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Effect of Monochromatic Infrared Energy on the Neuropathic Median Nerve Post Burn

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Abstract : Purpose: to evaluate the efficacy of monochromatic infrared energy on the neuropathic median nerve post burn. **Method of evaluation** (Measurement of the median nerve motor conduction velocity in meter/ second). **Methods:-** Thirty patients (18 males and 12 females) ranging in age from 20 to 35 years, they were selected from the out-clinics of Kasr-El-Aini (Cairo University hospitals) and Om-Al-Misrieen hospital (Ministry of Health). They were divided into two groups; One experimental group and one control group) the experimental group formed of 15 patients to which the monochromatic infrared energy in addition to the traditional physical therapy program (rest, ice and pulsed ultrasound therapy) were applied, while the control group was formed of 15 patients to which only the traditional physical therapy program was applied. Every patient was relaxed in a comfortable supine position with two therapy pads of the monochromatic infrared energy (MIRE) unit were applied for the experimental group as follow; one therapy pad was positioned on the cubital fossa (elbow level) and the other therapy pad was placed on motor point (middle of the muscle) of the abductor pollicis brevis (APB). Each session of the monochromatic infrared energy (MIRE) was done for 20 minutes 3 times per week for 2 months as a total period of treatment. Measurements were conducted before starting the treatment as a first record and at the end of the second month of treatment as a second (final) record. **Results and conclusion:-** Results showed that application of the monochromatic infrared energy had a valuable improving effects on the neuropathic median nerve post burn as evidenced by the highly significant increases in the median nerve motor conduction velocity in meter/ second.
Key words (Monochromatic infrared energy, Neuropathic median nerve post burn and Motor conduction velocity).

Introduction

Monochromatic infrared energy has been in use since 1994 when it was cleared for marketing by the Food and Drug Administration (FDA) for increasing local circulation and for reducing pain. The proposed mechanism of action for MIRE is to increase the microcirculation of the tissues under the diodes as a result of hemoglobin absorbing the infrared wavelength and releasing small amounts of nitric oxide (NO) in the blood vessels. This is believed by some to increase endothelial cell formation of NO, a powerful vasodilator and

angiogenesis mediator. Angiogenesis and vasodilatation increase circulating oxygen levels in the treated tissues. Scanning laser Doppler has been used in case studies demonstrating improved tissue perfusion, or microcirculation, in the tissues receiving MIRE treatment [1,2,3,4].

Monochromatic infrared energy therapy is a therapy that utilizes infrared light therapy through contact with the skin. This therapy may also be referred to as infrared therapy, near-infrared light therapy, and infrared light treatment. It is also known as monochromatic, near-infrared photo energy (MIRE™) (Anodyne Therapy LLC, Tampa, FL). The Anodyne® Therapy System (ATS) (Anodyne Therapy LLC, Tampa, FL) is one type of devices that utilizes MIRE. With Anodyne devices, light is emitted by an array of 60 superluminous gallium aluminum diodes located on a flexible pad. According to the manufacturer website the mechanism of action is a combination of topical heat and an increased local release of nitric oxide with use of wavelength (890nm) of near infrared light energy.[5,6,7.]

The Anodyne Professional Therapy System is a MIRE device that received marketing clearance from the U.S. Food and Drug Administration (FDA) in 1994 through the 510(k) process. MIRE therapy stimulate the endogenous purification by removal of infectious microorganisms and cell debris via the following; increasing stimulation of macrophages, increasing bacterial phagocytosis activity and bacterial phagocytosis capacity by increasing formation of the scavenger cells, increasing stimulation of neutrophils, increasing number of neutrophils and phagocytosis activity. The Anodyne® Therapy System is a noninvasive medical device that delivers Monochromatic Infrared Energy/ (MIRE™) through infrared light-emitting diodes. These diodes are mounted in flexible Therapy Pads, and emit infrared light at a wavelength of 890 nm, increasing local circulation and reducing pain, stiffness and muscle spasm where applied. [1,3,6,7.]

Burned patients suffer from many problems due to disruption of the normal protective functions of the skin, injury to the vascular tree and blood elements, severe metabolic stress with abnormal capillary permeability, protein-rich fluid extra-vascular oedema and low cutaneous blood flow. A burn injury can have devastating effects on the neuromuscular system. Patient complaints regarding weakness or lack of sensation often are rationalized as generalized sequelae of the burn injury and healing process. However, these symptoms may be due to peripheral neuropathies resulting from either impaired nerve axons, or myelin sheaths, or both, [8,9.]

Peripheral neuropathies have been observed following thermal injury and most often affect nerves under the area of the burn, peripheral neuropathies are usually seen in patients with burns greater than 20 % of total body surface area (TBSA). The number of nerves involved per patient ranged from 3 to 7. The source was believed to be due to a multiple crush syndrome, in which multiple different neuropathic factors in each patient summate to cause a multiple mononeuropathy. Burn-Associated polyneuropathy (BAPN) is common after thermal injury and the electrophysiological manifestations of BAPN are present within the first week (Post burn) in burned and even in unburned limbs, which, were attributed to an inflammatory cascade caused by thermal injuries and resulted in nerve function alterations. [10,11,12,13,14,15.]

Certain patients are more susceptible to peripheral neuropathies as alcoholics, diabetics and others, as well as the aged because they are less mobile and their nerves do not tolerate pressure well. Peripheral neuropathy can be either generalized or local in nature. Patients with a generalized peripheral neuropathy, termed polyneuropathy, often complain of fatigue and lack of endurance. The exact etiology of peripheral neuropathy is unknown, however, neurotoxicities from the numerous drugs used in the treatment of burn patients has been suggested. Also stretch or compression injury to a single nerve, resulting from improper treatment techniques applied to a burn patient, has been suggested. [16,17,18,19,20.]

Also the hypermetabolic response of the burned patients, has been suggested as a cause of the peripheral neuropathies, as the basal metabolic rate (B.M.R) of the burned patients increase in excess of 2 to 2.5 times normal (Normal B.M.R. equals 40 ± 10 % $C/m^2/hr$), which contributed to the excess of the circulating catecholamines, that increase the sympathetic tone in burned patients, leading to increased systemic vascular resistance, decreased cutaneous, muscular and endoneurial blood flow (Nerve blood flow) resulting in nerve function alterations.[9,21,22.]

Material and Methods

Subjects:

Thirty patients (18 males and 12 females) ranging in age from 20 to 35 years, they were selected from the out-clinics of Kasr-El-Aini (Cairo University hospitals) and Om-Al-Misrieen hospital (Ministry of Health). They were divided into two groups; One experimental group and one control group) the experimental group formed of 15 patients to which the monochromatic infrared energy in addition to the traditional physical therapy program (rest, ice and pulsed ultrasound therapy) were applied, while the control group was formed of 15 patients to which only the traditional physical therapy program was applied. Every patient was relaxed in a comfortable supine position with two therapy pads of the monochromatic infrared energy (MIRE) unit were applied for the experimental group as follow; one therapy pad was positioned on the cubital fossa (elbow level) and the other therapy pad was placed on motor point (middle of the muscle) of the abductor pollicis brevis (APB). Each session of the monochromatic infrared energy (MIRE) was done for 20 minutes 3 times per week for 2 months as a total period of treatment. Measurements were conducted before starting the treatment as a first record and at the end of the second month of treatment as a second (final) record.

Instrumentation:

In this study the measuring equipment was the Amplaid EMG12 machine to obtain an objective evaluation of the motor conduction velocity of the median nerve, while the therapeutic equipment was the Anodyne® Therapy Systems-Model 480 that delivers Monochromatic Infrared Energy/ (MIRE™) which received clearance from the US FDA in 1994.[2,4,11,16,17,19.]

Procedures

Evaluation:

Protocol of the motor conduction velocity measurement:

Position of subject and electrodes: Upon arrival, the subject was asked to lie in supine position on a therapeutic plinth for approximately 5 minutes for rest and relaxation and to be familiar with the environment. **Recording electrodes:** The active recording electrode was placed over the main bulk of the abductor pollicis brevis (APB) muscle (one-half the distance between the metacarpophalangeal joint of the thumb and the midpoint of the distal wrist crease). The reference electrode was placed distally on the thumb. The ground electrode was placed over the palmar aspect of the ulnar border of the hand. **Stimulating electrodes: In the distal stimulation:** the stimulating cathode was placed 8 cm proximal to the active recording electrode to provide a standardized distal latency segment and located between the palmaris longus and flexor carpi radialis tendons. **In the proximal stimulation:** The stimulating cathode was placed in the cubital fossa (just medial to the pulsation of the brachial artery). Recording electrodes as well as the ground electrode were moistened with jelly and firmly fixed in their places by an adhesive plaster. [10,12,22,23.]

Experimental technique of the median nerve MCV measurement: The MCV recording technique was conducted in an air conditioned room, where a thermometer was available during the whole time of the experiment to notice the ambient testing room temperature which was adjusted within a comfortable, reasonable and narrow range between 24 ^{o}C to 28 ^{o}C by setting the thermostat of the air-condition, and thus the temperature gradient along the course of the tested nerve was minimized. The controlled temperature of the testing room and warming the tested extremity by a deep stroking massage for 5 minutes was conducted, and thus the temperature-related variability was eliminated. Subject was asked to lie supine for approximately 5 minutes for arm and forearm massage, rest and relaxation as well as to be familiar with the environment of the testing room. The on-off switch of the Amplaid EMG12 machine was turned on. The Amplaid EMG12 program disk was inserted to load the EMG12 software program. All needed electrodes for the test were applied. Electrodes were connected to the positive and negative input of the active channels of the EMG12 head-box as well as the common electrode. The program was moved automatically to the ready condition of the neurography 1 motor NCV test after pressing any key for one time plus the enter key for 2 times on the alphanumeric keyboard. The electrode impedance for the active channels was checked, to be less than 5 k Ohm. The data acquisition process was started after pressing the run key on the keyboard. The stimulus was gradually increased till the figure (trace) has been obtained. Then the stop key was pressed and the cursor was moved to

measure latency. Print key was pressed as well as the paper key that allow paper to be advanced. During all recordings the sensitivity used was 1 mv / division, and the sweep speed was 3 ms, while the stimulus intensity was ranged from 0.1 to 99 mA and stimuli were used to gain supramaximal recording. Recording of the segmental distances between the points of stimulation (marked by a marker pen midway the 2 stimulating electrodes) were measured by a tape measure.[13,14,15,17,22,23.]

Nerve conduction velocity was calculated by the following formula:

$$\text{Conduction velocity} = \frac{L1 - L2}{\text{Distance in (cm)} \times 10}$$

Where: L1 = proximal latency and L2 = Distal latency. [15,18.]

Data collection procedures: Before the beginning of the experiment, an initial recording of the MCV of the median nerve for every subject was performed in all the two groups as a first record while the second record was done after 2 months of treatment. To eliminate the effect of one variable that could contribute to slight changes in the required data for any individual subject, the two tests were administer approximately during the same time of the day. [3,13,18,23,24,25.]

- Treatment protocol including position of patient and therapy pads placement of the monochromatic infrared energy (MIRE) in the study group (A): Patients were treated as outpatients, patients received full explanation about the purpose of the treatment, the therapeutic and physiological benefits of the monochromatic infrared energy (MIRE).Subject was relaxed in a comfortable supine position with two therapy pads of the monochromatic infrared energy (MIRE) unit were applied for the experimental group as following; one therapy pad was positioned on the cubital fossa (elbow level) and the other therapy pad was placed on motor point (middle of the muscle) of the abductor pollicis brevis (APB). Each session of the monochromatic infrared energy (MIRE) was done for 20 minutes 3 times per week for 2 months as a total period of treatment. [2,3,4,6,7.]

Data analysis:

The motor conduction velocity (MCV) of the median nerve in meter/ second, was measured pre-treatment as a first record and after two months as a second final record in both groups. Collected data were fed into computer for the statistical analysis; descriptive statistics as mean, standard deviation, minimum and maximum were calculated for each group. The t-test was done to compare the mean difference of the two groups before and after application and within each group. Alpha point of 0.05 was used as a level of significance.[24,25.]

Results

As shown in table (1) and figure (1), the mean value of the MCV before treatment was (39.3 ± 2.6) meter/ second in the study group (MIRE group), while after treatment was (58.5 ± 3.8) meter/ second. These results revealed a highly significant increase in MCV ($P < 0.0001$). While in the control group (Traditional physical therapy group), the mean value of the MCV before treatment was (39.2 ± 2.4) meter/ second, while after treatment was (39.4 ± 2.5) meter/ second. These results revealed non-significant reduction in the MCV ($P > 0.05$).

Table (1): Comparison of the mean values of the motor conduction velocity (MCV) in meter/ second before and after treatment in the study and control groups

	Before treatment		After treatment		Mean difference	T-value	P.value	Level of significance
	Mean	± SD	Mean	± SD				
Study group (MIRE group)	39.3	2.6	58.5	3.8	-19.2000	-16.15	0.0001	Highly significant decrease
Control group (Traditional physical therapy group)	39.2	2.4	39.4	2.5	-0.20000	-0.22	0.825	Non-significant

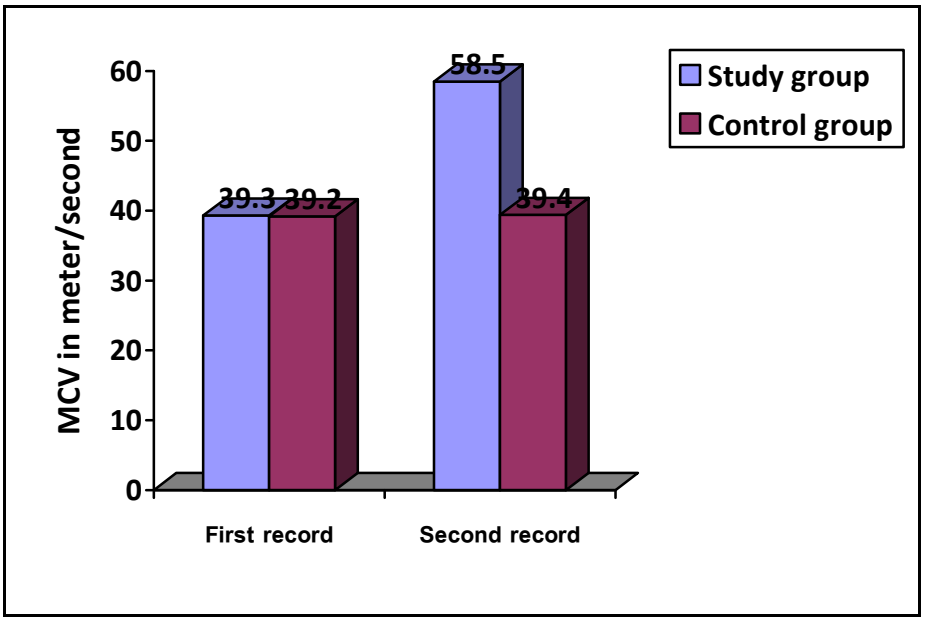

Fig (1): Mean values of the motor conduction velocity (MCV) before and after treatment in both groups.

Discussion

The basic pathophysiological consequence of the burn injury is the loss of the capillary integrity, localized increase in the micro vascular permeability, generalized impairment in the cell membrane resulting in cell swelling and increase osmotic pressure of the burned tissues leading to further fluid accumulation and oedema formation, which is a result of the outpouring of the intravascular fluid into the interstitial spaces. This process occurs at all areas of partial skin thickness burns and at the areas which are adjacent to and subjacent to the full skin thickness burns.[6,8.]

Burn patients suffer from many problems due to disruption of the normal protective functions of the skin, injury to the vascular tree and blood elements, severe metabolic stress with abnormal capillary permeability, protein rich fluid extra vascular or edema and low cutaneous blood flow. A burn injury can have

devastating effects on the neuromuscular system. Patient's complaints regarding weakness or lack of sensation often are rationalized as generalized sequelae of the burn injury and healing process. However, these symptoms may be due to peripheral neuropathies and entrapment syndromes resulting from impaired nerve axons, or myelin sheath or both.[8,22.]

Mononeuropathies and entrapment syndromes have been observed following thermal injury and most often affect nerves under the area of the burn, and they are usually seen in patient with burn greater than 20% of total body surface area (TBSA). The occurrence of entrapment syndromes or multiple mononeuropathies after thermal burns covered greater than 20% of TBSA is common and the number of nerves involved per patient ranged from 3 to 7 nerves. The source was believed to be due to multiple crush syndromes, in which multiple different neuropathic factors in each patient summate to cause a multiple mononeuropathies or entrapment neuropathies.[10,1113,15.]

Electrodiagnostic studies (e.g., EMG, nerve conduction studies) remain the criterion standard for objective evaluations of neuropathic conditions. These studies are not without flaws; they are highly operator-dependent and the results do not always correlate with the severity of symptoms or patient outcomes. Despite these drawbacks, they may help confirm equivocal physical examination findings or help isolate the specific site of compression preoperatively. EMGs also may be used to verify progression or resolution in neurophysiology following surgical release. Common compressive neuropathies affect the median nerve in the form of pronator syndrome, anterior interosseous syndrome and the carpal tunnel syndrome; also the common compressive neuropathies affect the ulnar nerve in the form of cubital tunnel syndrome and ulnar tunnel syndrome. Common compressive neuropathies also affect the radial nerve in the form of radial tunnel syndrome, posterior interosseous syndrome and superficial radial nerve syndrome. Sites of compression of the pronator syndrome as an example of the median nerve entrapment syndrome includes the lacertus fibrosus, bicipital aponeurosis, superficial forearm fascia , the Struthers ligament, thickened or aberrant origin of pronator teres from distal humerus, the pronator teres (musculofascial band or compression between 2 muscular heads), and the proximal arch or the flexor digitorum superficialis. [17,18,19.]

MIRE therapy increases microcirculation in lower-leg chronic ischemia, improves myocardial perfusion in patients with severe coronary artery, decreases pain and enhances re-epithelialization in chronic leg ulcers and, it may pervert bone vascular disorders such as disorders such as osteonecrosis and may have a bactericidal effect against Staphylococcus aurous. MIRE therapy increases vascular endothelial growth factor (VGEF), eNOS and proliferating cell nuclear antigen (PCNA), enhances perfusion to ischemic tissues (myocardial and skin flaps) and stimulates angiogenesis. One proposed mechanism of action of(MIRE) modality is that it increases nitric oxide (NO) in the blood and plasmas of normal adult subjects (authors' unpublished research). An elevation in NO has been suggested to be the basis of improved rates and quality of healing during L-arginine or nitroglycerin therapy in patients with wounds. Dietary L-arginine, a source of NO via the constitutive is form of the enzyme nitric oxidesynthase (cNOS), increases the rate of wound healing following traumatic, thermal, and fracture injuries. It has been proposed that through this NO-mediated process, MIRE might prove beneficial in patients with venous and diabetic ulcers and in patients who exhibit slow rates of post amputation wound closure. [2,3,4,5.]

The second proposed mechanism of action MIRE therapy is the absorption of energy by mitochondria; this may cause a chain of reactions on the molecular level, leading to an increase in cell energy and activation of the nucleic acid synthesis, which is essential for wound repair. The third proposed mechanism is obtained by the infrared like energy; this initiates the response at the membrane level, probably through photo physical effects on CA++ channels. MIRE therapy has been shown to stimulate the release of the growth factors from the irradiated cells. Growth factors stimulate angiogenesis, extracellular matrix production and degradation as well as cytokine release. The key cells in skin ulcer contraction and collagen synthesis are fibroblasts and keratinocytes. Nitric oxide, known as the 'endothelium-derived relaxing factor', or 'EDRF', is biosynthesized endogenously from L-arginine, oxygen, and NADPH by various nitric oxide synthase(NOS) enzymes. Reduction of inorganic nitrate may also serve to make nitric oxide. The endothelium (inner lining) of blood vessels uses nitric oxide to signal the surrounding smooth to relax, thus resulting in vasodilatation and increasing blood flow. Nitric oxide is highly reactive (having a lifetime of a few seconds), yet diffuses freely across membranes. These attributes make nitric oxide ideal for a transient paracrine (between adjacent cells) and autocrine (within a single cell) signaling molecule. [1,5,6,7.]

The findings of the present study showed non-significant differences in the pre-treatment records of MCV between the mean values of the study and control groups.

Results of the study group revealed a highly significant increase in the mean values of MCV, after application of the MIRE, when compared against the pre-application results. But results of the control group revealed non-significant difference in the mean values of MCV, after application of the traditional physical therapy, when compared against the pre-application results.

Significant differences showed in the study and control groups were consistent with those observed and recorded by Buchthal et al., 2004; Burke, 2003 and 2005; Clifft et al., 2005; Helm et al., 2001; Jianping et al., 2005; Kochman, 2004; Liu et al., 2005 and Volkert et al., 2005.

Results of this study support the expectation that application of MIRE had a valuable improving effects on the peripheral circulation via lowering vascular resistance and increasing skin, muscle and nerve circulation in cases of neuropathic median nerve post burn as evidenced by the highly significant increases in the MCV.

Conclusion

Application of MIRE had a valuable improving effects on the peripheral circulation via lowering vascular resistance and increasing skin, muscle and nerve circulation in cases of neuropathic median nerve post burn as evidenced by the highly significant increases in the MCV.

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