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Ultrasound Thermotherapy Effect on Motor Recovery in Children with Bell's Palsy

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Abstract:
Facial nerve paralysis has a tremendous impact on the patient as well as the family, particularly when a pediatric patient is involved. The purpose of this study was to evaluate the effect of ultrasound thermotherapy on the regeneration rate and acceleration of recovery of the facial nerve in children with Bell's palsy. Thirty patients with Bell's palsy from both sexes aged between 8 and 12 years participated for two weeks in this study and were divided randomly into two groups; of equal number control group (A) and study group (B). The study group received continuous ultrasound therapy with low intensity of 0.5w/cm^2 and frequency of 1MH$_Z$ applied below the inferior lobe of the ear at the stylomastoid foramen behind the mastoid process. Ultrasound therapy was applied for 5 minutes per session, 5 days a week for two successive weeks. Placebo ultrasound treatment (0.0w/cm^2) was applied for the control group. The two groups received the same designed physical therapy program for Bell's palsy. Each child was evaluated individually both clinically and electrophysiologically using electronurography (which include facial nerve motor distal latency, amplitude of the evoked compound muscle action potential and the percentage of degeneration) before and after the suggested period of the treatment. Statistically, significant improvement was obtained in clinical parameters in the control and study groups when comparing their pre and post treatment mean values. However, significant difference was recorded between the two groups after treatment in favor of the study group in all parameters. The obtained results strongly support the introduction of ultrasound thermotherapy as an adjunct to the treatment program of Bell's palsy in children.

Key words: Facial palsy, Bell's palsy, Ultrasound, Electroneurography.

INTRODUCTION:

Paralysis of the seventh cranial nerve, the facial nerve is usually immediately obvious. Whether it develops in a child or an adult. **(Evans et al., 2005)**. Ultrasound is a physical therapy agent commonly used to increase temperature in deep tissue. The biologic effects observed when mammalian tissues are exposed to ultrasound include changes in: blood flow rates, tissue metabolism, nerve function, the extensibility of connective tissue and the permeability of biologic membranes **(Oztas et al., 1998)**. The rate of peripheral nerve regeneration in response to ultrasonic irradiation has been studied. It was concluded that the rate of nerve regeneration can be influenced by deep heat and application of heat has been recommended for the treatment of peripheral neuropathy **(Hong et al., 1988)**. In animal study, ultrasound deep heat was found to have a therapeutic effect on the recovery of nerve conduction in compression neuropathy of the rat tibial nerve when a low dose (0.5 watt/Cm^2) was used. An adverse effect resulted when a higher dose (1.0 watt/Cm^2) was applied **(Oztas et al., 1998)**. In the present study, we hypothesized that therapeutic ultrasound will accelerate facial nerve regeneration with proper parameters. We therefore, aimed to investigate the effect of ultrasound thermotherapy on motor recovery in children with Bell's palsy.

Methods

Subjects

After approving this study by the research ethical committee of Faculty of Phyical Therapy, Cairo University , patient blinded, placebo controlled before-after treatment study of thirty children with Bell's palsy from both sexes ranged in age from 8 to 12 years. All of the subject's parents signed an informed consent form developed by the investigator that explained the purpose and procedures of this study. The patients were selected from the out patient clinic, Faculty of Physical Therapy, Cairo University, Egypt and from the out-patient clinic, National Institute for Neuromotor system, Embaba City, Giza, Egypt. Selection criteria for this study as follows:

Inclusive criteria:

1- Children with unilateral lower motor neuron facial paralysis (right or Left) due to peripheral facial nerve lesion at the level of stylomastoid foramen (Bell's palsy). 2- Their age ranging from 8 to 12 years. 3- Onset of the lesion was two weeks.

Exclusive criteria:

1- Children who had complete transection of the facial nerve. 2- Children who had bilateral facial palsy. 3- Children who had recurrent facial palsy. 4- Children who had facial palsy caused by tumor or by congenital etiology. 5- Children with upper motor neuron facial palsy. 6- Children with history of epilepsy, cardiac pacemaker or other medical complications. 7- Children with history of allergic reaction to conductive gel. 6- children with lower motor neuron facial palsy before the level of stylomastoid foramen (other than Bell's palsy)

The patients included in this study were divided randomly into two groups of equal number. Control group (A) received placebo ultrasound in addition to the designed physical therapy program. Study group (B) received continuous ultrasound thermotherapy in addition to the same designed physical therapy program giving to group (A).

Evaluative Procedure

Electroneurography

A computerized electromyographic apparatus (Neuroscreen plus – four channel – version 1.59 produced by TOENNIES, 97204 Hochberg, Germany) was used with surface electrodes. To prepare the patient, he was placed in sitting position comfortably in a chair. The examiner used medical cotton damped with alcohol to clean the stimulating and the recording sites to reduce skin impedance. Ky gel was used on the recording cup electrodes then electrodes were fixed firmly by using plaster straps. An active (negative) recording electrode was placed on the frontalis muscle at mid-point above the eye brow with a reference electrode on the tip of the nose. The ground electrode placed between the stimulating and the recording electrodes. Stimulation and recoding from the non- affected side: facial nerve of the non-affected side was stimulated using a bipolar surface electrode near the mastoid foramen. The cathode placed distally below the ear just in front of the mastoid process and the anode placed more posterior. The stimulus delivered is 0.1m sec duration and intensity varied from 0-50mA to obtain supra maximal stimulation that is the level of stimulus intensity 25% above maximal stimulus. The maximal stimulus is the stimulus at which the response doses not increase in amplitude on increasing stimulus intensity. Stimulation and recording from the affected side: The procedure of stimulation and recording used for non-affected side was repeated on the affected side.

Electroneurography (ENoG) schedule as suggested by **(Shin, 2003):** Facial nerve latency, amplitude and the percentage of degeneration were used pre- treatment to select the subjects of this study and post- treatment to follow up: a)Facial nerve latency: was measured to the onset of the initial deflection from the base line. b)Amplitude: was assessed as following upward deflection was recorded as negative and downward deflection was recorded as positive. Peak to peak amplitude measurements were used. c)Percentage of degeneration: the side to side amplitude percentage indicates the degree of axonal loss, the percentage of degeneration was calculated according to the following

formu

$$\frac{(\text{Amplitude of healthy side} - \text{Amplitude of affected side})}{\text{Amplitude of healthy side}} \times 100$$

Treatment procedures

Ultrasound treatment

Phyaction 190I (Manfacturer Uniphy B.V.; Address Ekkersr ijt4401, 5692 DL Son, The Netherlands) ultrasound machine with a coupling media of aquasonic ultrasound transmission gel were used. The transmission gel and ultrasound facial sound head were at room temperature before treatment. Continuous ultrasound therapy with intensity of 0.5 W/Cm^2 and frequency of 1MHz was applied for the study group (below the inferior lobe of the ear at the stylomastoid foramen behind the mastoid process). Placebo ultrasound treatment (0.0 W/Cm^2) was applied for the control group. Ultrasound therapy was applied for 5 minutes per session, 5 days a week for two weeks.

Physical therapy treatment program:

All patients in the two groups were treated by physical therapy program applied on the affected side of the face. 1- Massage, it was given for about 5 minutes until hyperemia appears without exacerbating pain, types of massage include effleurage (half circular and longitudinal), deep friction massage (transverse and vertical), and kneading were used. 2-Faradic stimulation, one surface electrode was placed on the nerve trunk on the affected side and the other was connected to the skin overlying the facial muscles (Frontalis, and Orbicularis oris) and used to stimulate the motor belly of each one of them separately. Time of stimulation was 20 minutes. 3.Proprioceptive neuromuscular facilitation (P. N.F): Facial muscles act in three directions. a)For muscles acting in the upward direction, e.g. frontalis, levator labi, zygomaticus and levator anguli oris; resistance was given on the occipit during neck extension. B) For muscles acting in the downward direction e.g. corregator, procerus, orbicularis occuli and platysma, resistance was given on frontal bone during neck flexion. C)For muscles acting in the side way direction e.g. depressor labi inferior, and depressor anguli oris, resistance was given during the lateral bending and rotation of neck at the affected side. 4.Splinting: It can be used with grown up children in the form of hook connected between the affected mouth angle and the ipsilateral ear. In small babies a plaster may be used. Splinting is used to prevent muscles over stretch and contractures. 5.Facial neuromuscular reeducation: it is a process of relearning facial movements using mirror feedback.6.Gradual strengthening exercises, assistive, free and resistive exercises were applied according to the muscle's grades. All exercises were in the form of play and games like exercises that were performed in front of a large mirror. 7.Home routine and advices.

Data Statistical Analysis

The obtained data was statistically analyzed using:

Descriptive Statistics:

The mean ± standard deviation ($\overline{X} \pm$ SD) were calculated for each variable, for the two groups (study and control) before and after treatment.

Inferential Statistics:

- The presence of the significant differences between the pre and post training effects within each group were evaluated using **paired t-test**.
- The between group differences in the studied variables were carried out before and after treatment using **unpaired t-test**.
- The probability (P) was obtained from the distribution of the tables. The difference between proportions was considered statistically significant if the p value is 0.05 or less.

RESULTS

This study was conducted to study the effect of ultrasound thermotherapy on motor recovery in children with Bell's palsy.

Data were obtained from both groups, control group (A) and study group (B) before and after the treatment programs. The obtained data within this study were

I. Facial nerve latency of the affected side

I-1. Comparison between the distal latency mean values of the two groups (A & B) on the effected side pre-treatment:

As presented in table (4) and demonstrated in figure (4), when comparing the pre-treatment mean values of the two groups (A and B), concerning latency of affected side, the mean values ± SD were 3.906 ± 0.76 and 3.94 ± 0.63 (Milliseconds) for the two groups respectively which indicated no significant difference (P > 0.05).

Table (4): Pre treatment mean values of latency (Milliseconds) of affected side for control and study groups (A and B):

Item	Patient's groups	$\overline{X}$ ± SD	MD	t value	p value	Significance
Latency (Milliseconds)	Control Group (A) Pre treatment	3.906 ± 0.76	0.34	0.595	< 0.05	NS
	Study Group (B) Pre treatment	3.94 ± 0.63				

$\overline{X}$: mean.SD: Standard Deviation, MD: Mean difference, p value: Probability value, t value: Unpaired t value, NS: Non significant

Figure (4): Pre treatment mean values of latency (Milliseconds) for control and study groups (A and B)

I-2. Comparison between pre and post treatment mean values of latency in control and study groups (A and B):

A- Pre and post treatment distal latency mean value of the control group:

Data presented in table (5) and illustrated in figure (5) showed that, in control group A, the pre and post treatment mean values of latency were 3.906 ± 0.76 and 2.846 ± 0.66 (Milliseconds) respectively, which suggest significant difference (p < 0.05).

Table (5): Pre and post treatment distal latency mean values of control group (A) **Control group**

Item		$\overline{X}$ ± SD	MD	t value	p value	Significance
Latency (Milliseconds)	Pre	3.906 ± 0.76	1.066	5.055	> 0.05	S
	Post	2.84 ± 0.66				

$\overline{X}$: mean.SD: Standard Deviation, MD: Mean difference, p value: Probability value, t value: Unpaired t value, NS: Non significant

Figure (5): Pre and post treatment mean values of latency of control group (A)

B. Pre and post treatment distal latency mean values of the study group (B):

Data presented in table (6) and illustrated in figure (6) showed that, in the study group (B) before and after

treatment, the mean values ± SD of latency were 3.94 ± 0.63 and 1.94 ± 0.51 (Milliseconds) respectively, which suggest significant difference (P < 0.05).

Table (6): Pre and post treatment mean values of latency of study group (B)

Item		$\bar{X}$ ± SD	MD	t value	p value	Significance
Latency (Milliseconds)	Pre	3.94 ± 0.63	2	6.33	< 0.05	S
	Post	1.94 ± 0.51				

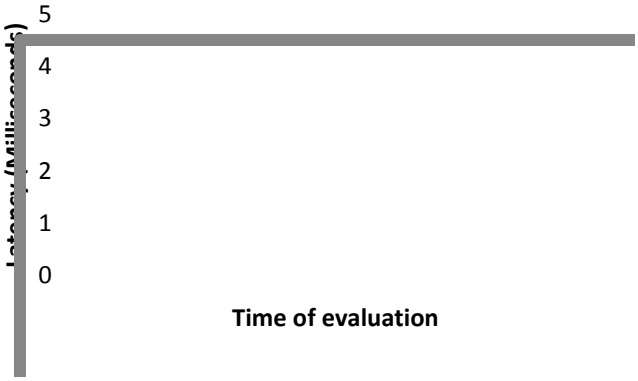

Figure (6): Pre and post treatment mean values of latency of the study group (B)

I-3. Comparison between the distal latency post treatment mean values in the control and study groups (A and B):

It's evident from table (7) and demonstrated in figure (7) that, the mean values ± SD of latency for control and study groups (A and B) were 2.84 ± 0.66 and 1.94 ± 0.51 (Milliseconds) respectively. The difference between both groups in their post treatment mean values ± SD of latency was significant (P < 0.05). The percentage of improvement was 31.69 in favor of the study group B.

Table (7): Post treatment mean values of latency for both groups (A and B)

Item	Patient's groups	$\bar{X}$ ± SD	MD	t value	Improv. (%)	P value	Significance
Latency (Milliseconds)	Control Group (A) Post treatment	2.84 ± 0.66	0.9	3.605	31.69	< 0.05	S
	Study Group (B) Post treatment	1.94 ± 0.51					

$\bar{X}$: mean.SD: Standard Deviation, MD: Mean difference, p value: Probability value, t value: Unpaired t value, NS: Non significant

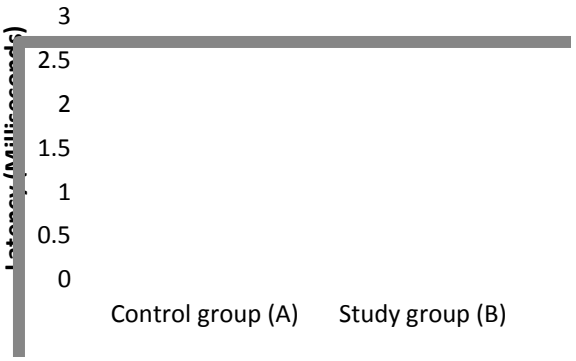

Figure (7): Post treatment mean values of latency for both control and study groups (A and B).

II. Facial nerve amplitude of the affected side

II-1. Comparison between the amplitude mean values of the control and study groups (A & B) pre-treatment:

As presented in table (8) and demonstrated in figure (8), when comparing the amplitude mean values of the two groups (A and B), concerning amplitude of affected side, the mean values ± SD were 0.285 ± 0.33 and 0.272 ± 0.37 (Millivolts) for both groups respectively which indicated no significant difference (P > 0.05).

Table (8): Pre-treatment mean values of amplitude (Millivolts) of both groups (A and B):

Item	Patient's groups	$\bar{X}$ ± SD	MD	t value	p value	Significance
Amplitude (Millivolts)	Control Group (A) Pre treatment	0.285 ± 0.33	0.01	0.022	> 0.05	NS
	Study Group (B) Pre treatment	0.275 ± 0.37				

$\bar{X}$: mean.SD: Standard Deviation, MD: Mean difference, p value: Probability value, t value: Unpaired t value, NS: Non significant

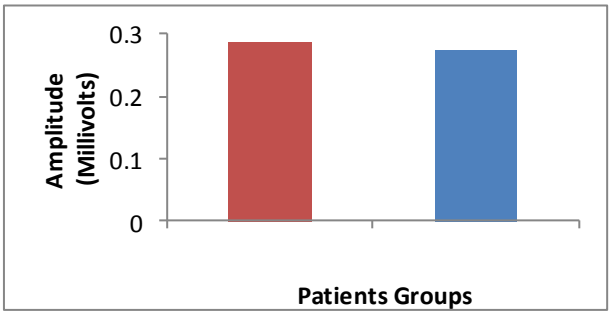

Figure (8): Pre-treatment amplitude mean values (Millivolts) of both groups (A&B)

II-2. Comparison between pre and post treatment mean values of amplitude of both groups (A and B):

A. Pre and post treatment amplitude mean values of control group A:

Data presented in table (9) and demonstrated in figure (9) showed that, in control group A, the pre and post treatment mean values ± SD of amplitude of affected side were 0.285 ± 0.33 and 0.753 ± 0.51 (Millivolts) respectively, suggesting significant difference (P < 0.05).

Table (9): Pre and post treatment amplitude mean values of control group (A):

Item		$\overline{X}$ ± SD	MD	t value	p value	Significance
Amplitude (Millivolts)	Pre	0.285 ± 0.33	0.468	1.93	< 0.05	S
	Post	0.753 ± 0.51				

$\overline{X}$: mean.SD: Standard Deviation, MD: Mean difference, p value: Probability value, t value: Unpaired t value, NS: Non significant

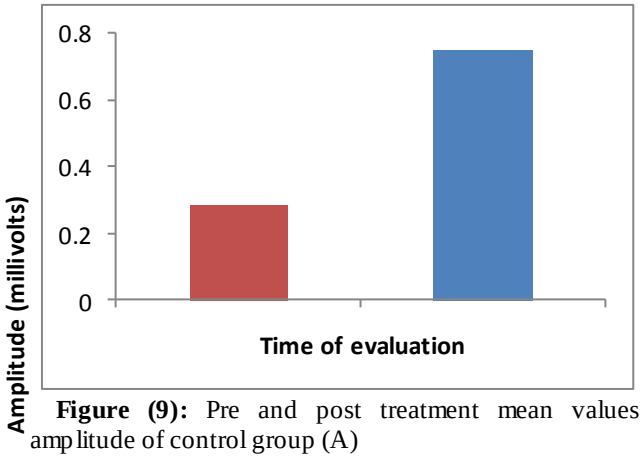

Figure (9): Pre and post treatment mean values of amplitude of control group (A)

B. Pre and post treatment amplitude mean values of the study group (B):

Data presented in table (10) and demonstrated in figure (10) showed that, in the study group (B) the amplitude mean values ± SD of the affected side were 0.275 ± 0.37 and 1.346 ± 1.01 (Millivolts) respectively, which suggesting significant difference (P < 0.05).

Table (10): Pre and post treatment mean values of amplitude of study group (B).

Item		$\overline{X}$ ± SD	MD	t value	p value	Significance
Amplitude (Millivolts)	Pre	0.275 ± 0.37	1.071	3.905	< 0.05	S
	Post	0.753 ± 0.51				

$\overline{X}$: mean.SD: Standard Deviation, MD: Mean difference, p value: Probability value, t value: Unpaired t value, NS: Non significant

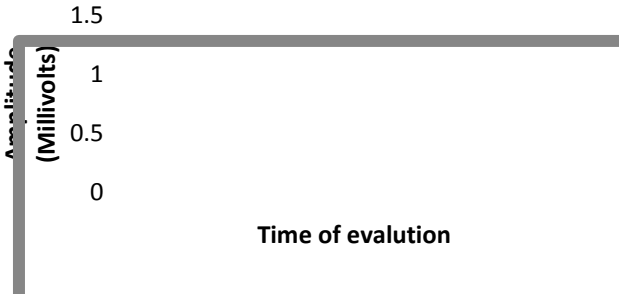

Figure (10): Pre and post treatment mean values of amplitude of study group (B).

III. Comparison between the amplitude mean values of the two groups (A & B) post treatment:

It's evident from table (11) and demonstrated in figure (11) that, the post treatment mean values ± SD of amplitude of the two groups (A and B) were 0.753 ± 0.51 and 1.346 ± 1.01 (Millivolts) respectively suggesting significant difference (P < 0.05). The percentage of improvement was 78.8% in favor of the study group B.

Table (11): Post treatment amplitude mean values of the two groups (A and B):

Item	Patient's groups	$\overline{X}$ ± SD	MD	t value	Improv. (%)	p value	Significance
Amplitude (Millivolts)	Control Group (A) Post treatment	0.753 ± 0.51	0.593	3.461	78.8	< 0.05	S
	Study Group (B) Post treatment	1.346 ± 1.01					

$\overline{X}$: mean.SD: Standard Deviation, MD: Mean difference, p value: Probability value, t value: Unpaired t value, NS: Non significant

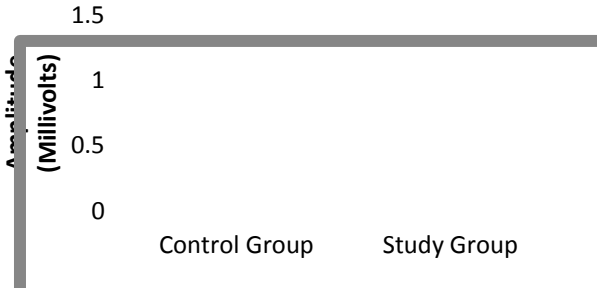

Figure (11): Post treatment mean values of amplitude of control and study groups (A and B).

III. PERCENTAGE OF DEGENERATION OF THE AFFECTED FACIAL NERVE

III-1. Comparison between pre treatment mean values of percentage of degeneration of the control and study groups (A and B):

As presented in table (12) and demonstrated in figure (12), when comparing the pre-treatment mean values of both groups (A and B), Concerning percentage of degeneration, the mean values ± SD were 82.53 ± 2.619 and 82.93 ± 3.833 % of both groups respectively which suggest no significant differences (P > 0.05).

Table (12): Pre-treatment mean values of percentage of degeneration (%) for the two groups (A and B):

Item	Patient's groups	$\overline{X}$ ± SD	MD	t value	p value	Significance
Percentage of degeneration	Control Group (A)	82.53 ± 2.619	0.4	0.339	> 0.05	NS
	Study Group (B)	82.93 ± 3.83				

$\overline{X}$: mean.SD: Standard Deviation, MD: Mean difference, p value: Probability value, t value: Unpaired t value, NS: Non significant

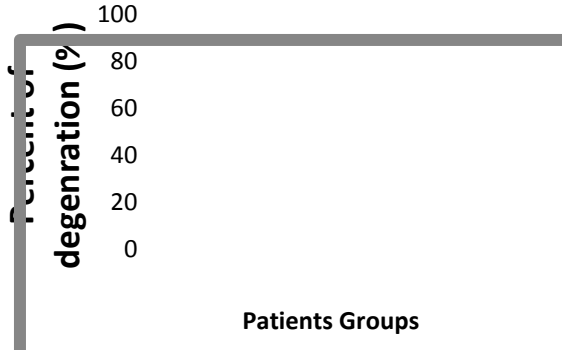

Figure (12): Pre-treatment percentage of degeneration mean values of control and study groups (A and B).

III-2. Comparison between pre and post treatment mean values of percentage of degeneration of the two groups (A and B):

A) Pre and post treatment mean values of percentage of degeneration of control group (A):

Data presented in table (13) and demonstrated in figure (13) showed that, in control group A, the pre and post treatment mean values ± SD of percentage of degeneration were 82.53 ± 2.619 and 54.346 ± 9.237 respectively, suggesting significant difference (P < 0.05).

Table (13): Pre and post treatment percentage of degeneration mean values in the control group (A):

Item	Patient's groups	$\overline{X}$ ± SD	MD	t value	p value	Significance
Percentage of degeneration	Pre	82.53 ± 2.619	28.184	11.574	< 0.05	S
	Post	54.346 ± 9.237				

$\overline{X}$: mean.SD: Standard Deviation, MD: Mean difference, p value: Probability value, t value: Unpaired t value, NS: Non significant

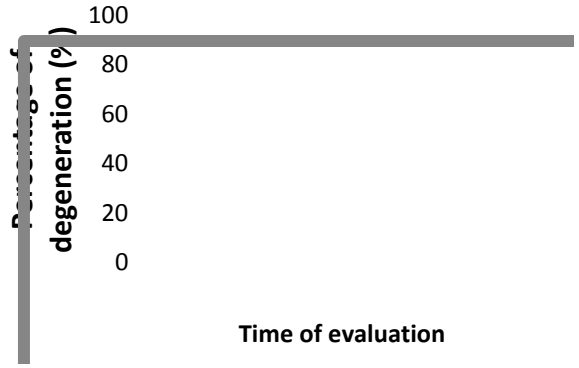

Figure (13): Pre and post treatment mean values of percentage of degeneration of control group (A)

B. Pre and post treatment percentage of degeneration mean values of the study group (B):

Data presented in table (14) and demonstrated in figure (14) showed that, in group B before and after treatment, the mean values ± SD of percentage of degeneration were 82.93 ± 3.833 and 23.99 ± 5.929 respectively, suggesting significant difference (P < 0.05)

Table (14): Pre and post treatment percentage of degeneration mean values of the study group (B)

Item	$\overline{X}$ ± SD	MD	t value	p value	Significance

Percentage of degeneration (%)	Pre	82.93 ± 3.833	58.49	27.3	< 0.05	S
	Post	23.99 ± 5.929				

$\overline{X}$: mean. SD: Standard Deviation
MD: Mean difference. p value: Probability value.
t value: Unpaired t value S: Significant.
%: Percentage of change.

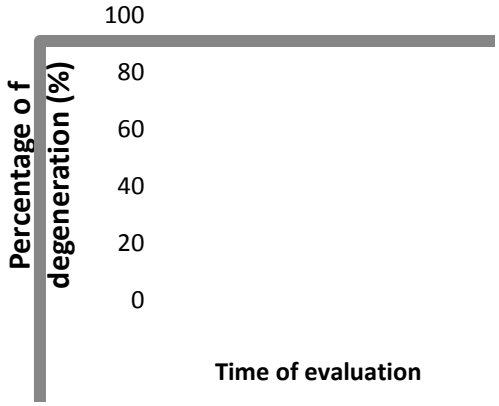

Figure (14): Pre and post treatment mean values of percentage of degeneration of study group (B).

III-3. Comparison between the percentage of degeneration mean values of control and study groups (A & B) post treatment:

As presented in table (15) and demonstrated in figure (15), the percentage of degeneration mean values of both groups (A & B) post treatment were 54.346 ± 9.237 and 23.99 ± 5.929 respectively, suggesting significant difference in favor of the study group ($P < 0.05$).

Item	Patient's Groups	$\overline{X}$ ± SD	MD	Improv (%)	t value	p value	Significance
Percentage of degeneration	Control Group (A) Post treatment	54.346 ± 9.237	30.356	55.9	10.711	< 0.05	S
	Study Group (B) Post treatment	23.99 ± 5.929					

$\overline{X}$: mean. SD: Standard Deviation
MD: Mean difference. P Value: Probability value.
t Value: Unpaired t value S: Significant
%: Improvment percentage

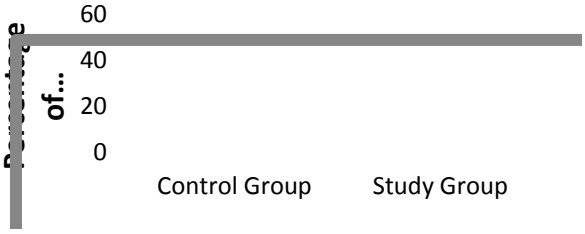

Figure (15): Post treatment mean values of percentage of degeneration of control and study groups.

Discussion

The facial nerve has motor, sensory and parasympathetic fibers. Among its functions are the vital control of the facial expression, taste to the anterior two thirds of the tongue and salivary and lachrymal gland secretion. The main sign for Bell's palsy is a distorted facial expression but patients can experience symptoms eg. taste loss, pain around the ear or hearing problems **(Fontana and Bagnato, 2012)**.

Although there is a controversy about the role of alternative treatment methods (e.g physical therapy, heat therapy, massage, exercises, electrical stimulation, acupuncture and laser therapy) all of these have been used with different degrees of success **(Teixeria et al., 2008)**. The objective of this study was to apply ultrasound thermotherapy to accelerate the recovery process of a child patient with Bell's palsy. Research has clearly demonstrated that ultrasound has an effect on nervous tissue and its ability to propagate action potential. Ultrasound has been reported to increase or decrease the nerve conduction velocity of peripheral nerves according to ultrasound intensity **(Baker et al., 2001)**.

Thirty children (boys and girls) ranging in age from 8 to 12 years with unilateral Bell's palsy palsy were assigned randomly into two groups, control group (A) and study group (B) of equal number each group included 15 patients. For each subject of the two groups, compound motor action potential (CMAP) of facial nerve was measured by using electrtoneurography (ENoG) before and after treatment to determine latency, amplitude and percentage of degeneration. The study group received continuous ultrasound therapy with low intensity of 0.5w/cm^2 and frequency of 1MHz applied below the inferior lobe of the ear at the stylomastoid foramen behind the mastoid process. Ultrasound therapy was applied for 5 minutes per session, 5 days a week for two successive weeks. The control group received placebo ultrasound treatment (0.0 w/cm^2). The two groups received the same designed physical therapy program. Reevaluation was performed after the final session. Data were collected and statistically analyzed to compare between pre-treatment differences between the two groups, pre and post treatment differences of each group and

post treatment differences between the two groups. Before the starting of the treatment program there was non-significant difference between control and study groups regarding clinical assessment and facial nerve latency, amplitude, and percentage of degeneration. Regarding the effect of ultrasound thermotherapy on CMAP latency of the facial nerve, the results of the present study showed that, after two weeks of treatment, the motor conduction latency was decreased indicating significant improvement in nerve conduction velocity.

The use of motor conduction latency as an objective indicator of the degree of innervation of paretic facial muscles has been demonstrated and confirmed by **Skevas et al., 1990, and Ruboyianes et al., 1994.**

Regarding the effect of ultrasound thermotherapy on CMAP amplitude of the facial nerve, the results of the present study showed that after two successive weeks of treatment, there was a significant increase in CMAP amplitude of the affected facial nerve.

Byrne et al., (2005) reported that there is a direct correlation between increasing the amplitude and the rapid rate of nerve regeneration as axon regeneration and remyelination proceed, more muscle fibers are recruited and their responses become increasingly more synchronized there by increasing the amplitude of the CMAP. The value of peak to peak amplitude of the CMAP is a function of 3 factors as follows (1) the population of motor nerve fibers responding to the stimulus (2) the synchronization of their responses (3) the size of the motor unit innervated by the axons.

The results of the present study are in agreement with findings of **(Hong et al., 1988 and Cullum et al., 2001)** who stated that ultrasound deep heat treatment was found to have a thermal effect on the recovery of nerve conduction in compression neuropathy of the rat tibial nerve when a low dose (0.5 watt/cm^2) was used. An adverse effect resulted when a higher dose (1.0 watt/cm^2) was used. Higher doses of ultrasound may cause a decrease of conduction velocity or even a conduction block caused by overheating or a mechanical obstruction effect.

Regarding the effect of ultrasound thermotherapy on the percentage of degeneration of the facial nerve, the results of the present study showed that after two weeks of treatment of children with facial play, there was a significant decrease in the percentage of degeneration of the affected facial nerve toward the end of the treatment program indicating significant improvement of the facial movement and facial symmetry with complete reestablishment to normality.

After axotomy of the facial nerve, a series of intrinsic and extrinsic neuronal events occurs. Intrinsically, these events include the following (1) anterograde "Wallerian degeneration" at 1 to 14 days in which distal myelin swells, retracts, and fragments: (2) retrograde signaling of injury in the form of calcium and sodium leakage from the site of axotomy and generation of an "injury current". Increases in cytokine production, and loss of trophic signals from target – derived sources: and (3) retrograde degeneration marked by neuronal cell body swelling and nuclear eccentricity, sealing of the seeping cut proximal end, and formation of an axonal bulb from which the proximal axon will regrow. Extrinsically, Schwann cells are activated by cytokines, trophic factors, and macrophage factors. They have phagocytic activity for myelin and cell debris; produce cytokines and growth factors, including brain- derived neurotrophic factor (BDNF) and align themselves along the chains of Bungner to form a guiding scaffold for regenerating axons to their targets **(Byrne et al., 2005)**. Regeneration occurs in many tissues, but in the nervous system it has the special meaning of axon growth, not cell replacement. Axon regeneration is a motile process, and growth cones at the elongating axon tip express receptors that mediate responses to environmental signals **(McKerracher 2002)**. Regeneration is the process by which damaged axons regrow and re-establish their original connections. Axon sprouting refers to the process whereby axons from undamaged neurons form new branches to replace those of the lost neurons. This process includes growth of axons and formation of new synapse. Synapse formation that occurs in reaction to an abnormal stimulus such as an injury, termed reactive synaptogenesis. If the sprouting is from homologous neurons, the outcome is likely to maintain or improve the rate of recovery. The sprouts can also come from heterologous neurons. However, in this case the outcome is difficult to predict **(Siegel, 1999)**.

There are many factors that affect neuronal growth: 1-Growth factors: Neurotrophins (NTS) are a family of proteins comprising nerve growth factor (NGF), brain – derived neruotrophic factor (BDNF), NT-3, NT- 4/5 and NT-6. These endogenous soluble proteins have been shown to inhibit neuronal apoptosis and to promote neuronal survival, development,regeneration, plasticity, and neuron related enzyme synthesis **(Flanders and Burmester, 2003)**. 2-Cell adhesion molecules: There are several cell adhesion molecules that can support and stimulate neuronal growth. Isoforms of the neural cell adhesion molecules (NCAM) have been shown to mediate axonal outgrowth after peripheral nerve injury and during reinnervation through interactions with other cell- surface molecules **(Siegel, 1999)**. 3-Growth-associated protein-43 (GAP-43)**:** It is the marker for the plastic potential of neurons. Expression of GAP-43 seems to correlate with a more rapid nerve recovery during the earlier stages of nerve regeneration. The current consensus view is that GAP -43 is involved in axonal sprouting and in the response of growth cones to guidance cues **(Zhang et al., 2005)**. 4-Schwann cells proliferation: schwann cells differentiation and proliferation are essential for establishing the environment for motor neuron regeneration. Once activated, Schwann cells appear to provide an appropriate extracellular matrix and neurotrophic factors to neurons **(Siegel, 1999)**. 5-α calcitionin gene-related peptide (α- CGRP):Calcitionin gene- related peptide (CGRP) is a 37 amino acid peptide found in both sensory and motor neurons in two forms; α- CGRP and β- CGRP. The majority of motorneurons that supply skeletal muscle contain α-CGRP immunoreactivity **(Snyder, et al., 2002)**. 6-Inflammatory cells activity: Inflammatory cells play an important role in axonal regeneration following injury, as they produce factors that stimulate growth and re-myelination, as well as remove axonal and myelin debris from the area. Also fibroblasts are responsible for the generation of collagen fibrils that form the scaffold of the nerve **(Anders et al, 2004)**. There is evidence to show that therapeutic ultrasound can interact with cell types (e.g platelets, mast cells, macrophages, and neutrophils) influencing their activity and leading to the acceleration of repair **(Watson, 2006)**.

Therapeutic ultrasound can alter membrane permeability to various ions. The ability to affect calcium transport through cell membranes is of considerable clinical significance as calcium in its role as an intracellular or second messenger can have a profound effect on cell activity, for instance by increasing synthesis and secretion of wound factors by cells involved in the healing process. This has been shown to occur in macrophages in response to therapeutic levels of ultrasound and these are one of the key cells in the wound healing system, being a source of numerous wound factors **(Leung et al., 2004)**.

Dinno et al., (1989) demonstrated that ultrasound can induced reduction in the sodium / potassium ATPase pump activity. A decrease in pump activity, if it occurs in neuronal plasma membranes may inhibit the transduction of noxious stimuli and subsequent neural transmission which may account in part for the pain relief that is often experienced following clinical exposure to therapeutic ultrasound. **Hart (1993)** also found that following the in vitro exposure of macrophages to ultrasound, a wound factor was released into the surrounding medium that was mitogenic for fibroblasts which are responsible for the generation of collagen fibrils that form the scaffold of the nerve.

Watson and Stephen, (2008) Summarized the effects of ultrasound on nervous tissues as follows: it selectively heats peripheral nerves; may alter or block impulse conduction and may increase membrane permeability and tissue metabolism. They pointed out that any of the above-mentioned mechanisms may be due to the thermal effect of ultrasound and may cause pain relief.

From previous studies, it can be concluded that nerve regeneration may be affected by a thermal agent. Ultrasound thermotherapy biological effects have been recognized, these include, enhanced blood flow, increased membrane permeability and nerve conduction, stimulation of macrophages and fibroblasts proliferation and increased the inflammatory cells activity. All of these extra cellular nerve components play an important role in accelerating the axonal regeneration.

Conclusion:

According to the results of the present study, it is possible to conclude that ultrasound thermotherapy increases the regeneration rate of the facial nerve. The effects of the modality appear to be dose dependent (Continuous ultrasound therapy with low intensity of 0.5 w/cm^2 and frequency of 1 MHz applied below the inferior lobe of the ear at the stylomastiod foramen for 5 minutes per session, 5 days a week for successive two week. It is important therefore to apply the energy at the right time and the right dose in order to gain maximal benefit.

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الملخص العربي

تأثير العلاج بالموجات الفوق الصوتيةالحرارية على تحفيز شفاء العصب السابع عندالأطفال المصابين بالشلل الوجهي الطرفي

يهدف هذاالبحث إلى دراسة فاعلية الموجات الفوق الصوتية الحرارية فى علاج الشلل الوجهي الطرفي عند الأطفال، وقد أجري هذ البحث على 30 مريضاً تم تقسيمهم عشوائياً إلى مجموعتين متساويتين، مجموعة تحكم ومجموعة دراسة، وشمل البرنامج العلاجي لمجموعة الدراسة علاج العصب السابع باستخدام الموجات الفوق الصوتية الحرارية،وتم علاج مجموعة التحكم بوضع جهاز الموجات الفوق الصوتية فى حالة عدم بث للموجات، تم علاج المرضي فى المجموعتين باستخدام برنامج العلاج الطبيعي للعصب السابع ، أظهرت النتائج تحسننا في التقييم الكهربائي للعصب السابع في المجموعتين وكانت نسبة التحسن أعلي في مجموعة الدراسة عن مجموعة التحكم .

الكلمات الدالة: الشلل الوجهي الطرفي عند الأطفال – الموجات الفوق الصوتية الحرارية – التقييم الكهربائي للعصب السابع