"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".

By

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A dissertation submitted to the
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In partial fulfillment
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Master of Physiotherapy (M.P.T)

In

PAEDIATRICS

Under the guidance of
Mr. S.PADMAKUMAR

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DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled

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is a bonafide and genuine research work carried out by me under the guidance of **Mr.S.PADMAKUMAR, PRINCIPAL,** Shree Devi College of Physiotherapy.

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ABSTRACT

"A comparative study to assess the effectiveness of cryotherapy with that of the superficial heat in combination with electrical and stretching in reducing spasticity of plantar flexors in children with spastic cerebral palsy."

Back ground: The most common movement disorder in posture and movement dependent tone regulation disorder. The clinical manifestation of spastic paresis varies widely, depending on various impairments of muscle function that can be distinguished. The functional ability of the child with spasticity often deteriorates during development. To reduce spasticity and prevent contractures various interventions in physiotherapy are given. Electrical stimulation, cryotherapy, superficial heat and stretching are also the treatment approaches in physiotherapy used to reduce spasticity in children with spastic cerebral palsy.

Method: 60 patients from special schools and from Govt. Wenlock hospital, with the age group between 5-15 years were randomly divided into two groups: Group -A received electrical stimulation for tibialis anterior 30 min, cryotherapy for 15 min, and stretching for planar flexors. Group-B received electrical stimulation for tibialis anterior, superficial heat for 15 min, and stretching on spastic plantar flexors for 3 days in 4 weeks. The pre and post treatment assessment was done by using Modified Ashworth scale and ROM measurement.

Result: There was a very highly significant increase in ROM in group A receiving electrical stimulation, ice, stretching as compared to group B receiving electrical stimulation, heat, stretching which showed slight increase in ROM. This study is highly significant in favour of electrical stimulation, ice, stretching.

Conclusion: This study concludes that both the protocols reduce symptoms and increase the functional status. But electrical stimulation, ice, stretching (Group A) is more effective in reducing spasticity and increasing ROM than Electrical stimulation, superficial heat, stretching (Group B)

Key words: spasticity, electrical stimulation, ice, superficial heat, stretching, physiotherapy

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INTRODUCTION

Cerebral palsy (CP) is a well recognized neurodevelopmental condition beginning in early childhood and persisting through the life span. Originally reported by Little in 1861.

CP describes a group of disorder of the development of movement and posture, causing activity limitation, that are attributed to non progressive disturbances that occurred in the developing fetal or infant brain. The motor disorders of the cerebral palsy are often accompanied by disturbances of sensation, cognition, communication, perception and/or behaviour, and/or by a seizure disorder.[1]

In the industrialized world, the **incidence of CP** is about 2 per 1000 live births. There are an estimated 4-8 millions children and people in India with CP. The incidence is more in males than females. The surveillance of CP in Europe reports a M:F ratio of 1.33:1. In the United States, approximately 10,000 infants and babies are diagnosed with CP each year and 1200-1500 are diagnosed at preschool age. Overall advances in case of pregnant mothers and their babies has not resulted in a noticeable decrease in CP.

Prevalence of CP is best calculated around the school entry age of about 6 years, the prevalence in the US, is estimated to be 2.4 out of 100 children Apgar scores have sometimes been used as one factor to predict whether or not an individual will develop CP.[2]

The **etiology** of CP is very diverse and multifactorial. The causes are congenital, genetic, inflammatory, infectious, anoxic, traumatic and metabolic. The injury to the developing brain may be prenatal, natal or postnatal. As much as 75%-80% of the cases are due to significant trauma or asphyxia. The most important risk factor seems to be prematurity

and low birth weight. CP occurs more commonly in children who are born very prematurely or at term.

Prenatal Chloriaamnionitis is also a significant **risk factor** accounting for as much as 12% CP in term infants and 28% in premature infants. Cystic Periventricular Leukomalacia (CPVL) is a risk factor with 60%-100% of patients with CPVL developing CP.

Prenatal risk factor includes intrauterine infections, teratogenic exposures, placental complications, multiple births, and maternal conditions such as mental retardation, seizures or hyperthyroidism.

Perinatal risk factors are infections, intracranial hemorrhage, seizures', hypoglycemia, hyperbilirubinia, perinatal arterial ischemic stroke and significant birth asphyxia.

Postnatal causes include tonic, infectious meningitis, and encephalitis, traumatic such as drowning. There is also a relation between coagulopathies causing cerebral infarction and particularly hemiplegic type of CP. Postnatal events accounts for 12-25% of CP but in a large no of cases, the cause of CP remains unknown.[3]

The **topographic classification** of CP is monoplegia, hemiplegia, diplegia, triplegia and quadriplegia. Monoplegia and triplegia are relatively uncommon. Diplegia is the commonest form (30-40%) hemiplegia is 20-30% and quadriplegia 10%.

Quadriplegic CP: This is the most severe forms involving all four limbs, and the trunk. Upper limbs are more severely involved than the lower limbs, associated with acute

hypoxic intrapartum asphyxia. Voluntary movements are few, vasomotor changes of the extremities are common. Most children have pseudobulbar signs with difficulties in swallowing and recurrent aspiration of food material; intellectual impairment is severe in all cases.

Hemiplegic CP: Spastic hemiparasis is a unilateral paresis with upper limbs more severely affected than the lower limbs. Voluntary movements are impaired with hand function being most affected. Pincer grasp of the thumb, extension of the wrist and supination of the forearm are affected. In the lower limbs dorsiflexion and eversion of the foot are most impaired. There is increased flexor tone with hemi paretic posture, flexion at the elbow and wrist, knees and equines position of the foot. Palmar grasp may persist for many years. Sensory abnormalities in the affected limbs are common. Steriognosis impaired most frequently. Two point discrimination and position sense is also defective. Seizures occur in more than 50% visual field defects, homonymous hemianopia, cranial nerve abnormalities most commonly facial nerve palsies are seen.

Diplegic CP: Spastic diplegia is associated with prematurity and low birth weight. Nearly all preterm infants with spastic diplegia exhibit CPVL on neuroimaging. Intellectual impairment, visual disturbances due to the site of injury affecting the descending corticospinal tracts and visual radiations. Lower limbs are more severely affected than the upper limbs. Toe walking is present due to impaired dorsiflexion. In severe cases there is flexion at hips, knees, and to lesser extent elbows. When the child is held vertically rigidity of lower limbs is most evident and adductor spasm of the lower extremities causes scissoring of the legs.

Based on the type of neuromuscular deficits CP is classified into (1) Spastic (2) Dyskinetic (inclusive of choreo athetoid and dystonic) (3) Ataxic (4) Hypotonic
(5) Mixed.

Spastic CP is the commonest and accounts for 70-75% of all cases. Dyskinetic 10-15% and ataxic <5%. Spastic type exhibit pyramidal involvement with upper motor neuron signs, weakness, hypertonia, hyperreflexia, clonus and +ve babinski.

Dsykiesia is characterized by exrapyramidal involvement in which rigidity, chorea, choreoathetosis, athetoid and dystonic movement are seen. This type of CP is associated with birth asphyxia. The severity of dystonic postures may vary with body position, emotional state, and sleep. Clonus and Babinski are absent. Primitive reflexes are more prominent and persist for a longer time in dyskinetic CP.

Ataxic CP is causing by damage to the cerebellum. Some of these individuals have hypotonia and tumors. Motor skills such as writing, typing or using scissors might be affected, as well as balance and co-ordination is impaired especially while walking. It is common for individuals to have difficulty with visual and/or auditory processing.

Hypotonic CP is characterized by generalized muscular hypotonia that persists beyond 2 to3 years of age that does not result from a primary disorder of muscle or peripheral nerve. The deep tendon reflexes are normal or hyperactive.

Mixed CP is the mixture of all the above types.[3]

Spasticity is a physiological consequence of an injury to the nervous system. It is a complex problem which can cause profound disability, alone or in combination with the other features of an upper motor neuron syndrome and can give rise to significant difficulties in the process of rehabilitation. This can be associated with profound restriction to activity and participation due to pain, weakness and contractures.

Spasticity is a common symptom seen as a consequence of an injury to the brain (stroke, trauma, hypoxia, infection, CP, post surgery) spinal cord injury or multiple sclerosis.

It is **defined** as a "disordered sensory- motor control, resulting from an upper motor neuron lesion presenting as intermittent or sustained involuntary activation of the muscle". It is associated with both positive and negative components of upper motor neuron syndromes. **Positive components** include muscle over activity, flexor and extensor spasm, hyperreflexia, athetosis, spastic dystopias, clonus, and an extensor plantar response. Common **negative symptoms** comprise weakness/paralysis, early hypotonia, fatigue, loss of dexterity.

Spasticity does not always cause harm and can occasionally assist in the rehabilitation process by enabling a patient to stand when their limb weakness would not allow it.

Assessment of spasticity can be done by Ashworth scale, Goniometry, Tardieu scale, Goal attainment scale, Spasm scale, Penn spasticity scale.[4]

Spasticity has been characterized as an abnormal muscle state in which phasic and tonic stretch reflexes are usually hyperactive, flexion reflexes are hyperactive and dexterity and strength are decreased.

The precise **nature** of the neurological changes producing spasticity in various disorders has yet to be elicited. In general, spasticity is thought to result from a disruption of the normal balance of neural inputs to the alpha motor neurons. Central nervous system disorders may

result in an increase in the central or peripheral excitatory inputs to the alpha motor neurons, a decrease in inhibitory inputs to alpha motor neurons, or some combination of these factors. The net effect of such input imbalances is increased alpha motor neuron excitability, increased muscle tone, and disordered motor control. In more mild cases, spastic muscle may not be spontaneously active but exhibit hyper reflexia, which resist either passive or active stretch of the muscle. In more severe cases, spastic muscles may be in a state of nearly constant contraction lead to permanent joint contracture.[5]

The **management of spasticity** is carried out by a co-ordinated multidisciplinary team, which allows more timely intervention and close monitor of the progress.

The therapeutic and physical interventions include

(a) Physical modalities-stretching, cooling the muscles, heat includes fluidotherapy, paraffin, superficial heat and whirlpools.

-Orthosis/equipment aids, e.g.: ankle foot orthosis, insole, ankle supports, wrist/hand/elbow splints, knee splints, spinal brace, hip brace etc.

-Positioning e.g., T soles, wedges, cushions, foot straps

-Massage

-Splinting

-Dynamic physiotherapy techniques-Bobath technique, PNF, Brunnstrom technique

(b) Electrical therapy

-Functional Electrical Stimulation

-NMES

-Therapeutic Electrical Stimulation

(c) Pharmacological

Oral Drugs:-Beclofin, benzodiazepines, siluzole, beta blockers, thymoxamine, Orphenadrine.

Intrathecal Pump-Baclopfen.

D) Nerve Block:-Peripheral, regional, Neurolytic blockage.

E) Botulinum toxin injection.

The effect of toxin is to inhibit the release of acetylcholine at NMJ.

F) Surgical Techniques.

-Neuromuscular techniques:-Anterior and Posterior rhizotomy, pheripheral neurotomy, drezotomy, percutanous radiofrequency rhizotomy, spinalcord and deep cerebellar stimulation of the superior cerebellar peduncle. Functional neurosurgery.

-Orthopaedic Procedures:- muscles or tendon lengthening, operations tenotomy, neuroctomies, transfer of tendons.[4]

Electrical stimulation was used for the control of spasticity. As far back as 1871 Levin and co-workers in their study, they found that antagonist to spastic musculature were stimulated with uninterrupted "Faradic" current at a frequency of 100 pps using a monopolar electrode configuration. The electrode placed over the motor point for the muscle stimulation was applied at an amplitude to evoke a maximum contraction. Regardless of the underlying pathology the authors reported a relaxation of hypertonicity within several seconds of initiating stimulation as evidenced by reduction in opposition to passive stretch of the spastic muscle. Reduction in spasticity was accompanied by improvements in function associated with self-care activities, mobility, and posture.

As the stimulation applied to the peripheral nerve leading to antagonist muscle, the large diameter Ia muscle spindle afferent fibres originating in these fibres are transmitted to the spinal cord and excite spinal interneurons, which in turn inhibit the activity in the motor neurons to spastic muscle. The activation of such a reciprocal inhibition pathway may lead to an immediate reduction of the activity in the spastic muscle.[5]

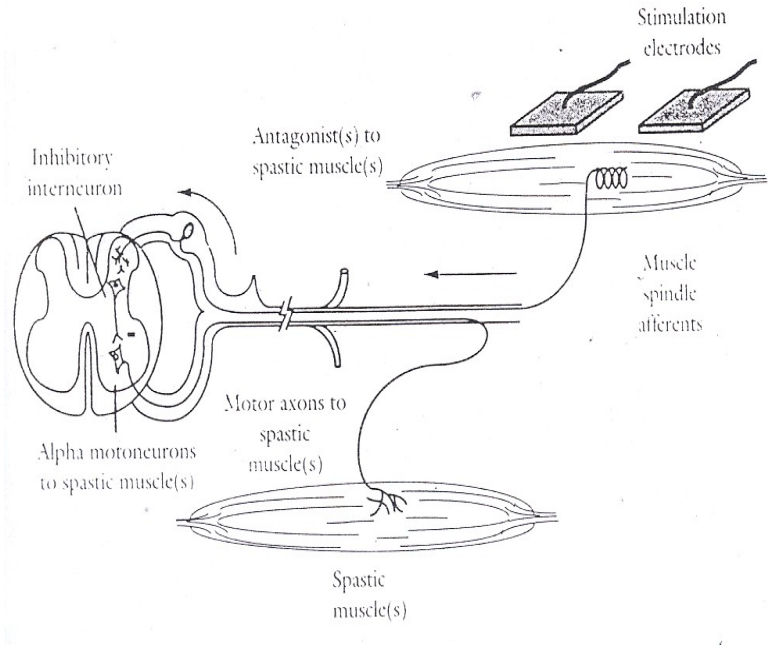


FIG. 1.1 Proposed mechanism of action of antagonist stimulation to control spasticity.

During cryotherapy heat from the body tissue is transferred to the cold modality via conduction provided. There is direct contact between the two bodies. The greater the temperature differences between the body and modality quicker the transfer of heat.

Regeneration of body heat and modality cooling as the body tissue gives up heat, the heat lost is replaced by circulating blood and surrounding tissues. Cold penetrates more deeply than superficial heating modalities. For example after 20 minutes of ice pack application to the gastronomies muscle the intra muscular temperature declined more slowly than the subcutaneous temperature (fig) when ice pack was removed the subcutaneous

temperature rose steeply where as the intra muscular temperature continued to decline for a period of time until both tissues were within 2^0 to 3^0c of each time.[6]

Cooling has a dual action as it inhibits the mono synaptic stretch reflects and lowers the receptor sensitivity after it has been removed. Cooling can be used in different ways. It causes gradual switching of the activity of individual muscle.[7]

Stretching, the process of elongation is one currently used technique in the management of spasticity. Stretches can be applied mechanically (i.e. with a dynamometer or an intelligent feedback controlled device) manual stretching is better in clinical practice. During stretching tension is applied to the soft tissue structure. Structures that are put under tension consist of muscle, tendon, and connective tissue, vascular, dermal and neural tissue. Stretching may change the muscle's viscoelastic structural and excitability properties. However many neural and non neural responses to stretch especially in spasticity. The aims of stretching in spasticity may be to normalize muscle tone, to maintain or increase soft tissue extensibility, to reduce pain and to improve function. Stretching as a treatment can vary in intensity of stretch is the amount of tension that applied to the structure. Stretching can be static, dynamic, prolonged and ballistic stretching.

Slow sustained stretching forms the basis of spasticity treatment. It activates the muscle spindles [Ia and II endings] Golgi tendon organs [Ib endings] which are sensitive to length changes. It inhibits or dampens muscle contraction and tone largely due to peripheral reflex effects. It can be more effective in extensor muscle than flexors due to added effects of II inhibition.[8]

Superficial heat and cold have been used for centuries to manage soft tissue and joint injuries with specific goal of relieving pain, altering the physiological process underlying the

tissue healing, and affecting the plasticity of the connective tissue, including muscle, tendon, legs and joint capsules.

Electrotherapy agents which may be used for cryotherapy are cold packs, ice massage, cold whirlpool, vapour coolant sprays, or cold compression units. Or thermotherapy by using moist heat or hydrocollator packs, paraffin baths, warm whirlpool, infrared lumps, US and SWD.

Superficial heat is best applied before performing flexibility exercises to take advantage of the benefits of tissue temperature elevation, including vasodialatation that increases the tissue oxygenation and transport of metabolites to exercising tissue, increasing the rate of enzymatic and biochemical reaction that may facilitate tissue healing and altered viscoelastic properties leading to increased soft tissue extensibility, decreased joint stiffness, and increasing range of motion.

The neuromuscular effect of heat, in contrast to those of cold includes increased nerve conduction velocity and decreased latency time for both sensory and motor nerves. Nerve conduction velocity increases 2m/sec for every 1^0c (1.8^0F) increase in temperature .muscle relaxation occurs as a result of a decreased firing rate of type II muscle spindle afferents and gamma afferents and an increased firing rate of type II fibers of golgi tendon organs. These, in turn contribute to a decrease in firing of the alpha motornueron to the extrafusal muscle fiber, resulting in muscle relaxation. Heat lowers the stimulus threshold for muscle spindle activity.

Heat causes increased connective tissue extensibility if the tendon, ligament, scar tissue or joint capsule tissues are superficially located. For maximum connective tissue plastic deformation to occur, the tissue temperature must be maintained in 40^0 to 45^0c (104^0 to 113^0F) for a minimum of 5 to 10 minutes while applying the stretch. In cases where the viscoelastic properties of connective tissue are to be altered, the tissue must be subjected to

adequate concurrent heat and stretch for a sufficiently long period of time to result in permanent tissue elongation. Superficial heat may be successfully applied to reduce joint stiffness and increases the elasticity of superficial joint capsular structures to facilitate exercise.[9]

The application of two therapeutic modalities at the same site is described as combination therapy. Ultrasonic therapy is frequently used with other modalities including hot packs, cold and electrical stimulation. This can be done because the ultrasonic transducer provides low resistance electrical contact with skin. Use of low intensity SWD (placebo) intermittent lumbar traction and exercise were found superior to that when they are used alone in chronic low back pain. The justification for the use of combination therapy is there will be beneficial effect of both modalities, making the therapy efficient also there may be an enhancing effect of one therapy upon the other making the combination more effective than each therapy alone.[10,11]

As per the available literature it has been found that electrical stimulation, ice, heat and stretching is effective in spasticity.

However there has been no study has been designed to investigate the effectiveness of combination therapy of electrical stimulation, ice, heat and stretching methods on spasticity in children with cerebral palsy.

HYPOTHESIS

Research question of the study:

- Whether the application of electrical stimulation Ice and stretching on spasticity in ankle plantar Flexors are effective?
- Whether the application of electrical stimulation, heat and stretching on spasticity in ankle plantar flexors is effective?

The null hypothesis is as follows:

- There will be no significant difference in the effectiveness of electrical stimulation, ice, stretching and electrical stimulation, heat, stretching on the spasticity of plantar flexors in spastic CP children.

The alternate hypothesis is as follows:

- There will be significant difference in the effectiveness of electrical simulation, ice and stretching versus electrical stimulation, heat and stretching on spasticity of plantar flexors in spastic CP children.

OBJECTIVES

The study purpose is to achieve the following objectives

- To evaluate the effect of electrical stimulation, ice, stretching on the spasticity of plantar flexors in children with spastic CP.
- To evaluate the effect of electrical stimulation, heat, stretching on the spasticity of plantar flexors in children with spastic CP.
- To compare the effect of electrical stimulation, ice, stretching and electrical stimulation, heat, stretching on the spasticity of plantar flexors in children with spastic CP.

REVIEW OF LITERATURE

A study conducted to find out the effect of NMES to gastronomies or to the gastronomies/ Tibialis anterior in gait and functional activity of children with spastic CP, had 14 children aged b/w 4-14 (4 with hemiplegia and 10 ē diplegia). The study has conducted through for four weeks phases. Treatment 1 had only gastronomies stimulation and treatment 2 had both Gastrocnemies/ Tibialis anterior stimulation. The children were divided into two groups and were received one treatment at the time were received one treatment at the time and all the children in two groups received both treatment 1&2 and was observed for the performance of the treatment. It was concluded that there an increase in ankle ROM. But it did not show whether gastronomies alone stimulated or gastronomies and tibialis anterior stimulated gave a higher result. But there was no dominance b/w gastronomies or gastronomies/tibialis anterior stimulation. Hence NMES is effective.[12] A prospective study was designed to determine whether the improvement in the hand function of children with spastic hemiplegic up is due to the effect of NMES, dynamic bracing, or a combination of the two methods. 24 patients met the inclusion criteria out of 31 b/w the age 3 to 18 years. Patients were randomly allocated to three groups by a closed envelop method, group 1 had two 30 minute sessions of NMES a day applied on the antagonist extensors without bracing, group 2 had two 30 min session of dynamic bracing per day and group 3 had two 30 min session of NMES and dynamic bracing every day. Treatment was continued for 6 months in all groups. They were evaluated before therapy, and 3 months after completion of the therapy. The combined treatment group showed significant improvement during the treatment in Melbourne score, grip strength and hand posture deformity. They found out the combined use of NMES with dynamic bracing is more effective than either treatment alone.[13] A study done to investigate and compare the effects of NMES on strength of tibialis anterior muscle and on active and passive range of motion of ankle joint dorsiflexion. Ten children

aged 7 to 15 years were chosen, 8 with CP and 2 with stroke. They were divided into two groups of five. Their children having physical therapy twice a week placed in group 1 and once a week in group 2. The patient underwent NMES sessions for 7 weeks. Each session had a length of 30 min. There were significant increase in muscle strength, gross motor function and passive ROM of ankle dorsiflexion in both groups and active dorsiflexion in the first group. The study suggested that NMES may be a useful the therapeutic tool, even when applied once a week.[14]

A randomized with in participant controlled trial was done to assess the effect of electrical stimulation in addition to passive stretching reduce spasticity and contractive more than passive stretching alone in children with cerebral palsy. The experimental intervention consisted of 30 min of electrical stimulation of the quadriceps 3 times per week and passive stretching of the hamstring 5 times per week. At the end of 4 weeks outcome measure were collected. The result showed impro0vement in MAS score and improvement in knee extension.[15] A systematic review of literature was conducted to determines whether static easy muscle stretching increases ankle dorsiflexion. Three metaanalysis were planned on the basis of duration of stretching interventions provided in each trial. Stretching time periods ≤ 15 min; ≥ 15 min; > 30 min. Trials were assessed in population who met the inclusion criteria. Ankle dorsiflexion was measured both activity and passively. The result showed easy muscle stretches provide a small but statistically significant increase in ankle dorsiflexion, particularly after 5-30 min of stretching.[16] A study was done to compare the effectiveness of NMES and cryotherapy on reduction of specificity and improvement of hand function in patients with spastic CP of the upper extremity. Only 20 out of 38 subjects met the inclusion criteria and divided into 2 groups. Group A treated with passive stretching and cryo therapy and group B with passive stretching and neuromuscular electrical stimulation. Passive

stretching for 60 sec with a duration of five min about. NMES for 30 min and ice for 20-30 min. Pre and post results showed that two modalities used to reduce specificity and enhance hand function and that none of the two modalities (NMES or cryotherapy) is superior to the other.[17]A study done to determine the effect of continuous cold air therapy in relieving specificity. Spastic paraplegia was induced by transaction of spiral cord in 46 rabbits. Cold air was applied to triceps surae muscle for 30 min at 3 different intramuscular temperatures (25, 30, 32.5° C). The electro physiological result showed to relieve spasticity with cold air therapy, the intramuscular temperature should he maintained at 30°C. The duration of treatment was between 30-60 min of the cold air therapy.[18] A study was conducted to assess the effect of cryotherapy on masseter spasticity on mouth opening. 24 patients with CP who had spasticity of tetra paresis type were included and ice was applied on the skin surface with sliding movement over the masseter bilaterally for one min. The result was the temporary reduction in spasticity.[19]

A study was done to empirically test the benefits of heat application on hamstring flexibility. 30 undergraduate student athletes were selected and participated in a 2 (treatment; heat V_s stretching) by 2 (counterbalanced order; heat first V_s stretching first). The results indicate that a 20 min moist heat application produced significantly more hamstring flexibility than 30 sec of static stretching, when goniometry is used as a tool to measure the range.[20] Study was done to determine the effect of superficial heat, deep heat, and active exercise. Warm up on the extensibility of the plantar flexor muscles. 97 subjects with limited dorsiflexion (≤ 20°) ROM ,were randomly assigned to 1 of 5 groups. Group 1 as control group with no stretching, group 2 with stretching protocol only, group 3 active heel raises to warm up the muscle prior to stretching, group 4 received superficial moist heat prior to stretching to the plantar flexion, group 5 with continuous US with a frequency of 1Hz and

intensity of 1.5w/cm^2 for 7 min. Dorsiflexion range of motion measurements were taken initially and after 2, 4, and 6 weeks. All experimental groups increased active and passive ROM. The group receiving US before performing the stretching protocol displayed the greatest in both AROM & PROM.[21]

A study to investigate whether appropriate inter-rater reliability of goniometric measurements could be achieved in children with cerebral palsy by a less experienced and more experienced therapist within the same session in a clinical setting. 25 children with spastic dipleigia underwent goniometry measurements of their right and/or left legs. Study showed that intrarater goniometric reliability can be established between a less experienced and more experienced therapist within same session.[22] A prospective, cross sectional, observational study done to determine the reliability of goniometric measurement in children with spastic cerebral palsy. Passive ROM of hip extension, abduction and external rotation, hip flexion with knee extended, and ankle dorsiflexion was measured using universal goniometry. Each child was assessed by three physical therapists once in each session on two different sessions a week apart. The result from this study encouraged the use of goniometric measurements in assessing children with spastic diplegic CP.[23]

A study was done to find out the reliability of MAS in assessment of plantar flexor muscle spasticity in 30 patients with traumatic brain injury. Results found that MAS is minimally adequate to support its continued use.[24] A study done to assess the intra and interater reliability of the AS and MAS, and to examine the reliability both scales in the lower extremities in children with spastic CP. 30 children participated in the study out of 38 children. The functional level of participants was classified according to the GMFCS. All measurements were taken in supine except hip external rotation in sitting position. In this

study reliability in hip flexors, adductors, internal rotators, hamstrings and gastronemius muscle groups were investigated. The result showed the interrater reliability scores of both AS and MAS ranged from moderate to good and inter rater reliability scores ranged considerably from poor to good.[25]

METHODOLOGY

60 spastic CP children of 5-15 years of age were taken from the some special schools and were served by using a questionnaire given to their parents for their inclusion in the study. Those who were eligible according to the questionnaire were then assessed for spasticity by using MAS children having grade I and II were included in the study.

These eligible participants were randomly divided in to two groups. Group A (electrical stimulation, ice, stretching) and group B (electrical stimulation, heat, stretching), the total number of eligible participants during the study was 60 (boys and girls). The parents of both the groups were explained about the respective procedure which their children would be undergoing for a period of 4 weeks and were made to sign consent form before their participation in the study.

Subjects:

Group A: consist of 30 subject 12 boys and 18 girls with the mean age of 9 years, participated in the study for electrical stimulation, ice and stretching.

Group B: consisted of 30 subjects 15 boys and 15 girls with the mean age of 9 years, participated in the study for electrical stimulation, heat, and stretching.

Inclusion criteria:

- Patients with age group 5-15.
- Patients with spastic cerebral palsy.
- Patients with spasticity grade I and III of MAS.

- Patients who are able to walk with assistance.

Exclusion criteria:

- Children with developed deformation of lower limb.
- Skin infection and fractures.
- Unco-opertative children.
- Patients with cognitive impairments.
- Patients undergone botulinum toxin injection any time in the past 6 months.

Source of data collection:

- Govt. wenlock district hospital Mangalore
- Shree Devi college of physiotherapy
- St.Agnes special school of disabled children
- Chetana child development centre

Tools used:

- Electrical stimulator
- Ice packs
- Moist heat pack
- Towels
- Vegetable oil
- Goniometry
- Exercise mat

Procedure:

All the patients were screened on the day one of the treatment according to the general neurological assessment format and MAS. The assessment was taken on the first day of session of 4 weeks before the treatment and on the last day of 4 weeks after the treatment session. The movement of dorsiflexion was checked in order to check the spasticity of plantar flexors. The treatment was given to both the group for 4 weeks. The selected patients were divided into 2 groups, Group A and Group B on a random basis.

- Group A –electrical stimulation followed by ice and then stretching.
- Group B- electrical stimulation followed by heat and then stretching

Electrical stimulation and stretching administered to both groups A and B.

Electrical stimulation of the patients affected leg was carried out by making the patients sit on the chair or in supine lying position. Proper measures were taken to reduce the resistance of the skin over the sight of stimulation (that is the anterior aspect of leg). After the skin preparation 2 surface electrodes will be placed over the anterior aspect of the leg and adjusted so that visible movements occur at the ankle joint towards dorsiflexion. The NMES of the dorsiflexors will be carried out for half an hour (30 minutes) thrice a week.

The type of current selected for the stimulation of the dorsiflexors will be characteristics of bi-phasic waveform with an amplitude ranging between 300 msec pulse width 0.3 msec frequency of 30 hz with an intermittent rest period set to 4sec after each pulse. The current amplitude and stimulus frequency will be adjusted to subjects comfort.

Stretching of the plantar flexors is done. Patient in supine position with the knee extended. Grasping the patient's heel with one hand, and forearm positioned along the plantar

surface of the foot. The anterior aspect of the tibia will be stabilized with other hand. Dorsiflexion of the talocrural joint of the ankle will be done by pulling the calcaneus in pressure in a superior direction just proximal to the heads of the metatarsals with forearm. Stretching was sustained for one minute and was given at a repetition of ten.

Ice given to Group A:

In supine position, the patient was given ice pack on the plantar flexors for 15 minutes. After the application of ice, stretching of plantar flexors was done.

Moist heat given to Group B:

In supine position, moist heat was kept on the plantar flexors for 10-15 minutes followed by which stretching was given.

FIGURE: 4.1 ELECTRICAL STIMULATION APPLICATION

FIGURE: 4.2 STRETCHING FOR THE PLANTAR FLEXORS

FIGURE: 4.3 MEASURING THE ANGLE OF DORSIFLEXION

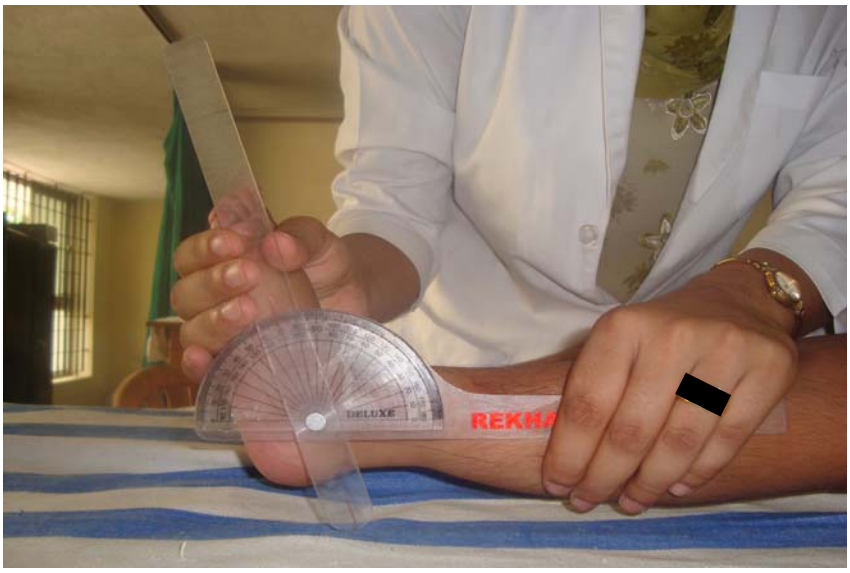


FIGURE: 4.4 MOIST PACK APPLICATION

FIGURE: 4.5 ICE PACK APPLICATION

FIGURE: 4.6 TOOLS USED

RESULTS

The study was conducted on 60 children with CP who were divided into two groups, ae A and group B consisting 30 subjects each. The children in group A were given ice and group B superficial heat. Electrical stimulation and stretching were given in both the groups in common.

As the outcome measure, MAS was used to assess the spasticity in plantar flexors in children with spastic CP. The assessment was done on 1st day and last day of the 4 weeks of treatment session. The grade were tabulated and subjected to statistical analysis. The analysis of data was done by t test and Mann-Whitney, Wilcoxon signed ranks test.

The ROM and MAS scores were subjected for statistical analysis to know the effectiveness of the technique.

AGE DISTRIBUTION

TABLE 5.1 AGE DISTRIBUTION

	Group	N	Minimum	Maximum	Mean	Std. Deviation	Median	t value	p value
Age	Group A	30	5	15	9.53	3.048	9.00	.568	.572
	Group B	30	5	15	9.10	2.857	9.00		NS

The Mean age in group A was 9.53±3.04, and that of in group B 9.10±2.85, t test shows that there is no significant difference between the groups with respect to age

GENDER DISTRIBUTION:

TABLE 5.2 GENDER DISTRIBUTION

		Group		
		Group A	Group B	Total
Sex	F	18	15	33
		60.0%	50.0%	55.0%
	M	12	15	27
		40.0%	50.0%	45.0%
Total		30	30	60
		100.0%	100.0%	100.0%

x2 = 0.606, p=0.436, NS

60% in group A and 50% in group B were females. 40% of group A and 50% of group B were males. Chisquare test shows that there is no significant difference between the groups with respect to genderwise ratio.

ROM

TABLE: 5.3
PRE AND POST ROM COMPARISON WITH IN THE GROUPS.

Group		N	Minimum	Maximum	Mean	Std. Deviation	Median	t value	p value
Group A	Pre ROM	30	6	14	9.47	2.145	9.00	8.532	p<0.001
	Post ROM	30	9	14	11.57	1.547	12.00		
Group B	Pre ROM	30	8	13	10.10	1.668	10.00	8.449	p<0.001
	Post ROM	30	9	13	11.17	1.341	11.00		

In group A mean ROM before the treatment was 9.47±2.1, and after the treatment 11.57±1.5, t test shows that there is significant increase in ROM after the treatment in group A

In group B mean ROM before the treatment was 10.10±1.6, and after the treatment 11.17±1.3, t test shows that there is significant increase in ROM after the treatment in group B.

ROM

TABLE: 5.4
PRE AND POST ROM COMPARISON BETWEEN THE GROUPS.

	Group	N	Minimum	Maximum	Mean	Std. Deviation	Median	t value	p value
Change in ROM	Group A	30	0	5	2.10	1.348	2.00	3.736	p<0.001
	Group B	30	0	2	1.07	.691	1.00		HS

It shows the comparison of effectiveness of ROM between group A and B. The t value 3.736 p<0.001 shows that there is highly significant difference between group A and group B. It shows that treatment given to group A is better than group B.

MAS

TABLE: 5.5
PRE AND POST MAS COMPARISON WITH IN THE GROUPS.

	Group	N	Minimum	Maximum	Mean	Std. Deviation	Median	Wilcoxon Signed Ranks Test	p value
Pre MAS	Group A	30	1	3	1.72	.583	1+	4.513	p<o.oo1
	Group B	30	1	3	1.88	.678	2.00		
Post MAS	Group A	30	0	2	.82	.482	1.00	4.110	p<o.oo1
	Group B	30	0	3	1.33	.562	1.00		

In group A mean MAS before the treatment was 1.72 ±0.583, and after the treatment 0.82 ±0.482 with p <0.001. It shows that there is significant increase in MAS after the treatment in group A

In group B mean MAS before the treatment was 1.88 ±0.678, and after the treatment 1.33 ±0.562 with p<0.001, test shows that there is significant increase in MAS after the treatment in group B.

MAS

TABLE: 5.6
PRE AND POST MAS COMPARISON BETWEEN THE GROUPS.

	Group	N	Minimum	Maximum	Mean	Std. Deviation	Median	Mann-Whitney Test	p value
Change in MAS	Group A	30	0	2	.90	.635	1.00	2.108	.0350
	Group B	30	0	2	.55	.461	.50		

It shows the comparison of effectiveness of MAS between group A and B. The test value 2.108 p<0.0350 shows that there is highly significant difference between group A and group B. It shows that treatment given to group A is better than group B.

FIGURE 5.2
AGE DISTRIBUTION

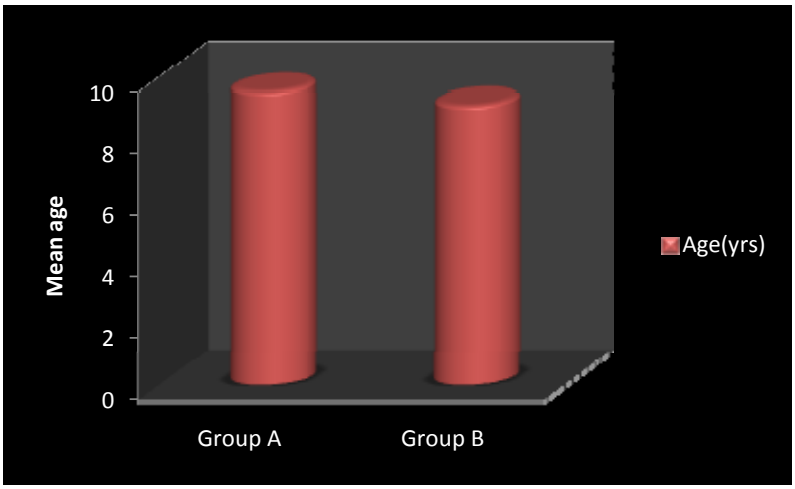


FIGURE 5.3
GENDER DISTRIBUTION

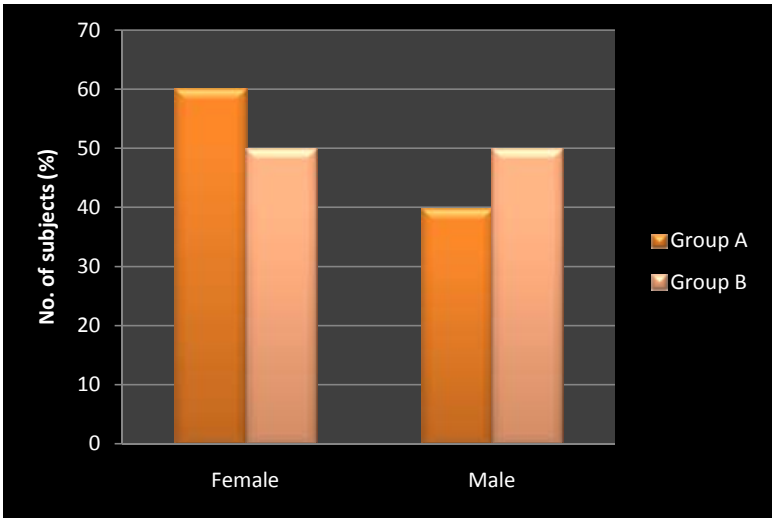


FIG. 5.4
COMPARISON BETWEEN MEAN OF PRE AND POST TREATMENT ROM.

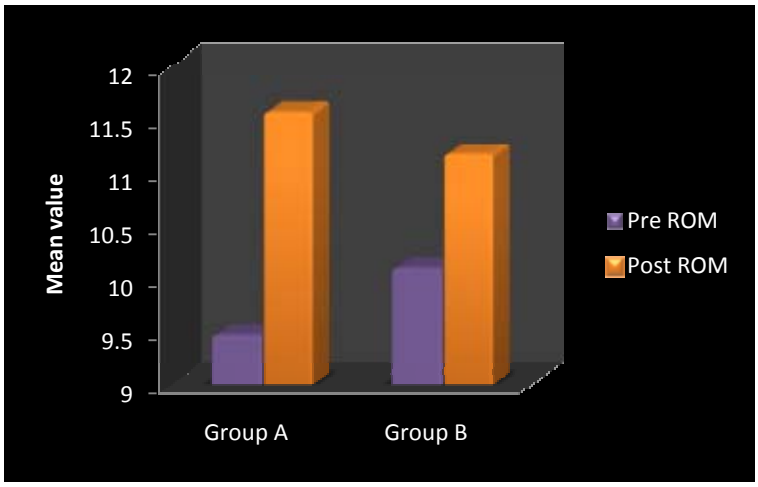
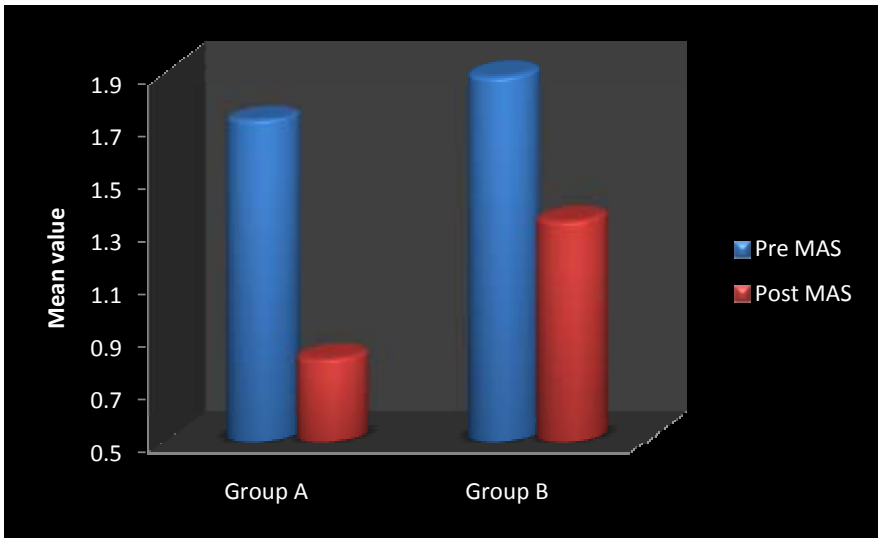


FIG. 5.5
COMPARISON BETWEEN MEAN OF PRE AND POST TREATMENT MAS.



DISCUSSION

Cerebral palsy is not a disease but is a rather a category of disabilities. The anatomic sites of involvement, degree of motor disability, associated dysfunction and causes are heterogenous.

The spastic types are the most common, followed by ataxic and athetoid types. Spasticity leads to number of problems in the cerebral palsy children such as leg muscle tightness, leading to shortening of the muscles and finally contractures.

There are various modalities used for reducing the spasticity. This study was done to examine the effect of electrical stimulation, ice, superficial heat and stretching on spasticity in children with spastic CP. The study was done on 60 CP children of spastic type of cerebral palsy. The subjects were divided in to two groups, consisting 30 subjects each. Group-A was treated with ice and Group B with superficial heat and both groups were treated with electrical stimulation in common for duration of 4 weeks. The results demonstrated that subjects treated with both the interventions were significant in improving ROM and reducing MAS score. However, statistically the group receiving electrical stimulation, ice, stretching showed very highly significant increasing improving ROM than the group receiving electrical stimulation, heat, stretching. The comparison of ROM between the groups was done by t test the improvement from pre treatment to 4 weeks post treatment showed t value 3.736 with $p<0.001$.

The comparison of MAS between the groups showed significant difference during pre and post MAS 4.513, 4.100 respectively with $p <0.001$

According to this study electrical stimulation, ice, stretching is effective in treating spastic cerebral palsy children this is concomitant by the previous studies.

Andrew J. Robinson, reports stimulation applied to the nerve leading to the antagonist muscle they inhibit the activity in the motor neurons to spastic muscle. The activation of such a reciprocal inhibition pathway may lead to an immediate reduction of the activity in the spastic muscle.

Cryothrerapy is highly effective in improving the ROM thus this may be due to decrease in the spasticity, preventing the chances of muscle tightness which leads to shortening and contractures. It is supported by the results of the study done by **Semonova KA, Bubnova VA**, et al, in which spastic diplegia, hemiplegia and hemikinetic forms of cerebral palsy were treated with cryo application on hands which caused the reduction in muscle tone. Cooling has a dual action as it inhibits the monosynaptic stretch reflexes and lowers the receptor sensitivity after it has been removed. Cooling can be used in different ways causes gradual switching off the activity of individual muscle.

According to **Thomas J Bevend Eerdt** Stretching, the process of elongation is one currently used technique in the management of spasticity. Stretching may change the muscle's visocoelastic structural and excitability properties. However many neural and non neural structures respond to stretch especially in spasticity. The aims of stretching in spasticity may be to normalize muscle tone, to maintain or increase soft tissue extensibility, to reduce pain and to improve function. Stretching as a treatment can vary in intensity of stretch which is the amount of tension that applied to the structure. Stretching can be static, dynamic, prolonged and ballistic stretching.

Slow sustained stretching forms the basis of spasticity treatment. It activates the muscle spindles [Ia and II endings] Golgi tendon organs [Ib endings] which are sensitive to

length changes. It inhibits or dampens muscle contraction and tone largely due to peripheral reflex.

According to **Kristinn Heinrichs** The neuromuscular effect of heat, in contrast to those of cold include increased nerve conduction velocity and decreased latency time for both sensory and motor nerves. Nerve conduction velocity increases 2m/sec for every 1^0c (1.8^0F) increase in temperature .Muscle relaxation occurs as a result of a decreased firing rate of type II muscle spindle afferents and gamma afferents and an increased firing rate of type II fibers of golgi tendon organs. These, in turn contribute to a decrease in firing of the alpha motornueron to the extrafusal muscle fiber, resulting in muscle relaxation. Heat lowers the stimulus threshold for muscle spindle activity. Superficial heat applied to muscle will alter its visocoelastic property leading to increased soft tissue extensibility, decreased joint stiffness, and increased ROM.

Thus overall analysis showed that group A receiving electrical stimulation, ice, stretching showed very significant increase in ROM and reduced MAS score, indicating reduction in spasticity than group B receiving electrical stimulation, heat, stretching.

Limitations of the study:

- The study was done on small size.
- The study was conducted in short duration and no follow up was done with the patients.
- The study did not include long term follow up. Thus the results cannot tell us about the effectiveness of both interventions in long term.

Scope of further study:

- Further study can be done check the combined effect of combination therapy.
- The study of same treatment approaches with the inclusion of control group can be done.
- A long term follow up study should be done to check the long term effect of the both intervention.
- The study on the same treatment approaches with large treatment group can be done.
- The study can be done on other group of spastic muscle.

CONCLUSION

The results of the study indicate that both the protocols electrical stimulation, ice, stretching and electrical stimulation, superficial heat, stretching are effective in reducing the spasticity.

The protocol of electrical stimulation, ice, stretching has a highly significant value than the protocol electrical stimulation, superficial heat, stretching in treating children with spasticity.

Further research is required to find out the efficacy of both interventions in different age groups and different type of CP patients.

SUMMARY

The study is aimed to compare the effect of combination therapy on spasticity in children with CP. CP children of 5-15 years of age were taken from the special schools screened by using a questionnaire given to their parents for their inclusion in the study and spasticity was assessed by using MAS scale. The eligible participants were randomly divided into two groups, Group-A and Group-B. Each group received the intervention according to their group for 4 weeks. Both are effective in reducing the spasticity as there was improvement in all the treatment parameters. Group A has a highly significant value than Group B over a period of 4 weeks.

Further research is recommended.

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ANNEXURE 10.1

CP QUESTIONNAIRE

1. NAME:

2. AGE:

3. SEX:

4. D.O.B:

5. ADDRESS:

6. Is the child is cooperative with others?
   Yes No
7. Is the child is able to communicate with other people?
   Yes No
8. Is the child able to follow what is told to him?
   Yes No
9. Is there presence of altered sensation in lower limb?
   Yes No
10. Is the child sensitive to cold, heat, electrical stimulation?
    Yes No
11. Any case of fracture in the lower limb?
    Yes No
12. Any case of dislocation in the lower limb?
    Yes No
13. Is there presence of deformity in the lower limb?
    Yes No
14. Is there presence of any abnormal movement?
    Yes No

ANNEXURE 10.2

ASSESSMENT CHART

Name:
Age:
Gender:
Address:
Weight:
Height:
Date of assessment:
Chief complaints:

History:

I) History of present illness
II) Prenatal history
III) Perinatal history
IV) Postnatal history
V) Medical history
    Medical records
    i) Precaution
    ii) Patient status
    iii) medication
VI) Genetic history

On Observation:

Attitude of child:
In supine
In prone
In sitting
In standing
Posture:
Skin:
Built:
Debilitating deformity:
Visual contractures:
External appliances:
Mode of ambulation:
Involuntary movements:

Balance in:
Sitting
Standing
Walking
Midline activities:
Transfer of objects:

On Palpation:
Vital parameters:
HR:
RR:
Tenderness
Odema
Temperature
Tightness

On Examination:
i) Gross examination
Recognition:
Social smile:
Vision:
Speech:
Hearing:
Cognition (attention, co-operation):

ii) Sensory examination
Fine touch:
Crude touch:
Temperature:
Pain:
Pressure:
Steriognosis:
Proprioception:

iii) Developmental milestones
Neck control:
Rolling over:
Sitting:
Crawling:
Kneeling:
Standing:
Walking:

iv) Tone
Hypertonia:
Hypotonia:

v) Motor examination;

Range of motion:

	AROM			PROM
Hip joint				
Flexion				
Extension				
Abduction				
Adduction				
Medial rotation				
Lateral rotation				
Knee joint				
Flexion				
Extension				
Ankle joint				
Plantar flexion				
Dorsi flexion				
Inversion				
Eversion				

a) Limb Length:

Lower Limb	Right	Left
True length		
Apparent length		

b) Limb girth:

Lower limb	Right	Left
Thigh		
Calf		

c) Reflexes

Deep (stretch)

Lower limb	Right	Left
Knee		
Ankle		

Superficial

	Present	Absent
Plantar		

e) Any associated problems:
f) Gait:
g) ADL's
Feeding
Dressing
Toileting
Hygiene
Self maintenance

Provisional diagnosis

ANNEXURE 10.3

MODIFIED ASHWORTH SCALE

Grade	Tone
0	No increase in muscle tone
1	Slight increase in muscle tone, manifested by a catch and release or minimal resistance at the end range of movement (ROM) when the affected part 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	Slight increase in muscle tone throughout most of the ROM, but affected part easily moved.
3	Considerable increase in muscle tone throughout most of the ROM
4	Affected parts rigid in flexion or extension.

ANNEXURE 10.4

RECORDING FORM

1) NAME:
2) AGE:
3) SEX:

ROM		MAS	
PRE	POST	PRE	POST

ANNEXURE 10.5

CONSET FORM

I, …… , F/O……. hereby agree to provide my fullest conset and co-operation to Ms Jyothi Pinto as a subject for her dissertation work entitled as **"A comparative study to assess the effectiveness of cryotherapy with that of the superficial heat in combination with electrical and stretching in reducing spasticity of plantar flexors in children with spastic cerebral palsy."** The researcher has explained me about the study, risk and benefits of participation and has answered my all questions and queries regarding the study to my satisfaction. I am also aware that I can withdraw from the study anytime I wish to do so.

Place:
Date:

Signature of the parent:

Signature of the researcher:

GROUP A: ELECTRICAL STIMULATION, ICE, STRETCHING

SL.NO	AGE/SEX	ROM		MAS	
		PRE	POST	PRE	POST
1	5/F	8	10	3	1
2	7/M	9	12	2	1
3	6/F	7	10	1+	0
4	8/F	10	11	2	1
5	9/F	9	10	1	1
6	11/M	8	11	1+	1
7	15/F	12	13	2	2
8	5/M	13	13	3	1+
9	7/M	9	11	1	1
10	9/M	8	9	2	1+
11	7/F	11	13	1	0
12	9/F	14	14	1+	1
13	8/F	8	9	1+	1
14	9/M	6	10	2	2
15	14/M	7	9	1	1
16	15\M	8	10	3	1+
17	12/M	9	12	1+	1
18	7/F	10	11	2	1+
19	8/M	11	13	1+	1
20	12/F	13	13	2	1
21	11/F	12	14	2	2
22	14/F	8	10	1	1
23	11/F	6	9	2	1
24	6/F	9	11	2	1+
25	10/F	8	10	1+	1
26	6/F	9	10	1	0
27	7/M	11	12	2	1+
28	14/F	10	11	1+	1
29	12/M	13	13	1+	1
30	12/F	8	9	1	1

GROUP B: SUPERFICIAL HEAT, STRETCHING AND ELECTRICAL STIMULATION

SL.NO	AGE/SEX	ROM		MAS	
		PRE	POST	PRE	POST
1	6/F	8	9	2	1+
2	11/M	9	11	3	2
3	5/F	8	10	2	1
4	9/F	11	11	1	1
5	5/M	10	11	1+	1
6	7/M	12	13	1	1
7	15/F	11	12	3	2
8	9/F	13	13	2	1+
9	9/F	9	10	2	1+
10	7/F	8	10	1	1
11	9/M	9	10	1+	1
12	7/M	10	12	1+	0
13	14/M	11	12	2	1
14	8/F	8	9	1	1
15	9/M	9	11	3	2
16	10/M	9	10	2	1+
17	12/F	10	11	3	3
18	7/F	9	11	1+	1
19	8/M	12	12	2	2
20	13/M	11	12	1+	1
21	9/F	13	13	2	1
22	12/F	12	13	3	1+
23	8/M	11	12	2	2
24	12/F	8	9	1+	1
25	6/M	9	10	2	1+
26	5/M	9	11	1	1
27	12/M	12	12	3	2
28	14/F	13	13	1	1
29	6/M	8	9	2	1
30	9/F	11	13	1+	1