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Effect of Translatoric Joint Mobilization on Knee Joint Range of Motion in Spastic Diplegic Children: A Clinical Trial

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ABSTRACT

Background

Cerebral palsy (CP) is a non progressive but often changing, motor impairements due lesions of the Central Nervous System (CNS). Knee flexion contractures are a common secondary complication experienced by spastic children. The purpose of this study was to examine the effects of Translatoric Joint Mobilization (TJM) program to address bilateral knee flexion contractures in diplegic Cerebral Palsy (CP) children. The use of translatoric joint mobilization along with other common Physical Therapy (PT) interventions in this population remains controversial.

Objectives

1. To establish the effectiveness of Kaltenborn's Translatoric Joint Mobilizationin improving knee joint range of motion in spastic diplegia and
2. To see improvement in functional mobility as measured by GMFM (88)scoring.

Methodology

Examination of function and impairment of 30 children with spastic diplegia included Popliteal Angle, Gross Motor Function Measure (GMFM)1 total score and dimension D and E of GMFM. Physiotherapy intervention consisted of Kaltenborn's Translatoric knee joint mobilization and NDT for group A (experimental), and for group B (control) only NDT. The treatment was given for 6 days a week for 3 weeks.

Results

After 3 weeks of mobilization, there was significant improvement in poplitealangle and GMFM scoring seen in group A as compared to group B.

Conclusion

Translatoric joint mobilization proved to be a safe and effective addition to standard PT interventions for

knee flexion contractures for the children in this study. The functional impact of improved knee extension was seen as an improvement in GMFM scores.

Keywords: joint mobilization; knee flexion contractures; cerebral palsy; NDT;GMFM; Popliteal angle

INTRODUCTION

Cerebral Palsy (CP) is defined as a non progressive insult to a developing or immature Central Nervous System (CNS).[1]Cerebral palsy (CP) is a common neurodevelopmental disability. It is considered as one of the 3 most common lifelong developmental disabilities; others being autism and mental retardation which causes considerable hardship to the affected individual and their families.[2]

A widely quoted definition was proposed by Bax, which describes CP as "A group of disorders of movement and posture due to a defect or a lesion of theimmature brain".[3] The most recent and most widely accepted definition proposed at the 1990 international meeting on epidemiology held in Brioni, Yugoslavia definedCP as an "umbrella term covering a group of non-progressive, but often changing, motor impairment syndrome secondary to lesion(s) or abnormalities of the brain arising in early stages of development".[4] This term is commonly used name for a group of conditions characterized by motor dysfunction due to non progressive brain damage early in life. It is stated as a static encephalopathy in which even though the primary lesion, anomaly or injury is static, the clinical pattern of presentation may change with time due to growth and developmental plasticity and maturation of the central nervous system (CNS).[5] The way the damage is apparent, changes as the child grows and develops and thus the therapist is faced with a complex situation ofpathological symptoms within the context of a developing child.

Although the lesion itself is non-progressive, the resulting impairments, disabilities and handicaps can be progressive. Cerebral palsy was defined in the literature by Dr. William Little, who described it in detail in a paper presented to the London Obstetrical Society in 1861. The disability was first called “Little’s disease” because of his extensive descriptions and early attempts to determine the causes of what he called spastic infantile paralysis. Motor disorder(s) caused due to Cerebral Palsy are accompanied by "disturbances of sensation, cognition communication, perception, and/or behavior, and/or by a Seizure disorder" [6].

The current over all worldwide prevalence in children ages 3 to 10 years is 2.4per 1000 children[3] with incidence being 2 to 2.5 per 1000 live births.[7] The male female ratio is 2.42:1, with males being more severely disabled.[5]Etiology of CP is very diverse and multi-factorial. The causes are congenital, genetic, inflammatory, infectious, anoxic, traumatic and metabolic with the mostimportant risk factors being prematurity and low birth weight. Injury to the developing brain may be during the prenatal (75% to 80%), perinatal (congenital CP) and postnatal period i.e. up to 2 years of life (acquired CP).[4, 7]

Prenatal risk factors –

Umbilical arterial base deficit less than 13 mmol/L or pH greater than 7, infants who have major or multiple congenital or metabolic abnormalities, central nervous system or systemic infection, infants with signs of intrauterine growth restriction, early imaging evidence of long-standing neurologic abnormalities, such as porencephaly, extensive chorioamnionitis, microcephaly at birth (head circumference less than the third centile), major prenatal placental abruption, reduced fetal heart rate variability from the onset of labor, congenital coagulation disorders in the child, presence of other major prenatal risk factors for cerebral palsy (such as preterm birth at less than 34 weeks' gestation, multiple pregnancy or an

autoimmune disease), presence of major postnatal risk factors for cerebral palsy (such as postnatal encephalitis, prolonged hypotension, or hypoxia due to severe respiratory disease), a sibling with cerebral palsy, especially of the same type.

Perinatal risk factors–

Prematurity and intrauterine growth rate restriction: 40% to 50%, Birth asphyxia or birth trauma: 25% to 30%, Neonatal stroke: 5% to 10%, Toxoplasmosis, rubella, cytomegalovirus, herpes simplex, other infections, 5% to 10% Chromosomal abnormalities: 5% to 10% chorioanmionitis, intrauterine infections, teratogenic exposure, placental complications, multiple births, maternal conditions (mental retardation, seizures, hyperthyroidism) that may lead to foetal deprivation of blood supply. Birth process itself may cause CP, others include maternal bleeding, problem with the placenta, maternal infections and obstetrical complications such as prolonged labour, use of forceps, prolapsed cord, abnormal presentation of foetus.

Postnatal Factors-

responsible for motor control and muscle tone of legs, injury results in spastic diplegic CP.[11] Spastic diplegia is the commonest in India i.e. 30% to 40%. In a retrospective study based on the clinical profile spastic diplegia was found to be most common in North India i.e. 54%.[6]

Classification based on their functional limitation divides CP into mild, moderate and sever.

Cerebral Palsy is divided into four major classifications to describe the different movement impairments[6]. These classifications reflect the area of brain damaged. The four major classifications are:

1. Spastic (damage to the corticospinal tracts that descend from the motor cortex)
2. Athetoid /Dyskinetic (putamen of the basal ganglia)
3. Ataxic (cerebellum) and
4. Mixed.

Spastic Cerebral palsy is by far the most common type, occurring in 70% to 80% of all cases. A person with spastic cerebral palsy develops tight muscles in some parts of the body that are unable to relax. Affected joints become stiff and difficult to move.

Usually, a person has problems controlling movements, poor coordination and balance, and difficulty talking and eating.

There are four types of spastic Cerebral Palsy, grouped according to topographical classification they are[12].

1. **Monoplegia-**
   Only one arm or leg is affected.
2. **Hemiplegia-**
   - One half of the body is involved
   - They usually have hemi anopsia
   - Most of the hemiplegics learn to walk
3. **Diplegia-**
   Both the upper limbs and lower limbs are affected but lower limbs are moreaffected.
4. **Triplegia-**
   Both the lower limbs and one upper limb are affected.
5. **Quadriplegia-**
   - Both upper limbs and lower limbs are equally affected

- May have problem in sucking and swallowing
- Have a weak shill cry
- Usually have a opisthotonic posture

The non spastic forms of cerebral palsy include dyskinetic cerebral palsy(subdivided into athetoid and dystonic forms) and ataxic cerebral palsy.

Dyskinetic cerebral palsy-
It is associated with muscle tone that fluctuates between being loose and tight. In some cases, rapid and jerky or uncontrolled slow continuous movements occur involuntarily. These movements most often affect the face and neck, hands, feet,arms, legs, and sometimes the torso.

Athetoid (hyperkinetic) cerebral palsy-
This includes relaxed and limp muscles during sleep, with some involuntary jerking (chorea) or writhing (athetosis). If the face and mouth muscles are affected, problems may develop related to unusual facial expressions, drooling, speaking, and choking when sucking, drinking, and eating.

Dystonic cerebral palsy-
There is a state of abnormal tonicity in any of the tissues. They usually have dyskinetic movements due to disordered tonicity of muscle.

Ataxic cerebral palsy -
It is the rarest type of cerebral palsy and involves the entire body. Abnormal body movements affect the trunk, hands, arms, and legs. Ataxic cerebral palsy causes problems with: balance, precise movement e.g, the person may reach too far or too close to touch objects and may also have poor hand control (intention tremor). The person may not be able to button clothes, write, or use scissors, often only one hand isable to reach for an object; the other hand may shake with attempts to move it.

Person with ataxic Cerebral Palsy may walk with the feet unusually far apart. Some children have symptoms of more than one type of cerebral palsy. For example, spastic legs (symptoms of spastic diplegic cerebral palsy) and problems with facial muscle control (symptoms of dyskinetic cerebral palsy) may both develop. Total bodycerebral palsy affects the entire body to some degree. Complications of cerebral palsy and other medical problems are more likely to develop when the entire body is involved rather than isolated parts. Total body cerebral palsy may include: Spastic quadriplegic Cerebral Palsy, Dyskinetic Cerebral Palsy and Ataxic Cerebral Palsy.

Of all the different types, spastic diplegia is the most common. In spastic diplegic children, the lower extremities are more affected than the upper extremities. Spastic diplegic type of Cerebral palsy is caused by irreversible damage usually to the motor cortex area of the brain ensuring that the potential for recovery does not exist. Most victims of this type of Cerebral Palsy do not suffer from intellectual disabilities. Spastic diplegic type of Cerebral Palsy, in common with all other forms of Cerebral Palsy is usually the result of naturally occurring phenomena that cause what is commonly known as a birth defect. Because of the lesions that cause diplegia, there is a marked discrepancy between control of the upper body and that

of the lower body. The lower extremities show limited synergies for use in function namely:

- Poor ability to grade co-contraction and reciprocal inhibition with excessive co- contraction often used.
- Poor ability to terminate sustained muscle activity
- Abnormal timing of muscle contractions
- The alignment and control of sitting and standing can be very abnormal, so that gravity and external forces quickly deform bones, help shorten muscles and misalign joints. The hips are almost always pulled into flexion, adduction and internal rotation. This narrows the base of support in all positions, which makes learning upright trunk control more difficult.

The medial hamstrings are the muscles that seem most responsible for the hip internal rotation which are also knee flexors. Therefore, it is not uncommon to see children in some degree of knee flexion in all their postures. Most children with spastic diplegia do eventually walk.

Early in 18th century, Cerebral Palsy was treated using primarily an orthopedic approach. Surgery that was used for children with polio was used for children with cerebral palsy. In 1940's new approaches for treatment emerged in the world of therapy. New treatment ideas by the Bobaths, Margaret Rood, and others also came into being. These approaches attempted to understand the problems in sensory and motor control and the role that development played in attaining skills, as well as the secondary orthopedic impairments caused by Cerebral Palsy[6].

There are different treatment approaches used in cerebral palsy, such as Physical therapy, Occupational therapy (OT), Speech and language therapy,Surgeries, Botulinum Toxin injections etc. Different types of surgeries like tenototmy,selective rhizotomy, derotation osteotomy, loosening tight muscles and releasing fixed joints have been carried out. In Speech and language therapy, the therapists work with children on communication skills. Occupational therapist usually works with children on better ways to use their arms, hands, and upper body. They may teach children better or easier ways to write, draw, cut with scissors, brush their teeth, dress and feed themselves, or control their wheelchair. Occupational therapists also help children find the right special equipment to make some everyday jobs a little easier. Orthotic management: in conjunction with other interventions orthoses continue to play an important role in management of Cerebral Palsy. Orthoses are designed with one of the two primary aims-either to affect the body structure or to assist function or both.

The other aims of lower limb orthotic management for Cerebral Palsy are-

- To correct and/or prevent deformity,
- To provide a base of support,
- To facilitate training in skills and to improve the efficiency of gait.

The job of the physical therapist is to help a child's mobility to develop and to teach and practice exercises designed to avoid contractures, bone deformity andunwanted movement.
A physical therapy (PT) program may consist of one or more of the following—

Stretching-
Stretching of muscles is performed by moving the arms or legs in a way that produces a slow and steady

pull on the muscles opposite to the direction of itscontraction, to keep them loose and prevent contractures

Strengthening-
Strengthening exercises work specific muscle groups to enable them tosupport the body better and increase function.

Positioning-
The body is placed in a specific position to attain long stretches. Some positions help to reduce unwanted tone. Positioning can be done in a variety of ways, including bracing, abduction pillows, and knee immobilizers, wheelchair inserts, sitting recommendations and handling techniques. Physical therapists usually focus onactivities involving the legs, such as walking, braces, using crutches and rehabilitation after a surgery.

Recreational therapists-
They help kids with Cerebral Palsy have fun. They work with children on sports skills or other leisure activities. In recreational therapy kids may work on dance, swimming or horseback riding. They may also work on art or horticulture (growing and taking care of plants) or almost any other hobby they are interested in.

Neuro Developmental Treatment (NDT)-
It is one of the most common technique used the treatment approach of cerebral palsy. Neuro Developmental Treatment (NDT) is a therapeutic approach usedin working with people who have central nervous system insult that create difficulties in controlling movement. Berta and Karel Bobath of London, England, a therapist/physician team, developed the NDT approach in the 1940's. Originallyworking with children with cerebral palsy and adults with stroke, they believed in treating the many complex problems with control of movement caused by central nervous system insult. Persons having minimal to severe motor difficulties can benefitfrom the NDT approach[6].

There are currently four broad statements of belief in Neuro Developmental Treatment theory. A person's ability to function is a result of their many body systems working together smoothly and efficiently. Additionally, parts of those systems such as a muscle, sensory process or respiratory pattern assist with the movements in the person's unique dailyenvironment. Those who use the Neuro Developmental Treatment approach base efficient movement on the study of motor control, motor development and motor learning. There are consistent ways that people learn and lose motor skills throughout their lives. This can be studied and compared to people who have movement problemsthat interfere with learning motor skills. Treatment begins with an assessment of the person's functional performance. It then focuses on building the person's abilities while addressing the movement problems. The goal of Neuro Developmental Treatment is to help the person achieve functional skills by addressing movement difficulties. Along with these approaches, management by MANUAL THERAPY (MT) plays an important role in improving flexibility and control of the affected limbs and joints. Manipulation is defined by Dorland's Pocket Medical Dictionary as 'skilful or dexterous treatment by the hands. The word 'therapy' is derived from the Greek therapeuein, meaning 'to take care of, 'therapon' meaning' an attendant', a living person.' Therapeutic' signifies the curative and healing potential of one person towards

another. **Manual therapy**[13] is therefore the use of hands in a curative and healing manner, and can be defined as the use of manipulation with therapeutic intent.The aim of manual therapy is to influence the reparative and healing capacity of the individual. Manual therapy has been stated to be the most effective when directed at "mechanical joint dysfunction in which there is restriction of accessory motion due to capsular or ligamentous tightness or adherence."[14]

Assessment, therefore, includes testing of the accessory movements particular to that joint to determine the presence of pain or resistance, or both, to movement. Resistance to movement is typically produced by either capsuloligamentous tightness (stiffness) or muscle activity (spasm).[15] The resistance produced by stiffness is described as being consistent in strength and position in the range of movement, whereas that produced by muscle spasm varies in response to the speed and method ofthe examination movement.[15]

Manual therapies commonly used in the treatment of spastic diplegia areStretching, Myofacial release, joint mobilization. etc.

Joint mobilization-

It is a manual technique given to restore the range of motion in a joint and/ or to increase joint mobility. The mechanism by which joint mobilization or manipulation "work" are not known, although many hypotheses have been proposed as the knowledge of articular and soft tissue neurology, biomechanics, and pathology has expanded[16]. Articular neurology has provided much of the background to understanding the effect of passive movement in modulating pain. The type I, II and III mechanoreceptors located in joint capsules and ligaments are stimulated by active and passive joint movement[17]. Passive mobilization techniques may be a means of activating type I and II mechanoreceptors thereby reducing pain and reflex muscle spasm.[18] Type III mechanoreceptors (found only in capsules and ligaments of peripheral joints) may be activated by strong stretch or thrust techniques and may have an inhibitory effect on surrounding muscle. However, there are certain absolute contraindications given by Hertling and Kesler[14] for mobilization. They are Bacterial infection Neoplasm and Recent fracture. There are a number of treatment approaches given by leading proponents of manual therapy—Cyriax, Maitland, Kaltenborn, Mulligan etc.

Mulligan:

In this the patient performs a sustained active movement along with the therapist and takes the joint to its full range of motion. But, children with spasticity cannot do this. Thus, this method cannot be applied in cerebral palsy children.Another type of joint mobilization technique is Kaltenborn's

TRANSLATORIC JOINT MOBILIZATION (TJM).

Kaltenborn's Translatoric Mobilization:

Translatoric Joint Mobilization was developed by Freddy M. Kaltenborn in 1954[19]. He introduced the concept of translatoric linear bone movements, in the form of linear translatoric traction and gliding in relation to a treatment plane, to further reduce joint compression forces. The mobilization is based on the Convex-Concave rule[19]. It is based on the relationship between normal bone rotations and the gliding component of the corresponding joint movements (roll-gliding). This approach is useful for joints with very small ranges of movement, when severe pain limits movement, or for novice practioners. The

therapist moves a bone with convex joint surface opposite to the direction of restricted movement in the distal aspect of the bone, and a concave joint surface in the same direction as the direction of the restricted bone movement. In every joint there are positions in which looseness or slack in the capsule andligaments allows small, precise movements of joint play to occur as a consequence of internal and external (e.g., passive) movement forces on the body. These joint play movements are an accessory movement not under voluntary control, and are essential to the easy, painless performance of active movement.

The purpose of joint mobilization in restricted joints involves the restorationof joint play to normalize the roll-gliding that is essential to active movement.

There are 3 different grades of Translatoric mobilization.

Grade I

It helps to relieve pain. It is an extremely small traction force which produces no appreciable increase in joint separation. Grade I traction nullifies the compressive forces acting on the joint.

Grade II

Relieves pain and increases joint movement. Grade II movement first takes up the slack in the tissues surrounding the joint and then tightens the tissues. In the slack zone at the beginning of the Grade II range there is very little resistance to passive movement. Approaching the end of the Grade II range a marked resistance is felt called the first stop.

Grade III

It helps in maintaining and increasing joint movement. This movement is applied after the slack has been taken up and all tissues become taut. At this point Grade III stretching force applied over a sufficient period of time can safely stretch tissues crossing the jointDue to the scarce literature of this relatively new approach, information available pertaining to its effects on joint range of motion is limited.

Joint mobilization in children with spasticity:

Capsular tightness may be the primary findings that indicate treatment by jointmobilization in persons with musculoskeletal disabilities but it is not the whole concern for the child with spastic cerebral palsy. When the associated findings of muscle shortening, hyperactive stretch reflexes, skeletal deviations, and muscle weakness are considered, the use of mobilization to enhance or restore joint mobilityis not a straightforward[16].

The predisposition of immature growth plates to injury-particularly during growth spurts-suggests the need to be cautious when using joint mobilization inchildren. There are few CNS disorders in which joint mobilization is contraindicated. They are:

In the child with pure athetoid and ataxic forms of cerebral palsy, joints tendto be hypermobile.[20] Hypermobility of the spine in children with athetoid cerebral palsy may lead to cervical instability. Another common neurodevelopmental disability in which joints are hypermobile secondary to lax ligaments is Down syndrome.

Other, less common, neurodevelopmental disabilities, such as Pader-Willi syndrome, may be characterized by general hypotonia and hypermobile joints.[21] For these children as well joint mobilization is contraindicated.

Very few studies have been done on the use of joint mobilization in cerebral palsy children. Hence this study is intended to determine the effect of Translatoric type of joint mobilization on knee joint range of motion (ROM) in spastic diplegic children.

OBJECTIVES

3. To establish the effectiveness of Kaltenborn's Translatoric Joint Mobilizationin improving knee joint range of motion in spastic diplegia and
4. To see improvement in functional mobility as measured by GMFM (88)scoring.

HYPOTHESIS

Null hypothesis: There will be no improvement in knee joint range of motionfollowing TJM in children with spastic diplegia.

Alternative hypothesis: There will be improvement in knee joint range of motionfollowing TJM in children with spastic diplegia.

REVIEW OF LITERATURE

A review article written by **Freeman MA**, **Pinskerova V** describes the anatomy of the articular surfaces and their movement in the normal tibio-femoral joint. With the aid of computer-imaging, the movements were related to an anatomy- based co-ordinate system to avoid kinematic cross-talk. The shapes of the articular surfaces were reported. The movements of the condyles were described from hyperextension to full passive flexion. Medially the condyle hardly moves antero- posteriorly from 0 degrees to 120 degrees but the contact area transfers from an anterior pair of tibio-femoral surfaces at 10 degrees to a posterior pair at about 30 degrees. Thus because of the shapes of the bones, the medial contact area moves backwards with flexion to 30 degrees but the condyle does not. Laterally the femoral condyle and the contact area move posteriorly but to a variable extent in the mid- range causing tibial internal rotation to occur with flexion around a medial axis. From 120 degrees to full flexion both condyles roll back onto the posterior horn so that the tibio-femoral joint subluxes.[22]

Ziv et al. In their experimental studies in mice who have a genetic spasticity of lower limbs suggested that muscle growth occurs at the musculotendinous junction which they termed the 'growth plate' of the muscle. New sarcomeres are added in response to the stretching of growth by the bone. When the muscle is spastic it cannot keep pace with the bone growth by adding sarcomeres. The muscle is not shortened, but merely fails to grow as rapidly as the bone. In spastic mice muscle growth was reduced by 45 per cent, resulting in contractures.[23]

A study was done by **Pierce SR**, **Barbe MF**, **Barr AE**, **Shewokis PA**, **Lauer RT** to examine the frequency of co-contraction and the relationship between reflex activity and co-contraction during passive movements of the knee joint in children with cerebral palsy. Twenty children with cerebral palsy participated in this study. One set of ten continuous passive movements from 90 degrees of knee flexion to 25 degrees of knee flexion and from 25 degrees of knee flexion to 90 degrees of knee flexion was completed at 15 degrees /s, 90 degrees /s, and 180 degrees /s. The mean percentage of the range of motion of each movement and mean percentage of the number of movements which exhibited electromyographic activity of the vastus lateralis, medial hamstrings, and co-contraction were calculated for each set of movements. Thus it was concluded that co-contraction commonly occurs during passive movements of the knee in children with cerebral palsy. The presence of co- contraction may influence measurements of

spasticity which use passive movements to assess spasticity.[24]

A study was done by **H.Kuno** to define a geometrical hexagon model for representing the sagittal range of motion (ROM) of hip and knee joint. The effect of both monoarticular and biarticular muscles on joint mobility in children with spastic cerebral palsy (SCP) were analyzed by this geometrical method. The ROM in normal children indicated age dependent reduction of maximal hip flexion and shortening of hamstring and rectus femoris muscles. A number of SCP children showed greater reduction of both hip flexion and extension and shortening of hamstring and rectus femoris muscles, whereas the ROM of knee joint was similar to that in normalchildren. The deteriorated hip joint mobility seems to be associated with shortening ofmuscle due to their intrinsic spasticity. The impaired ROM was more noticeable in SCP non-ambulatory child than in independent ambulator. Thus, more extended rangeand frequent opportunity of joint motion may play an important role in improving the joint mobility in this patient group.[25]

A study was done by **Susan R Harris, Lundgren BD** to find the indications and precautions for use of joint mobilization in children with CNS disorders. He concluded that although there is limited research support for the use of mobilization inadults who have musculoskeletal disorders, there have been no published studies examining its efficacy for its use in children with CNS disorders. Hence, physical therapists need to be cautious in the use of mobilization.[26]

A study was conducted by **Ben-Sorek s, Davis CM** to collect survey data regarding the education of physical therapists in mobilization techniques, to examine quantitative changes in entry-level curricula from 1970 to 1986, and to examine basic and continuing education opportunities and determine whether physical therapists are making use of these opportunities. According to the results, it seemed to indicate that mobilization techniques are becoming more widely used by physical therapists to treat joint dysfunction.[27]

A study was conducted by **Kluding, Patrica et al** to determine if joint mobilizations improve ankle mobility and sit-to-stand function in 5 subjects with hemiplegia following stroke (7 to 11 months post-CVA). Ankle range of motion, ankle kinematics during sit-to-stand, and time to complete the task were measured during every session.The intervention consisted of joint mobilizations to increase ankle dorsiflexion. All subjects demonstrated a statistically significant increase in passive ankle ROM. No consistent trends were found for ankle excursion during sit- to-stand or time to perform the task. Analysis of ankle kinematics revealed varying patterns of change for the individual subjects. [28]

A review article was written by **Carol Giddings Cochrane** to explore the use of mobilization in the treatment of capsular dysfunction of the shoulder joint in children with central nervous system dysfunction. A review of functional anatomy and of normal and abnormal development of the shoulder was presented. Basic mobilization principles and application of joint mobilization techniques to children with long-standing shoulder hypomobility and spasticity were discussed. He suggested joint mobilization as an appropriate form of treatment for some of the joint restrictions that occur in children with CP.[29]

A study was done by **RP Di Fabio** to establish objective criteria for judging the validity of manual therapy research, to identify and discuss the results of those trials that were determined to be valid demonstrations of treatment efficacy or valid demonstrations of non useful therapy and to determine whether patients who benefit from manual therapy have common characteristics. There was a paucity of valid explanatory research in all areas and a particular absence of controlled trials involvingmanual therapy applied to the peripheral joints.[30]

End range passive movements may reduce peripheral input to the CNS through adaptation of the encapsulated endings of joint nerves to the mechanical stimulus of prolonged stretch of the particular soft

tissue[r]. This is supported by a studydone by **P. Grigg and B. J. Greenspan** on response of primate joint afferent neurons to mechanical stimulation of knee joint. Responses of afferents were studied in relation to both passive manipulations of the knee and active contractions of quadriceps, semi-membranosus, and gastrocnemius muscles.The results support the hypothesis that joint afferents function as capsullar stretch receptors, responding to those mechanical events which result in loading of the capsule.[31]

In a case study done by **Kondratek Melodie et al** to find the effectiveness of translatoric joint mobilization on a 15 year old patient with bilateral knee flexion contractures, it was found that there was noticeable increase in knee joint range of motion and the child's gait pattern changed from shuffling her feet to consistent heel strike bilaterally.[32]

A study was done by **Creighton D et al** to determine the effectiveness of Tibio-Femoral joint traction mobilization (Translatoric) in improving knee flexion while not significantly increasing pain in 14 patients with limited passive knee flexion. Outcome measures included passive knee flexion measured with a goniometer, knee pain measured with a 10-cm VAS, and function measured with Patellar Disability Index. There was an improvement in knee joint passive flexion motion, reduced pain at the end range available passive knee flexion and improved functional status.[33]

A study conducted by **Brandt, G. Sole, M. Krause, M. Nel** to find an evidence-based review on the validity of the Kaltenborn rule as applied to the glenohumeral joint. Evidence-based systematic review was conducted to summarize and interpret the evidence on the direction of the accessory gliding movement of the head of the humerus (HOH) on the glenoid during physiological shoulder movement. Five hundred and eighty-one citations were screened. Data from 30 studies were summarized in five evidence tables with good inter-extracter agreement. The quality of the clinical trials rated a mean score of 51.27% according to the Physiotherapy Evidence Database scale (inter-rater agreement: κ=-0.6111κ=-0.6111). Evidence, however, indicated that not only the passive, but also the active and control subsystems of the shoulder may need to be considered when determining the directionof the translational gliding of the HOH. The indirect method, using Kaltenborn's convex–concave rule as applied to the glenohumeral joint, may therefore need to be reconsidered.[34]

In a study done by **Adams, Marjorie A et al** to ascertain if gait characteristicsof a group of children with various types of cerebral palsy would more closely approximate normal after being treated during a neurodevelopmental treatment (NDT)course, significant improvements were found in the whole group for stride and step length, foot angle, and velocity. The children with spastic diplegia seemed to benefit most from treatment showing gains in stride and step length as well as velocity and base of support measures.This study revealed efficacy of neurodevelopmental treatment in improving gait in children with cerebral palsy over a period of sixweeks.[35]

A study done by **Clopton N, Dutton J, Featherston T, Grigsby A, et.al** on children with hypertonia found that interrater reliability of the Modified Ashworth Scale may be lower than desired for clinical use for muscles other than hamstrings and elbow flexors, and intrarater reliability may also be lower than desired for muscles other than the hamstrings.[36]

A prospective cross-sectional observational study was planned to determinethe reliability of the Ashworth Scale (AS) and the Modified Ashworth Scale (MAS) inchildren with spastic cerebral palsy (CP) by **Mutlu A.** The study included 38 children with spastic diplegic CP. The mean age for the children was 52.9 months (SD: 19.6) ranging from 18 to 108 months. The functional levels of children were classified

according to the Gross Motor Function Classification System. Spasticity in hip flexors, adductors, internal rotators, hamstrings, gastrocnemius were assessed by AS and MAS. According to ICC scores, interrater reliability of AS and MAS varied from moderate to good. ICC scores of AS were between 0.54 and 0.78 and MAS were between 0.61–0.87. Test-retest results of AS and MAS varied from poor to good. ICC values were between 0.31 and 0.82 for AS and between 0.36 and 0.83 for MAS. It

was concluded that these scales are not very reliable and assessments of spasticity using these scales should be therefore interpreted with great caution.[37]

A study was done by **Thompson, Neil Stuart M.D., F.R.C.S. (Trand Orth); Baker et al** on relevance of the popliteal angle to hamstring length in cerebral palsy with crouch gait. The maximum hamstring length of each subject during gait was determined by three-dimensional modelling of their lower limbs. The modified popliteal angle measurements of the most repeatable observer demonstrated an inverse relationship between modified popliteal angle and maximum hamstring length($p < 0.01$) and muscle excursion ($p < 0.01$).[38]

A study was done by **Katz K**, **Rosenthal A**, **Yosipovitch Z** to find the normal ranges of popliteal angle. Measurement of the popliteal angle is used to assess hamstring contracture in children with cerebral palsy. The popliteal angle in 482 normal children, 1-10 years of age, was measured. Using a360 degrees goniometer, the popliteal angle was measured with the hip held at 90 degrees flexion to indicate hamstring muscle tightness. Between the ages of 1 and 3 years, the mean angle was 6 degrees (range, 0-15). At age 4, the angle rose to 17 degrees in girls and 27 degrees in boys (range, 5-45). At greater than or equal to 5 years the mean angle was 26 degrees with little change (range, 0-50). A popliteal angle of greater than 50 degrees in the above age groups indicates abnormal hamstring tightness.[39]

Russell et al states that 88-item Gross Motor Function Measure (GMFM) is a criterion-referenced observational measure that was developed and validated to assesschildren with CP. Evidence of the reliability of measurements obtained with GMFM has been established for children with CP, also evidence of construct validity of the measures's capacity to detect change in motor function over time has been

established. The GMFM scores of children who were young (<3years of age) and had mild CP, changed more than the scores of children who were older (>6years of age) and had severe CP.[40]

A study was done by **Josenby AL**, **Jarnlo GB**, **Gummesson C**, **Nordmark E**to study the longitudinal construct validity of 3 scoring options: the 88-item GMFM (GMFM-88) total, the GMFM-88 goal total, and the 66-item GMFM (GMFM-66). A clinical measurement design was used in this study. Forty-one children with CPdiplegia who were undergoing selective dorsal rhizotomy (SDR) were monitored withthe GMFM for 5 years. Two subgroups for gross motor function before surgery was created according to the Gross Motor Function Classification System (GMFCS) GMFCS levels I to III and GMFCS levels IV and V. This study included results obtained before SDR and at 6, 12, and 18 months and 3 and 5 years after SDR. The effect size (ES) and the standardized response mean (SRM) were calculated. The GMFM-88 total and goal total scores showed large changes in ES values (range=0.8- 0.9) and SRM values (range=0.9-1.3) at 12 months postoperatively, whereas theGMFM-66 scores showed lower ES values (range=0.3-0.4) and SRM values (range=0.7-0.8) for both subgroups. Later postoperatively, larger values for longitudinal construct validity were found. The ES and SRM values generally were lower for the GMFM-66 scores than for the GMFM-88 total and goal total scores.[41]

A study was done by **McWhirk LB**, **Glanzman AM** to investigate whether appropriate inter-rater

reliability of goniometric measurement could be achieved in children with cerebral palsy (CP) by a less-experienced and more-experienced therapist within the same session in a clinic setting. Twenty-five children with spastic CP underwent goniometric measurement of their right and/or left legs (46 legs). Two pediatric physical therapists, with 10 and one year of experience measured five lower- extremity motions within the same session. Standard positioning, stabilization, and bony landmarks were reviewed before the study. Interclass correlation coefficients ranged from 0.582 (hip extension) to 0.929 (popliteal angle). Data was in agreement with the literature on the reliability of range of motion measurements in children with CP. Hence, a result, it was concluded that inter-rater goniometric reliability can be established between a less-experienced and more-experienced therapist within the same session.[42]

A study was done by **Gajdosik RL, Bohannon RW** to review the related literature on the reliability and validity of goniometric measurements of the extremities and concluded that clinicians should adopt standardized methods of testing and should interpret and report goniometric results as ROM measurements only, not as measurements of factors that may affect ROM.[43]

A study was done by **Mutlu A, Livanelioglu A, Gunel MK** to determine the reliability of goniometric measurements in children with diplegic cerebral palsy and found that results from this study encourage the use of goniometric measurements in assessing children with spastic diplegic CP.[44]

A research study was done by **Gogia PP, Braatz JH, Rose SJ, Norton BJ** to assess the intertester reliability and validity of goniometric measurements at the knee, and found that the goniometric measurements of the knee joint are both reliable and valid.[45]

METHODOLOGY

Study Design:

The study design was a clinical trial.

(Ethical clearance was obtained from the Institutional Ethical Committee, J. N. Medical College, Belgaum, before commencement of the study)

Sample Size:

The sample size for this study was thirty (30) subjects/ participants in the age group of 18 months- 15 years.

Study Sample:

Sample included both male and female children with a clinical diagnosis of spastic diplegic Cerebral Palsy.

Sampling Design:

Convenience sampling with systematic allocation

Group A: Conventional therapy and Kaltenborn's Translatoric Joint Mobilization(n==15)

Group B: Conventional therapy (n=15)

Participants:

Male and female children with clinical diagnosis of spastic diplegia who were referred to the Pediatric Physiotherapy department and whose parents were willing to let their children participate in the study

Source of Data:

Children reporting to Pediatric Physiotherapy OPD or referrals from Department of Pediatrics and Child Development Clinic (CDC), K.L.E.S' Dr. Prabhakar Kore Hospital and Medical Research Center and children reporting toK.L.E.S' B.M Kankanwadi Aurvedic Hospital Belgaum-590 010, Karnataka, India

during the study period from April 1st to December 31st 2008.

Inclusion Criteria:

1. Both male and female participants with clinical diagnosis of spastic diplegia.
2. Age: 18 months to 15 years
3. Modified Ashworth scale for spasticity, grade 3 and 4 for hamstring group ofmuscles.
4. Parents willing to let their children participate in this study.

Exclusion Criteria:

1. Children who have been given Botulinum Toxin in preceding 6 months.
2. Any orthopaedic surgeries performed in preceding 6 months.
3. Children with fixed bony deformities of lower limbs.
4. Children of mental age less than 2 years (who can't follow instructions).
5. Children with visual, hearing and cognitive deficits.

Materials

Materials used for this study consisted of the following**,**

1. Consent Form – An informed consent form signed by the parents/ guardian ofthe children to allow their inclusion in the study.
2. Data collection sheet.
3. Treatment table/ plinth
4. Sand bag
5. Stopwatch
6. Toys

Apparatus and Equipments: Following apparatus were used for the study.

1. Weighing Machine:

A standard bathroom weighing machine with 1 kg increment was used to measure the weight of each participant in kilograms.

2. Goniometer:

Universal goniometer was used to measure knee joint range of motion.

3. Gross Motor Functional Measure (88) scale

GMFM is a criterion-based observational measure consisting of 88 itemsdistributed into 5 dimensions: A, B, C, D and E. It samples motor skills that are typical of a normal developing milestone.

MAIN OUTCOME MEASURES:

1. Popliteal Angle:

For measurement of popliteal angle the starting position of the child is in supine lying. The examination hip and knee was put in 90 degrees of flexion each andthen the knee was passively extended until a moderate resistance was met in the muscle. Popliteal angle was detrmined by measuring the angle that the tibia subtends with the extended line of the femur. The opposite leg was kept in extended on the couch. The angle was noted in degrees with goniometer. An angle less than 125 degrees suggests significant hamstring tightness.

2. GMFM (88)

It contains 88 items that are explicitly defined, scored using a four-point scale and organized into five dimensional categories called GMFM dimensions (A-E): GMFM-A (Lying and Rolling); GMFM-B (Sitting); GMFM-C (Crawling and Kneeling); GMFM-D (Standing); GMFM-E (Walking, Running and Jumping). The GMFM-total score is derived by averaging all GMFM (A-E) percentage scores.

INTERVENTIONS:

The interventions included in the present study were TJM and NDT.

Translatoric Joint Mobilization:

It is a type of joint mobilization proposed by Freddy M Kaltenborn. It workson the principle of convex-concave rule. The mobilization given to the knee joint in the present study consisted of 6 types of glides. Grades used were II to III

Neuro Developmental Treatment:

Developed by Berta Bobbath, NDT is a therapeutic approach used in working with people who have central nervous system insult. The techniques used in this present study are- activities in sitting, sit to stand and activities in standing All the participants with spastic diplegia who reported to the physiotherapy outpatient department were screened clinically. By considering inclusion and exclusion criteria, they were requested to participate in the study. Those willing to participate in the study were given brief idea about the nature of the study and the intervention. Passive knee extension range motion was measured bilaterally by Universal Goniometer and functional outcome was scored using GMFM as an interventional outcome measure.

Then participants were allocated systematically into 2 groups:
Group A: Translatoric Joint Mobilization + Neuro Developmental Treatment
Group B: Neuro Developmental Treatment

Both groups recieved the treatment for six times / week for three weeks.

PROCEDURE:

Parents of the participants were requested to sign a consent form prior to allocation. Participants who met the inclusion criteria were allotted to either group A or group B. Group A which was the experimental group included participants undergoing Kaltenborn's Translatoric Joint Mobilization (TJM) along with conventional therapy. Group B participants which were the control group were only given conventional therapy. The demographic data was obtained. Pre-test data included Popliteal angle and GMFM scores, post-test data included popliteal angle at
the end of every week for 3 weeks and GMFM scoring at the end of 3rd week or 21st day. Most of the children with diplegia are able to sit or stand independently andhave problem with ambulation.

Group A: Experimental group (TJM and Conventional Physiotherapy):

Instructions and details about the procedure administered, was explained to parents of the participants prior to the start of the study. Before administering TJM, the participants were given Conventional

Physiotherapy which included Neuro Developmental Treatment (NDT). The treatment techniques given in NDT were- sit tostand, activities in standing, and walking for 30 minutes. A rest interval was given for 15 minutes followed by 15 minutes of TJM. The participant began by lying supine followed by measurement of the Popliteal angle bilaterally, with a Universal Plastic Goniometer, later followed by mobilization.

The mobilization techniques include:

1. **Knee Traction:**

The patient lies prone with knee in its resting position. Participant's distalthigh is fixed against the treatment surface. The therapist grips the patient's ankle joint with both hands and Grade III (movement applied after the slack has been taken up) traction movement is applied in line with the lower leg.

2. **Knee Anterior Glide:**

The patient lies prone with knees near the edge of the table. His thigh is fixed against the table. The patient's lower leg is held against the therapist's body with bothhands. The medial side is gripped with left hand proximal to the ankle and right hand distal to the knee. Right hypothenar eminence is placed on the tibia.

Grade III anterior glide movement is applied to the proximal medial tibia by the therapist leaning through right forearm and bending his knees.

3. **Knee Lateral Glide:**

The patient is in side-lying position with the lateral side of the leg to be treatedon the treatment surface & knee in its resting position. His distal thigh is fixed on the table with the sand bag proximal to the joint space. The therapist holds the patient's lower leg from the medial side with both his hands, with left hand proximal to the ankle and right hand distal to the knee with his hypothenar eminence on the medial tibia just distal to the joint space. Grade II and III lateral glide movement is applied.

4. **Knee Medial Glide:**

The patient lies in side lying with lateral side of his leg on the surface andknee in resting position. The therapist places his hand proximal to the patient's ankle and fixes it against the table. With his other hand he grips around the medial side of the patient's thigh just proximal to the knee. A relative medial glide is applied to the tibia with grade I and III lateral glide to the femur.

5. **Patella Medial and Lateral Glide:**

The patient lies supine with knee in resting position. The therapist grips the thigh just above the patella from the anterior side. With the heel of his hand the therapist grips the lateral edge of the patella. Grade II or III medial glide movement is applied.

Same technique is applied for the lateral glide with grade II or III lateral glide.

The dosage of mobilization given per technique was 30-40 seconds with the entire treatment session extending up to 10-15 minutes. Intervention was given for 6 days/ week for 3 weeks.

Group B Control group (conventional physiotherapy):

The parents of participants in this group received prior instruction regarding the procedure. The therapy administered included Neuro Developmental Treatment(as described above). Conventional therapy was given for a total of 30 minutes, 6 days/ week for 3 weeks.

The outcome measures measured pre and post treatment were subjected to statistical analysis.

RESULTS

The results of this study were analyzed in terms of improvement in Popliteal angle which was measured using universal goniometer and improvement in Function using GMFM scale. Dimension D and E of GMFM were calculated separately. Intra and inter group differences were compared so as to evaluate the effectiveness of Kaltenborn's joint mobilization under consideration in the present study.

Statistical Analysis

Statistical analysis for the present study was done manually as well as usingthe statistics software graph MedCalc 8.2 version so as to verify the results obtained. For this purpose data was entered into an excel spread sheet, tabulated and subjected to statistical analysis. Various statistical measures such as mean, standard deviation and tests of significance such as paired and Unpaired't' tests were utilized for this purpose for all the available scores in all the participants. Comparison of the pre interventional and post interventional outcome measures and within the groups was done by using one student paired t test while Unpaired 't' test was used to measure thedifference and improvement seen between the two groups. Improvement scores were measured in terms of difference between pre intervention and post intervention valuesfor both the groups. These scores were subjected for statistical analysis using unpaired't test. Probability values less than 0.05 were considered statistically significant and probability values less than 0.0001 were considered highly significant.

DEMOGRAPHIC PROFILE

Age Distribution

The average age of the participants in group A subjects was 6.60±3.25 years (min=3yr and max=14yr) and group B subjects was 5.53±2.90 years (min=2 and max=12) years. There was no significant difference between the mean ages of the subjects in the two groups. (p= 0.3580)

Sex Distribution

Total 30 children participated in the study. Out of which 16 were males and 14were females. There were 9 males and 6 females in group A and 7 males and 8 females in group B.

OUTCOME MEASUREMENTS:

1.Popliteal Angle (PA)

In group A, the pre interventional and post interventional PA scores for right knee were 50.20 ± 20.06 and 61.13±20.61 respectively whereas pre interventional andpost interventional PA scores for left knee were 48.87±16.37 and 58.67±17.64 respectively. In group B, the pre interventional and post interventional PA scores for right knee were 61.60±12.76 and 62.60±12.80 respectively whereas pre interventionaland post interventional PA scores for left knee were 61.20±12.42 and 62.20±11.92 respectively. Within group changes in the Popliteal Angle for both right and left knee revealed statistically significant increase in knee joint range of motion (extension)post interventionaly for the two groups. This was done using paired't' test. The between group analysis for Popliteal Angle showed statistically significant difference between group A versus group B ($p<0.0001$) for right knee whereas for left knee there was no significant difference found ($p=0.5256$). This was done by using unpaired't' test.

Average improvement score seen in right knee was 10.93±5.61 (min=2; max=20) and1.00±0.76 (min=0.00; max=2.00) for group A and B respectively whereas average improvement score seen in left knee was 9.80±5.49 (min=2; max=20) and 1.00±1.07 (min=0.00; max=4.00) for group A and B

respectively.Statistically significant difference in improvement between group A and B was found for both right (p=<0.0001) and left (p=<0.0001) knee using unpaired‘t’ test.

2.Dimension D and E of Gross Motor Functional Measurement (GMFM)

In group A, the pre interventional and post interventional scores for dimensionD were 3.08 ± 1.26 and 5.93±1.75 respectively whereas pre interventional and post interventional scores for dimension E were 10.47±8.09 and 13.80±8.32 respectively. In group B, the pre interventional and post interventional scores for dimension D were 1.47±1.36 and 2.27±1.67 respectively whereas pre interventional and post interventional scores for left E were 7.53±3.11 and 8.07±3.33 respectively. Within group changes for dimensions D and E revealed statistically significant increase in thevalues post interventionaly for the two groups. This was done using paired‘t’ test. Thebetween group analysis for dimensions D and E showed statistically significant difference between group A versus group B (p<0.0001). This was done by using unpaired’ test.

Average improvement score seen in dimension D was 21.13±0.92 (min=0.00; max=3) and0.80±0.86 (min=0.00; max=3.00) for group A and B respectively whereas average improvement score seen in dimension E was 3.20±1.26 (min=2.00; max=6.00) and 0.53±0.52 (min=0.00; max=1.00) for group A and B respectively.

Statistically significant difference in improvement of dimension D (p=0.000315) and E (p=<0.0001) between group A and B was found using unpaired‘t’ test.

i) Gross Motor Function Measure (GMFM) total score

In the present study pre-interventional values of GMFM total score were 66.74 ± 11.25 and 57.63 ±10.80 in group A and B respectively whereas post-interventional values for GMFM total score were 70.61 ± 11.15 and 59.10 ±10.59 in group A and B respectively. Within group changes in GMFM revealed statistically significant increase in the values post interventionaly for the two groups. This was done using paired‘t’ test. The between group analysis for GMFM showed statistically significant difference between group A versus group B (p<0.0001). This was done by using unpaired’ test.

Average improvement seen in GMFM total score was 3.86±2.82 (min=0.98; max=10.913) and 1.47±0.92 (min=0.02; max=2.97) for group A and B respectively. Statistically significant difference in improvement between group A and B was found (p=0.004119) using unpaired‘t’ test.

DISCUSSION

Spastic diplegia is the most prevalent form of CP. Spastic diplegic children were found to be ideal for inclusion in the present study as they have obviousspasticity in the lower limbs and none in the upper limbs except for the fine motor co- ordinations defects. Their prognosis for life function is generally good.[46] Other forms of CP were not included in the study because children with Quadriplegia have total body involvement and hence usually get wheelchair bound. In case of athetoid, ataxic and hypotonic forms of CP, joint mobilization has been contraindicated due to unstable and lax joints.[47]

The application/ use of joint mobilization in children with spastic diplegia is controversial and hardly few studies have been done. It has been stated that until moreresearch on joint mobilization in children with CNS disorders been conducted, this therapy should be used cautiously.

Studies were done on mobilization for children with CNS disorders. Carol Giddings Cochrane wrote a

review article to explore the use of mobilization in the treatment of capsular dysfunction of the shoulder joint in children with central nervous system dysfunction. A review of functional anatomy and of normal and abnormal development of the shoulder was presented. Basic mobilization principles and application of joint mobilization techniques to children with long-standing shoulder hypomobility and spasticity were discussed. He suggested joint mobilizationas an appropriate form of treatment for some of the joint restrictions that occur in children with CP.[48] In a study conducted by Kondratek on knee joint ROM in an adolescent CP child, TJM was found to be effective.

In the present study, statistically significant improvement in popliteal angles were found in both right and left knee in both the groups- experimental (TJM+NDT) and control (Only NDT).

Prolonged spasticity in the muscle of a growing child often leads to shorteningof the muscle (myostatic contracture). When this is unrelieved, joint capsule contractures get superimposed on myostatic contractures.[1]Thus, inspite of availability of some hamstring extensibility, freedom of knee extension gets limited due to shortening of periarticular structure.

Kaltenborn proposed that the mobilization should be given in a direction basedon analysis of the restriction in range of motion and the articular surface anatomy.[49] The primary indication for use has been mechanical joint dysfunction in which thereis restriction of joint play leading to pain or limitation of active physiological movement. Pathological changes that occur at the microscopic level in the joint are decrease in intracellular water content, resulting in a decrease in the distance between the fibers constituting the joint capsule, which inturn results in an increase in fiber cross-link formation that produces adhesions[49] leading to ROM and functional limitations. TJM is thought to reverse these changes by promoting movement betweencapsular fibers. This is believed to result in an increase in interstitial water contentand inter fiber distance. Mobilization helps to break intra capsular fibrofatty adhesionsand increase in length of capsular fibers. Thus it can be speculated that increase in popliteal angle was due to correction of arthrokinematic restrictions leading to increased ROM.

Knee flexion and extension are more important and major determinants of activities like coming up to stand, standing, walking, jumping and running.[50] Knee
flexion contractures increase the energy requirement and limitations for these activities.

A study was conducted by Kondratek to find the effectiveness of TJM on a 15 year old patient with bilateral knee flexion contractures.[32]

The results of the present study are comparable to the above study in terms of improved knee ROM following TJM. The functional outcome was measured byKondratek in terms of improvement in gait pattern i.e., the child's gait changed from shuffling her feet to bilateral heel strike. However, in the present study, GMFM scoring was included as a measure of functional outcome and not the gait pattern. Thus the functional outcome cannot be compared to the above study.

In the present study, GMFM total score was improved statistically in both the groups. Two children showed drastic increase in GMFM total score. One child showed increase from 54% to 65 % and the other showed increase from 68% to 76%. One child showed least improvement of 54.25% to 55.23%. This could be due to severe contractures (PA<20). Significant changes were also seen in individual scores of dimensions D and E. Dimensions D and E consisted of items like standing, walking, running etc.

Thus in the present study, increase in knee extension ROM has probably helped in reducing the limitations caused due to a flexed knee and improvement couldbe seen in the results. However, no clinically or functionally evident improvement could be seen.

To conclude, TJM along with conventional therapy can be used as an effectiveintervention for improving knee joint restrictions and improve gross motor functions in children with spastic diplegic CP.

However, until more research on joint mobilization in children with CNSdisorders been conducted, this therapy should be used cautiously.

LIMITATIONS:

- The sample size taken in the study was small due to limited study period.
- Few dimensions of GMFM included items like hopping, jumping, climbing steps up and down with alternating feet etc which cannot be suitably administered to children between ages 2-3 years.

RECOMMENDATIONS:

- To use randomized controlled trial as study design to get more generalizableanswers.
- Treatment of hip and ankle along with knee joint should also be considered.
- Further studies should be carried out with aim to overcome the limitations of thepresent study.
- Use of moist therapy before mobilization can give better results by making themuscles pliable.

CONCLUSION

A complex multifaceted disorder such as CP most likely requires a multifaceted approach to rehabilitation. Factors that can be remedied need to beidentified and more effective strategies for producing positive functional outcomes must be developed. The present study focused on knee flexion contractures as one component of the motor dysfunction seen in children with CP. Translatoric joint mobilization proved to be a safe and effective addition to standard PT interventionsfor knee flexion contractures for the children in this study. The functional impact of improved knee extension was the improvement in GMFM scores.

SUMMARY

This research study was done to find the effectiveness of Kaltenborn's Translatoric Joint Mobilization along with Neuro Developmental Therapy on knee joint ROM in children with spastic diplegic CP. Thirty participants were allocated systematically into two groups i.e. group A and B, each group comprising of 15 participants. Group A was treated with TJM and NDT and group B received only NDT. Outcome was measured in terms of Popliteal angle measured by universal goniometer and functional outcome was measured using GMFM scale with D and E dimensions scored separately and total score. Paired and unpaired't' test were donefor statistical analysis. In present study with-in group and between group comparisonsshowed statistical improvement in terms of knee ROM and functional outcome for both the groups.

Hence, based on the results, it can be concluded that the present study focused on knee flexion contractures as one component of the motor dysfunction seen in children with CP. Translatoric joint mobilization proved to be a safe and effective addition to standard PT interventions for knee flexion contractures in spastic diplegic children. The functional impact of improved knee extension was the improvement in GMFM scores.

TABLES

Table 1: Age Distribution

	Experimental	Control	p value	Inference
Mean	6.60±3.25	5.53±2.90	0.3508	NS
Minimum	3.00	2.00		
Maximum	14.00	12.00		

Table 2: Right Popliteal angle (Mean)

	Experimental	Control	p value	Inference
Pre-test	50.20±20.06	61.60±12.76	0.0739	NS
Post-test	61.13±20.61	62.60±12.80	0.0276	S
p value	<0.0001	<0.0001		
Inference	S	S		

Table 3: Improvement of right knee

	Experimental	Control	p value	Inference
Mean	10.93±5.61	1.00±0.76	<0.0001	S
Minimum	2.00	0.00		
Maximum	20.00	2.00		

Table 4: Left Popliteal angle (Mean)

	Experimental	Control	p value	Inference
Pre-test	48.87±16.37	61.20±12.42	0.8165	NS
Post-test	58.67±17.64	62.20±11.92	0.5256	NS
p value	<0.0001	0.001385		
Inference	S	S		

Table 5: Improvement of left knee

	Experimental	Control	p value	Inference
Mean	9.80±5.49	1.00±1.07	<0.0001	S
Minimum	2.00	0.00		
Maximum	20.00	4.00		

Table 6: GMFM dimension D (mean)

	Experimental	Control	p value	Inference
Pre-test	3.08±1.26	1.47±1.36	<0.0001	S
Post-test	5.93±1.75	2.27±1.67	<0.0001	S
p value	<0.0001	0.00146		
Inference	S	S		

Table 7: Improvement for D

	Experimental	Control	p value	Inference
Mean	2.13±0.92	0.80±0.86	0.000315	S
Minimum	0.00	0.00		
Maximum	3.00	3.00		

Table 8: GMFM dimension E (Mean)

	Experimental	Control	p value	Inference
Pre-test	10.47±8.09	7.53±3.11	0.200538	NS
Post-test	13.80±8.32	8.07±3.33	0.019469	S
p value	<0.0001	0.000658		
Inference	S	S		

Table 9: Improvement for E

	Experimental	Control	p value	Inference
Mean	3.20±1.26	0.53±0.52	<0.0001	S
Minimum	2.00	0.00		
Maximum	6.00	1.00		

Table 10: Mean GMFM total score

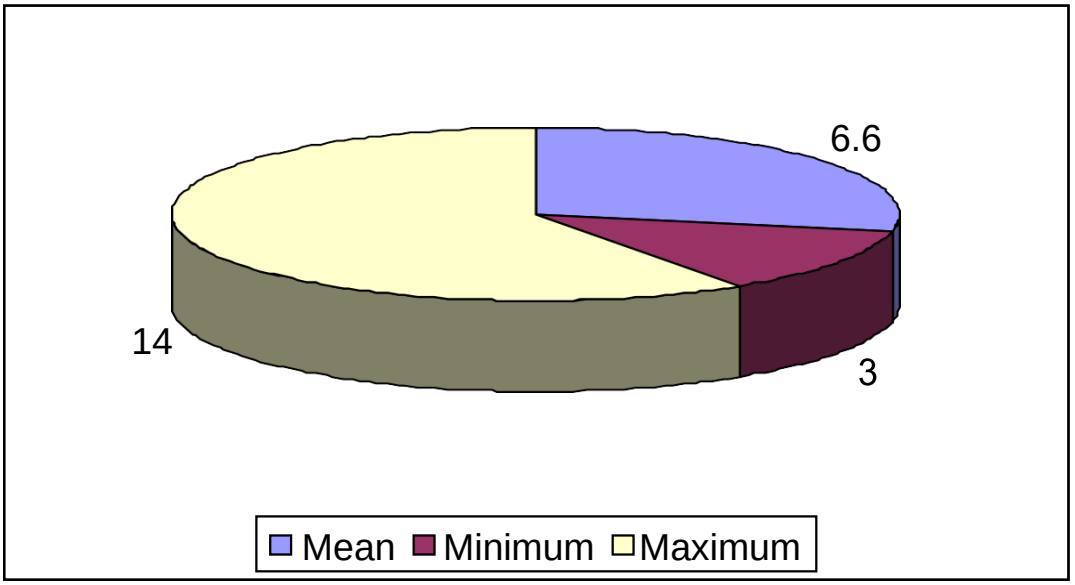
	Experimental	Control	p value	Inference
Pre-test	66.74±11.25	57.63±10.80	0.031563	S
Post-test	70.61±11.15	59.10±10.59	0.0072012	S
p value	<0.0001	<0.0001		
Inference	S	S		

Table 11: Improvement for GMFM

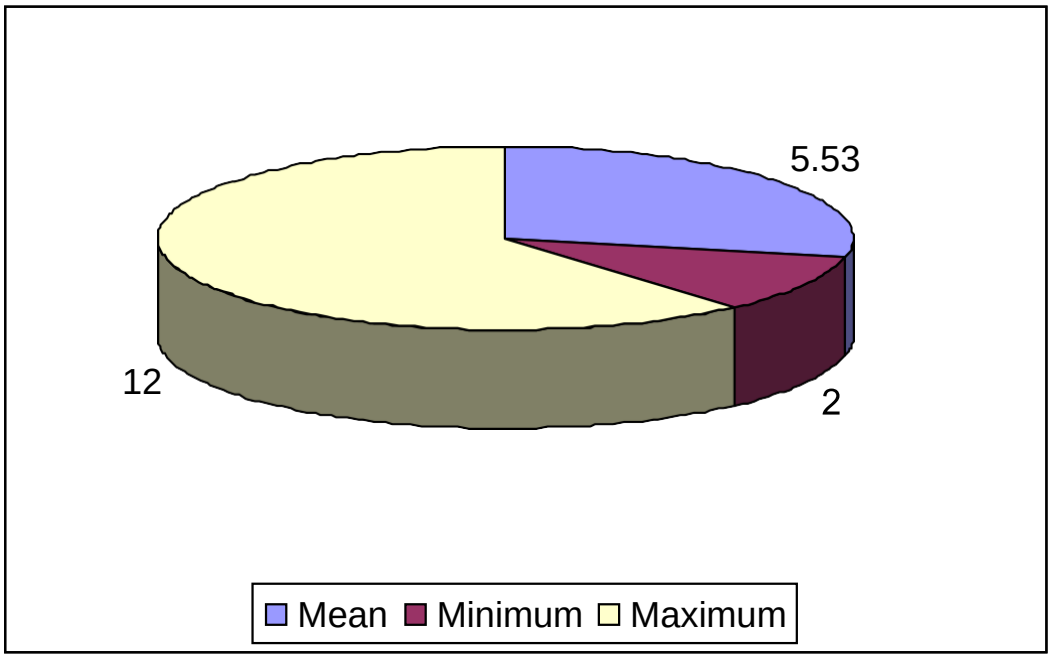
	Experimental	Control	p value	Inference
Mean	3.86±2.82	1.47±0.92	0.004119	S
Minimum	0.98	0.02		
Maximum	10.91	2.97		

GRAPHS

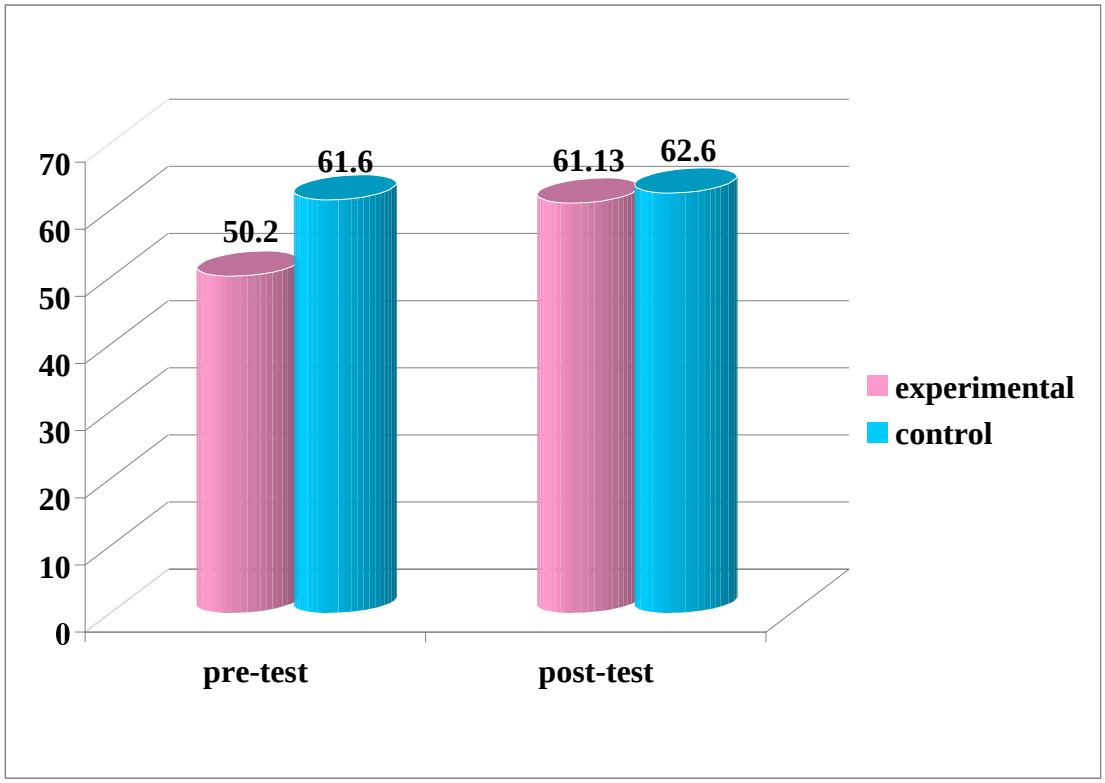
Graph No.1: Mean Age (Experimental)



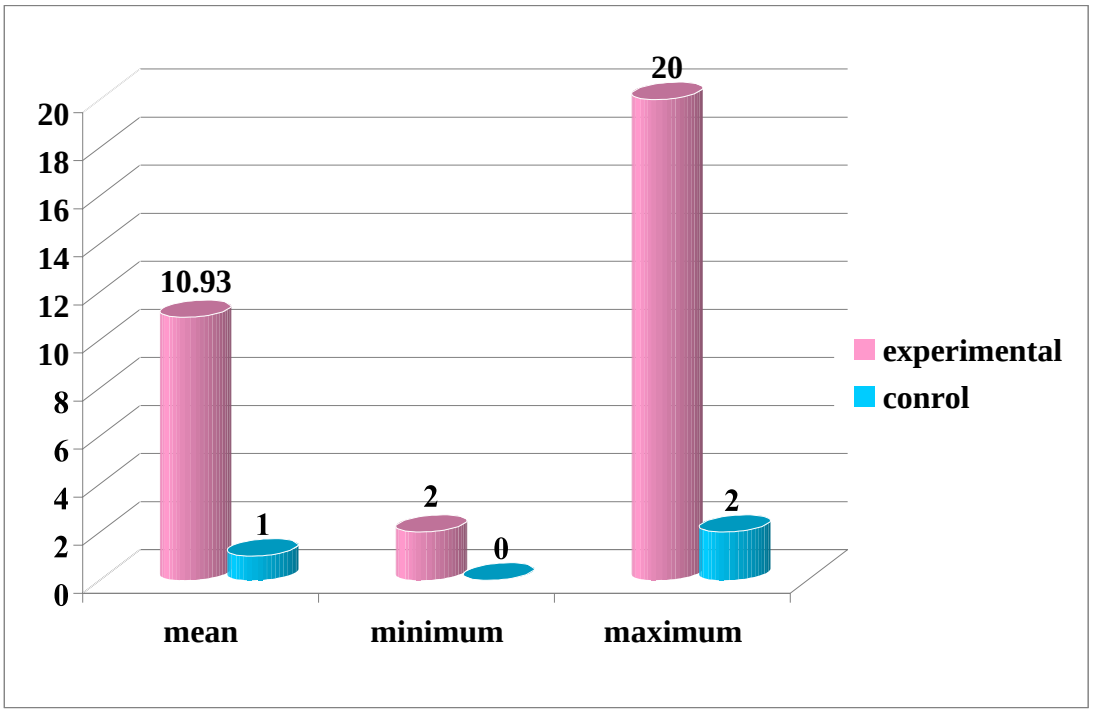
Graph No.2: Mean Age (Control)



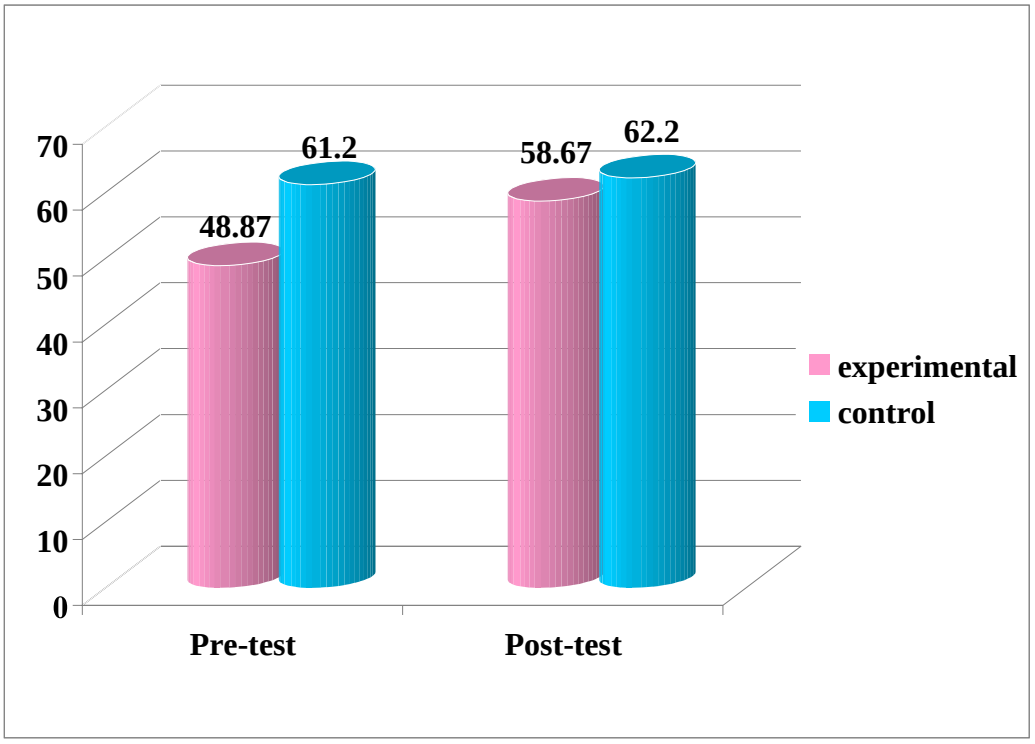
Graph No.3: Right Popliteal angle (Mean)



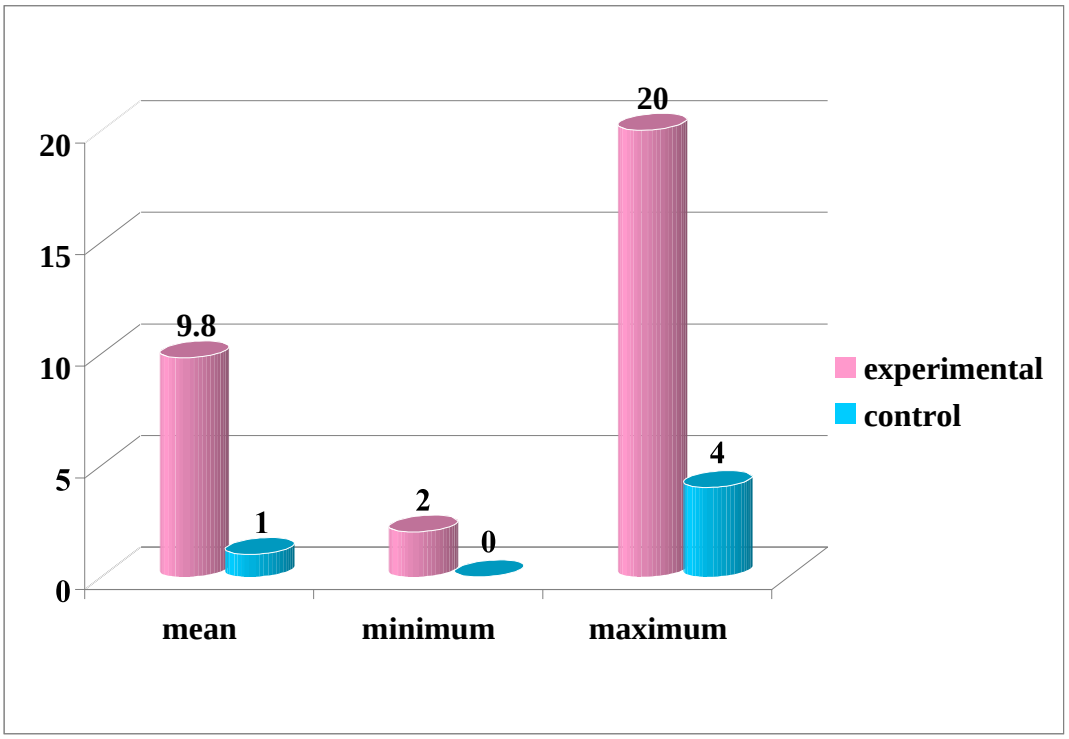
Graph No.4: Improvement of right knee



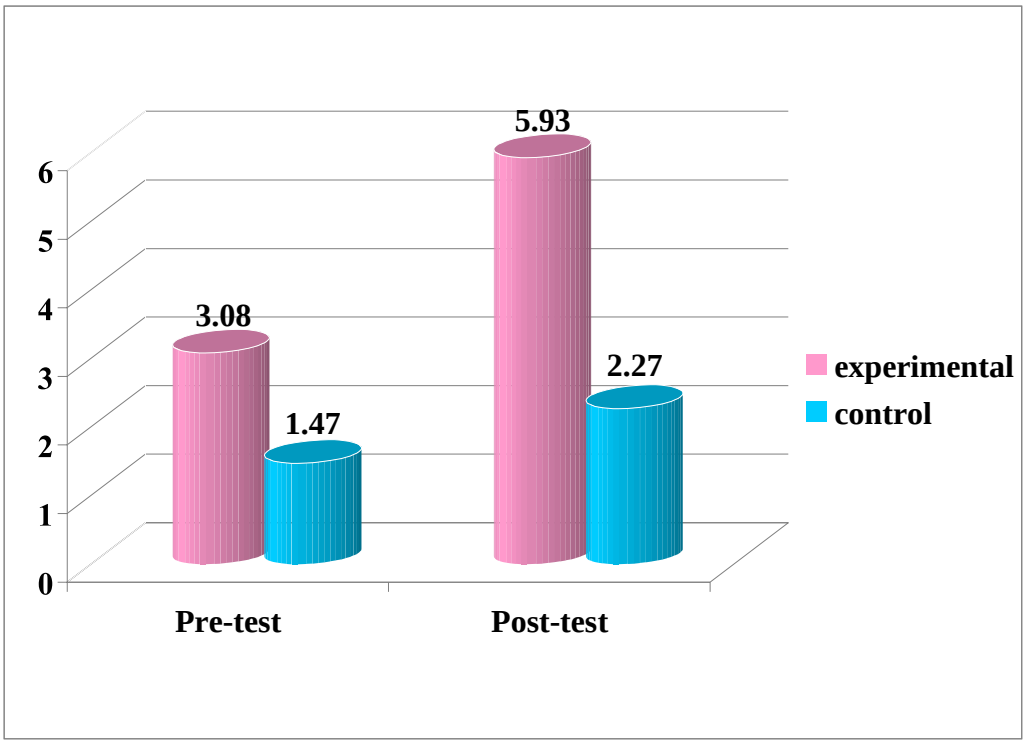
Graph No.5: Left Popliteal angle (Mean)



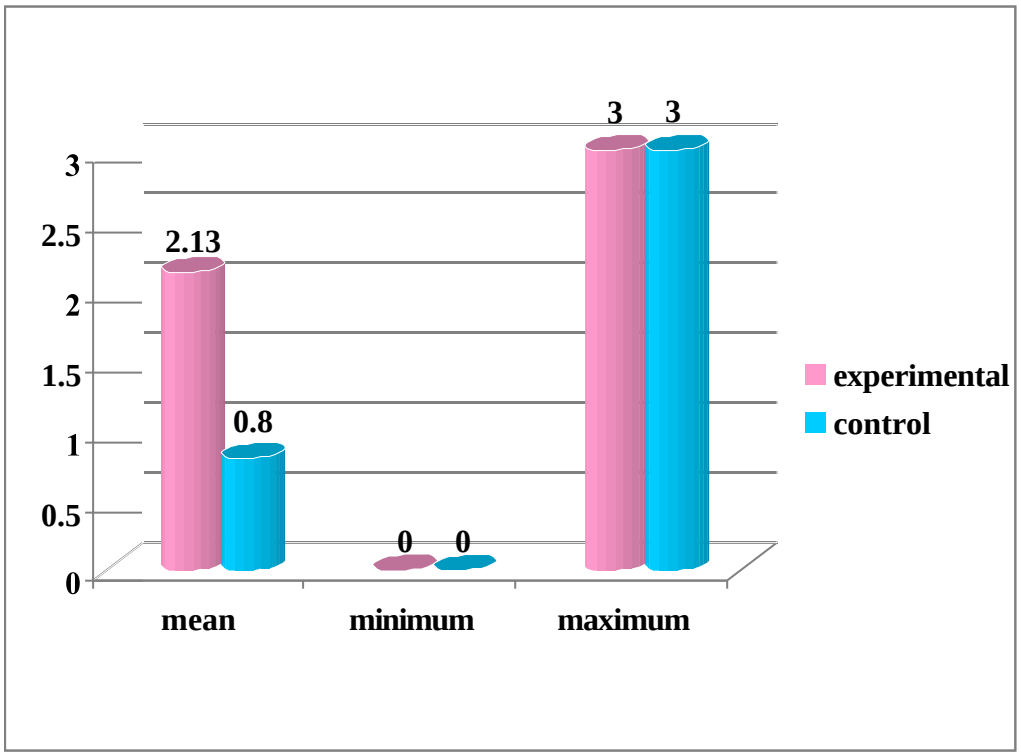
Graph No.6: Improvement of left knee



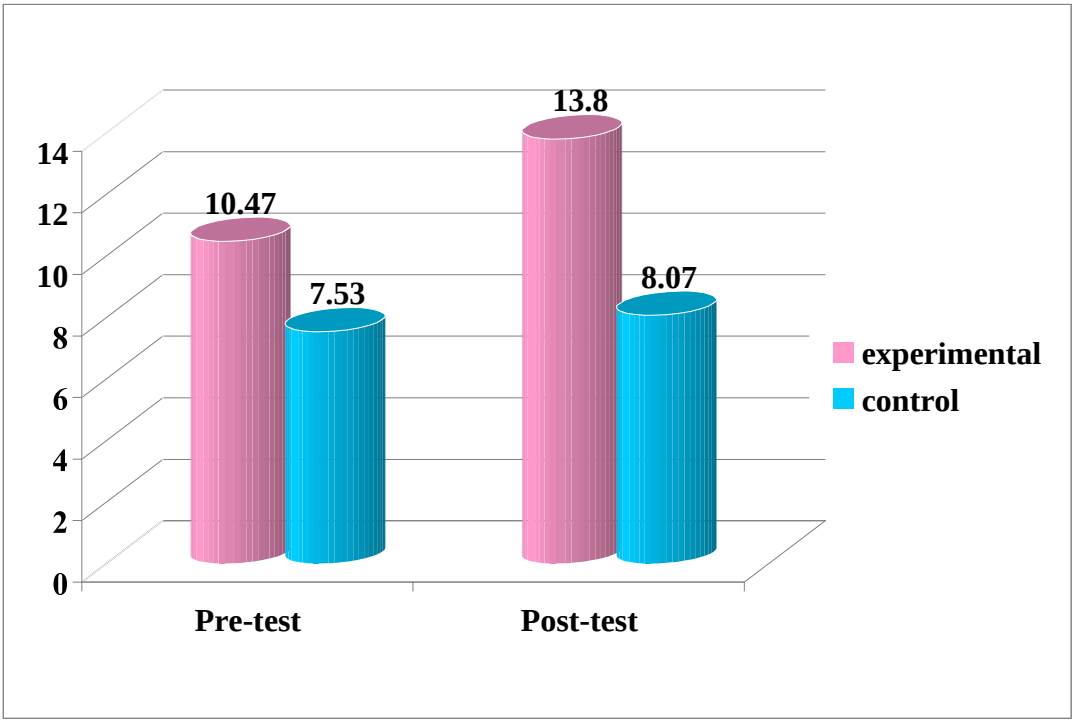
Graph No.7: GMFM dimension D (mean)



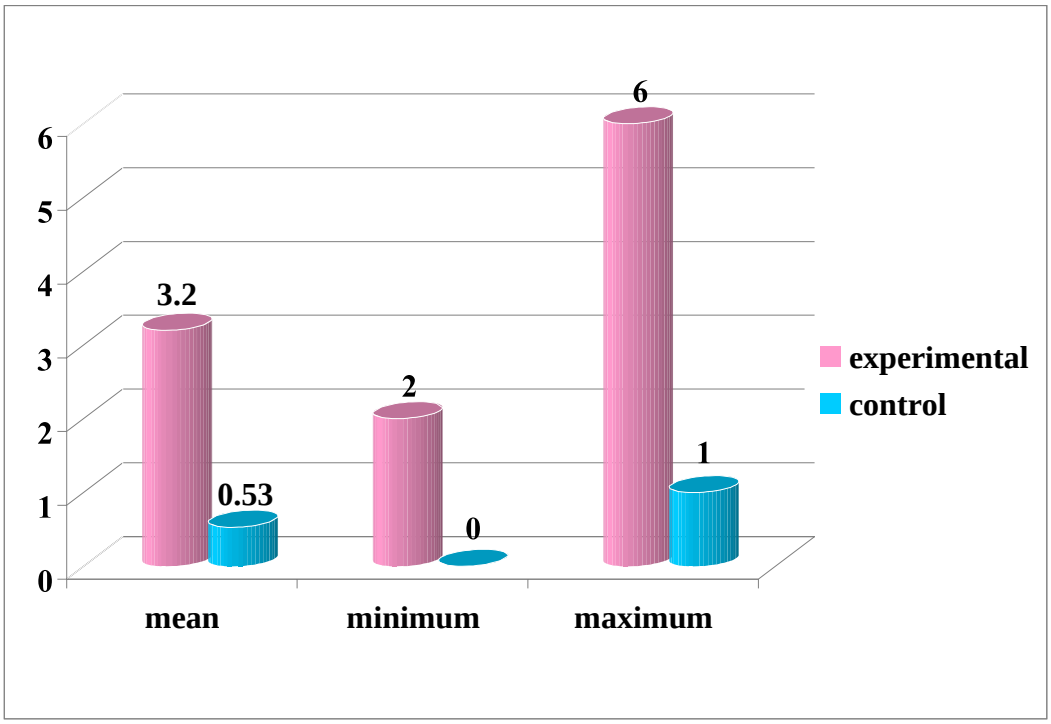
Graph No.8: Improvement for D



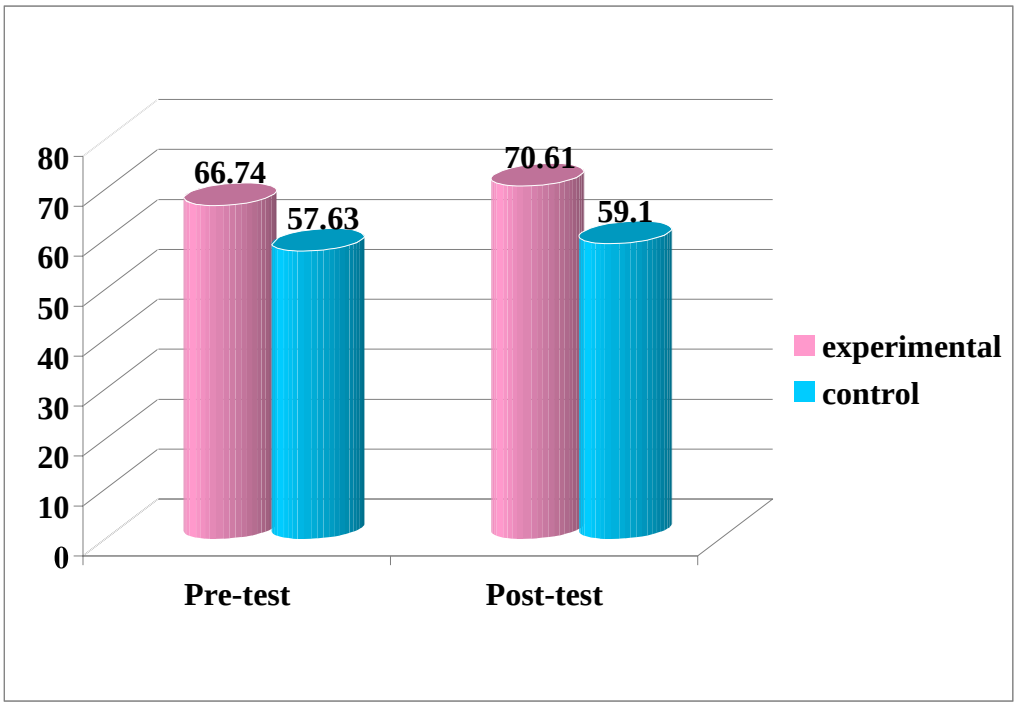
Graph No.9: GMFM dimension E (mean)



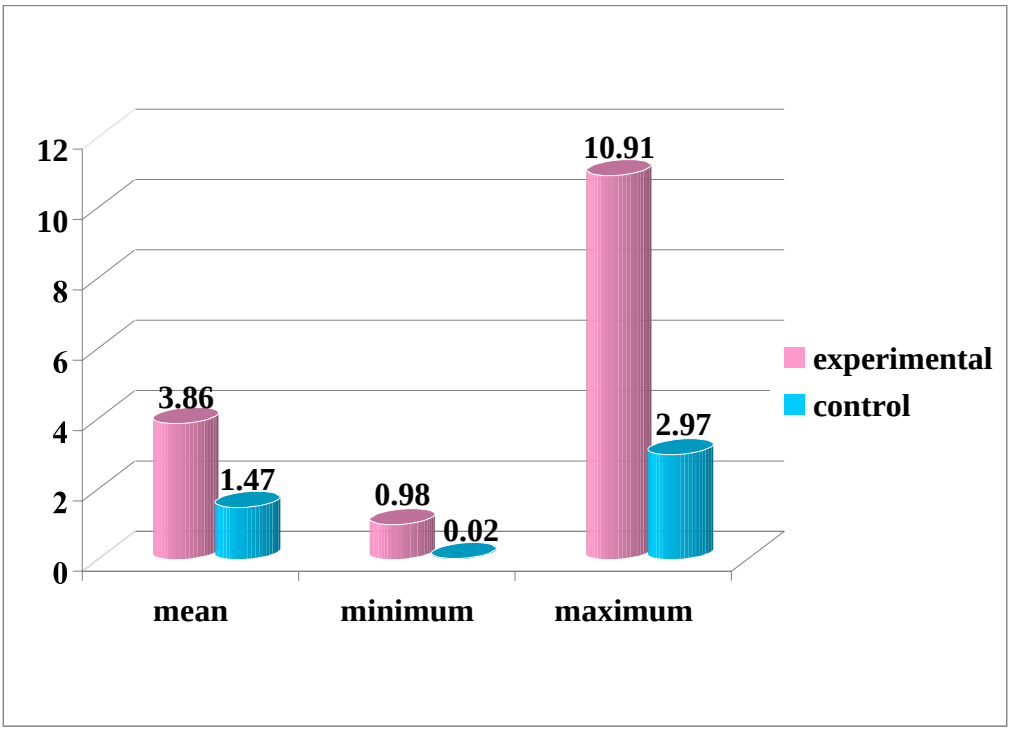
Graph No.10: Improvement for E



Graph No.11: Mean GMFM total score

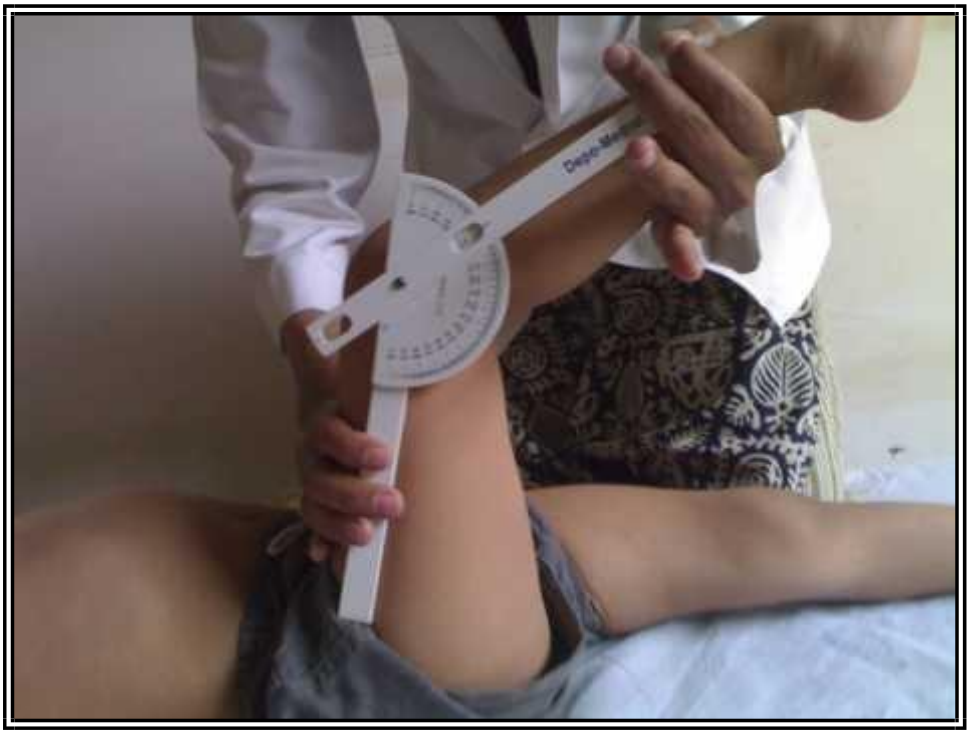


Graph No.12: Improvement for GMFM

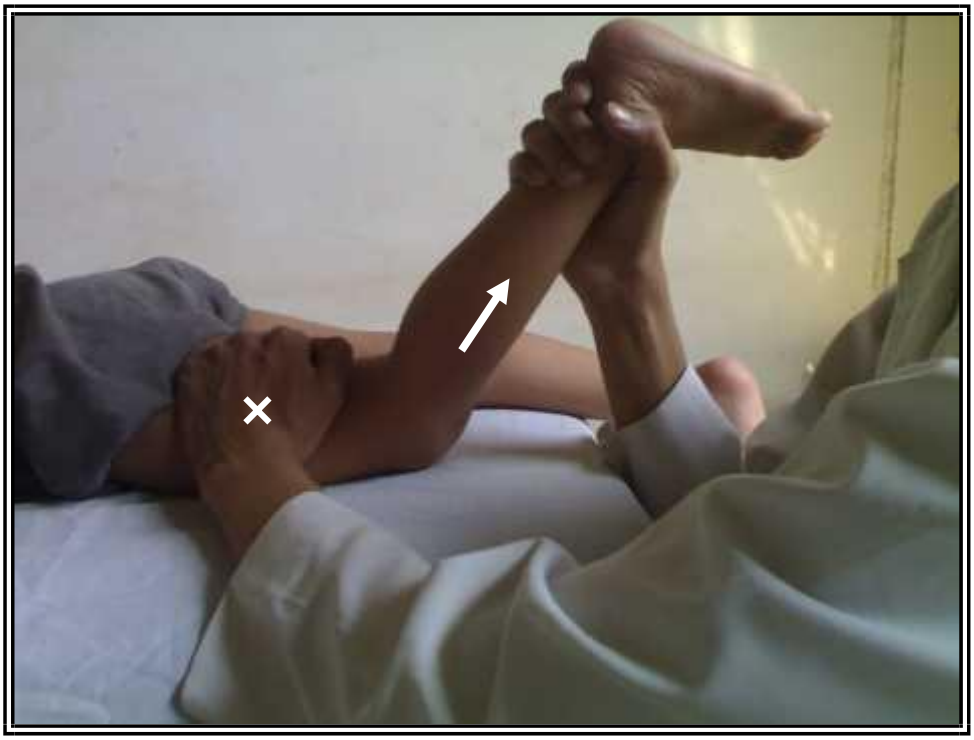


PHOTOGRAPHS

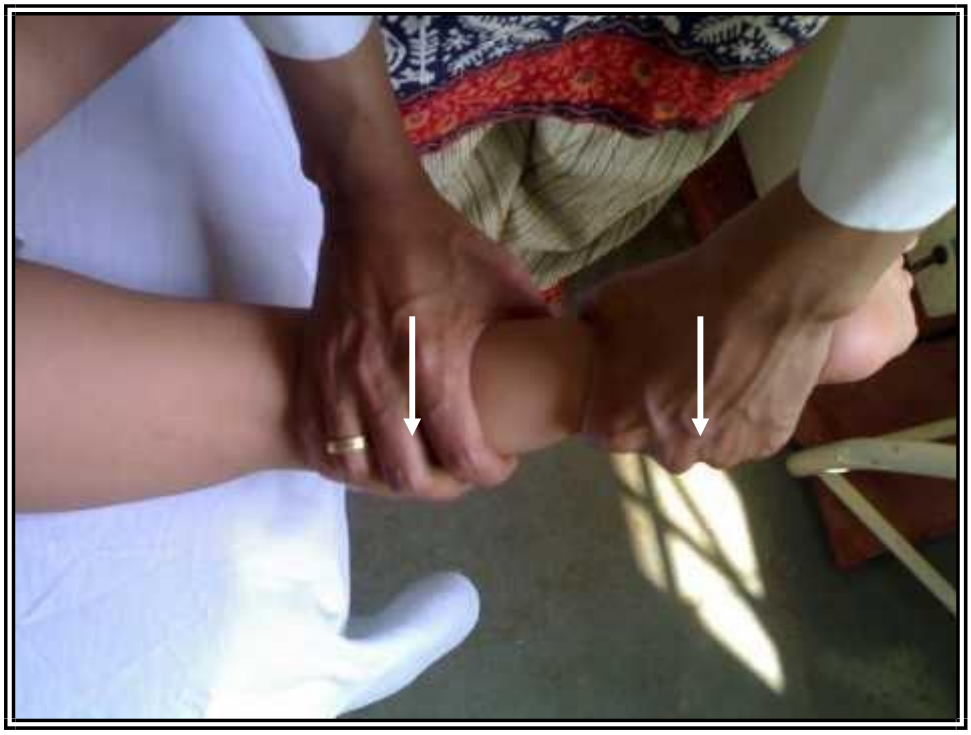
Photograph No.1: Instruments



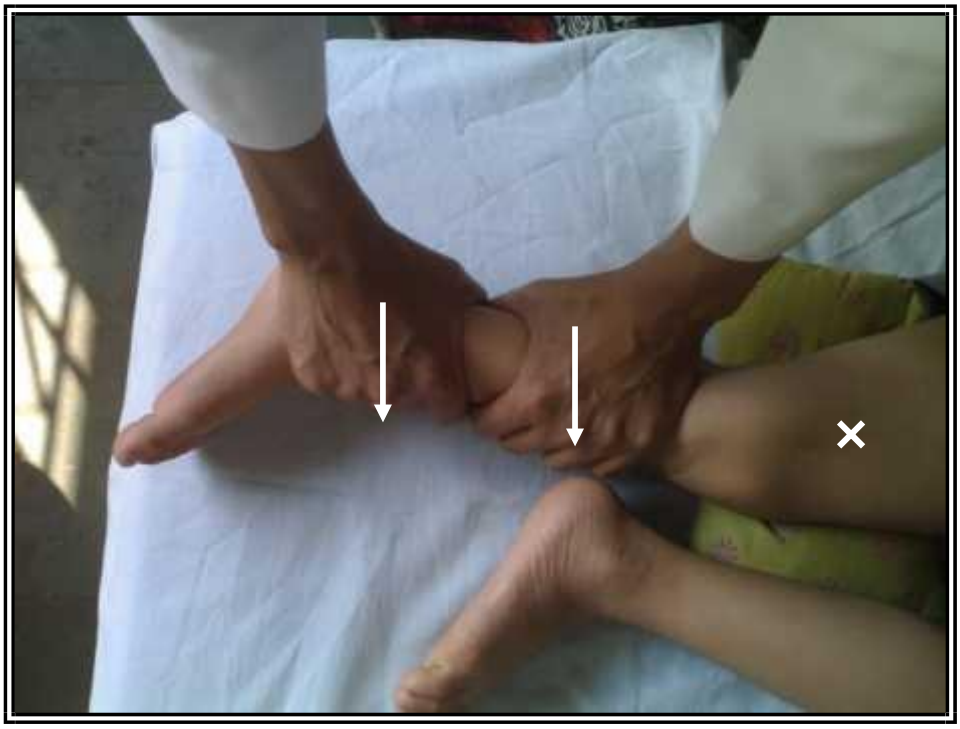
Photograph No.2: Measurement of Popliteal Angle



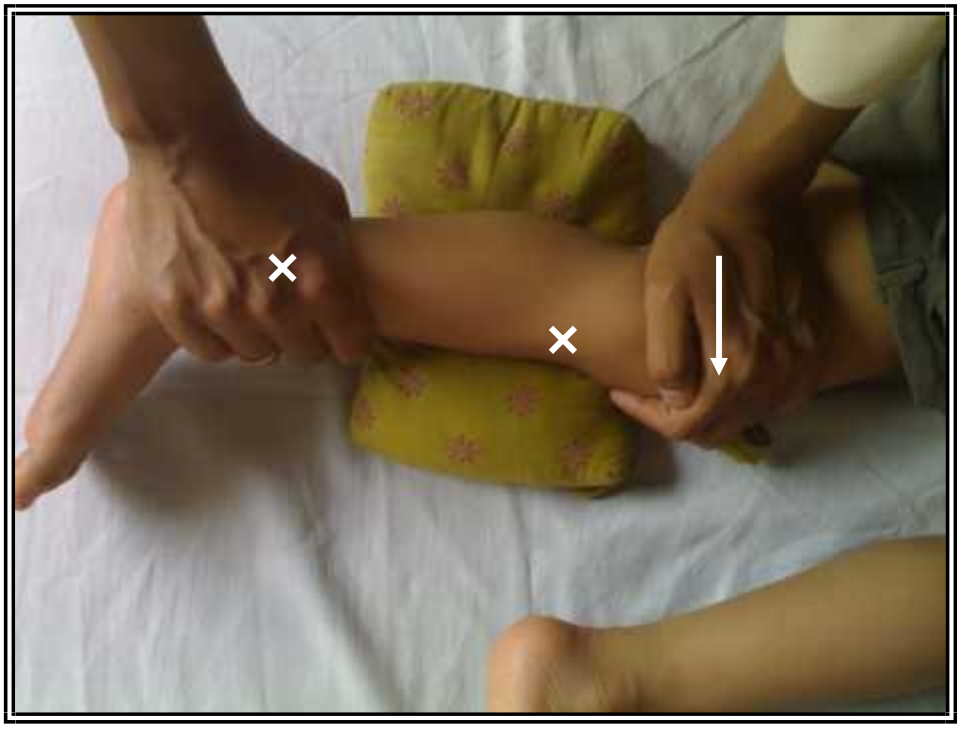
Photograph No.3: Knee Traction



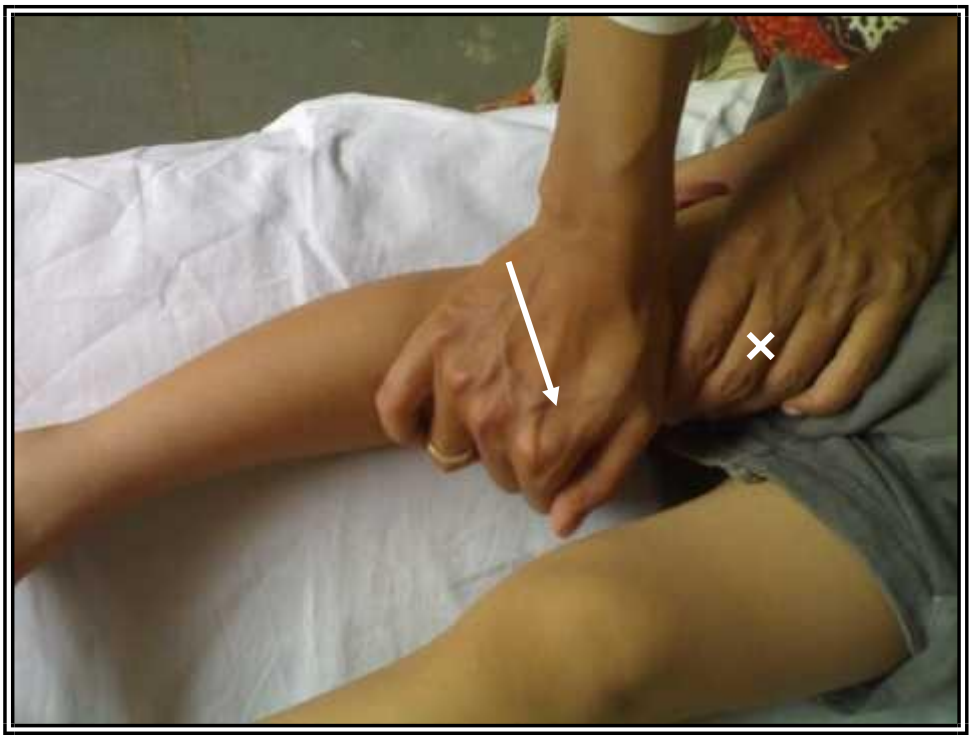
Photograph No.4: Knee Anterior Glide



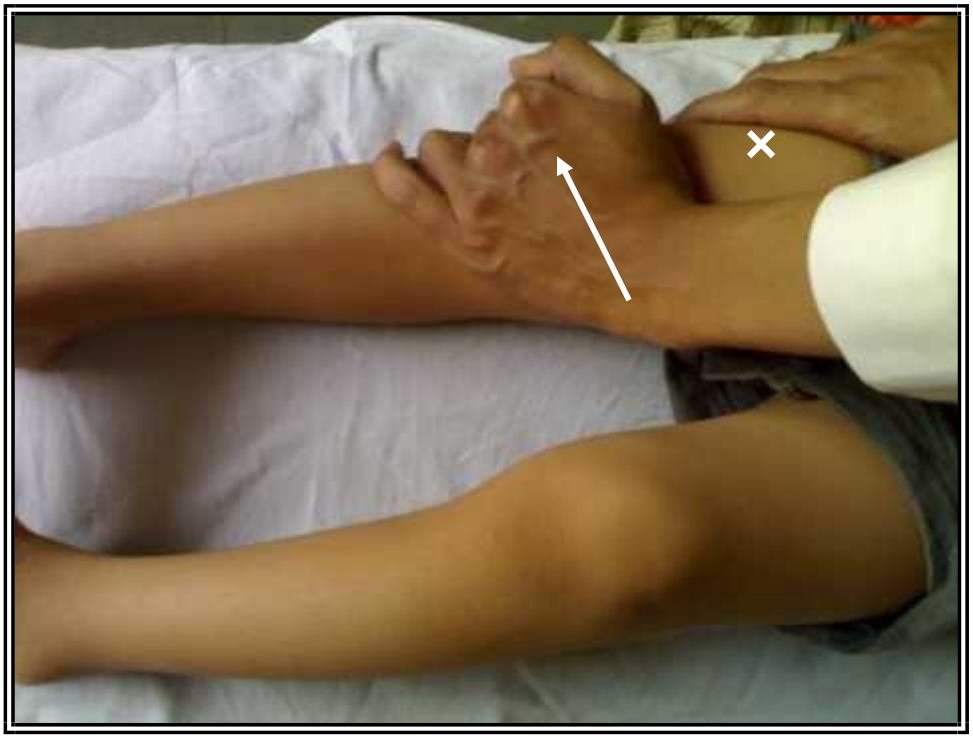
Photograph No.5: Knee Lateral Glide



Photograph No.6: Knee Medial Glide



Photograph No.7: Patella Medial Glide



Photograph No.8: Patella Lateral Glide

Photograph No.9: Neuro Developmental Treatment

MASTER CHART

Experimental Group

Sr No	Age	Sex	Popliteal Angle				IMPROVEMENT		GMFM dimensions				IMPROVEMENT		GMFM Total score		IMPROVEMENT
			Pre-test		Post-test				Pre-test		Post-test				Pre-test	Post-test	
			R	L	R	L	R	L	*D*	*E*	D	E	D	E			
1	8.00	M	62	53	80	60	18	7	*2*	*8*	4	10	2	2	63.2	65.7	2.5
2	4.00	M	15	13	20	18	5	5	3	7	3	10	0	3	54.25	55.23	0.98
3	4.00	M	50	50	70	70	20	20	4	0	6	2	2	2	57.8	65.3	7.5
4	3.00	F	60	45	68	50	8	5	3	0	4	2	1	2	52.32	55.54	3.22
5	4.00	M	85	70	87	72	2	2	3	4	6	9	3	5	82.3	85.57	3.27
6	14.00	M	20	30	25	37	5	7	4	13	6	15	2	2	77.8	81.32	3.52
7	11.00	M	50	55	70	75	20	20	2	23	5	26	3	3	78.86	80.77	1.91
8	7.00	F	60	50	70	55	10	5	4	17	5	20	1	3	69.88	71.45	1.57
9	5.00	F	20	30	35	42	15	12	3	3	5	5	2	2	52.22	53.43	1.21
10	7.00	M	47	53	52	59	5	6	5	25	7	28	2	3	62.23	64.34	2.11
11	3.00	F	54	53	65	64	11	11	4	6	7	12	3	6	84.43	87.34	2.91
12	5.00	F	75	77	84	89	9	12	7	10	10	15	3	5	76.55	80.76	4.21
13	7.00	M	40	40	52	51	12	11	4	6	6	9	2	3	54.43	65.34	10.91
14	6.00	F	48	47	62	63	14	16	5	21	8	24	3	3	66.34	70.43	4.09
15	11.00	M	67	67	77	75	10	8	4	14	7	20	3	4	68.56	76.56	8

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