electrode placement area) was degreased for proper transmission of laser. A 904nm diode laser was chosen to irradiate the ulnar nerve behind the medial epicondyle of the humerus for 20 secs through the overlying skin. The laser delivered continuous energy at 4.0 J/cm². Antidromically action potential, peak to peak amplitude, onset latency, nerve conduction velocity were recorded before and after irradiation of the ulnar nerve.

Results: The study revealed significant difference in peak-to-peak amplitude (P=0.0021) and distal peak latency (P=0.0100) but there was no significant difference in nerve conduction velocity (P=0.2738) between laser group and sham laser group.

Conclusion: Low-level laser irradiation to the ulnar nerve resulted in increase in peak to peak amplitude and distal latency in this study. This increase corresponds to a decrease in sensory nerve conduction velocity and further studies may reveal its analgesic effects which can be induced in inflammatory conditions of the Nerve.

Keywords: Low intensity laser irradiation; neural conduction; ulnar nerve; physiotherapy; phototherapy; Biostimulation.

INTRODUCTION

Laser therapy is widely used over the globe in treating various ailments in health care. It is used by physiotherapists in the treatment of pain and inflammation. Quiet recently it has further widened its horizon in providing multifaceted health care in a physiotherapeutic perspective (1). Various research has been done to prove its efficacy in altering or providing beneficial effects in neural adaptations and modification in terms of peripheral and central nervous system disorders (2). Our team has done studies to prove its neuroregenerative effect in the past and has shown promising results. Laser has evolved and transformed itself from bigger units which require HeNe (Helium Neon gas) to produce laser to small handy devices which nowadays use only a diode chip to produce laser. The use of laser in health care from therapy to surgery has increased its scope and widened its horizons in understanding its clinical significance with scientific relations (3).

Ulnar nerve entrapment or neuropathy can be involved at elbow, upper arm, wrist. The recognition of distinctive clinical and neurophysiological features is important for localization. Ulnar entrapment and neuropathy at elbow is the second most type of focal neuropathy (4). Ulnar nerve lesions at elbow may occur in condylar groove is quite shallow, therefore ulnar nerve is vulnerable to injury following repeated minor pressure, chronic compression by callus and general anesthesia (5). During flexion or extension of elbow, ulnar nerve moves and back forth out of ulnar nerve. Alteration of carrying angle of the elbow and limitation of full extension predispose to ulnar neuropathy at the elbow (6).

Recent research investigated that the effect of pulsed laser therapy upon nerve conduction and evoked potential recorded for dorsal roots and dorsal spinal cord. The result of this study confirmed the hypothesis selective blocking small diameter fibers activity with this slow conducting fibers being found to be most affected by such laser irradiation.

Application of LPL might cause presynaptic inhibition of those different fibers that signal pain to the central nervous system (7).

Application of low power laser increase the latency sensory branches of radial nerve in human. Hence this study is designed to examine the nerve conduction velocity and electrophysiological parameter of ulnar nerve. Pain and the nerve injury are the common problem. These two will alter the functional activity of the individuals (8). In these situation there is some alteration in the physiological activity of the nerve. Hence this study designed to examine the nerve conduction velocity and electrophysiological parameter of ulnar nerve. If the conduction velocity increases it will be used for regeneration of damaged nerve or it decreases it can be used for pain relief. The aim is to analyse the neurophysiological effect of laser on ulnar nerve. Ulnar neuropathy is the second commonest problem that will alter the functional activities. In neuropathies there would changes in the nerve conduction velocity, peak to peak amplitude, onset latency, action potential. This study is designed to determine the neurophysiological changes occurring in the ulnar nerve after the application of low power laser (9, 10). The selection of laser in this study is because it is portable, reliable, precise and less time consuming, does not possess any harmful effects to the patient. This study aims to analyze the recordings of electrophysiological changes occurring in healthy human Ulnar nerve after laser irradiation.

METHODOLOGY

This is an experimental trial conducted in a private medical college hospital. The participants are grouped as Group I -30 individuals (experimental group), Group II -30 individuals (Sham group).

Inclusion criteria - Normal individuals without having any neurological deficit, Age 20-30 years, both gender, Asymptomatic individuals, Individuals without pain in the upper limb.

Exclusion criteria - Individuals with neuropathies, Brachial plexus injury, Nerve compression, Orthopaedic problems.

Procedure

Sixty healthy subjects were recruited for the study. Each Participant was screened for neurological deficits in the upper extremity. The procedure was clearly explained and informed consent obtained. Randomization of the participants were done to place them in either of the groups. Group 1 served as treatment (Laser) group and they received gallium arsenide laser diode irradiation for 20 Seconds. Group 2 served as placebo group and they did not receive any energy output from the laser but measurement for nerve conduction was done similar to the other group. Nerve conduction velocity were

performed antidromically on the subject's dominant hands. For this study the subjects randomly assigned to the relevant groups. All the subject's were positioned in supine lying with the irradiated dominant hand was in 135° of flexion. The recording electrode was fixed to the hypothenar muscle and the stimulating electrode was placed 4cm distal to the medial epicondyle of humerus and secure the electrode with tape. Ground electrode was placed over the thenar eminence and secured firmly with tape. Antidromical testing procedure was performed and all the subjects received gallium arsenide laser treatment. Group 1 was irradiated with the treatment probe of 904nm diode laser and was set to deliver continuous energy of 4.0J/cm² on the skin overlying the ulnar nerve behind the medial epicondyle of humerus for 20 secs. Group 2 was irradiated with similar procedure but with sham irradiation (11-15).

Onset latency, peak to peak amplitude, Nerve conduction velocity was recorded as pretest value and stored. The treatment area was irradiated for 20 secs with gallium arsenide diode laser application or placebo application. The posttest onset latency, peak to peak amplitude, nerve conduction velocity was recorded and stored immediately after the treatment.

The Outcome measures includes Nerve conduction velocity, Distal peak latency, Peak to peak amplitude. The statistical analysis was performed with intragroup and intergroup associations assessed by t tests.

RESULTS

The t-test results suggests significant difference between groups of pretreatment latency difference existed between the placebo and laser group. The pretest and posttest samples for laser and placebo groups suggests a significant difference in peak to peak amplitude ($P < 0.0001$) and Distal peak latency ($P = 0.0100$) and there is no significant difference in nerve conduction velocity ($P = 0.2738$).

Peak to Peak amplitude – LASER group: $t = 9.9474$, $df = 29$, $SED = 0.032$, The two-tailed P value is less than 0.0001, statistically significant. (Table 1).

Table 1: Peak to Peak amplitude – LASER

S. No.		Pre test	Post Test
1.	Mean	6.683	7.000
2.	SD	0.376	0.358
3.	SEM	0.069	0.065
4.	n	30	30

Peak to Peak amplitude – Placebo group: $t = 0.7328$, $df = 29$, $SED = 0.027$, The two-tailed P value equals 0.4695, not statistically significant (Table 2).

Table 2: Peak to Peak amplitude - Placebo

S. No.		Pre-test	Post Test
1.	Mean	6.657	6.677
2.	SD	0.434	0.417
3.	SEM	0.079	0.076
4.	n	30	30

Peak to Peak Amplitude – LASER and Placebo comparison: $t = 3.2234$, $df = 58$, $SED = 0.100$. The two-tailed P value equals 0.0021, statistically significant (Table 3).

Table 3: Peak to Peak amplitude – Laser and Placebo comparison

S. No		LASER	PLACEBO
1.	Mean	7.000	6.677
2.	SD	0.358	0.417
3.	SEM	0.065	0.076
4.	n	30	30

Distal Peak Latency - LASER Group: $t = 1.7834$, $df = 29$, $SED = 0.024$, The two-tailed P value equals 0.0050 statistically significant (Table 4).

Table 4: Distal Peak Latency - LASER Group

S. No		Pretest	Post Test
1.	Mean	3.733	4.177
2.	SD	0.326	0.408
3.	SEM	0.051	0.047
4.	n	30	30

Distal Peak Latency - Placebo Group: $t = 0.1364$, $df = 29$, $SED = 0.049$. The two-tailed P value equals 0.8925 not statistically significant (Table 5).

Table 5: Distal Peak Latency - Placebo Group

S. No.		Pretest	Post Test
1.	Mean	3.770	3.777
2.	SD	0.737	0.786
3.	SEM	0.135	0.144
4.	n	30	30

Distal Peak Latency - LASER & Placebo Group comparison - $t = 0.0000$, $df = 58$, $SED = 0.206$. The two-tailed P value equals 0.0100, statistically significant (Table 6).

Table 6: Distal Peak Latency - LASER & Placebo Group comparison

S. No		LASER	PLACEBO
1.	Mean	4.177	3.777
2.	SD	0.308	0.486
3.	SEM	0.047	0.044
4.	n	30	30

Nerve conduction velocity - LASER & Placebo Group comparison: $t = 1.1048$, $df = 58$, $SED = 0.875$. The two-tailed P value equals 0.2738, not statistically significant (Table 9).

Table 7: NCV-LASER & Placebo Group Comparison

S. No.		LASER	PLACEBO
1.	Mean	58.3970	59.3637
2.	SD	3.3955	3.3821
3.	SEM	0.6199	0.6175
4.	n	30	30

DISCUSSION

The findings of this investigation back up the theory that using a gallium arsenide laser increase sensory nerve onset latency while decreasing nerve conduction velocity. Analgesic treatments, such as the therapeutic use of ice, have been proven by other researchers to reduce nerve conduction velocity. The findings of this study point to nerve transmission interference as a plausible mechanism for the analgesic effects of gallium arsenide laser irradiation. Although nerve conduction continues after laser irradiation, it is much slower (16-19).

The mechanisms of pain perception are complex. Some researchers believe that each synapse in the medulla spinalis is controlled by spinal and supraspinal mechanisms. The idea that the brain doesn't just passively accept information but also decides what it "wants" is crucial to our understanding of the significance of those outcomes. Timing is critical in these complex relays. Any alteration in sensory transmission patterns, no matter how minor, can have an impact on the precise circuitry of relays at the spinal cord level. (20-22).

Application of Low level laser had increased Onset and peak latency of SNAPs. This findings shows that nerve conduction is affected with laser beam. This might be due to indirect effect of laser on increment of ATP production in the cells and corresponding activation of Na^+ ion channels. Alternatively bioelectric effects of laser on ions transition through the cell membrane can affect cell membrane potential. Indeed, there some reports regarding hyperpolarisation of the cell membrane and increment of threshold of afferent fibres (23-25).

Although a 20-second exposure showed a substantial increase in start latency and peak-to-peak amplitude, more research is needed to establish long-term consequences. We recommend that the effects of gallium arsenide laser irradiation on neuronal membranes be investigated under more rigorous electrophysiological circumstances employing intracellular recording, voltage clamping, and patch clamping methods. Further research is needed to see if similar physical results lead to gallium arsenide laser therapy of pain sufferers, as well as to look at the link between our findings and patients' subjective pain reduction claims. Our team has done studies to prove its neuroregenerative effect in the past. Similar electrophysiological studies have been done with Ultrasound therapy on ulnar nerve and has shown promising results (26, 27).

CONCLUSION

Low level laser irradiation to the ulnar nerve resulted in increase in peak to peak amplitude and distal latency in this study. This increase corresponds to a

decrease in sensory nerve conduction velocity and further studies may reveal its analgesic effects which can be induced in inflammatory conditions of the Nerve.

CONFLICT OF INTEREST

The authors declare no conflict of interest in the study.

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