

"Efficacy of Phonophoresis on Spastic Ankle Plantar Flexors Control in Diplegic Cerebral Palsy Children"

By:

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Abstract:

Purpose: The aim of the study was to show the efficacy of phonophoresis in combination with traditional physiotherapy for reducing calf muscle spasticity and tightness in diplegic cerebral palsy patient .

Subjects and Methods: The study included 30 patients with spastic diplegic cerebral palsy, with age ranged from 2 to 6 years old, with different grade of MAS 1,1+, divided into two groups. The first group with a mean age (3.87+1.25) years, followed phonophoresis plus traditional physiotherapy training ,3 sessions per week for 8 weeks, the second group with a mean age (4.33+1.35) years followed traditional physiotherapy training only,3 sessions per week for 8 weeks. Outcome measures were modified ashworth scale (MAS) that measure spasticity and universal standard goniometer to measure joint range of motion.

Result: A significant improvement was recorded in spasticity control and tightness release of calf muscles in the experimental group.

Conclusion: Phonophoresis in combination with traditional physiotherapy was more effective in reducing mild spasticity and tightness of calf muscle in diplegic cerebral palsy children than using traditional physiotherapy program alone.

Keywords: Phonophoresis - Ankle Plantar – Spastic - Diplegic Cerebral - Palsy Children







Introduction:

Spastic diplegia, the most prevalent form of CP, is characterized by motor incoordination, originally in the lower extremities, that impairs many functional abilities (Binder,1989). The lesions associated with spastic diplegia may produce any or all of the following symptoms: (1) increased muscle tone, or a velocity-dependent resistance to passive muscle stretch, in synergistic muscle groups; (2) a loss of motor muscle control; (3) deficient equilibrium reactions; and (4) relative imbalance of muscle forces in lower limb across the joints (Davis III,1991).

CP affects motor function and daily activities in children. These disabilities include spasticity. Spasticity can cause contractures due to a loss of sarcomeres, increased muscular rigidity, and alterations in connective tissues. CP care includes maintaining or increasing contractile and connective tissue length (stensj, 2004). Spasticity is linked to segmental spinal neurophysiological abnormalities. Increased a motor neuron excitability, decreased presynaptic and reciprocal inhibition, and decreased 1A facilitation. Passive resistance measures spastic muscle tone. Due to improved stretch reflexes. Spastic and neurologically damaged people have tight gastrocnemius-soleus complexes (Bakheit, et, al., 2003 & DiGiovanni, et, al., 2002).

Spasticity requires adequate treatment because the increased muscle tone inhibits vertical growth of muscle leading to permenant contracture and disability. Spasticity of ankle plantar flexors are frequently seen in patients with central nervous system disorders that lead to disability. Stroke, spastic diplegic and tetraplegic cerebral palsy, spinal cord injury and traumatic brain injury are the most common causes of spasticity (Yoo, et,al., 2002).

There are various spasticity treatments. Ultrasound is used to improve muscle flexibility and speed healing. Phonophoresis uses therapeutic ultrasonography to deliver pharmacologic agent such as anti-infammatory and analgesic drugs through intact skin into subcutaneous tissues. Phonophoresis' physical process is unknown. Several processes have been proposed: raising tissue temperature and hydration, audio streaming, radiation pressure wave, and modifying membrane potential. Phonophoresis depends on frequency, intensity, duty cycle, treatment time, and medication molecule (Michlovitz, et al., 2011; Byl, et al., 1995; Bare, et al., 1996; Nanda, et al., 2008).

Purpose of study:

To investigate whether children with diplegic CP, who had undergone eight weeks of Phonophoresis show change on calf muscle tone and dorsiflexion range of motion at ankle joint.

Hypothesis of the study:

Children with diplegic CP will show improvement in the calf muscle tone and active dorsiflexion range of motion following eight weeks of Phonophoresis application more than who received traditional exercise alone.

Significance of the study:

By 2020, Saudi Arabia could have a million handicapped children (Harrison, 2004). Spasticity is a sign of muscular dysfunction and gait deviation. In CP children, therapeutic ultrasonography has been proven to reduce calf spasticity. This study's use of phonophoresis on spasticity will help physiotherapists treat CP patients with spastic plantar flexors to improve gait.







CHAPTER II: Literature Review

Cerebral palsy:

Cerebral palsy (CP) is a static encephalopathy. It is a disorder of posture and or movement. It is defined as an "umbrella" term covering a group of non-progressive but predominating changing motor impairment syndromes secondary to lesions or anomalies of the brain arising in the early stages of its development. CP usually causes various common clinical features, in particularly:

- 1. Spasticity and Muscle tightness.
- 2. Involuntary movement.
- 3. Difficulty with "gross motor skills" such as walking or running.
- 4. Difficulty with "fine motor skills" such as writing or buttoning .
- 5. Difficulty in perception and sensation.

The severity and presence of these features depend on the location and the extent of damage in the brain (Krigger, 2006).

Epidemiology:

The incidence of cerebral palsy is 2-2.5 per 1000 live births. The incidence of spastic cerebral palsy 50% of identified cases which more common than the other types of cerebral palsy. The incidence of CP is expected to be high because of a high incidence of consanguineous marriages. Some studies showed that physical disabilities in Saudi Arabia are estimated to be 25.8% of overall disabilities. In fact, 21.8% of children with physical disabilities had CP (Rosen, et al., 1992; A.H.M.A, 1999).

Etiology:

It has been documented that some causes of CP are evident, they are very low birth weight with immaturity of the central nervous system, prenatal hypoxia or anoxia, or postnatal meningitis, vascular injuries to the brain, child abuse, or near drowning. Other causes are less obvious, such as exposure to toxic substances or undesignated genetic factors. However, any damage to the developing brain, whether caused by genetic or developmental disorders, injury or disease, may lead to CP (Sankar, et al., 2005)

Classification and Types of Cerebral Palsy:

Many classification system of cerebral palsy exist.

A. Classification according to the site of lesion:

A simplified three-group model: Pyramidal, Extrapyramidal and Mixed Type (Perret, et al., 1992)

• Pyramidal:

Children with the pyramidal form of cerebral palsy have experienced damage to their motor cortex or to the pyramidal tract. Damage to any part of this pathway leads to spasticity (Perret, et al., 1992)

• Extrapyramidal:

In this type, the damage occurs to the pathways outside the pyramidal tract. These extra-pyramidal tracts pass through the basal ganglia or emanate from the cerebellum. The most common type of extrapyramidal cerebral palsy is called athetoid cerebral palsy (Denhoff, et al., 1960).

• Mixed-type cerebral palsy:

The mixed-type of cerebral palsy includes elements of both the pyramidal and extrapyramidal forms. The most common types of mixed cerebral palsy are athetoid and spastic hemiplegic and athetoid and spastic-diplegic (Glenn, et al., 1990)







There are mainly other two methods of classifying CP. The first method is classification by movement disorder (Rosenbaum, et al., 2007) and the second is classification by the number of limbs involved (Minear, 1956).

B. Movement disorder classification:

This method divides CP into four types, which are:

- Spastic CP This type is represented by tight and stiff spastic muscles, and an increased resistance to passive stretch. Damage to the brain's cerebral cortex is generally the main caused of spastic CP. This is present in 75% 88% of people with cerebral palsy.
- Athetoid CP This type is associated with difficulty in controlling and coordinating movements. It results from damage to the basal ganglia in the midbrain. Present in about 15% of people with cerebral palsy
- Ataxic CP This type is the least common form of CP. It is characterized with a disturbed sense of balance and depth perception, impaired muscle tone (hypotonic), and a staggering gait. Ataxia results from damage to the cerebellum. This is present in about 4% of people with cerebral palsy.
- Mixed CP This type is found in 20% of CP cases, with any combination of the above types (Singhi, 2004).

C. Topographic classification:

This method classifies CP into five different types, which are as follows: (Minear, 1956)

- Quadriplegia all four limbs are involved.
- Diplopia all four limbs are involved, but lower limbs are more severely affected than the upper limbs.
- Hemiplegia one side of the body is affected. The upper limb is usually more involved than the lower limb.
- Triplegia three limbs are involved, usually both upper limbs and a lower limb.
- Monoplegia only one limb is affected, usually an upper limb.

Clinical manifestation:

A. Neurological:

• Loss of motor control:

The child with CP has abnormalities of muscle tone and reflexes, shows delay in developmental milestones, and presents with posture and movement problems. When he tries to move, muscle contractions cannot be effectively controlled. This is a result of many factors:

- The muscles are weak and cannot generate the appropriate force necessary for movement.
- Spasticity does not allow the muscle to relax. It causes unnecessary contractions during movement.
- The coordinated contraction and relaxation of many muscles is necessary for a smooth movement. Certain muscles need to relax while others contract. The cerebral centers controlling this complex selective motor control are disturbed in CP. The child is unable to relax certain antagonist muscles and contract the agonists necessary for a specific task.
- Primitive reflexes interfere with the development of gross and fine motor control.
- Advanced postural reactions for balance and equilibrium that are a prerequisite for sitting and walking are either delayed or nonexistent. When the child cannot sustain balance, movement becomes more difficult.
- Superficial sensation is generally normal, cortical sensation, proprioception and sensation of movement may be impaired (Berker, et al., 2005).
- Abnormal muscle tone:

Tone is assessed clinically using passive movements about a joint to determine muscular resistance. Tone is perceived by an examiner but not directly perceived by the patient. Assessment should include palpation of muscles to estimate the resting (baseline) state of muscle activation. Note, however, that tone is not rated by the presence or absence of muscle contraction at rest. Hypertonia is defined as abnormally increased resistance to externally imposed movement about a joint. It may be caused by spasticity, dystonia, rigidity, or a combination of features, while hypotonia is decreased muscle tone the baby may seem flaccid and relaxed, even floppy (Sanger, et al., 2003)







• Babiniski sign:

Plantar stimulation, a noxious stroking of the lateral sole of the foot, produces downward deflection (or plantar flexion) of the great toe in adults with normal upper motor neuron function and upward deflection (or dorsiflexion) of the great toe and fanning of the other toes in the presence of an upper motor neuron lesion. Most newborn babies are not neurologically mature so they normally show a Babinski sign. Upon stimulation of the sole, they extend the great toe. Many young infants do this, too, and it is perfectly normal. However, in time during infancy the Babinski response vanishes and, under normal circumstances, should never return. A Babinski sign in an older child or adult is abnormal. It is a sign of a problem in the central nervous system (CNS), most likely in a part of the CNS which called the pyramidal tract (Miller, et al., 2005).

• Hyperreflexia:

Certain abnormal reflexes may indicate cerebral palsy. Hyperreflexia are excessive reflex responses that cause twitching and spasticity.

B. Muscuskeletal:

• Contractures:

Muscles can become painfully fixed into abnormal positions, called contractures, which can increase muscle spasticity and joint deformities in people with CP.

• Spinal deformities:

Deformities of the spine—curvature (scoliosis), humpback (kyphosis), and saddle back (lordosis) are associated with CP. Spinal deformities can make sitting, standing, and walking difficult and cause chronic back pain. Pressure on and misalignment of the joints may result in osteoporosis (a breakdown of cartilage in the joints and bone enlargement)

• Atrophy:

Can occur due to neglect and bad position.

| Upper extremity | Lower extremity | | |
|-------------------------|----------------------|--|--|
| Pronator | Hip adductor-flexor | | |
| Wrist and finger flexor | Knee flexor | | |
| Thumb adductor | Ankle plantar flexor | | |
| | | | |

Table (1) The most common sites of contracture in the upper and lower extremity

| Spine Scoliosis, kyphosis | | | |
|-----------------------------------|------------------------------|--|--|
| Hip | Subluxation, dislocation | | |
| Femur& tibia | Internal or external torsion | | |
| Foot | Equinus, valgus, varus | | |

Table (2) The most common sites for deformity in human body

• Spastic diplegia:

The involvement of the limbs is symmetrical with lower ones being more severely involved. It has been reported that the most common type of CP is spastic diplegia. The pathology in premature infants with diplegic CP is found in periventricular area. In particular, periventricular leukomalacia(PVL) and intraventicular haemorrhage with enlargement of ventricular system lead to diplegia. Children with spastic diplegia are facing challenging problems of spasticity.

Spasticity prevents muscles and tendons from growing at the same rate as lengthening bones. Also, spasticity forms contractures, makes ambulation and fine- or gross-motor movements difficult, that will lead to muscle ache. Spasticity tends to adduct, internally rotate and flex the hips during walking. Flexion occurs excessively in the hips and knees as the leg is swinging through and/or on the weight bearing (stance) to allow a plantar-flexed foot to swing and clear the







ground, and to push the heel to the ground. This causes a characteristic walking rhythm, known as the crouched gait (Panteliadis, et al., 2004; Kyllerman, et al., 1982).

• Calf Muscle Anatomy:

The term "calf muscles" is often used in reference of the gastrocnemius and soleus. These muscles allow for simple functions such as the lowering of the foot at the ankle joint, which is also known as plantar flexion.

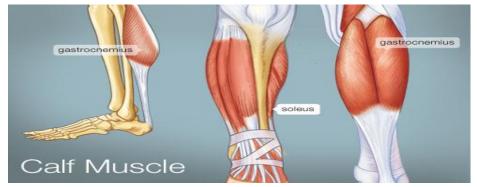


Figure (1) Calf muscle (gastocnemius & soleus)

The gastrocnemius muscle consists of two heads (lateral and medial) and together with the soleus muscle it forms a large muscle group of the ankle plantar flexor muscles: the triceps surae.

• Possible mechanisms underlying the reduced ankle mobility in children with SCP:

An important factor contributing to impaired ankle joint mobility in children with SCP is the hyperactivity of spastic triceps surae muscle (a large muscle group of the ankle plantar flexors) in response to fast stretching. This hyperactivity stems from an upper motor neuron lesion in the central nervous system. Due to the disinhibition of the stretch reflex and abnormal timing of activation, the triceps surae, involved in generating a plantar flexion moment at the ankle, is involuntary actively contracted (unless when lengthened at low speed). As a result, the ankle is predominantly maintained in plantar flexion (equinus). Also, the range of ankle movement is reduced (Bénard, 2011)

Pioneering dynamometry studies performed over three decades ago (Tardieu et al., 1981) indicated that in SCP, shortness of the triceps surae is an important factor in the decreased ankle dorsal flexion.

Physical Problems:

1. Spasticity:

Following an upper motor neurone (UMN) lesion, a patient can present with a variety of sensory motor and cognitive problems. The sensory motor problems in general classified as "positive features" (i.e. abnormal reflex responses, spasticity, spasms, clonus ...etc) and "negative features" (i.e. muscle weakness, loss of dexterity and fatigability).

Although both positive and negative features contribute to the resulting functional loss, in patients with an UMN lesion, there is a substantial focus on one particular positive feature "spasticity". This focus on spasticity results from the preamble that spasticity interferes with functional recovery and lead to secondary complications such as, pain, weakness, and contractures. Spasticity could be defined as "a motor disorder characterized by a velocity dependent increase in tonic stretch reflexes (muscle tone) and increased tendon jerks resulting from disinhibition of the stretch reflex, as one component of an upper motor neurone lesion" (Malhotra, et al., 2009).







Pathogenesis

The pathogenesis of spasticity is presumed to be due to disturbance between inhibitory and facilatory impulse from pyramidal and extra-pyramidal system lead to hyperexitability of alpha, gamma motor neuron of stretch reflex. Many hypotheses explain this hyper excitability. One suggests a change in the balance of excitatory and inhibitory inputs to the motor neuron pool. When the inhibitory inputs are interrupted, the interneuron's send excitatory impulses to the lower motor neurons and they become hyper excitable (Sheean, 2002).

Assessment of Spasticity:

Spasticity has been assessed with various scales:

The first group is referred to as the Ashworth-like scales, after Ashworth, who first described the principle of muscletone assessment. He developed a 5-point scoring scale that determined the degree of resistance encountered in a specific muscle group by passively moving a limb at one (non-) specified velocity through its ROM. This is called the original Ashworth scale (AS). The AS has three modifications, all of which share the same principle (Thibaut, 2013)

The second group is the modified Tardieu scale which described the principle of spasticity assessment by joint-angle measurement at different velocities of muscle stretch. It assesses the range of motion of the ankle and knee: The dynamic component, R1 or angle of the overac5ve stretch reflex is defined at Tardieu velocity of stretch V3 and the slow PROM or degree of muscle contracture R2 is graded as the angle at V1. The score is recorded as R1/R2. Boyd found that a large difference between the two measures characterizes a large dynamic component (Thibaut, 2013)

The third group King's hypertonicity scale could be a good alternative to measure spasticity and other symptoms of upper motor neuron syndrome because it assesses four areas: presence of increased tone, active range of motion, alternating movements and resistance to passive movement. Each component is evaluated separately and has a score of 1 (normal) to 5 (worst), which gives a total score ranging from 4-+20)Thibaut, 2013)

2. Tightness:

In normal muscle, collagen is highly organized around fascicles or groups of myofibres in a structure known as the perimysium and around individual myofibres known as the endomysium. Both the perimysium and the endomysium play important roles in force transduction and muscle stiffness. It is possible that if connective tissue accumulates within spastic muscle, the muscle's mechanical properties may be affected such that this accumulation contributes either directly or indirectly to the development of contractures and secondary bony abnormalities thus playing a major role in mobility problems observed in CP.

Collagen is increased in the spastic muscles of children with CP and that the amount of total collagen correlates with the severity of their disorder. The distribution of this increased collagen is consistent with it playing a role in increased muscle stiffness, formation of contractures and together with the relative lack of growth in length of spastic muscle, may ultimately lead to the formation of bony abnormalities. Once severe fibrotic changes have occurred in CP, muscle function will be impaired and cannot be reversed. (Booth, et al., 2001)

Muscles maintained in shortened positions demonstrate fewer series sarcomeres, reduced length, and increased passive stiffness. In contrast, when muscle is maintained in a lengthened position, the number of series sarcomeres increases and the muscle becomes longer (Tabary, et al., 1972; Williams, et al., 1978)

3. Balance:

The impairment of gross motor function can affect a child's ability to balance. Signs become recognizable as a child learns to sit, rise from a sitting position, and begins crawling or walking Balance impairment is most often associated with ataxic, and to a lesser degree, spastic cerebral palsy.







4. Coordination:

All children with cerebral palsy will likely experience some effect on motor control and coordination due to impaired muscle tone. Muscle control deficiencies can cause persistently stretched, constricted, rhythmic, or spastic limbs. Stress can exacerbate some indications. Some involve tasks, like reaching for something. Coordination and control vary by limb.

The impairment of coordination and control fall under the following types:

- Spastic movements hypertonic movements where the muscles are too tight resulting in muscle spasms, scissoring of the legs, clonus, contracture, fixed joints, and over-flexed limbs.
- Athetoid or dyskinetic movements fluctuating muscle tone causing uncontrolled, sometimes slow, writhing movements which can worsen with stress
- Ataxic movements poor coordination and balance making tasks such as writing, brushing teeth, buttoning shirts, tying shoes, and putting keys into slots difficult
- Mixed movements a mixture of movement impairments, most commonly a combination of spastic and athetoid types, affecting different limbs.

5. Deformity:

CP impairs posture and balance. As a baby sits up and moves, signs may arise. Symmetrical posture is normal. Sitting babies usually have both legs in front. When twisted, they look alike. Right and left limbs don't match in asymmetrical posture. Cerebral palsy commonly affects the hips. One hip-bent leg and one outward.

6. Gait:

The diplegic children begin standing with the hips, knees and ankles extended and the legs crossed. Later, hip and knee flexion and ankle plantar flexion occur. Walking patterns are established at approximately 5 to 7 years of age (Lin, et al., 2000).

In the sagittal plane, look for three types of pathologically abnormal gait:

• Jump gait:

The child walks with hips in flexion, knees in flexion and ankles in plantar flexion as if getting ready to jump. This is typical for diplegic and ambulatory total body involved children when they begin to walk. The reason is spasticity of hip and knee flexors and ankle plantar flexors (Lin, et al., 2000).

• Crouch gait:

Increased knee flexion and ankle hyper dorsiflexion occur during stance phase. They occur in older children and after isolated triceps lengthening's that have been performed without addressing the spastic hamstrings. Hip flexors and hamstrings are tight, and quadriceps and triceps are weak (Lin, et al., 2000).

• Stiff knee gait:

Decreased knee flexion occurs during swing phase. The rectus femoris muscle is spastic and does not allow the knee to flex in initial and midswing phases. Limitation of knee flexion causes difficulty in foot clearance and stair climbing (Lin, et al., 2000).

The abnormal gait in the frontal and transverse planes are:

• Scissoring gait and internal hip rotation

Scissoring gait is defined as crossing over of the legs during gait. The cause is hip adductor and medial hamstring spasticity combined with excessive femoral anteversion (Lin, et al., 2000).

• Trunk lurching

Trunk lurching is an increase in the side-to-side movement of the trunk during walking. It is caused by deficiency of balance. It may become worse after surgery and during periods of rapid growth (Lin, et al., 2000).







7. Mile stone

A child's gross motor function may be hindered or delayed as they grow. Gross motor function is the ability to coordinate big, multi-limb movements. Hypertonia or hypotonia can impede gross motor skills. Impaired gross motor functions limit walking, running, jumping, and balancing.

• Delayed gross motor functions – physical skills developed later than expected; often used in conjunction with developmental milestones for predictable stages of development.

Significant milestones of gross motor function include:

• Rolling Sitting up Crawling Standing Walking.

8. Hand function:

Executing precise movements defines the category of fine motor function. Fine motor control encompasses many activities that are learned, and involve a combination of both mental (planning and reasoning) and physical (coordination and sensation) skills to master.

9. Reflex:

Reflexes are involuntary body motions. Certain basic reflexes are present at or shortly after birth but decrease with maturity. Hyperreflexia causes spasms and jerking. In children with cerebral palsy, abnormal primitive reflexes may not operate properly or may not vanish at key stages of development.

Common primitive reflexes that may improperly function or persist include, but are not limited to:

- Asymmetrical tonic reflex : When the head turns, the legs on the same side will extend, and the opposite limbs contract like in a fencing pose. Asymmetrical tonic reflex should disappear around six months of age.
- Symmetrical tonic neck reflex: The infant assumes a crawling position when the head is extended. Symmetrical tonic neck reflex should disappear between eight and 11 months.
- Spinal gallant reflexes: When the infant lies on its stomach, the hips will turn towards the side of the body that is touched. Spinal gallant reflexes should disappear between three and nine months.
- Tonic labyrinthine reflex: When the head is tilted back, the back arches, the legs straighten, and the arms bend. Tonic labyrinthine reflex should disappear by three-and-a-half years of age.
- Palmer grasp reflex: When stimulating the palm, the hand flexes in a grasping motion. Palmer grasp reflex should disappear around four to six months.
- Placing reflex: When an infant is held upright and the back of a foot touches the surface, the legs will flex. Placing reflex should disappear by five months.
- Moro (startle) reflex: When the infant is tilted so his or her legs are above their head, the arms will extend. Moro reflex should disappear by six months.

10. weakness:

Weakness is the inability to produce or retain force. Maximum muscular force is highly associated with physiological and, to a lesser extent, anatomical cross-sectional area. Reduced tension shows muscle weakening. Reduced strength in CP children may be due to proportional changes in muscle cross-sectional area or reduced specific tension (Elder, 2003)

Management of CP:

CP cannot be cured, but selective management programs can often improve a child's capabilities. There is no standard management program that works for all children with CP. However, drugs can be used to control seizures and muscle spasms. Surgery and special orthosis help to compensate for muscle imbalance and overcome impairments. Counseling can help with emotional and psychological needs. Physical, occupational, speech, and behavioral therapies are other approaches that should be considered to help manage CP impairments and disabilities (Singhi,2004)

There are various methods used to improve functional activity through spasticity management:





• Pharmacological Management

Neuromuscular blockade interrupts neuron, neuromuscular junction, or muscle function. Baclofen, diazepam, and dentrolene are commonly used for spasticity. Baclofen prevents muscle-contracting spinal cord signals. Brain and body relaxant. Dantrolene hinders muscular contraction. Drugs can temporarily alleviate spasticity. Alcohol "washes" or muscle injections are used to reduce CP stiffness. This method corrects contractures. Injecting alcohol into a short muscle temporarily weakens it so therapists can stretch it. Phenol denatures injected protein like alcohol. Children with a growing nervous system should not receive phenol nerve blocks. Skin necrosis, muscular atrophy, paraesthesias, wound infection, and post-injection discomfort are phenol complications.

Botulinum toxin injection is considered to be an appropriate method to control spasticity. It is injected directly into the muscle. It blocks acetylcholine and alleviates muscle spas5city for 3-6 months.

It is indicated to help patients with CP by: (1) improving motor function by balancing muscle forces across joints (2) improving health-related quality of life by 24 decreasing spas5city and/or decreasing caregiver burden (3) decreasing pain from spasticity it has been observed that botulinum toxin improves (Grazko, et al., 1995).

• Surgical Management

Spasticity mismanagement is a major cause of CP contracture. This contracture is surgically treated. Orthopedic and neurosurgery are used to treat spasticity and contractures. The procedure can avoid additional spinal and limb deformities, but it requires competence and a rehabilitation regimen. Mainly used to lengthen muscles and tendons. Surgical methods for equinus deformity in cerebral palsy include gastrocnemius and achilles tendon lengthening. Neurosurgery, specifically dorsal root rhizotomy, regulates leg spasticity by cutting nerves that overstimulate leg muscles.

The procedure is controversial, but it can benefit some CP children, particularly those with spastic diplegia. Unfortunately, Dorsal root rhizotomy is costly, invasive, and painful. It requires expertise, and can cause fibrosis. In addition, the child must undergo general anesthesia (Cottalorda, et al., 1998; Engsberg, et al., 2002).

• Physical Therapy:

Physiotherapy plays a central role in managing the children with CP and it focuses on function, active movement and optimal use of the child's potential. The aims of rehabilitation in children with CP are to minimize the effect of physical impairments, to help the child gain independence in the community and to improve the quality of life of the handicapped children and their families who have a major role to play in the process (Cusick, et al., 2006)

• Bobath Concept (NDT):

Bobath NDT is the most popular CP physiotherapy method (Scrutto, 1984). Normal motor development and function, and contractures and abnormalities were the goals. NDT focused on muscular tone, reflexes, aberrant movement patterns, postural control, sensation, perception, and memory. NDT aimed to transform the nervous system's neural-based motor responses. Bobath is now considered a "idea" rather than a technique. The method combines facilitation, stimulation, and communication to enhance children's motor-sensory problems and functional independence (Bleck, 1975; Kerem, et al., 2003; Tsorlakis, et al., 2004).

• Phonophoresis:

Def.: Phonophoresis is a technique whereby the mechanical effects of ultrasound are used to enhance percutaneous absorption of anti-inflammatory drugs (e.g. hydrocortisone) and local analgesics (lidocaine), through the skin to the underlying tissue (Rennie, 1996).







Physiologic effect of Ultrasound therapy:

- Increased collagen extensibility
- Increased nerve conduction velocities.
- Increased metabolism of edema and exudates
- Decreased joint stiffnes
- Increased pain threshold
- Decreased muscle spasm
- Releases histamine

At an intramuscular depth of 3 cm, a 10-minute hot pack treatment yielded an increase of 0.8°C, whereas at this same depth, 1 MHz ultrasound has raised muscle temperature nearly 4°C in 10 minutes.

• Clinical use of Phonophoresis:

PHP used to manage several soft-tissue conditions, such as tendinitis, bursitis, and muscle spasm; reabsorb calcium deposits in soft tissue, and reduce joint contractures, pain, and scar tissue (as the box below). Continuous ultrasound is used because tissue permeability is increased by the thermal effects, so the medication is more easily absorbed. Treatment occurs at a lower intensity (1 to 1.5 W/cm) for 5 to 15 minutes (Prentice, et al., 1 9 9 9 8)

| Indication | Contraindication | | |
|--|--|--|--|
| Increase deep tissue heating | Hemorrhage | | |
| Decrease inflammation | Infection | | |
| and resolve hematomas | Thrombophlebitis | | |
| Decrease muscle spasm/spasticity | Over suspected malignancy/cancer | | |
| Chronic soft-tissue conditions | Areas of impaired circulation or Sensation | | |
| Decrease pain of neuroma | Over stress fracture sites | | |
| Increase extensibility of collagen tissue | Over epiphyseal growth plates | | |
| Decrease joint adhesions and/or joint contractures | Over the eyes, heart, spine, genitals | | |
| Treat postacute myositis ossificans | | | |

Table (3) Indication and Contraindication of Phonophoresis

Precautions:

- The use of PhP over metal implants is not contraindicated (Keep the sound head moving)
- PhP contraindicated over epiphyseal plates of growing bone (Prentice, et al., 1998)

CHAPTER III METHODOLGY

Subjects:

The study included 30 spastic diplegic children. Their age ranged from 2-6 years old, selected randomly from outpatient rehabilitation clinic in Maternity and Children Hospital in Jeddah City.

Inclusive criteria:

- 1. 2-6 years old with diplegic CP (18 male & 12 female).
- 2. Mild spasticity grade 1& 1+ regarding to MAS.
- 3. Can stand alone or with support.





Exclusive criteria:

Subjects with the following were excluded from participating in this study:

- 1. Severe mental retardation.
- 2. Severe cognitive, visual or auditory disorder.
- 3. Orthopedic surgery as release calf muscle.
- 4. Children who received botulinum toxin in the lower extremity musculature during the past 6 months or who wish to receive it within the period of study.
- 5. Severe spasticity.

The patients divided into two groups (15) patient for each group.

• Group A (Experimental):

Included 15 patient with different grade of MAS 1 ,1+ were received (phonophoresis plus traditional physiotherapy training).

• Group B (Control):

Included 15 patient with different grade of MAS 1,1+ were received (traditional physiotherapy training only).

Instrumentation:

This study was a randomized clinical trial continued 8 weeks at frequency 3 sessions per week.

A. for evaluation:

- The patient referred by neurologic clinic.
- All parent of children informed about the study and take their consent.
- All participants had an initial baseline assessment and final assessment.
- The participants divided into two groups randomly and neither group was aware of the treatment that the other group was receiving.

1) Spasticity Assessment:(Appendix I)

The Modified Ashworth Scale(MAS) is a widely used qualitative scale for the assessment of spasticity (Bohannon, et al., 1978). The scores range from 0 to 4

Modified Ashwarth Scale(MAS)

0 No increase in muscle tone

1 Slight increase in muscle tone, manifested by a catch and release or by minimal

resistance at the end of the range of motion when the affected part(s) is moved in flexion or extension

1+ Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM

2 More marked increase in muscle tone through most of the ROM, but affected part(s) easily moved

3 Considerable increase in muscle tone, passive movement difficult

4 Affected part(s) rigid in flexion or extension

2) Geniometry:

Spasticity limits joint movement. A standard universal goniometer Plastic- ISOM 12 (30 CM) - 360° was used to measure dorsiflexion (DF). Active motion (subject contracting muscles to move) was measured.

Supine, knee flexed 20-30 degrees (supported with pillow or towel).

Goniometer: Axis was over the fibula's lateral malleolus. A stationary arm was inserted lateral to the midline of the fibula's head. Moving arm parallel to calcaneal lateral midline









Figure (2) Universal goniometer

B. For treatment:

- Hot packs
- Assistive device as walker
- Ultrasound therapy device (figure 3).



Figure (3) Ultrasound Therapy Device

Procedure:

A.FOR EVALUATION: 1. Muscle Tone:





All spastic cerebral palsy children were subjected to passive movement for detecting of spasticity 3 times then we take the average also in detecting of ROM measure 3 times then take its average. The intensity of the muscle tone elicited at very slow and at rapid passive joint movement is compared and graded by modified ashwarth scale (Scholtes, et al., 2006).



Figure (4) Spasticity assessment MAS

2. Flexibility:

All spastic cerebral palsy children were recieved passive stretching for improving the mobility of tight muscles in lower extremities i.e. calf muscles, hamstring, hip flexor and hip adductors (Berker, et al., 2005)

3. Postural reaction

Postural reactions emerge in the first year of life and provide the foundation for functional motor skills. Changes in muscle tone in response to body and part posture help keep the body upright. Righting reactions align the head, trunk, and limbs.

Equilibrium reactions provide balance when the centre of gravity is disturbed. They are more mature responses to regain balance than righting reactions, and include counter-rotation of the head and trunk away from the direction of displacement, and the use of the extremities. Protective reactions are required to prevent injury if the equilibrium reactions are unable to restore balance. Protective reactions emerge first to the front, then the side and then backwards.

The Postural Reaction Score Sheet comprises three components for assessing postural reactions. Equilibrium, Righting, and Protective Reactions. Six subcomponents make up righting reaction. Prone, lateral, flexion, neck, labyrinthine, and optical righting. Protective reaction component consists of 4 subcomponents: Prone, Forward, Side, and Backward.

The scoring is done on a 7-point ordinal scale and Equilibrium reaction is assessed for slow and fast perturbation at two different tilt angles, in three different testing positions (lying, sitting and quadruped). The scoring is done on a 9-point ordinal scale using perturbation board (Zafeiriou, 2004).

| Name | Procedure | | |
|---|-----------|---|--|
| Vertical Righting Reactions – Lateral, Anterior & Posterior | | Procedure: Support child under axilla and tilt 35-40° laterally, anteriorly and posteriorly Resonse: Alignment of head with vertical is complete response, maintaining head in alignment with body is partial response | |







| Vertical Righting Reactions – Prone | -Procedure: Place child prone and observe cervical and trunk extension Response: Head lifted to 45° is appropriate response for 4 month old, by 6-7 months child should be able to extend entire trunk and pelvis so an upward concavity is observed (Landau response) |
|-------------------------------------|---|
| Vertical Righting Reaction – Supine | -Purpose: To restore body parts to normal alignment following rotation of some body segment -Types: Non-segmental rolling ('log rolling'), segmental rolling |
| Downward Protective Reaction | -Procedure: Hold child in vertical position, thrust feet first toward surface -Response: Extension and abduction of both legs in preparation for weight bearing |
| Forward Protective Response | -Procedure: Hodl child securely about chest, move infant forward toward surface in head-first position - Response: Arm extension and abduction bilaterally |
| Protective Extension in Sitting | Procedure: With child positioned in sitting gently push laterally, forward or backward to elicit protective response Response: Child extends arm to side, front or behidn to prevent self from falling |
| Equilibrium Reactions | -Prone: Abduction and extension or extremities, with curvature of trunk Sitting: Trunk extension or flexion to A/P displacement, abduction and extension of extremities to lateral displacement with trunk rotation - Quadruped: Similar to responses in prone -Standing: Ankel dorsi/plantarflexion, hip flexion or extension, trunk flexion or extension |

4. Developmental Age:

The Denver Developmental Screening Tool (DDST) is a norm-referenced screening tool designed to be a quick assessment to identify children from 1 month to 6 years of age with developmental delay. The DDST was measured 4 domains of development (personal-social, fine motor-adaptive, language, and gross motor) (Anttila, et al., 2008).

5. Reflexes:

The absence of advanced postural reactions and the presence of primitive reflexes beyond 6 months of age is a sign of poor prognosis (Berker, et al., 2005).

| Signs of | poor prognosis |
|-----------------|-------------------------|
| Present | Absent |
| ASTNR | Parachute response |
| STNR | Neck righting reactions |
| Moro | |
| Extensor thrust | |
| Stepping reflex | |

Table (5) Sign of poor prognosis related to primitive reflex and postural reaction

B. For Treatment:

• Applications

Physiotherapists emphasize the need for the practice to be evidence-based whenever possible. Recent reviews have addressed the effectiveness of physiotherapy interventions for children with CP focusing on neurodevelopmental therapy (NDT), training on muscle strengthening exercises, various physiotherapy interventions, and orthotic devices. Recently, methods such as biofeedback and electrical stimulation have been accepted as adjunct therapies (Anttila, et al., 2008).







• <u>**Traditional Physical Therapy:** Both</u> experimental and control group were received: - Prolonged stretch to spastic muscle to gain relaxation via:

First, a rapid stretch stimulates gamma fibers, which trigger contractile intrafusal muscle fiber and no contractile stretch receptors that provide afferent signals to PHC. Then AHC and alpha motor neuron contract extrafusal muscle fibers. At step 2, a single or repetitive contraction stimulates GTO, sending 1b afferent to PHC, then 1b inter neuron, which reverses the stimulated signals into inhibitory signals, inhibiting PHC. Inhibit AHC, alpha motor neuron, then extrafusal muscle fibers. (Positioning, night splint, reflex inhibiting pattern, Bobath technique) (Latash, 2008). Facilitating anti-spastic muscles (tapping, movement, fast stretch, triggering mass flexion, biofeedback, weight bearing, clenching to toes, rapping the muscle) (Azam, 2010).

- Passive stretching to tight muscles to destruct adhesions in muscles and sheath (most common tight muscles are; calf muscles, hamstring, hip flexor and hip adductor). It must be decent gentle gradual stretch not over stretch at all (Steultjens, et al., 2003).
- Gait training using aids as walker
- Hot packs to improve circulation and relax muscle tension.
- Balance training program which include static and dynamic training.

• Static balance training:

- Ability to maintain posture in different position about 20 second.

- Maintain quadruped position, kneeling, half kneeling, standing; standing with step forward, with large BOS then narrow BOS, shifting to one limb.

• Dynamic balance training:

- Ability to control the body when the support surface is moving or when the body is moving on a stable surface.
- Special graduated active ex. started from tapping followed by minimal resistance exercise to avoid the transient increase of spasticity caused by increased resistance (Azam, 2010).

• Experimental group were received Phonophoresis:

Frequency: 1 MHZ

Types: Continuous

Treatment time: 5 minutes

Technique application:

A-Treatment area:

The patient's skin needs to be carefully evaluated to minimize the natural, internal barriers to transcutaneous drug delivery (eg, dry skin, thick skin, dehydration, poor circulation, poor metabolism) and to maximize the effectiveness of phonophoresis treatments natural enhancers.

B-Application Procedure:

Step 1: A generous amount of coupling medium was applied to cleaned the dry skin.

Step 2: Transducer was moved slowly in a circular pattern that was in direct contact with the patient's skin on bulky spastic calf muscle as far away from epiphyseal plate of growing bone by combination of a small amount of the drugs (2.5 % Lidocaine cream & 2.5 % Hydrocortisone cream) (Stackhouse, et al., 2005; Jaskoviak, et al., 1993; Prentice, et al., 1998).

Step 3: Intensity was turned up to treatment level (1.w/cm2).



ISSN-E: 2617-9563





Figure (5) Prepared position for treatment



Figure (6) Aplication of Phonophoresis on standing table





| | Experimental group (n=15) | | | Control group (n=15) | | |
|--------|------------------------------|------|--|-------------------------|-------------------|--|
| Sex | <u>N %</u> | | | <u>N</u> | <u>%</u> | |
| Male | 7 46.7 | | | <u>11</u> | <u>73.3</u> | |
| Female | <u>8</u> | 53.3 | | <u>4</u> | <u>26.7</u> | |
| Age | 3.87 <u>+</u> 1.25 | | | 4 | .33 <u>+</u> 1.35 | |



Figure (7) Application of Phonophoresis on prone position

Statistical analysis:

The data of the sample were normally distributed. After collecting the data, an SPSS statistics program used to calculate the following values: mean, Standard Deviation(SD) and P value, accepted P value in 0.05

Paired –Samples t –test procedure used to compare the mean of two variables MAS and ROM (pre- post) for a single group and the independent –Sample t- test procedure used to compare the mean of two groups (experimental and control).

CHAPTER IV Result

Table (6) show the characteristics of the study group

The cerebral palsy children were divided into two equal groups:

• Group A (Experimental):

Included 15 patient with different grade of MAS 1 ,1+ were received (phonophoresis plus traditional physiotherapy training).

• Group B (Control):

Included 15 patient, with different grade of MAS 1,1+ were received (traditional physiotherapy training only). Table (6) show the characteristics of the study group, which can be summarized in the sex and age, the experimental group about 7 males and 8 females, male accounted for 46.7% and female 53.3%, while control group was 11 males and 4 female's patients, male accounted for 73.3% and female 26.7%, The mean value of age in experimental group was (3.87+1.25), while the mean of control group was (4.33+1.35), and no significant difference between the two groups regarding age.







• Result of the experimental group were received Phonophoresis plus traditional physiotherapy training)

Table (7) comparison of the mean values of MAS and ROM for the experimental group (pre and post) treatment.

| Ν | | Mean <u>+</u> SD | P-value |
|----|--------------------------|--------------------|---------|
| 15 | Pre –test scores of MAS | 1.87 <u>+</u> 1.19 | |
| 15 | Post- test scores of MAS | 1.27 <u>+</u> 1.67 | .0004 |
| 15 | Pre –test scores of ROM | 9.53 <u>+</u> 4.61 | |
| 15 | Post-test scores of ROM | 12.8 <u>+</u> 6.85 | .0003 |

Table (7) display the result of the experimental group in (pre and post) stages regarding (MAS and ROM). A significant difference was observed for both variables (P- value was .0004 for MAS), while (P- value WAS .0003 for ROM). For MAS the pre – test mean score was 1.87 and for post – test the score was 1.27 with improvement ratio 32.14%. For ROM the pre – test mean score was 9.53 and for post – test the score was 12.8 improvement ratio was 34.27%.

So, there is a significant difference between pre- test and post – test scores in both variables which indicating improvement as regard to MAS and ROM.

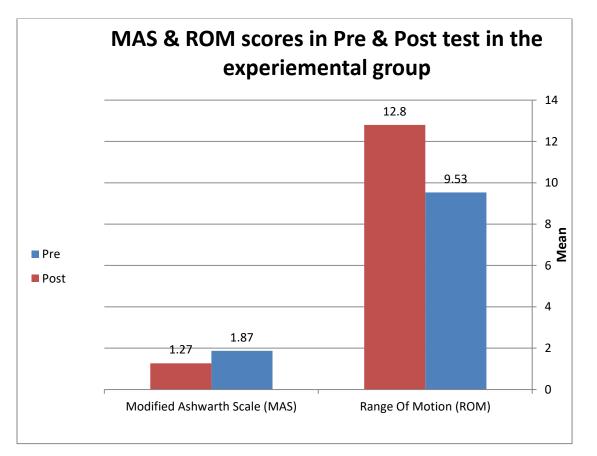


Figure (9) Showed the comparison of the mean values of MAS and ROM for the experimental group (pre and post) treatment

• Result of control group were received traditional physiotherapy training only:

Table (8) comparison of the mean values of MAS and ROM for the control group (pre and post) treatment.





| Ν | | Mean <u>+</u> SD | P-value |
|----|--------------------------|---------------------|---------|
| 15 | Pre -test scores of MAS | 2 <u>+</u> 1.19 | |
| 15 | Post- test scores of MAS | 1.93 <u>+</u> 1.28 | .33 |
| 15 | Pre -test scores of ROM | 9.47 <u>+</u> 4.97 | |
| 15 | Post-test scores of ROM | 10.93 <u>+</u> 6.04 | .02 |

Table (8) display the result of the control group in (pre and post) stages regarding (MAS and ROM), which compared the mean test scores pre-test and post-test in the subject received traditional physiotherapy training only. For MAS the pre – test mean score was 2 and for post – test the score was 1.93 with improvement ratio was 3.33%. For ROM the pre – test mean score was 9.47 and for post – test the score was 10.93 with improvement ratio was 15.49%. A significant difference between pre-test and post-test scores for ROM P value =.02, while there was no significant difference for MAS P value =.33.

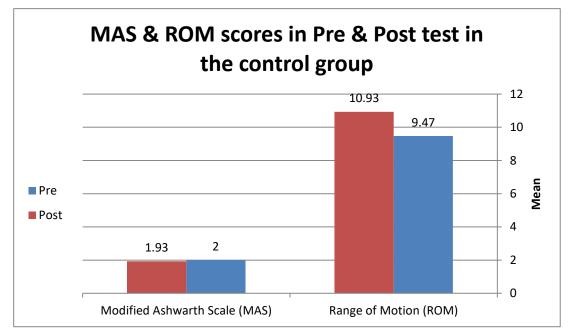


Figure (10) Showed the comparison of the mean values of MAS and ROM for the control group (pre and post) treatment.

| | | Control Group | | | | | |
|--------------------|--------------------|--------------------|--------------------|--------|-----------|-----------|------------|
| Pre. Post. | | Pre. | | Post. | | | |
| MAS | ROM | MAS | ROM | MAS | ROM | MAS | ROM |
| 1.87 <u>+</u> 1.18 | 9.53 <u>+</u> 4.61 | 1.27 <u>+</u> 1.67 | 12.8 <u>+</u> 6.85 | 2+1.19 | 9.47+4.97 | 1.93+1.28 | 10.93+6.04 |

Table (9) comparison between groups (Exp. & control) in pre and post regard to MAS & ROM scores.

Figure (11, 12) showed the comparison between experimental group and control group in the pre – test and post – test regarding MAS and ROM. A significant difference was observed for both variables in experimental group.





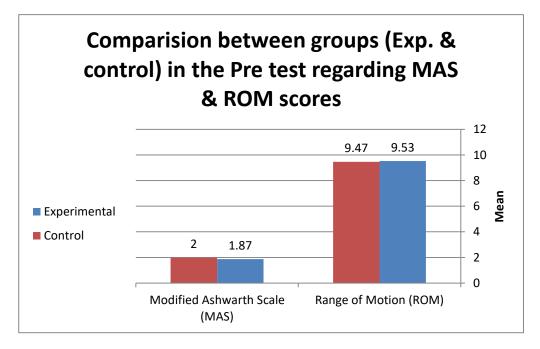
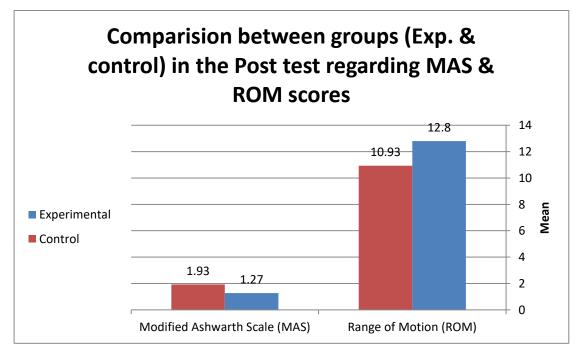


Figure (11) Showed the comparison between experimental group and control group in the pre – test regarding MAS and ROM



0...Figure (12) Showed the comparison between experimental group and control group in the post – test regarding MAS and ROM





CHAPTER V DISCUSSION

A course of phonophoresis plus traditional physiotherapy training were significantly more effective for reduction of spasticity and improvement of ROM than traditional physiotherapy only. The spasticity was reduced by 32.14% in experimental group that mean and SD was (3.87+1.25) in comparison with control group was (4.33+1.35) which reduced by 3.3% also the improvement of range of motion increased by 34.27% in comparison with control group which increased by 15.49%.

A significant difference was observed for both variables in experimental group (P- value was .0004 for MAS) and (P- value was .0003 for ROM) which mean decrease of spasticity and tightness in comparison with control group a significant difference was observed in ROM (P- value was .02), while there was no significant difference for MAS (P- value was .33) which mean decrease of tightness only. In experimental group, the patients who were received PhP have been remained the benefit throughout the most period of sessions.

Underlying mechanism of decrease spasticity and tightness:

In clinical trials and research, thermotherapy increases tissue extensibility. UMNS uses heat to control spasticity. Uncertain how heat affects spasticity. Phonophoresis uses ultrasound to deliver a topical medication formulation. This ultrasound enhances drug distribution via the skin for a local or systemic effect. Heat lowers the stimulation threshold for muscle spindle activity, which increases soft tissue flexibility, decreases joint stiffness, and increases ROM (Wessling, et al., 1987; Lin, 2003).

High-frequency sound waves can promote cell permeability and enhance topically administered medicines. US heating raises the kinetic energy of medication and cell membrane molecules, dilates entrance sites like hair follicles and sweat glands, and enhances circulation to the sonicated area. These physiological changes increase dermal capillary drug collection. Phonophoresis allows viscoelastic change in spastic muscles and decreases alpha moto neuron activity by increasing tissue temperature (Hufschmidt, et al., 1985; Smania, et al., 2010).

Topically applied drugs are categorized according to their intended site of action systemic or local. Systemic drugs diffuse through the epidermis to the dermis to reach the capillary network. Drugs with local targets diffuse into the area immediately below the administration site: subcutaneous tissue, muscle, synovium, ligaments, tendons, and joints. For years it was thought that topically applied drugs all entered the capillary network, became systemic, and then returned to the local area through the bloodstream. A system of local delivery that is separate from systemic delivery. The local and systemic delivery systems can be distinguished by noting the time required for peak absorption of the drug in the target tissue (drug concentration) (Byl, 1995)

Local anaesthetics reduce nerve conduction spasms by interfering with membrane depolarization-induced Na+ permeability. It can be used to test lidocaine's efficacy in treating spasticity. Ultrasonic waves push hydrocortisone through the epidermis in an unknown way. Ultrasonic energy's capacity to increase cell permeability allows more of the active hydrocortisone molecule into the cell, making it more effective as an anti-inflammatory. Ultrasound's phonophoretic impact on hydrocortisone molecules reduced discomfort and increased range of motion (GRIFFIN, et al., 1963; Wing, 1982).

The result of current study was similar to Ansari et al.67 which confirmed the effect of continuous ultrasounds in reducing spasticity. significantly reduced alpha moto neuron excitability (as measured by Hmax/Mmax ratio) and ankle plantar flexors spasticity (as measured by the Ashworth score), this study supports the previous report on the positive effect of phonophoresis in management of spasticity of improvement ROM and MAS.

Okita et al. found that continuous ultrasound treatment may prevent muscular contracture worsening. The latest study confirms earlier findings. Most studies demonstrated ultrasonography improved ROM, or decreased tightness, compared to stretch exercise alone. The current study supports previous findings. In their study, hydrocortisone plus ultrasound significantly increased range of motion. Al ansari et al 2007 evaluated the effect of physiotherapy ultrasound on muscle spasticity in hemiplegic patients. They found an improvement in ROM and MAS, but no significant decrease in spasticity. Large patient and control groups would clarify the effect of US on spasticity.

Studies by sahin et al (Sahin, et al., 2011) about the effect of ultrasound (US) on the spasticity occurring in the ankle plantar flexor muscles, it showed no significant in grade 2 and 3 regarding MAS, this support our study regarding that grade no improvement was found in two groups





Studies by nakhostin et al (Nakhostin, et al., 2009) about the effect of ultrasound and infrared in the management of muscle spasticity, it showed no improvement and no significant between ROM and MAS.

The limitation of the study was the number of subjects were few. there was reducing of mild spasticity only. Also there was no placebo treatment of phonophoresis to give better result.

Recommendations:

We recommend:

The use of phonophoresis during physical therapy sessions for children with spastic diplegic cerebral palsy.

Suggestions:

We suggest for future studies for researcher as follow:

- 1-Increase sample of the study.
- 2-Decrease number of the sessions.
- 3-Conduct using other tools of measurement.

Conclusion:

The combined effect of phonophoresis plus traditional physiotherapy program was more effective in reducing mild spasticity and tightness of calf muscle in diplegic cerebral palsy children than using traditional physiotherapy program only.





References:

- 1. Binder, H., & Eng, G. D. (1989). Rehabilitation management of children with spastic diplegic cerebral palsy. *Archives of physical medicine and rehabilitation*, 70(6), 482-489.
- 2. Davis III, R. B., Ounpuu, S., Tyburski, D., & Gage, J. R. (1991). A gait analysis data collection and reduction technique. *Human movement science*, *10*(5), 575-587.
- 3. Østensjø, S., Carlberg, E. B., & Vøllestad, N. K. (2004). Motor impairments in young children with cerebral palsy: relationship to gross motor function and everyday activities. *Developmental medicine and child neurology*, 46(9), 580-589.
- 4. Bakheit, A. M. O., Maynard, V. A., Curnow, J., Hudson, N., & Kodapala, S. (2003). The relation between Ashworth scale scores and the excitability of the α motor neurones in patients with post-stroke muscle spasticity. *Journal of Neurology, Neurosurgery & Psychiatry*, 74(5), 646-648.
- 5. DiGiovanni, C. W., Kuo, R., Tejwani, N., Price, R., Hansen Jr, S. T., Cziernecki, J., & Sangeorzan, B. J. (2002). Isolated gastrocnemius tightness. *JBJS*, *84*(6), 962-970.
- 6. Yoo, W. K., Chung, I. H., & Park, C. I. (2002). Anatomic motor point localization for the treatment of gastrocnemius muscle spasticity. *Yonsei medical journal*, 43(5), 627-630.
- 7. Michlovitz, et al. (2011). Modalities for therapeutic intervention, (FA Davis, 2011).
- 8. Byl, N. N. (1995). The use of ultrasound as an enhancer for transcutaneous drug delivery: phonophoresis. *Physical therapy*, 75(6), 539-553.
- Bare, A. C., McAnaw, M. B., Pritchard, A. E., Struebing, J. G., Smutok, M. A., Christie, D. S., ... & Seal, L. A. (1996). Phonophoretic delivery of 10% hydrocortisone through the epidermis of humans as determined by serum cortisol concentrations. *Physical therapy*, 76(7), 738-745.
- 10. Nanda, B. K. (2008). *Electrotherapy simplified*. Jaypee Brothers Publishers.
- 11. Harrison, R. (2004). A Cutting-Edge Rehabilitation Facility in Saudi Arabia Is Now a City of Hope. *WASHINGTON REPORT ON MIDDLE EAST AFFAIRS*, 23(5), 52-53.
- 12. Krigger, K. W. (2006). Cerebral palsy: an overview. American family physician, 73(1), 91-100.
- 13. Rosen, M. G., & Dickinson, J. C. (1992). The incidence of cerebral palsy. *American Journal of Obstetrics and Gynecology*, 167(2), 417-423.
- 14. A.H.M.A. (1999). National survey to study disability in children of Saudi Arabia. Riyadh: Prince Salman Center for Disability Research.
- 15. Sankar, C., & Mundkur, N. (2005). Cerebral palsy-definition, classification, etiology and early diagnosis. *The Indian Journal of Pediatrics*, 72(10), 865-868.
- 16. Perret, Y. M., & Batshaw, M. L. (1992). CHILDREN WITH DISABILITIES: A MEDICAL PRIMER.
- 17. Denhoff, E., & Robinault, I. P. (1960). Cerebral palsy and related disorders: a developmental approach to dysfunction. McGraw-Hill.
- 18. Glenn, M. B., & Whyte, J. (1990). *The practical management of spasticity in children and adults*. Lippincott Williams & Wilkins.
- 19. Rosenbaum, P., Paneth, N., Leviton, A., Goldstein, M., Bax, M., Damiano, D., ... & Jacobsson, B. (2007). A report: the definition and classification of cerebral palsy April 2006. *Dev Med Child Neurol Suppl*, 109(suppl 109), 8-14.
- 20. Minear, W. L. (1956). A classification of cerebral palsy. Pediatrics, 18(5), 841-852.
- 21. Singhi, P. D. (2004). Cerebral palsy-management. The Indian journal of pediatrics, 71(7), 635-639.
- 22. Berker, N., & Yalçın, S. (2005). The HELP guide to cerebral palsy. Global-HELP publication.
- Sanger, T. D., Delgado, M. R., Gaebler-Spira, D., Hallett, M., Mink, J. W., & Task Force on Childhood Motor Disorders. (2003). Classification and definition of disorders causing hypertonia in childhood. *Pediatrics*, 111(1), e89-e97.
- 24. Miller, T. M., & Johnston, S. C. (2005). Should the Babinski sign be part of the routine neurologic examination?. *Neurology*, 65(8), 1165-1168.
- 25. http://cerebralpalsy.org/about-cerebral-palsy/symptoms/eight-clinicalsigns-of-cerebral-palsy/
- 26. <u>http://www.ninds.nih.gov/disorders/cerebral_palsy/detail_cerebral_palsy.hm</u>.
- 27. Panteliadis, C. P., & Strassburg, H. M. (2004). Cerebral palsy: principles and management. George Thieme Verlag.
- 28. Kyllerman, M., Bager, B., Bensch, J., Bille, B., Olow, I., & Voss, H. (1982). Dyskinetic cerebral palsy: I. Clinical categories, associated neurological abnormalities and incidences. *Acta Paediatrica*, *71*(4), 543-550.
- 29. Bénard, M. R. (2011). Analysis of 3-D ultrasound of calf muscle geometry in children: growth, spasticity, mechanisms and treatment.





- 30. Tardieu, C., Colbeau-Justin, P., Bret, M. D., Lespargot, A., & Tardieu, G. (1981). Effects on torque angle curve of differences between the recorded tibia-calcaneal angle and the true anatomical angle. *European Journal of Applied Physiology and Occupational Physiology*, *46*(1), 41-46.
- 31. Malhotra, S., Pandyan, A. D., Day, C. R., Jones, P. W., & Hermens, H. (2009). Spasticity, an impairment that is poorly defined and poorly measured. *Clinical rehabilitation*, 23(7), 651-658.
- 32. Sheean, G. (2002). The pathophysiology of spasticity. European journal of neurology, 9, 3-9.
- 33. Thibaut, A., Chatelle, C., Ziegler, E., Bruno, M. A., Laureys, S., & Gosseries, O. (2013). Spasticity after stroke: physiology, assessment and treatment. *Brain injury*, 27(10), 1093-1105.
- 34. Booth, C. M., Cortina-Borja, M. J., & Theologis, T. N. (2001). Collagen accumulation in muscles of children with cerebral palsy and correlation with severity of spasticity. *Developmental medicine and child neurology*, 43(5), 314-320.
- 35. Tabary, J. C., Tabary, C., Tardieu, C., Tardieu, G., & Goldspink, G. (1972). Physiological and structural changes in the cat's soleus muscle due to immobilization at different lengths by plaster casts. *The Journal of physiology*, 224(1), 231-244.
- 36. Williams, P. E., & Goldspink, G. (1978). Changes in sarcomere length and physiological properties in immobilized muscle. *Journal of anatomy*, *127*(Pt 3), 459.
- 37. Lin, C. J., Guo, L. Y., Su, F. C., Chou, Y. L., & Cherng, R. J. (2000). Common abnormal kinetic patterns of the knee in gait in spastic diplegia of cerebral palsy. *Gait & posture*, *11*(3), 224-232.
- Elder, G. C., Kirk, J., Stewart, G., Cook, K., Weir, D., Marshall, A., & Leahey, L. (2003). Contributing factors to muscle weakness in children with cerebral palsy. *Developmental medicine and child neurology*, 45(8), 542-550.
- 39. <u>http://www.ofcp.on.ca/aboutcp.html##A</u>.
- 40. Grazko, M. A., Polo, K. B., & Jabbari, B. (1995). Botulinum toxin A for spasticity, muscle spasms, and rigidity. *Neurology*, 45(4), 712-717.
- 41. Cottalorda, J., Gautheron, V., Metton, G., Charmet, E., Maatougui, K., & Chavrier, Y. (1998). Predicting the outcome of adductor tenotomy. *International orthopaedics*, 22(6), 374-379.
- 42. Engsberg, J. R., Ross, S. A., Wagner, J. M., & Park, T. S. (2002). Changes in hip spasticity and strength following selective dorsal rhizotomy and physical therapy for spastic cerebral palsy. *Developmental medicine and child neurology*, 44(4), 220-226.
- 43. Cusick, A., McIntyre, S., Novak, I., Lannin, N., & Lowe, K. (2006). A comparison of goal attainment scaling and the Canadian Occupational Performance Measure for paediatric rehabilitation research. *Pediatric rehabilitation*, 9(2), 149-157.
- 44. Scrutton, D. (Ed.). (1984). *Management of the motor disorders of children with cerebral palsy* (Vol. 90). Cambridge University Press.
- 45. Bleck, E. E. (1975). Locomotor prognosis in cerebral palsy. *Developmental Medicine & Child Neurology*, 17(1), 18-25.
- 46. Kerem, M., & Livanelioglu, A. (2003). Effects of neurodevelopment therapy on motor development in children with cerebral palsy. *Physiotherapy Rehabil*, *13*(3), 117-123.
- 47. Tsorlakis, N., Evaggelinou, C., Grouios, G., & Tsorbatzoudis, C. (2004). Effect of intensive neurodevelopmental treatment in gross motor function of children with cerebral palsy. *Developmental medicine and child neurology*, 46(11), 740-745.
- 48. Rennie, G. A. (1996). Biophysical principles of heating and superficial heating agents. *Thermal agents in rehabilitation*.
- 49. http://www.nycc.edu/webdocs/ic/IQA/IQAFiles/Protocols/Chapter4/Therap uticUltrasound4_3.pdf.
- 50. Prentice, W. E., Quillen, W. S., & Underwood, F. B. (1998). *Therapeutic modalities for allied health professionals*. McGraw-Hill Companies.
- 51. Bohannon, R. W., Larkin, P. A., Smith, M. B., & Horton, M. G. (1987). Relationship between static muscle strength deficits and spasticity in stroke patients with hemiparesis. *Physical therapy*, 67(7), 1068-1071.
- 52. Scholtes, V. A., Becher, J. G., Beelen, A., & Lankhorst, G. J. (2006). Clinical assessment of spasticity in children with cerebral palsy: a critical review of available instruments. *Developmental Medicine & Child Neurology*, 48(1), 64-73.
- 53. Zafeiriou, D. I. (2004). Primitive reflexes and postural reactions in the neurodevelopmental examination. *Pediatric neurology*, *31*(1), 1-8.
- 54. DeLuca, S. C., Echols, K., Ramey, S. L., & Taub, E. (2003). Pediatric constraint-induced movement therapy for a young child with cerebral palsy: two episodes of care. *Physical Therapy*, *83*(11), 1003-1013.







- 55. Anttila, H., Autti-Rämö, I., Suoranta, J., Mäkelä, M., & Malmivaara, A. (2008). Effectiveness of physical therapy interventions for children with cerebral palsy: a systematic review. *BMC pediatrics*, 8(1), 1-10.
- 56. Latash, M. L. (2008). Neurophysiological basis of movement. Human Kinetics.
- 57. Azam, A.M. (2010). Efficacy of adjustable abduction spreader bar in correction of adduction deformity of the hip in spastic diplegic cerebral palsy children. Medical journal of cairo university 78.
- 58. Steultjens, E. M., Dekker, J., Bouter, L. M., van de Nes, J. C. M., Lambregts, B. L. M., & van den Ende, C. H. M. (2003). Occupational therapy for children with cerebral palsy.
- 59. http://dailymed.nlm.nih.gov/dailymed/archives/fdaDrugInfo.cfm?archiveid=12 808.
- 60. Stackhouse, S. K., Binder-Macleod, S. A., & Lee, S. C. (2005). Voluntary muscle activation, contractile properties, and fatigability in children with and without cerebral palsy. *Muscle & Nerve: Official Journal of the American Association of Electrodiagnostic Medicine*, 31(5), 594-601.
- 61. Jaskoviak, P. J., & Schafer, R. C. (1993). Applied physiotherapy: microcurrent therapy. J Chiropr, 381-400.
- 62. Prentice, W. E., Quillen, W. S., & Underwood, F. B. (1998). *Therapeutic modalities for allied health professionals*. McGraw-Hill Companies.
- 63. Wessling, K. C., Devane, D. A., & Hylton, C. R. (1987). Effects of static stretch versus static stretch and ultrasound combined on triceps surae muscle extensibility in healthy women. *Physical Therapy*, 67(5), 674-679.
- 64. Lin, Y. H. (2003). Effects of thermal therapy in improving the passive range of knee motion: comparison of cold and superficial heat applications. *Clinical rehabilitation*, *17*(6), 618-623.
- 65. http://www.strattonrehab.com/services-and-interventions/
- 66. Hufschmidt, A., & Mauritz, K. H. (1985). Chronic transformation of muscle in spasticity: a peripheral contribution to increased tone. *Journal of Neurology, Neurosurgery & Psychiatry*, 48(7), 676-685.
- 67. Smania, N., Picelli, A., Munari, D., Geroin, C., Ianes, P., Waldner, A., & Gandolfi, M. (2010). Rehabilitation procedures in the management of spasticity. *Eur J Phys Rehabil Med*, *46*(3), 423-38.
- GRIFFIN, J. E., & TOUCHSTONE, J. C. (1963). ULTRASONIC MOVEMENT OF CORTISOL INTO PIG TISSUES I. MOVEMENT INTO SKELETAL MUSCLE. American Journal of Physical Medicine & Rehabilitation, 42(2), 77-85.
- 69. Wing, M. Phonophoresis with hydrocortisone in the treatment of temporomandibular joint dysfunction. Physical Therapy 62, 32-33 (1982).
- 70. Okita, M., Nakano, J., Kataoka, H., Sakamoto, J., Origuchi, T., & Yoshimura, T. (2009). Effects of therapeutic ultrasound on joint mobility and collagen fibril arrangement in the endomysium of immobilized rat soleus muscle. *Ultrasound in medicine & biology*, *35*(2), 237-244.
- 71. Knight, C. A., Rutledge, C. R., Cox, M. E., Acosta, M., & Hall, S. J. (2001). Effect of superficial heat, deep heat, and active exercise warm-up on the extensibility of the plantar flexors. *Physical Therapy*, *81*(6), 1206-1214.
- 72. Rather Aijaz, Y., & Chaudhary, P. (2007). Ultrasound and prolong long duration stretching increase triceps surae muscle extensibility more than identical stretching alone. *Indian Journal of Physiotherapy and Occupational Therapy*, *1*(3), 07.
- 73. PINTO, J. (2010). A comparative study on the effectiveness of cryotherapy with that of the superficial heat in combination with electrical stimulation and stretching in reducing spasticity of plantar flexors in children with spastic cerebral palsy (Doctoral dissertation).
- 74. Nakano, J., Yamabayashi, C., Scott, A., & Reid, W. D. (2012). The effect of heat applied with stretch to increase range of motion: a systematic review. *Physical Therapy in Sport*, *13*(3), 180-188.
- 75. Khan, S., Shamsi, S., & Alyaemni, A. A. (2013). A comparison of superficial heat, deep heat and cold for improving plantar flexors extensibility. *Middle-East J. Sci. Res*, 13(4), 477-482.
- Draper, D. O., Anderson, C., Schulthies, S. S., & Ricard, M. D. (1998). Immediate and residual changes in dorsiflexion range of motion using an ultrasound heat and stretch routine. *Journal of Athletic Training*, 33(2), 141.
- 77. Fellinger, K., & Schmid, J. (1954). Klinik und Therapie des chronischen Gelenkrheumatismus. Verlag für Medizinische Wissenschaften.
- 78. Ansari, N. N., Naghdi, S., Bagheri, H., & Ghassabi, H. (2007). Therapeutic ultrasound in the treatment of ankle plantarflexor spasticity in a unilateral stroke population: a randomized, single-blind, placebo-controlled trial. *Electromyography and clinical neurophysiology*, *47*(3), 137.
- 79. Sahin, N., Ugurlu, H., & Karahan, A. Y. (2011). Efficacy of therapeutic ultrasound in the treatment of spasticity: a randomized controlled study. *NeuroRehabilitation*, 29(1), 61-66.
- 80. Nakhostin Ansari, N., Naghdi, S., Hasson, S., & Rastgoo, M. (2009). Efficacy of therapeutic ultrasound and infrared in the management of muscle spasticity. *Brain Injury*, 23(7-8), 632-638.

