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UNDER THE GUIDANCE OF DR.BRUNDA.S

LIST OF ABBREVIATIONS

CP- Cerebral palsy

CPVL- Carboxypeptidase vitellogenin like protein LSCS- Lower segment caesarean section

IUGR- Intra uterine growth restriction CFT- Capillary refill time

CNS- Central nervous system TB- Total bilirubin

DB- direct bilirubin LFT- Liver function test

DBF- Direct breast feeding FGR- Foetal growth restriction

GTCS- Generalized tonic colonic seizures LRTI- Lower respiratory tract infection BAER- brainstem auditory evoked potential CMV- Cytomegalovirus

CSF- Cerebrospinal fluid ROM- Range of motion

ABSTRACT

Background

Cerebral Palsy (CP) is a non-progressive neurological disorder affecting movement and posture, often accompanied by cognitive, sensory, and behavioural impairments Spastic diplegia, a common subtype, significantly affects mobility due to increased muscle tone in the lower limbs Surgical interventions like bilateral hamstring release are often necessary to address contractures and crouch gait However, postoperative rehabilitation is critical to functional recovery and long-term outcomes.

Objective

To evaluate the effectiveness of a structured physiotherapy rehabilitation program followmg bilateral hamstring release surgery in a child with spastic diplegic CP, using GMFM-88 and joint range of motion as outcome measures.

Methods

A case study was conducted on a male child diagnosed with spastic diplegic CP due to congenital CMV infection. The child underwent bilateral hamstring release, semitendinosus to adductor transfer, and patellar plication A tailored physiotherapy protocol was implemented postoperatively over 12 months. The program included progressive range-of-motion exercises, muscle strengthening, gait training, and balance activities Motor function improvement was measured using the GMFM-88 scale and ROM assessments at 1 day, 6 months, and 12 months post-surgery

RESULTS

Marked improvement was observed in GMFM-88 scores across all dimensions, particularly in lying & rolling (from ~35% to ~86%), sitting(~41% to~95%), and crawling & kneeling (~10% to ~88%) Standing and advanced mobility (walking, running, jumping) showed gradual but limited gains Hip and knee flexion improved from ~58° and ~55° at 1-3 months to~ 128° and
~125° at 9-12 months, indicating enhanced mobility and functional range

Conclusion

Surgical correction combined with early and sustained physiotherapy significantly improves functional mobility and joint flexibility in children with spastic diplegic CP. Although advanced motor skills develop more slowly, early intervention fosters independence and reduces long- term disability.

Keywords

Cerebral Palsy, Spastic Diplegia, Hamstring Release, Physiotherapy, GMFM-88, Rehabilitation, Motor Function, Range of Motion, Congenital CMV, Paediatric Neurology

I.INTRODUCTION

Cerebral palsy (CP) describes a group of disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing foetal or infant brain. The motor disorders of cerebral palsy are often accompanied by disturbances of sensation, cognition, communication, perception, and/or behaviour, and/or by a seizure disorder [1]
This definition recognizes that assessing the extent of activity restriction is part of the CP evaluation [7]A CP classification with four major components was also proposed **(l)** motor abnormalities, (2) accompanying impairments, (3) anatomical and neuro-imaging findings, and
(4) causation and timing [7]

Cerebral palsy (CP) is a well-recognized neurodevelopmental condition beginning in early childhood and persisting through the lifespan It is primarily a neuromotor disorder that affects the development of movement, muscle tone and posture. The underlying pathophysiology is an injury to the developing brain in the prenatal through neonatal period. Although the initial neuropathologic lesion is non-progressive, children with CP may

develop a range of secondary conditions over time that will variably affect their functional abilities[2]

This is not a single disease but rather a heterogeneous clinical syndrome resulting from injury to the developing brain. the clinical expression changes over time as the brain matures[3]

CP was first described by William Little in 1862 and initially was called Little's disease. lt was described as a disorder that appeared to strike children in the first year of life, affected developmental skill progression, and did not improve over time Little also connected this disorder to a lack of oxygen during birth Later, in 1897, Sigmund Freud suggested that CP might be rooted in the brain's development in the womb and related aberrant development to factors influencing the developing foetus (Accardo, 1982). Birth asphyxia alone was thought to be the cause ofCP until the 1980s, when biomedical research found this aetiology to be less likely and only one of many with potential to result in CP[4] Originally reported by Little in 1861 (and originally called 'cerebral paresis') Beginning at the end of the 19th century Sigmund Freudl and Sir William Osler2 both contributed important perspectives on the condition [1]

CP symptoms are heterogeneous, a child having Iimited brain injury may find difficulty in just one component of the musculoskeletal system, and another child with a broad range of symptoms may suffer from activities that hamper the activities of daily living of the child along with other life-threatening comorbidities like epilepsy however, its symptom may improve in due course of time owing to the maturity of the nervous system with age Damage to the developing brain before, during, or just after delivery affects both neurological and musculoskeletal systems of the body producing symptoms such as abnormal contraction of muscles, postural changes, and movement and activity limitation which are accompanied by sensory disturbances along with perceptual disorders and epilepsy

An epileptic seizure is a transient occurrence of signs and/or symptoms due to abnormal excessive or synchronous neuronal activity in the brain Epilepsy is "a disorder of the brain characterized by an enduring predisposition to generate epileptic seizures, and by the cognitive, psychological, and social consequences of this condition

Cerebral Palsy (CP) is the commonest cause of physical disability in early childhood

EPIDEMIOLOGY

There are two approaches to estimating the frequency of cerebral palsy Prevalence is the proportion of a population with the condition at a given point in time and is used to plan services Its estimation by surveys is straightforward but extremely labour-intensive on account of the size of the surveys required for this relatively rare condition. Incidence is the proportion of new cases arising in a population. It is used in aetiological studies seeking to understand the causal pathways to the responsible brain abnormality, for which it appears reasonable to consider that cerebral palsy exists when the brain abnormality exists.[9]

There have been radical changes in our understanding of aetiological factors over the past 20 years In 1862, Little reported that abnormal birth was a possible factor in spastic cerebral palsy. Although he was aware of other causes, his writings were interpreted to mean that abnormal birth was the primary cause of spastic cerebral palsy There was no recognition of Freud's converse view that intrauterine developmental abnormality was responsible (Scherzer and Tacharanite 1982). For over 100 years, most cases of cerebral palsy were thought to be caused by asphyxia during either labour or the perinatal period (Blair and Stanley 1988, Paneth 1986) Prevalence rates of cerebral palsy were used as outcome measures of obstetric practice and neonatal care and it was expected that improvement in

these areas would result in lower rates of cerebral palsy (Stanley and Blair 1991, Torr's et al 1990) As a result, there was increased

use of interventions such as electronic foetal monitoring and caesarean section. However, the role of perinatal asphyxia in the aetiology of cerebral palsy was challenged when the stillbirth and neonatal death rates declined but the cerebral palsy rate remained constant[10]

Overall, 86.6% children acquired CP prenatally and perinatally (e g. preterm birth, birth asphyxia, neonatal encephalopathy). Median age atCPdiagnosis was 3 years Moreover, 79.2% children had spastic CP and 73.3% were classified in Gross Motor Fw1ction Classification System levels III to V Notably, 473% of children never received rehabilitation services (median age at receiving rehabilitation services was 3y; 12.7% received assistive devices) and 75.6% of school-age children had no access to education. [11]

ETIOLOGY

The aetiology of CP is very diverse and multifactorial The causes are congenital, genetic, inflammatory, infectious, anoxic, traumatic and metabolic [12] It was found in some cases that not a single event, but a series of events, is responsible for the damage of motor nerves which finally results in this condition. [13]

The injury to the developing brain may be prenatal, natal or postnatal. As much as 75% - 80% of the cases are due to prenatal injury with less than 10% being due to significant birth trauma or asphyxia. The most important risk factor seems to be prematurity and low birth weight with risk of CP increasing with decreasing gestational age and birth weight. Cerebral palsy is seen in 10 - 18 % of babies in 500-999 grams birth weight CP occurs more commonly in children who are born very prematurely or at term. Although term infants are at relatively low absolute risk, term births constitute the large majority of all births, as well as approximately half of all births of children with cerebral palsy. Prenatal maternal chorioamnionitis is also a significant risk factor accounting for as much as 12% of cerebral palsy 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. [12]

Infection is also a significant risk factor for both preterm and term infants Premature infants who have evidence of post-natal systemic inflammation, such as from an infection, will have an increased risk of developing CP[14]

Genetics are now becoming more recognized as a contributing factor to CP Previously it was thought to contribute to 1-2% of cases that had familial links Recent studies utilizing exome sequencing show that 14% of cases have single gene mutations and 31% have clinically

relevant copy number variations The utilization of next generation sequencing in individuals with CP have demonstrated quite variable results, from 14-80% Although, it is difficult to understand the significance of these findings given the large variability, it is clear that the interaction of genetics may play a larger role in the manifestation of CP than previously thought [14]

CLASSIFICATION

This proposed definition of CP covers a wide range of clinical presentations and degrees of activity limitation, and it is, therefore, useful to further categorize individuals with CP into classes or groups The purposes of classification include the following.

1. Description: providing the level of detail about an individual with CP that will clearly delineate the nature of the problem and its severity
2. Prediction: providing information that can inform health care professionals of the current and future

service needs of individuals with CP

3. Comparison: providing sufficient information to permit reasonable comparison of series of cases of CP assembled in different places

4. Evaluation of change: providing information that will allow comparison of the same individual with CP at different points in time

Traditional classification schemes have focused principally on the distributional pattern of affected limbs (for example hemiplegia or diplegia) with an added modifier describing the predominant type of tone or movement abnormality (e g. spastic or dyskinetic), but it has become apparent that additional characteristics must be taken into account for a classification scheme to contribute substantively to the understanding and management of this disorder[1]

The topographic classification of CP is monoplegia, hemiplegia, diplegia and quadriplegia; monoplegia and triplegia are relatively uncommon There is a substantial overlap of the affected areas. In most studies, diplegia is the commonest form (30% - 40%), hemiplegia is 20% - 30%, and quadriplegia accounting for 10% - 15% In an analysis of 1000 cases of CP from India, it was found that spastic quadriplegia constituted 61% of cases followed by diplegia **22%** I

3. Gross Motor Function Classification System (GMFCS)

The GMFCS is the most established and recognized of the functional classification measures in CP The GMFC is a simple, five-level, ordinal grading system created to describe the gross motor function of an individual with CP. First described in 1997 by Palisano et al., the GMFCS provides a common language for a practitioner that is meaningful, quick and easy to use. The GMFCS describes self-initiated movement and use of assistive devices (walkers, crutches, canes, wheelchairs) for mobility during an individual's usual activity Following research to stratify typical motor function for children with CP, the authors concluded that a five-level classification system worked well to discriminate clinically meaningful distinctions in motor function This classification system was initially designed to be used with children 2-12 years of age. The GMFCS was later expanded and revised in 2007 to include ages 12-l 8, as well as to increase descriptors and differentiations for the levels based on the child's age, while taking into account developmental milestones. These more detailed and age-appropriate descriptors allow a more accurate classification[15]

An individual classified in GMFCS I is able to walk without limitations Individuals less than two years of age are able to crawl on hands and knees, pull to stand, cruise when holding onto furniture, and are able to attain independent walking between the age of 18 months and two years Between two and four years of age, skills include sitting independently and transitioning between sitting and standing independently Between age four and six years, the individual in GMFCS I is able to walk indoors and outdoors independently, climb stairs, and start to run and jump. Between the ages of six and twelve, additional abilities include walking up and down curbs, walk community distances, negotiate stairs without railings, and run and jump (which may include some limitations). Between age 12 to 18, the abilities are the same as age 6 to 12. [15]

Based on the GMFCS revised and expanded version, an individual classified in GMFCS I is able to walk without limitations Individuals less than two years of age are able to crawl on hands and knees, pull to stand, cruise when holding onto furniture, and are able to attain independent walking between the age of 18 months and two years. Between two and four years of age, skills include sitting independently and transitioning between sitting and standing

independently Between age four and six years, the individual in GMFCS I is able to walk indoors and outdoors independently, climb stairs, and start to run and jump Between the ages of six and twelve, additional abilities include walking up and down curbs, walk community

distances, negotiate stairs without railings, and run and jump (which may include some limitations) Between age 12 to 18, the abilities are the same as age 6 to 12.[15]

A child classified in GMFCS II can walk with limitations. Limitations may include balance or endurance, use of a hand-held mobility device prior to age 4, use of a railing on stairs, or an inability to run or jump GMFCS II functioning may result in the use of wheeled mobility for long distances A child before the age of two can sit with upper extremity support, crawl on their stomach, and may be able to pull-to-stand or cruise with support Between ages 2 and 4, the child can transition into and out of sitting without support, can sit without support (but may need to use their upper extremities for balance), can crawl on hands and knees, cruise with support and walk with a mobility device Between the ages of four and six, the individual can transition into and out of standing without support, walk short flat distances without an assistive device, do stairs with the railing, but is unable to run or jump From age 6 to 12, the individual can walk in most terrains but has limitations with distance or uneven surfaces, may use wheeled mobility for long distances, can do stairs with the railing, but is able to do minimal or no running and jumping Between ages 12 and 18, the abilities are the same as age 6 to 12, but a handheld mobility device may be used for safety.[15]

A child classified in GMFCS III can often walk with a hand-held mobility device indoors, but use wheeled mobility in the community and for longer distances. GMFCS III level functioning indicates the ability to sit with little to no external support and to stand to complete transfers Children in GMFCS Ill who are less than 2 years old can roll and occasionally crawl forward when lying on their stomach, as well as sit with some low back support Between ages 2 and 4, a child can "W" sit on the floor with some help getting into the position, crawl on his/her stomach or creep on all fours, and may pull-to-stand and walk short distances using a handheld mobility device (walker or gait trainer) with some assistance for manoeuvring. From age 4 to 6, a child can sit in a standard chair but may require extra support to allow full upper extremity function, walk with a handheld mobility device and do stairs with assistance; typically, wheeled mobility is used for longer distances. In GMFCS III, children aged 6 to 12 walks with a handheld mobility device indoors and use wheeled mobility (either manual or powered) for distance, require assistance to move between floors, sitting and standing, and negotiate stairs with assistance For ages 12 to 18, the abilities are the same as age 6 to 12, but more variability is demonstrated in primary mobility preferences.[15] An individual classified in GMFC IV can sit supported, but self-mobility is limited, often being transported in a manual wheelchair or using powered mobility. Children before the age of 2 have head control and can roll, but require truncal support to sit Between ages 2 and 4, a child in GMFCS IV can sit with upper extremity support, require assistance to transition into sitting, and may require adaptive equipment for sitting or standing. At this age, some self- mobility is possible through rolling or stomach crawling short distances, but reciprocal leg movement is not present From age 4 to 6, children require adaptive equipment for trunk control to allow sitting and assistance to move between positions Children may walk short distances with a mobility device and with assistance, and use wheeled mobility for distances, and/or be independent with powered mobility. Children in GMFCS IV and ages 6 to 12 require adapted seating and assistance with transfers, and utilize wheeled power mobility independently or manual mobility with assistance in most settings Many

children can have independent floor mobility with crawling or rolling, or may walk short distances with assistance. For ages 12 to 18, the abilities are the same as ages 6 to 12 [15]

Children classified in GMFCS V have more severe limitations with head and trunk control and self-mobility is only possible using a power wheelchair Children in GMFCS V before the age of 2 do not have independent head or trunk control and require assistance to roll Between ages 2 and 4, a child has no independent movement and requires assistance for transport using manual mobility devices. Adaptive equipment is required for sitting and standing, but function is still limited It is possible to become independent using power mobility with additional adaptations From age 4 onwards, the abilities of children in GMFC V are stable with a need for complete assistance with transfers emerging after age 6.[15]

CASE STUDY

A full-term male neonate, Baby, was delivered via Lower Segment Caesarean Section (LSCS) on 12/12/2009 at 10:04 PM at Sumathi Nursing Home The indication for LSC was foetal distress, oligohydramnios, and intrauterine growth restriction (IUGR) The baby was born in a vertex presentation and cried immediately after birth. The Apgar score was normal at both **1** mmute and 5 mmutes of life

On Day 3 of life, the baby developed jaundice, which progressively worsened By Day 5, the baby became lethargic and started feeding poorly, leading to referral to MS. Ramaiah Medical Teaching Hospital (MSRMH) for further evaluation and management On the day of admission, the baby's vitals were stable, with a heart rate of 136 bpm, respiratory rate of 42/min, capillary refill time (CFT) of 3 seconds, and warm peripheries. Hydration was assessed as fair On systemic examination, the cardiovascular and respiratory systems were normal, with no murmurs or adventitious lung sounds The abdomen was soft, with no hepatosplenomegaly, and the central nervous system (CNS) examination showed normal tone and activity.

Laboratory investigations revealed elevated bilirubin levels, with total bilirubin (TB) at 21.35 mg/dL, which later improved to 6 10 mg/dL after treatment Direct bilirubin (DB) was 179 mg/dL, which remained stable at 1.75 mg/dLLiver function tests (LFTs) showed mild abnormalities, including marginally elevated CRP (2 _9), normal haemoglobin (183 g/dL), total

count (6,400/mm^3), and platelet count (3,14,000/mm^3) The TORCH panel was sent for evaluation to rule out congenital infections, and an ultrasound abdomen showed no significant hepatobiliary abnormality

Based on the clinical findings and investigations, a fmal diagnosis of Neonatal Cholestatic Jaundice (likely due to early-onset sepsis and cholestasis) was made. The baby was managed with intravenous fluids to maintain hydration, supportive care, and Ursodeoxycholic Acid (UDCA) at 10 mg/kg/day to improve bile flow and reduce cholestasis. Exclusive direct breastfeeding (DBF) was encouraged, and weight monitoring was initiated Infection control measures were also implemented, with antibiotics considered if sepsis was suspected.

At discharge, the baby's weight was 1.76 kg, length 44 cm, head circwnference 33 cm, and chest circumference 26 cm The baby was active, pink, and feeding well Discharge advice included continuing exclusive breastfeeding, keeping the baby warm, continuing UDCA therapy, and regular weight monitoring.

After being managed with supportive care, Ursodeoxycholic Acid (UDCA), and exclusive breastfeeding, he showed significant improvement, and his bilirubin levels normalized However, despite initial stabilization, his low birth weight and perinatal complications raised concerns about potential long-term developmental effects. Over the following months, while his growth parameters were closely monitored, emerging signs of

neurodevelopmental delay became apparent.

At 9 months, He was readmitted with a history of febrile seizures and concerns regarding delayed milestones His birth history, including FGR and neonatal sepsis, along with his history of prolonged jaundice, suggested an increased risk for neurological impairments.

He was readmitted to M S Ramaiah Medical Teaching Hospital on 30/09/2010 with complaints of fever for one day and seizures since the morning of admission. The child had developed a moderate-grade fever the previous day, with no chills, rigors, or rashes. By the next morning, he experienced a generalized tonic-clonic seizure (GTCS) lasting approximately 15 minutes, characterized by limb convulsions, up-rolling of eyeballs, and frothing at the mouth The child was brought to the hospital in a febrile state

On the day of admission, he was conscious, alert, and active, with a pulse rate of 100/min, capillary refill time of less than 3 seconds, and afebrile temperature at the time of examination. Systemic examination findings were within normal limits, with no signs of meningeal irritation or neurological deficits, confirming the absence of CN infections Based on clinical evaluation, a diagnosis of Febrile Seizures with Lower Respiratory Tract Infection (LRTI) was made The child was managed with IV antibiotics, tepid sponging, and antipyretics, following which he remained seizure-free, and his vitals stabilized After five days of hospitalization, he was discharged in a stable condition on medication with instructions for tepid sponging and monitoring for recurrence of seizures Parents were advised to seek immediate medical attention if seizures recurred.

After his initial hospitalization on 30/09/2010 for febrile seizures and lower respiratory tract infection (LRTI), he was discharged in a stable condition with instructions for fever management and seizure monitoring However, over the following weeks, his parents noticed persistent concerns regarding his motor development. Despite recovering from the acute febrile episode, he exhibited delayed acquisition of developmental milestones, prompting further evaluation By 08/11/2010, he had not shown expected progress in motor skills, raising suspicions of an underlying neurological disorder. His parents reported reduced voluntary limb movements and an absence of independent sitting or crawling, leading them to seek medical attention once again This follow-up visit marked the beginning of a more in-depth investigation into his developmental delays and neuromuscular condition On 08/11/20 IO he presented with concerns of delayed developmental milestones, a history of febrile seizures, and signs of neurological impairment. he showed significant delays, achieving neck control at 6 months, rolling over at 8 months, and failing to attain independent sitting, crawling, or standing. Parents reported that he did not attempt to crawl and showed limited voluntary movements His history included a generalized tonic-clonic seizure (GTCS) episode lasting approximately 8 minutes, triggered by fever Neurological examination revealed microcephaly with a head circumference of 40 cm, mild suture overlapping, increased deep tendon reflexes, and global spasticity affecting all four limbs. His muscle tightness predominantly involved the adductors, hamstrings, and gastrocnemius muscles, indicating early dystonia He also exhibited irritability and difficulty with coordination

Investigations were conducted to determine the underlying cause of his condition. MRI and neuro sonograms showed no significant structural abnormalities, and BAER tests ruled out auditory impairments. However, a TORCH panel revealed cytomegalovirus (CMV) positivity, suggesting congenital CMV infection as the likely aetiology of his neurological deficits Cerebrospinal fluid (CSF) analysis indicated elevated protein levels (226 9 mg/dL), pointing to possible central nervous system involvement. Based on these findings, he was diagnosed with congenital CMV infection leading to microcephaly, mixed quadriparesis with early dystonia, and global developmental delay.

Management focused on seizure control, neuromuscular rehabilitation, and supportive care He was on medications to manage seizures and prevent recurrence. Physiotherapy was initiated at eleven months and continued regularly, emphasizing trunk control, muscle strengthening, and spasticity reduction through passive and active limb exercises His rehabilitation program aimed to prevent contractures and improve mobility. Additionally, he received calcium and vitamin D supplementation to support bone health Long-term follow-up was planned to monitor neurodevelopmental progress, seizure activity, and CMV-related comp!ications While cognitive and motor impairments were anticipated, early intervention and consistent phys10therapy were crucial for improving his functional independence and overall quality of Iife The prognosis depended on his response to therapy, with the primary goal being to enhance his adaptive capabilities and maximize his potential for independent movement Over the following years, He continued to receive multidisciplinary care, including regular physiotherapy, medical management, and developmental interventions His seizures remained under control, and his neuromuscular rehabilitation showed promising results With consistent therapy, he demonstrated gradual improvements in postural control, limb coordination, and voluntary movements. While he still faced challenges in achieving independent mobility, his physiotherapy regimen helped prevent contractures and optimize his functional abilities His caregivers closely monitored his neurodevelopmental progress, ensuring that he received adequate support to enhance his adaptive skills Despite the severity of his neurological impairments, early intervention significantly contributed to his ability to engage in daily activities with assistance However, his condition remained vulnerable to external triggers, and any disruption in his routine could impact his mobility

Years passed with steady progress, but an unexpected health episode disrupted his advancements. One day, he developed a fever, which was accompanied by an unusual hamstring stretch. This stretch appeared to have been forceful, likely straining his already spastic and tight muscles Almost immediately, he experienced severe pain in his legs, making any movement unbearable Over the next few days, his condition worsened he was unable to stand, and his mobility regressed significantly. His posture shifted into a persistent crouched position, a hallmark of worsening spasticity and muscle tightness in individuals with neurological impairments

Concerned about his sudden loss of mobility and increased pain, his parents sought medical attention Upon evaluation, the doctor noted a significant increase in hamstring tightness, exacerbated by his pre-existing spasticity The abrupt worsening of his gait and the onset of crouching raised concerns about musculoskeletal complications, possibly related to contractures or joint deformities After thorough assessment, the doctor recommended surgical intervention (bilateral hamstring release) to address his muscle tightness and improve his mobility The proposed surgery aimed to release the affected muscles, correct his posture, and enhance his ability to bear weight on his legs While surgery was a significant step, it was deemed necessary to prevent further deterioration and restore his potential for functional independence

On 3/04/2024 he underwent **bilateral hamstring release, bilateral semitendinosus to adductor transfer, and bilateral patellar plication.**

This combined procedure is performed in spastic diplegic cerebral palsy to reduce knee flexion contractures, improve hip stability, and correct patellar alignment, thereby enhancing mobility and reducing crouch gait

Surgical Procedure

Preoperative Preparation

The patient is placed under general anaesthesia.

The surgical site (posterior thigh and anterior knee) is sterilized and draped. A tourniquet may be used to minimize blood loss.

1. **Bilateral Hamstring Release**

Incision: A small 2-4 cm incision is made on the posterior thigh near the distal hamstring tendons.
Tendon Lengthening: The semitendinosus and semimembranosus tendons are partially released or lengthened using a Z-plasty or direct lengthening technique

2. **Bilateral Semitendinosus to Adductor Transfer**

Incision: A separate incision is made over the distal semitendinosus tendon near the knee

Transfer to Adductor Magnus: A new incision is made at the medial thigh, and the semitendinosus is sutured to the adductor magnus tendon, ensuring balanced tension.

3. **Bilateral Patellar Plication**

Objective: To correct patellar instability, improve knee alignment, and prevent excessive movement
Incision: A midline anterior knee incision is made.

Plication Procedure: The medial retinaculum and quadriceps tendon are tightened to reposition the patella. Excess soft tissue may be excised
Closure: The wound was sutured, and sterile dressing was applied

Therapy Goals:

I_ Restore joint mobility and prevent contractures

2. Strengthen weak muscles while managing spasticity
3. Improve postural stability and functional mobility.

4 Enhance gait efficiency and reduce compensatory movements

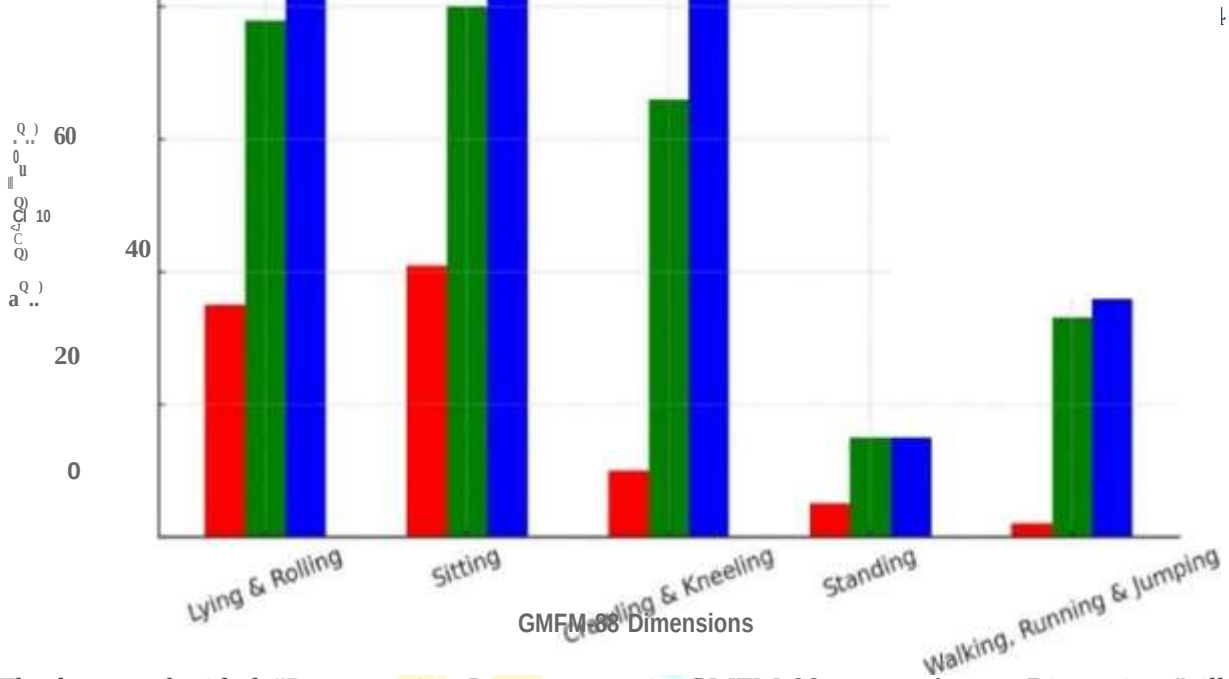
This structured approach ensures optimal recovery, minimizes complications, and helps the patient achieve functional independence within a year

REHABILITATION PROTOCOL

PHASE	GOALS	EXERCISES	PROGRESSION
(Day 1-3 months) Day I -2 Weeks	Pain control, joint mobility, prevention of complications	-Passive and assisted range of motion -Ankle pumps (Gastrocnemius, soleus) -Knee passive extension / Flexion (Hamstrings, Quadriceps) -Hip abduction and adduction (Gluteus Medius, Adductors) - sometric strengthening (Hold 5 sec, IO reps 2 sets) - Quadriceps sets, Hamstring sets, Gluteal sets. -Heel slides - eated ankle pumps (10-15 reps)	- Knee flexion and extension progress from 0-30° of knee flexion initially to 0-54° - Hip flexion: increase from 0-30° to 0-57° - Focus on gentle passive range of motion without overstretching the hamstring muscle - Gentle calf stretches and ankle pumps
(Dayl-3 months) Week 3-6	Gradual weight bearing, muscle activation, stability training	-Weight - bearing progression -parallel bar standing -Active assisted and active ROM (10 reps 3 sets) -Straight leg raise (quadriceps, hip flexors) - Hip extension and abduction (gluteus maximus and Medius)	-Knee flexion progress to 0-87° -Hip flexion 0-90° -Ankle ROM: -Focus on unprovmg dorsiflexion and plantarflexion for gait
		-Ankle dorsiflexion/plantarflexion -Functional training (sit to stand 5-10 reps 2 sets -Gait training with walker -Bridging (hold 5sec 10 reps)	training and weight bearing tolerance -Focus on progressing weight bearing tolerance with controlled knee and hip movements to avoid overstretching of hamstrings
Phase 2: 3-6 months	Strengthening, improving balance and co-ordination	-Strengthening exercises (10-15 reps 2sets) -Mini squats (with support) -bridging (hold 5sec) -Gait training -Parallel bar walking -Single leg standing (with support) -step-ups (on to a low step) (10 reps	-Knee flexion 0-110° by end of this phase to allow for proper squatting -Hip flexion 0-115° to facilitate dynamic movements -Ankle ROM: 0-10° dorsiflexion and full plantar

		per leg) -side lying raises (with resistance band) (hold 10- 15 sec) (3reps per leg)	flexion -Controlled progression of knee and hip ROM to ensure functional range for strengthening exercises like mini squats, bridging, and step ups
6-9 Months	Advanced strengthening, gait refinement and endurance	-Strength and endurance training (10-15 reps, 3 sets) -step-ups (quadriceps, glutes, hamstrings) -Cycling (10-15 mins) -Obstacle walking, incline walking -Proprioception and agility (balance board exercises)	-Knee flexion: 0-125° by end this phase for more advanced strengthening and cycling -Hip flexion: 0-128° -Ankle ROM: full dorsiflexion and plantar flexion
			-Full ROM at hip and knee 1s essential for more dynamic exercises. focus on joint mobility in functional movements
9-12 months	Functional independence and maintenance of strength and mobility	-Strength and mobility maintenance (squats, TheraBand exercises) -Gait and endurance training (walking and side walking)	-Knee flexion: 0-125° -Hip flexion: 0-130° -Ankle ROM: full range with no restrictions -Emphasize flexibility and dynamic stretches, including hamstring stretches to ensure proper lengthening after hamstring release

RESULT



The bar graph titled "Postoperative Improvement in GMFM-88 scores Across Dimensions" illustrates the progression of motor function in a child post-hamstring release surgery, measured using the GMFM-88 (Gross Motor Function Measure-88) scale. It compares scores in five functional dimensions at three time points:
Red bars: 1st Day Post-Cast Removal Green bars: 6 Months Post-Surgery Blue bars: 12 Months Post-Surgery

Observations by Dimension:

Lying & Rolling:

Steady improvement over time: ~35% (1st day)-+ ~78% (6 months)-+ ~86% (12 months)
ndicates early recovery in basic mobility.

Sitting:

Starts at ~41%, improves to ~80% at 6 months, and ~95% by 12 months Indicates major functional gain and trunk control improvement.

3. Crawling & Kneeling:

Significant increase: ~10%-----+ ~65%-----+ ~88%

Indicates gradual but strong progression in transitional movements

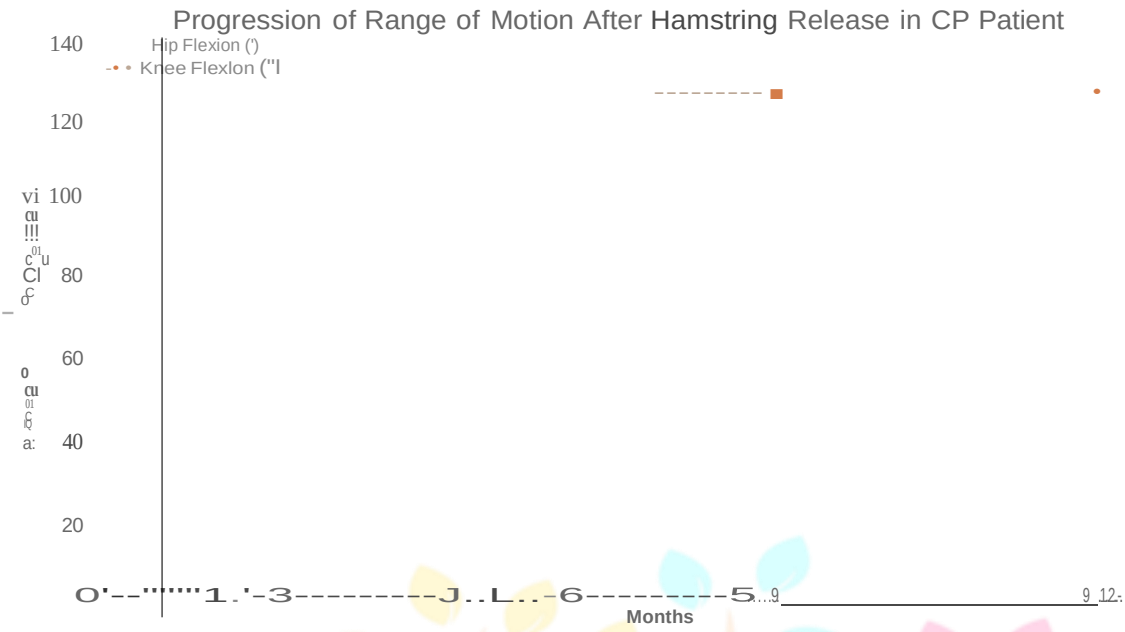
4. Standing:

Starts very low (~7%), improves to ~15%, and stabilizes at that level by 12 months Shows limited recovery, possibly due to persistent postural or strength challenges 5 Walking, Running & Jumping:

Begins almost at 0%, rises to ~33% at 6 months, and ~36% at 12 months

Reflects slow improvement in advanced mobility, often the most challenging area for post-op recovery.
Overall Interpretation:

The graph demonstrates significant functional recovery over time, especially in basic and intermediate motor tasks. While advanced skills like walking and jumping show improvement, progress is slower-likely due to higher coordination and strength demands



This line graph titled "Progression of Range of Motion After Hamstring Release in CP Patient" illustrates the improvement in hip and knee flexion range of motion (ROM) over a 12-month period in a cerebral palsy (CP) patient following hamstring release surgery.

X-Axis:

Time intervals in months:

1-3
3-6
6-9
9-12

Y-Axis:

Range of Motion in Degrees for flexion movements (from 0° to 140°) Lines:

Yellow solid line with circles: Hip flexion ROM Orange dashed line with squares: Knee flexion ROM

Observations:

1. 1-3 Months:

Hip flexion was observed to be 58° and knee flexion to be 55°

2. 3-6 Months:

Hip ROM was observed to be 115° Knee ROM to be 110°

Indicates significant early gains due to rehabilitation and soft tissue recovery.

3. 6-9 Months:

Hip was observed to be 125° and Knee to be 123°

Continued improvement, but at a slower rate than the initial months

4. 9-12 Months:

Hip flexion was observed to be 128° and Knee flexion to be 125° Gains plateau, indicating near-maximal recovery in passive ROM. Interpretation:

This graph shows substantial improvement in hip and knee flexion ROM within the first 6 months post-surgery, with a gradual plateauing thereafter. This pattern supports the importance of early and consistent physiotherapy intervention following hamstring release in CP patients

According to the article, the prescribed exercises have been found to be effective for individuals recovering from bilateral hamstring release and bilateral semitendinosus to adductor transfer and bilateral patellar plication surgery, The implementation of these exercises has consistently yielded positive clinical outcomes contributing to enhanced functional recovery and a marked improvement in the patient's overall condition [22]

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ANNEXURE

APPENDIX:]

GROSS MOTOR FUNCTION MEASURE (GMFM) SCORE SHEET (GMFM-88 and GMFM-66 scoring)

Child's Name: ID#:

Assessment Date: GMFCS Level"

year/ month/ day ☐ ☐ ☐ ☐ ☐

Date of Birth: year/ month/ day II Ill IV V

Chronological Age: year/ month/ day Evaluator's Name:

Testing Condition (e.g., room, clothing, time, others present):

Contact for Research Group:

CanChild Centre for Childhood Disability Research, Institute for Applied Health Sciences, McMaster University, 1400 Main St. W., Room 408, Hamilton, ON Canada LBS 1C7

Email: canchild@mcmaster.caWebsite:www.canchild.ca

[1]GMFCS level Is a rating of severity of motor function. DefInItions for Iha GMFCS-E&R (expanded & revised) are found In Palisano et al. (2008). Developmental Medicine & Child Neurology. 50:744-750 and in the GMAE-2 scoring software. http:/lmotorgroW1h.canchild.ca/en/GMFCS/resources/GMFCS·ER.pdf

Check (3) the appropriate score: if an item is not tested (NT), circle the item number on the right column

Item	A: LYING & ROLLING	SCORE	NT
6.	WITHEXTREMITIES SYI«METRICAL ..... oD 10		SUP,HEAD IN MIDLINE: MNS HEAD 20 30 1.
7.	THEOTHER..... o0 10		SUP: BRINGS HANDS TOMIOUI<E.FIIGERS OIIEv.TTH 20 30 2.
8.	45•..... o0 10		SUP. LIFTSHEA0 20 30 3.
9.	RANGE..... o0 10		SUP: FL.EXES R HP& Mt,EE THROUGHFULL 20 30 4.
10.	KNEETHROUGHFULLRANGE..... o0 10		SUP:FLEXES L HP& 20 30 5.
11.	AAM.11',NDCROSSESMIDLINE TOWAAD TOY..... o0 10		SUP:REACHES OIJTWITH R 20 30 6.
12.	TOWARD TOY..... oD	, O	SUP:REACHES OIJTWITH L ARM.HAM:!CROSSES MIOLINE 20 30 7.
13.	SIDE..... o0 10		SUP: ROUS TOPROVER R 20 30 8.
14.	SIDE..... o0 10		SUP:ROUS TOPROVER L 20 30 9.
15.	HEADUPRIGIfT..... o0 10		PR:uns 20 30 10.
16.	LIFTSHEADIJ'RIGHT.EUIOWS EXT, MST RAISED..... o0 10		PR ON FOREARMS: 20 30 11.
17.	FOREARM.FULLY EXTENDS OI'POSITE ARMFORWARD..... o0 , O 20		PR ON FOREARMS: WEIGHTOii R 30 12.
18.	LFOREARM.FUUyEXTENDS OPI'OSITE ARMFORWARD ..... o0 , O 20		PR ON FOREARMS: WEIGHTON 30 13.
19.	SIDE..... o0 , O 20		PR: ROUS TO SUP OVER R 30 14.
20.	..... o0 , O 20		PR:ROI.LS TOSUPMR L SIDE 30 15.
21.	..... o0 10		PR:PIVOTS TO R 90°USINGEXT!IE/,IITIES 20 30 16.
22.	..... o0 , O 20		PR:PIVOTS ro L 90• USING EXTIIDIITIES 30 17.

TOTAL DIMENSION A

Item	B:SITTING	SCORE	NT
18.	SUP,HANDS GRASPED BY EXAMINER:PIJLLSSELFTOSITTINGWITHIEADCONTROL..... o0 , O	20 30	18.
19.	SUP:ROUS TOR SDE:ATTAINS SITTING ..... o0 , O	20 30	19.
20.	SUP:ROUS TO L SIDE.ATTAINS SITTING ..... oD , O	20 30	20.
21.	SIT ON MAT, SUPPORTED ATTHORAX BYTHERAPIST:LIFTSHEADUPRIGHT, IMtll'AINS 3 SECONDS ..... o0 , O	20 30	21.
2.	SIT ON MAT, SUPPORTED AT THORAX BY THERAPIST: LfTSHEAD MIDLINE.MAINTAINS 10 SECONDS..... o0 , O	20 30	22.
23.	SIT ON MAT, ARM(S) PROPPING: MAIfIAINS,5 SECONDS ..... o0 , O	20 30	23.
24.	SIT ON MAT:MAINTAIN,ARM$fREE,3 SECCNJS o0 10	20 30	24.
25.	SIT ON MAT WITH SMALL TOY INFRONT:LEANS FORWNID, TOUCHESTOY, RE-ERECTS WITHOO'T ARMPROf'PI/KI ..... oD , O	20 30	25.
26.	SIT ON MAT:TOOCIES TOYPLACED 45• IIEHHJOULO'S R SOE. R TOSTART..... oD , O	20 30	26.
27.	SIT ON MAT:TOUCHES TOYPI.ACED 45°BEHTHJCHIID'SL SI>E.RETURNSTOSTART..... R o0 , O	20 30	27.
28.	SIDE SIT:MIJNTAINS,ARMSFRE£, 5 SECONDS..... o0 , O	20 30	28.
29.	L SIDE SIT: MAMAtlS,ARMS AIEE, 5 SECOIIDS ..... oD 10	20 30	29.
30.	SIT ON MAT:I.OYrU!S TOPR WITH CONTROL ..... o0 , O	20 30	30.
31.	SIT ON MAT WITH FEET IN FRONT: ATTAINS 4 POINTMR RsDE..... o0 , O	20 30	31.
32.	SIT ON MAT WITH FEET IN FRONT: ATTAINS 4 PONT OVER L SIDE..... o0 10	20 30	32.

33.	SIT ON MAT:PIVOTS 90°, \',INIQUT A!lt,ISASSISffIG	○0	10	20	30	33.
34.	SIT ON BENCH: MAINTAINS,ARIISANDFEETFREE,10SECONJS.....	○0	,○	20	30	34.
35.	STD: ATTAINSSITONSMAU 8ENCH.....	○0	10	20	30	35.
36.	ON THE FLOOR: ATTAINSSITONSMAUBENCH.....	○0	,○	20	30	36.
37.	ON THE FLOOR: ATTAINSSITOilLARGE BEHCIf	○0	,○	20	30	37.

TOTAL DIMENSION **B**

Item	C:CRAWLING & KNEELING	SCORE	NT
38.	PR: CIEEPS FORWARD 1.Bm(6')	DO 10 20 30	38.
39.	4 POINT:IIAINTAm. WEIGHT ON HAlfISAND KNEES. 10 SECOt«JS	oO 10 20 30	39.
40.	4 POINT:ATTAINS SITARMS FREE.	oO 10 20 30	40.
41.		oO	
42.	LEVEL	oO	
43.	LEVEL	oO	
44.	1.Bm(6')	oO	
45.	4 POINT:CRAWISREal'ROCAI.LYFORWAR01.8m (6')	oO	
46.		oO	
47.	STEPSONHANDSANOKNEESHET	oO	
48.	SECONDS	oO	
49.	USINGARMS.MAINTAINS.AAMFSREE. 10 SECONDS	oO	
50.	t.tAfflAINS,ARMSFREE. 10SECONDS	oO	
51.		oO	

PR: ATTAINS4 POtfl, WEIGHT ON w.NDS 00 IOEcS 10 20 30 41.
4 POINT:REACHES FORWARD WITH R ARM,HAND A80VE SHOLJIDER 10 20 30 42.
4 POINT:REAOES FORWARD WITH L ARM,IW(I AllOVESI()II.DER 10 20 30 43.
4 POINT:CRAWIS OR HITCHES FORWARD 10 20 30 44.
10 20 30 45.
4 POINT:CRAWIS UP 4 STEPS ONHANOSAND KNrnlfEET 10 20 30 46.
4 POINT:CRAWISBACKWAROS OOWN 4 10 20 30 47.
SIT ON MAT: ATTAINSHIGHKNUSNG/\RMS,MAINTAINS,AJWSfREE,10 10 20 30 48.
HIGH KN:ATTAINS HALF KNON R KNEE 10 20 30 49.
HIGH KN:ATTAINS HAIHNON L KNEEUSINGARMS. 10 20 30 50.
HIGH KN:KN WAI.XS FORWARD 10 STEPS,ARMSFRI:E 10 20 30 51.

TOTAL DIMENSION C

Item	D:STANDING	SCORE	NT
52.		ON THE FLOOR:PIIIS TOSTD1,T LARGE oO 1D 20 30	52.
53.	STD: MAINTAINS.ARMS FREE. 3 SECONDS	oO 1D 20 30	53.
54.	STD: OOLOOIG ONTDIAAGEBENCH WITHONE HAND, LFTS R FOOT, 3 SECONDS	oO 1D 20 30	54.
55.	STD:I«:JLOOIGONT0IAAGEBENCHWITHONEHAND,IFTS L FOOT, 3 SECONDS	oO ,□ 20 30	55.
56.		STD:MAINTAINS.ARMS FREE. 20 SECONDS oO 1D 20 30	56.
57.		sm:LFTs L Foor. ARMSFREE. 10sEOONDS	,□ 20 30 57.
58.		STD: LFTS R FOOT, ARMSFREE, 10 SECONDS oO 1D 20 30	58.
59.	SIT ON SMALL BENCH:AITAI<SSTOWITIIOUTUSINGARMS	oO ,□ 20 30	59.
60.	USIMGARMS	HIGH KN:ATTAINSSTOTHROUGHIW.F KIION R KNEE,WITH0UT oO 1D 20 30	60.
61.	I.51NGARMS	HIGH KN:ATTAi STD THROUGH HALF IOI ON L KNEE, wmrour oO 1D 20 30	61.
62.		STD:LOWE.RS TOSIT *OH* FLOOR WITH CONTROL. ARMS FREE 1D 20 30	62.
63.		STD:ATTAINS SOUI,T, AAMS FREE oO 1D 20 30	63.
64.		STD:PICI(S UP OBJECT FRON FLOOR, ARMS FREE,RfMNS TO ST'-'10 oO 10 20 30	64.

TOTAL DIMENSION D

34

Item E: WALKI G,**RUNNING** & **JUMPING** **SCORE** **NT**

65.

R.....

66.

STD, 2 HANDS ON LARGE BENCH: C1USES **5** fffil'STO

oD ,□ 20 30 65.

STD,2HANDS ON LARGE BENCH: CRI.ISES 5 STEPS TO L-·-

oO 10 20 30 66.

fil STO. 2 H DSHELO: FORW 10 STEPS .... oD 10 20 30 67

68.

STD, 1HAND HELD: MJCSFORWARD 10 STEPS

oO , O 20 30 68.

69. STD:

FORWARD 10 STEPS·····

-- oO ,0 20 30 **69.**

70. STD:

FORWARD 10 STEPS STOPS TURNS 180°, RETLRIIS .....

oD 10 20 30 **70**

71. STO:W ..... 8'aYtARD

10 STEPS ..... oD 10 20 30 71.

72. STO

fORWIJfD 10 STa>s CAARYtlG A I>AG£ OBLCT H 2 IWCIS.....

oD ,O 20 30 *n.*

73. STD: FORW 10 CONSEaJTMmPS BETWEEN PNW.LEl. I 20lm (8'")N>

oO , O 20 30 73.

74. STD.W RIIWAIID 10 CU1IYE STEPS CN A STIIAG(T I.Nc 2cm WD£ .....

oD 10 20 30 74.

75. STD: S1EPS ST AT LEVEL R

FOOTI.EADOtO ..... oD 10 20 30 75

76. STO: STEPSO'JER ATELEVEL L FOOTI..EADIHG ..... oD , O

20 30 76

n. STD.III.INS 4.Sm(15'1.STOPS&RETURNS ..... oO , O 20 30 77

8 STD SAU. TH RFOOT ○0 ,O 20 30 78

79. STD: IIAI.I. W!IH l FOOT
○0 , O 20 30 79.

80. STD. JJ1.1PS30cm (1T) IIGH, 110TH rm TNiEOUSL.Y ○0 ,O
20 30 80

81. STD JJI.IPS F 30 cm (12"), BOTH fEET TAIEOUSIY ○0 10
20 30 **81**

82 STDO R FOOT: fo>SCIII R FOOT 10 TIMESWIIltNA 60cm (24') CIRQ.E ○0 , O 20 30 82

83. STO ON L FOOT IG'SOHL FOOT 10n.ov.mt A 60cm (24") ClflQ.E ○0 , O 20 30 83.

84 STD,HOLDING 1 RAIL W SUP SIEPS HOU>ING 1 ML, TER Fm ○0 ,O 20 30 84.

85. STD,HOLDING 1RAJL: 4 STEPS HCJ..DlhO 1 RAI. TEANATHI A.IT ○0 , 0
20 30 85

86. STD W UP 4 STEJ'S. -.LTERMT FE£1
○D 10 20 30 86.

87. STO: DOWN 4 SIEPS.M. FEfl ○D ,O
20 30 87.

88 STD ON 15cm (6") STEP:...uFSorF aonmET TN€0USL.'1' ○0 10 20 30 88

TOTAL D MENSION E _____

Was this assessment indicative of is child's"reg!Aar" performance? YES D ○D
COMMENTS:

GMFM BSUMMARYSCORE

DIMENSION	CALCULATION OF DI	GOAL AREA
Total Dimension A	=	
L ng Rolling	x 100:::: %	A.☐
	51	

. Sitting	Tolal Dimer\Sion B 60	= 60	100=	%	B.D
C. Cra ng & Kneeling	T al D mansion C 42	= 42	100 =	%	.☐
Stanaing	Total Dimension D 39	= 39	100 =	%	.☐
E. Wa ing, Running J mp·ng	Total Dimension E 72	= 72	100=	%	.☐

TOTAL SCORE• %A D o/oE
ota ions

• = =
5

GOAL TOTALSCORE"' Sum of 1mension idenb ed as a go area
#Of goal areas

'' = %

_____ to _____
95% Confidence Intervals

ESTING WITH AIDS/ORTHOSESUSING THE GMFM-88

Indicate below wi1h e check (4) which aid/orthosis was used and what dimension it was rslapplied. (There mey be more than one). AID Olmen

Orthosis		Olmenmnnr---	
Rollalor/pusher	D	Hip Control	0
Walker	D	Knee Control	D
H Frame crutches	D	Ankle-foot Control	D
Crutches	D	Fool Control	D
QuadCane	0	Shoes	D
cane	D	None	D
None	D	Other	0
Other	D	(please !Jlllclfy)	

(please specify)

GMFM-88 SUMMARY SCORE USING AIDS/ORTHOSES

DIMENSION	CALCULATION OF DIMENSION%SCORES			AREA	GOAL
F. Lying& Rolling	Total Dimension A 51	= 51	x 100 = %	A.☐	
G. Silting	Total Dimension B 60	= 60	x 100 = %	B.☐	
H. Crawling & Kneeling	Total Dimension C 42	= 42	x 100: %	.☐	
I. Standing	Total DimensionD 39	= 39	x 100 = %	D.☐	
J. Walking,Running & Jumping	Total Dimension E 72	= 72	x 100 = %	E.☐	

TOTAL **SCORE=** $\frac{\%A + \%B + \%C + \%D + \%E}{\text{Total\# of Dimensions}}$

$= \frac{\quad}{\quad} = \quad \%5$

GOAL TOTAL **SCORE=** $\frac{\text{Sum of \%scores for each dimension identifiedas a goal area}}{\text{\# of Goal areas}}$

$= \frac{\quad}{\quad} = \quad \%$

PPENDIX:2

DIMENSION	MAXSCORE	DAY1SCORE	DAY 1%	6 MONTHSCORE	6 MONTHS %	12 MONTH SCORE	12 MONTHS%
l ngandrolling	51	18	35%	40	78%	44	86%
Siitting	60	25	41%	48	80%	57	95%
Craw ngandkneeling	42	0	0%	28	66%	37	88%
Standing	39	1	2%	6	15%	6	15%
Walking,runningandjumping	72	0	0%	24	33%	26	36%
TOTALSCORE		78	15%	146	5496	170	64%