Effect of LASER in Acute Lower Motor Neuron Lesion

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ABSTRACT

Background and purpose: The purpose of this study was to investigate the effects of laser on latency, amplitude and axonal loss of the facial nerve in acute Bell's palsy. Subjects: Forty patients of both sexes participated in this study, they were diagnosed as acute Bell's palsy with more than 30%axonal loss of the facial nerve, their age ranged between 25-45 years of old (X= 35.03, SD= ± 5.44). They were assigned into two groups: Group I: twenty patients received laser therapy for 15 minutes daily from the fifth to the fourteenth day from the palsy onset. Group II: twenty patients received daily placebo laser for 15 minutes from the fifth day to the fourteenth day from the palsy onset. Methods: All patients were subjected to electroneuronography (ENoG) on the fifth and the fourteenth day from the palsy onset to record latency, amplitude and axonal loss. Results: At the end of treatment, paired t-test revealed a significant decrease in motor distal latency after laser application (P<0.05), no significant increase in amplitude and a non significant decrease in axonal loss (P>0.05).Concerning the placebo group, there was an obvious deterioration in condition in the form of significant increase in motor distal latency and axonal loss (p<0.05) and a significant decrease in amplitude (P<0.05). Independent t-test between laser group and placebo group showed that there was a statistically significant increase in the improvement of laser group rather than the placebo group especially in the amplitude and motor distal latency (p<0.05).Discussion and conclusion: The study indicates that laser might tend to resolve oedema and inflammation of the facial nerve, manifested by tendency to decrease motor distal latency and axonal loss in acute Bell's palsy.

Key words: *Laser, Bell's palsy, Amplitude, Latency and Axonal loss.*

INTRODUCTION

LASER is an acronym for light amplification by stimulated emission of radiation (Baxter,1994). It has been used in various fields such as industry, film production, medicine and science (Rochkind et al., 1987). Soft Laser has been used in soft tissues inflammatory conditions (Fernands et al., 1991). Studies favor the use of cold laser as it has been found to be with no hazardous or complicated effects since it is operated in low power (Baxter,1994).Studies on the antiinflammatory effect of a low power laser are still at the stage where more experimental cases should be demonstrated. In a study of the effect of Ga-Al-As Laser irradiation on experimentally

induced inflammation in rats, the results indicated that in the acute stage laser has an inhibitory effect on the increased vascular permeability, exudation of plasma proteins and water in the second phase of inflammation. It may have exerted an inhibitory effect on granuloma formation by strongly affecting the inflammatory response, also exerted inhibitory effect on oedema (Honmura et al.,1993). The effects of laser biostimulation on prostaglandins was initiated by studying changes in E and F type (PG) in rat skin wounds subjected to 1 J/cm^2 He-Ne laser stimulation.Thus it was suggested that laser accelerates reabsorption of inflammatory phase rapidly by altering level of various PGs (Enwemeka, 1988).

Temporary mild compression of nerve will lead to conduction block called neuropraxia due to local edema and injury of myelin sheath. Thus a neuropraxia can be a partial block with some conduction across the affected region. If the compression injury is more severe it may cause sufficient damage to nerve axon so that is unable to support the metabolic process of its distal part resulting in degeneration of the whole length of the nerve fiber including the myelin sheath distal to the lesion. This prcess is called wallerian degeneration, it takes some days to extend throughout the distal part of the nerve about 3 to 4 days or more.

Wallerian degeneration occurs in the whole segment of the nerve distal to the injury (antrograde-degeneration) and proximally for only one cm to nearest proximal node of ranvier (retrograde-degeneration). The process occurs slowly over 10-21 days at the end of which the fibrous framework of the nerve and schwann sheath are all that remain.If the fibrous framework of the bundle of the nerve remains intact and fills with Schwann's cells so that ultimately nerve fibrils sprouting from the intact proximal part of the nerve this kind is called axonotemesis.The length of time needed for full recovery to occur will depend on the site of the lesion and the length of the nerve that has to regrow. The rate of growth being more rapid at first up to 5 mm/day, but it is considered to be an average 1-2 mm/day. If injury disrupts all tissues of the nerve fiber, then distal segment will degenerate, the lesion is called neurotemesis. Such lesion often require surgery to ensure that two cuts ends are approximated to allow regrowth. It can be seen that some nerve lesion may be mixture with some fibers being completely interrupted while others suffer only a neuropraxia, which leads to different effects on nerve conduction (Low et al., 1994).

When laser is applied to injured peripheral nerve prevents drop in the compound muscle action potential (CMAP), obviates scar formation and increases the vascularization, prevents degeneration of the surrounding muscle and accelerates regeneration of the injured nerve (Rochkind et al.,1988). Treatment of lower motor neuron lesion (LMNL) following injury remains a major clinical problem. One of the main acute injury of LMNL is idiopathic facial nerve paralysis "Bell's palsy" which is defined as an idiopathic paralysis to the facial nerve resulting in varying degrees of facial muscle paralysis with no suggestion of central nervous system or posterior fossa/ear disease (Williams et al.,1994). It usually involves all types of movements of face (Dimitru et al.,1988). The incidence of Bell's palsy is between 20and 30 per100.000 population. It is estimated that Bell's palsy affects someone every 12 minutes, men and women are affected approximately equally at any age (Dimitru et al., 1988, Schwarts et al., 1991). Some studies have shown the highest incidence to be in 20-35 years old with both sides equally

involved. Following Bell's palsy there are oedema, dilated capillaries and degeneration of medullary sheath and axis cylinders of the facial nerve. A minor swelling can compress the veins and deprive the nerves of their blood supply and finally results in ischaemia of facial nerve. The initial change following the onset of lesion is an interstitial oedema of the nerve. A conduction block occurs because of demylination in the region of damage. The fast conducting fibers are the first to be affected nerve fibers may degenerate and others remain intact of reversible block. Such reversible block may persists for as long as 6to12 months after the onset of facial paralysis (Wadsxorth et al., 1988). The etiology of Bell's palsy is almost obscure. It has been described as paralysis of the facial nerve with unknown cause. Inflammation seen in few patients may have been attributable to unrecognized Herpes Zoster (jankel,1978).It was believed that normal conduction remains on the third day indicates good prognosis. In Bell's palsy the onset is slower and few weeks go by before a bad prognosis is declared. Conduction measurement provides relatively early evidence of denervation since it has been found that all conduction ceases in the distal segment of the nerve by seventh day after nerve section. Latencies of 2.9 ± 0.4 (< 4.2) with a side to side difference of < 0.7 are considered normal. Prolonged latency indicates a poor prognosis, however persons with highly unsatisfactory recovery outcomes may have normal latencies throughout the course of the disease (Dimitru,1994). Evoking of facial nerve compound muscle action potential (CMAP) is known as electroneuronography (ENoG), it refers to stimulation of facial nerve and recording a compound muscle action potential from some facial muscle. Because the amplitude depends on the number of the conducting axons, the reduction in amplitude is suitable method in assessing the degree of damage (shin,1993). The side to side amplitude percentage indicates the degree of axonal loss, the best muscle for locating the active electrode was found to be naslis muscle in nasolabial fold. If 26% or more of axons are lost 88% of patients had incomplete recovery (Binnie et al., 1995). Since there is no report in the literature evaluating the effect of laser therapy on acute neuropathy in human-beings, we investigated the electrophysiological effect of laser application compared to placebo laser on a common acute peripheral motor nerve lesion of the facial nerve known as acute Bell's palsy.

MATERIALS AND METHODS

I) Patients

Forty patients (25 males and 15 females) with acute Bell's palsy participated in this study. Their age ranged beween 25-45 years old (X = 35.03, SD = ±5.44) for both sexes and with the following criteria: diagnosed by physician as acute Bell's palsy two or three days from the palsy onset, patients with more than 30% of axonal loss determined by (ENoG) (Shin, 1993) in the fifth day after palsy onset (Dimtru, 1994) and those who expressed their willingness to cooperate during different phases of the study by writing down an informed consent.

Exclusive criteria: Chronic cases of Bell's palsy, any other causes of facial palsy and subjects who did not attend for two sessions were excluded from this study. Participants were selected from El-Kaser El-ani teaching hospitals within two to three days from the onset of Bell's palsy presentation (Dimitru,1994). The total period of the treatment was two weeks from the fifth day to

the end of the second week. Recording of (ENoG) for all subjects was performed at the fifth day and the final record by the end of the second week.

Group I: (Laser group). They were twenty patients (13 males and 7 females) received two weeks laser therapy, six days per week.

Group II: (Placebo group). They were twenty patients (12males and 8 females) who received placebo laser for two weeks,six days per each week.

II) Instrumentation

Measuring instrument: The Amplaid EMG 12 which is a two channels electrodiagnostic system designed for clinical application was used to measure latency, amplitude and axonal loss. The EMG provides test menu for most electrodiagnostic tests as well as automatic measurements of clinical parameters. In the present study intrarater reliability was conducted to measure the repeatability of latency and amplitude of the EMG readings after 15 minutes for same patient by the same examiner. This was done by repeating the test for nine patients in amplitude.

Therapeutic instrument: Laser unit used in this study was Galium Aluminum Arsenide Laser (Ga-Al-As), with pulsed mode, wave length 830 nm and treatment head surface of 3.5cm and power of 40mW,6J and classified potential dangerous as class 3B, hazard to the eye but not to the skin (Salibo,1998).

III) Procedures

Evaluative Procedures: Before starting treatment the patients were observed for the presence or loss of symmetry, if there was drop in eye brow or deviated to one side, asked to close his/her eye and if there was Bell's phenomenon, whether lacrimation was affected or not, and taste sensation of anterior two thirds of the tongue was tested and if there was deviation in the angle of mouth. Parotid gland was palpated bilaterally and pressed at both stylomastoid foramen at the same time to examine pain at this area.

Measurement Procedures: EMG was used to measure latency and amplitude in the involved and noninvolved side. Electrodes gel was applied for good conduction and electrodes were fixed firmly by using plaster straps. Participant's face was cleaned thoroughly with alcohol to remove perspiration, oils, any type of make-up solutions and shaving in male. Then the electrodes were placed to test the patient. The active electrode (red) was placed on nasalis muscle just lateral to the nasal ala, reference electrode (yellow) was placed at the bridge of the nose and the ground electrode (green) was placed on the temple of the forehead. Latencies and amplitudes were saved. Percentage difference between the amplitude in the involved side and the noninvolved side was calculated as following: Percentage of spared axons= Amplitude of involved side/Amplitude of noninvolved side x 100. Percentage of axonal loss = 100 - Percentage of spared axons. (Dimitru, 1988).

Therapeutic procedures

Laser therapy: Group I received laser therapy . Before application the face was washed with water and soap to remove sweat. Both patient and therapist wear special eye goggles to protect eyes from the hazardous effect of the laser beam. The facial nerve was irradiated for 15 minutes using the contact technique; for 10 minutes below the inferior lobe of ear just infront of the mastoid process at stylomastoid foramen and 5minutes on the facial trunk at the angle of the jaw to deliver 6 J/cm^2 (Ebert and roberts, 1997).Patients attended for laser treatment six days per week for two weeks.

Placebo laser; Group II attended as same as group I ,but received placebo laser. All patients were unaware of treatment groups. Taking medications or physical therapy was not allowed during the study; therefore, patients were reviewed daily to determine any worsening in symptoms or desire to discontinue the study.

IV) Statistical Analysis

The means and standard deviations were calculated for all subjects in each group for each measuring parameter. The student t test was used to compare the value among group before and after treatment. One-way ANOVA was used to compare the differences among the values between groups. A $P<0.05$ was considered statistically significant.

RESULTS

Results of this study indicated high intrarater reliability. In latency coefficient of reliability was 0.83 and in amplitude coefficient of reliability was 0.31.

I) Laser group

As shown in table1 and fig. 1, comparing electrophysiologic parameters before and after treatment: there is a significant decrease in latency of facial nerve ($t=4.6$, $P=0.0001$), the mean value of motor distal latency before treatment was (2.9 ± 0.83), and after treatment was (2.2 ± 0.67). There was a nonsignificant increase in amplitude and also a nonsignificant decrease in axonal loss ($P>0.05$).

Table (1): Comparison of the electrophysiologic parameters before and after laser treatment.

Parameters	Before treatment	After treatment	t-Test	P
Amplitude (mv.)	1.09 ± 0.65	1.19 ± 0.71	1.98	0.06
Motor DL	2.9 ± 0.83	2.2 ± 0.67	4.6	0.0001
% axonal loss	59.45 ± 14.95	57.1 ± 18.02	1.08	0.29

All measures reported as mean (SD)
Abbreviation: DL ,distal latency

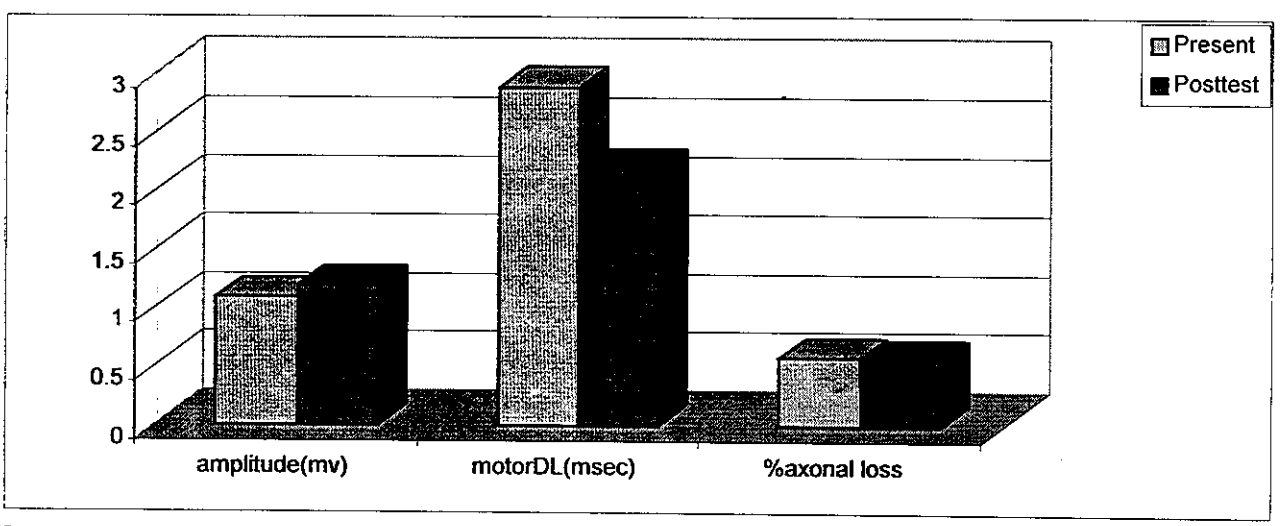

Fig. (1): Comparison of the electrophysiologic parameters before and after laser treatment.

II) Placebo group

Table 2 and fig. 2 comparing electrophysiologic parameters before and after placebo treatment :there was a significant increase in latency of facial nerve (t= -3.1, p=0.005), the mean value of motor distal latency before treatment was (2.98±0.96),and after treatment was (3.03±0.95).There was a significant decrease in amplitude and also a significant increase in axonal loss (p>0.05).

Table (2): Comparison of the electrophysiologic parameters before and after placebo treatment.

Parameters	Before treatment	After treatment	t-Test	P
Amplitude (mv.)	0.9±0.4	0.85±0.38	-4.39	0.0001
Motor DL	2.98±0.96	3.03±0.95	-3.1	0.005
% axonal loss	58.62±16.37	60.5±15.95	-4.2	0.0001

All measures reported as mean (SD)
Abbreviation: DL ,distal latency

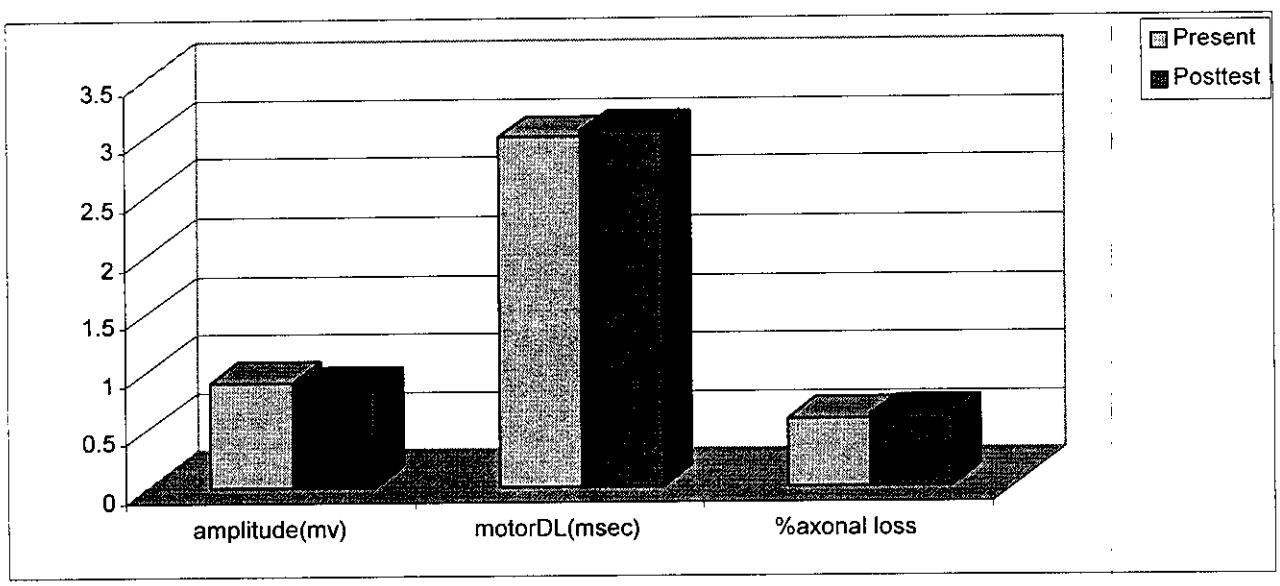

Fig. (2): Comparison of the electrophysiologic parameters before and after placebo treatment.

III) **Between groups:** Table 3 and fig. 3 showed the treatment group measures before and after treatment and compared the improvement between groups. It was found that there was no significant difference between groups in all parameters before treatment , but there was a significant difference between both groups in motor distal latency after treatment (P<0.05).As regard to improvement, laser group showed a significant improvement in amplitude and motor distal latency (P<0.05) and a non-significant improvement in axonal loss (P>0.05).

Table (3): Comparison of the electrophysiologic parameters before and after treatment in both groups.

Pretest Parameters	Laser group	Placebo group	t-Test	P
Amplitude (mv.)	1.09 (0.65)	0.9 (0.4)	0.96	0.35
Motor DL	2.9 (0.83)	2.98 (0.96)	0.18	0.86
% axonal loss	59.45 (14.95)	58.62 (16.37)	0.15	0.88
Post-test				
Amplitude (mv.)	1.19 (0.71)	0.85 (0.38)	1.6	0.13
Motor DL	2.2 (0.67)	3.03 (0.95)	3.18	0.005
% axonal loss	57.1 (18.02)	60.5 (15.95)	0.57	0.58
Improvement				
Amplitude (mv.)	0.09 (0.22)	-0.04 (0.45)	2.9	0.009
Motor DL	0.72 (0.7)	-0.05 (0.07)	4.97	0.0001
% axonal loss	2.3 (9.6)	-1.9 (2.05)	1.9	0.067

All measures reported as mean (SD)
Abbreviation: DL.,distal latency

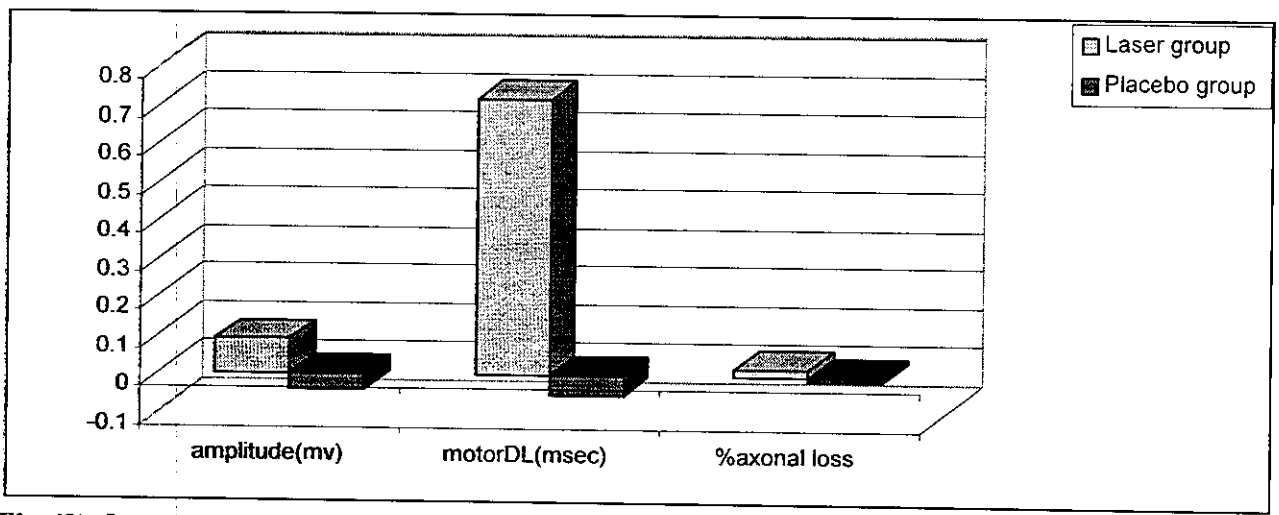

Fig. (3): Improvement in both groups.

DISCUSSION

Lower motor neuron lesion of the facial nerve is known as Bell's palsy occurs with different severity from one patient to another and is treated with corticosteroids and nonsteroidal anti-inflammatory drugs in the acute stage for about 50 years (zoorob, 1998). No single alternative treatment was suggested (Shafshak,1994). On the other hand, laser has an antiinflammatory effect (Honmure,1993 and Rochkind et al.,1987) stimulator (Ebert,1997) and biostimulator effect without hazards or complicated sequences since it is operated in low level (Baxter,1994). Ga-al-As laser was thought to have its penetrating effect up to 2 cm (Salibo et al,1998).Facial nerve is located at depth two cm at stylomastoid foramen, so it can affect the facial nerve easily without reflection. Wavelength with 820 nm was attributed to the beneficial effect of soft laser on nervous tissue and was diminished

with decreasing wavelength from 632 nm down to 465 nm. Six J of energy was delivered to facial nerve because energy under 3 J has been proven to produce no change in compound muscle action potential and energy higher than 8-9 J has an inhibitory effect. Meanwhile, energy levels less than 7J were effective in altering CMAP amplitude (Ebert and Roberts, 1997).

Evoking of facial nerve compound muscle action potential (CMAP) is known as electroneuronography (EnoG), it can monitor the function of the seventh cranial nerve (Harper, 1998). It refers to stimulation of the facial nerve and recording a compound muscle action potential from some facial muscle (Dimitru,1994). Because the amplitude depends on the number of the conducting axons, the reduction in amplitude is a suitable method in assessing the degree of damage (Shin, 1993). There is little agreement towards the best muscle for originally located the active electrode. It was found that nasalis muscle in nasolabial fold just lateral to nasolabial fold is an appropriate site because it has a well defined end plate zone. The reference electrode is located in nasolabial fold or bridge of the nose and the ground electrode on the chin or forehead with facial nerve stimulated at stylomastoid foramen or at the angle of the jaw or inferior to the zygomatic arch (Dimitru, 1994).

In laser group: Patients had a significant improvement in motor distal latency and marked improvement in amplitude which indicates the therapeutic effectiveness of repeated laser therapy as a conservative treatment agent in lower motor neuron lesion seen in cases of Bell's palsy. The results indicate that in acute stage of inflammation, the irradiation of low power laser has an inhibitory effect on granuloma formation. It exerts an inhibitory effect on edema more than half of inhibition exerted by mg/kg of indomethacin. In the acute stage of inflammation the first phase involving the peak of extravasion of highly contacted plasma proteins within 2 hours after inflammation this is due to the effect of some chemical mediators such as sertonin, histamine which is the cause of edema, kinins and prostaglandins. In the second phase there is increased permeability of water, edema and the volume of exudation increase. This is attributed to the mobilization of neutrophils by chemotactic factors release which is inhibited by low power laser (Honmura,1993). The net outcome was not to a significant level in all other measured parameters such as amplitude and axonal loss because this study was conducted through the acute stage (two weeks only) after the onset of paralysis.

In placebo group: There was a significant deterioration in all electrophysiologic parameters which may be attributed to the increased intraneural fluid, which causes the nerve to swell along significant portion of its longitudinal axis. This compression leads to further edema, resulting in cyclical pattern with net result of neural dysfunction associated with electrophysiological abnormalities of conduction block, demyelination and axonal loss. The junction between internal auditory meatus and the labrynthine segment is believed to be location of greatest neural compromise (Louis,1977). The swelling that is thought to occur within the facial canal and to generate neural compromise may begin to damage nerve by producing conduction block and axonal loss. Initially it produces partial loss of facial function over the coarse of the next several days, the pressure within the facial canal may continue to increase causing further neural injury and progressive loss of function. A CMAP percentage measurement 50% axonal loss implies 50% of sparing nerve

function. A CMAP percentage measurement 50% axonal loss implies 50% of sparing nerve regardless of the patient clinical function and this significant dysfunction is due to conduction block rather than axonal loss, as one cannot assess the status of the facial nerve at the time of examination. At obtaining CMAP OF 50% on day 5 does not imply that 50% is spared as neural loss may continue to be seen up to day 7 (Dimitru,1988 & Shin, 1993).

Possible explanation of these results is that following axonotemesis, the distal part must undergo wallerian degeneration then followed by regeneration, 2-3 weeks after onset of paralysis, with drop in CMAP. It is suggested that these sequences was masked by the effect of Ga-Al-As laser that might accelerate wallerian degeneration and enhance process of regrowth of nerve fibers (Rochkind, 1990).

Between both groups: Independent t-test showed significant improvement in motor distal latency and amplitude in the laser group, this may be attributed to the pathological changes which take place in the placebo group and masked by the effect of Ga-Al-As laser. Following Bell's palsy, there are edema, dilated capillaries and degeneration of the medullary sheath and axis cylinders of the facial nerve. In severe cases there is later degeneration of the schwann sheaths and finally fibrosis of the nerve, any minor swelling can compress the veins and deprive the nerves of their blood supply and finally results in ischaemia of facial nerve. The initial change following the onset of lesion is an interstitial edema of the nerve. A conduction block occurs because of demyelination in the region of damage. (Wadsxorth, et al., 1988). Since the underlying pathology in Bell's palsy is focal demyelination caused by compression, the myelinated part of the facial nerve was more sensitive to laser treatment.

From the above, null hypothesis is rejected as it was found that there was a significant difference between laser therapy and placebo therapy effect in acute lower motor neuron lesion found in patients with Bell's palsy.

Further research is needed to investigate the laser role in patients with bad prognosis of Bell's palsy and to examine modalities to replace medications that have side effects on patients.

CONCLUSION

Patients with acute lower motor neuron lesion due to focal demyelination caused by compression such as cases of Bell's palsy showed statistically significant improvement in amplitude and motor distal latency of the facial nerve after 2 weeks of laser treatment. Also preventing the expected deterioration which was seen in placebo group. The efficacy and safety of laser therapy in lower motor neuron lesion needs further investigations, the following studies were recommended:

1- The effect of laser on the function of the facial muscles.
2- Long term application of laser in patients with lower motor neuron lesion (extended to the subacute stage).
3- Another laser parameters to study alteration in latency, amplitude and axonal loss.
4- Other modalities feasibility to replace medications that have side effects on patients.

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الملخص العربي

تأثير الليزر على الإصابة الحادة للعصب الحركي السفلي

الغرض من الدراسة: الغرض من هذا البحث هو دراسة تأثير الليزر العلاجي علي زمن الإثارة و شدة الإثارة و نسبة فقد الألياف العصبية للعصب السابع في حالات شلل الوجه الحاد (بلز) و ذلك باستخدام جهاز رسم العضلات الكهربائي.

أفـراد العيـنة: أربعـون شخصـاً من الجنسين شاركوا في هذه الدراسة كلهم مصابون بإصابة حادة للعصب الحركي السفلي السابع من الأعصـاب المخيـة و المسـمي بشلل الوجه الحاد (بلز) بنسبة فقد للألياف العصبية أكثر من ٣٠% و تتراوح أعمارهم بين ٢٥و٤٥ سنة (متوسط الأعمار ٣٥,٠٣عاما ٥,٤٤) و قسمّوا إلي مجموعتين:

المجموعـة الأولـي:عشرون مريضاً تم علاجهم بالليزر العلاجي لمدة ١٥ دقيقة يوميا ابتداءً من اليوم الخامس إلي اليوم الرابع عشر من تاريخ الإصابة.

المجموعة الثانية: عشرون مريضاً تم علاجهم علاجاً وهمياً بالليزر لمدة ١٥ دقيقة ابتداءً من اليوم الخامس إلي اليوم الرابع عشر من تاريخ الإصابة بالمرض.

تـم استخدام جهاز رسم العضلات الكهربائي للقياس قبل و بعد التجربة في المجموعتين أي في اليوم الخامس قبل التجربة و اليوم الرابع عشر بعد التجربة .

النـتائج : بالنسـبة لـلمجموعة الأولى (مجموعة العلاج الفعلي بالليزر) أسفرت النتائج إحصائياً عن نقصان ذو مدلول إحصائي في زمن الإثارة للعصب السابع بعد استخدام الليزر العلاجي و زيادة ليست ذات دلاله إحصائية في شده الإثارة ونقصان ذو دلاله إحصائية في نسبه فقد الألياف العصبية .

بالنسـبة لـلمجموعة الثانية (مجموعة العلاج الوهمي بالليزر) أسفرت النتائج إحصائياً عن الآتي: تدهور للحالة في كل القياسات السابقة وذلـك في صورة زيادة ذات دلاله إحصائية في كل من زمن الإثارة و نسبة فقد الألياف العصبية و نقصان ذو دلاله إحصائية في شدة الإثارة .

و بالنسبة للمقارنة بين المجموعتين وجد أن هناك تحسن ذو دلاله إحصائية في كل المقاييس في مجموعة الليزر عنه في مجموعة العلاج الوهمي.

المناقشـة و المضـمون : يسـتخلص مما سبق أنه قد يكون العلاج بالليزر قادراً على إزالة التورم والالتهاب لعصب الوجه متضمناً ميلا لإنقاص زمن الإثارة للعصب. و من هذا المنطلق ينصح باستخدام الليزر في الإصابة الحادة للعصب الحركي السفلي في حالات شلل الوجه (بلز) مع الحاجة إلي مزيد من الأبحاث لدراسة استخدام جرعات مختلفة من الليزر و كذلك تأثير امتداد العلاج إلي ما بعد المرحلة الحادة .