Management of the Knee Problems in Spastic Cerebral Palsy

Abstract

Two common knee problems in cerebral palsy are increased knee flexion during stance phase and reduced knee flexion during the swing phase of gait. We reviewed the recent literature and based on that, we formed this review. Hamstring spasticity, quadriceps weakness, soleus weakness, and lever-arm dysfunction are few factors which lead to increased knee flexion during stance phase. Rectus spasticity diminishes knee flexion in the swing. Resulting gait-stiff knee gait interferes with ground clearance. Both gait patterns result into esthetically poor gait and increased energy consumption. Knee flexion gait may lead to pain in the knee. Natural history of knee flexion gait suggests deterioration over time. In the early stage, these gait abnormalities are managed by nonoperative treatment. Cases in which nonoperative measures fail or advance cases need surgical treatment. Various variables which are taken into consideration before selecting a particular treatment option are described. We also present an algorithm for decision-making. Nonsurgical options and surgical procedures are discussed.

Keywords: *Cerebral palsy, diplegia, knee flexion stiff knee*

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Introduction

Knee joint problems are common in cerebral palsy (CP).[1] Excessive knee flexion during stance phase and limited knee flexion during swing phase are two common clinical problems.[1]

This article discusses the causes and management of these gait deviations. In the past three decades, our understanding of biomechanics of these problems has resulted into better management strategies. Information which can be applied to clinical practice is included in this review.

Materials and Methods

We searched PubMed, Medline, Cochrane, CINAHL, and Google Scholar search of the English literature. We searched database from January 1988 to January 2018 (30 years) using following keywords CP, knee flexion, knee extension, flexion contracture, hamstring contracture, rectus contracture, crouch gait, stiff-knee gait, nonoperative treatment of cerebral palsy, hamstring lengthening, hamstring transfer, distal femur osteotomy, femur shortening osteotomy, complications, and functional outcome of crouch gait surgery. Cross-references of these articles were also read, and all those articles were also included in the study.

We have included Level IV and above original studies, review articles, systemic review, and meta-analysis for the study. We have excluded single case report, comments, letter to editor, and duplication of the article.

Results

We found 367 publications initially. Of these, 300 articles were found to be potentially pertinent. Finally, 289 original articles and 11 review articles were utilized for preparing this review (which fulfilled inclusion and exclusion criteria). These articles were utilized to delineate etiology, natural history of crouch gait, clinical examination and radiological investigations, important variables of decision-making, management of crouch gait and SKG, and complications of the various surgeries.

Knee movements are controlled by quadriceps and hamstrings. Imbalance between these two groups of muscles results into gait deviation. This may result into gait where there is more flexion during the stance phase-flexed knee gait (FKG) or diminished knee flexion during the swing phase-SKG.

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Flexed Knee Gait

At initial contact, the knee is slightly flexed approximately 5°. During weight acceptance, the knee flexes approximately 10°–15°. In middle stance phase, the knee gradually goes into extension. In late stance phase, the knee starts rapid knee flexion. Knee flexes to 60° which facilitates ground clearance of the limb. Knee extends again during the terminal swing preparing the limb for the next initial contact.

Causes of Flexed Knee Gait

FKG results when knee extensors are not able to overcome forces which produce knee flexion. Various mechanisms lead to FKG. They affect the knee directly or indirectly. Both types of mechanisms should be taken into consideration while managing the patient with FKG. Brief description about them will be useful to understand the management principles.

Hamstrings shortening

Hamstrings are biarticular muscles. It is responsible for the hip extension and knee flexion. The child with CP has spasticity of the hamstrings early in the life. Uncontrolled spasticity of hamstrings leads to the contracture (true shortening) of the hamstrings.[2,3] Short and spastic hamstrings contribute to the flexion contracture of the knee joint.

Fixed flexion deformity of the knee

During midstance phase, knee extends fully. Fixed flexion deformity (FFD) of the knee does not allow the knee to extend fully. When person with knee FFD tries to stand upright or walk, demand of quadriceps increases.[4]

Quadriceps weakness

Quadriceps is antigravity muscle which is responsible for the upright position in the human. When quadriceps is weak, knee tends to buckle during the stance phase.[5]

Patella tendon lengthening

FKG demands increased and continuous quadriceps demands which result in stretching of the patellar tendon over time.[6] Elongation of the patella tendon further increases the weakness of the quadriceps.[7]

Soleus weakness

Soleus is an important stabilizer of tibia over the foot. It pulls the tibia from behind and prevents excessive forward movement of the leg during a midstance phase.[4] When soleus is weak, tibia moves forward excessively over the ankle resulting into excessive dorsiflexion during a midstance phase. To maintain the body upright over the dorsiflexed ankle, more knee flexion takes place.

There are various mechanisms which can lead to soleus weakness.[8,9] Children with CP have less volume of muscle compared to typically developing children.[10] As the amount of muscle is directly proportionate to the strength of the muscle, these children have less strength. Ankle-foot orthosis may provide stability at the ankle but restricts movement at ankle joint and may lead to disuse weakness of soleus.[11] Surgical lengthening of soleus before the age of 7 years may be iatrogenic cause for crouch gait.[12] Soleus requires lever arm of the foot for its efficient function. When a child has planovalgus deformity of the foot, this lever arm is reduced, and this makes soleus functionally less efficient. External tibial torsion also reduces the length of the lever arm.[4] Planovalgus foot and tibial extortion are called lever-arm disorders.[4]

Effect of Flexed Knee Gait on Energy Consumption

In normal gait, during midstance, ground reaction force (GRF) passes anterior to the center of the knee joint. When GRF is anterior to the center of the knee joint, knee remains stable by the posterior capsule and no muscle action is required[4] [Figure 1a]. This is energy-saving mechanism. As knee starts remaining inflexion, GRF passes posterior to the center of the knee joint [Figure 1b]. Now, the knee cannot achieve passive stability by posterior capsule. Quadriceps has to stabilize the knee so that person can remain upright. In normal person, quadriceps work only for small portion of the stance phase. However, when knee remains flexed during stance phase, quadriceps has to work during the whole of the stance phase. Both of these factors increase quadriceps demand and the energy expenditure while walking. Due to high energy consumption, the person walking with FKG gets fatigued faster compared to their normal peers.[13]

Excessive quadriceps action results into repetitive microtrauma at the insertion sites of the quadriceps and patellar tendons, leading to fragmentation of the inferior pole of the patella or tibial tubercle [Figure 2].

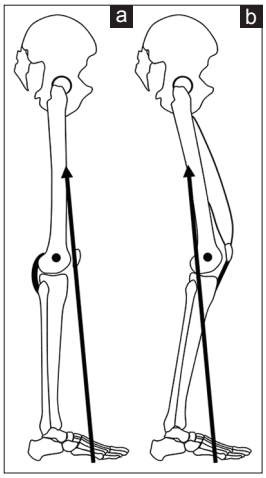

Figure 1: (a) When knee can achieve full knee extension, ground reaction force passes anterior to knee joint. (b) When knee has fixed flexion deformity, ground reaction force passes posterior to center of knee joint. Quadriceps action is required to maintain upright posture

Loading over the patellofemoral joint increases when one walks with FKG. This results in anterior knee pain. One out of five children with CP will have knee pain.[14] Knee pain can also be due to patellar instability because of increased femoral anteversion or tibial extortion.[14] The patella alta decreases the moment arm of quadriceps during terminal knee extension, which contributes to the further weakening of extensor mechanism.

Natural History

Insight into the natural progression of gait function in CP is essential. However, there is a paucity of information available in the literature about the natural progression of the condition, due in part to the fact that children presenting to the health-care system commonly receive treatment. Longitudinal assessment of children with CP using 3-dimensional gait analysis over a period of about 4 years showed deterioration in knee function.[12] Hamstring contracture also increases over time.[15,16] Healthy children, on the other hand, do not deteriorate over time.

Clinical Examination

At the initiation of clinical evaluation, Gross Motor Function Classification System (GMFCS) level and topographic pattern should be identified.[17] As the outcome of intervention in the presence of spasticity and dystonia is very different, every effort should be invested to check associated involuntary movement in the child with CP. A child should be asked for knee pain, recent deterioration of the function, and decreased walking distance.[14,18]

In most of the children, the problem at the knee is associated with problems with hip and ankle; hence, the child is evaluated at all the three levels. These joints are assessed in all the three planes. The range of motion, muscle length estimation, and strength evaluation are carried out. Torsion of femur and tibia is observed. The foot is evaluated for a midfoot break.

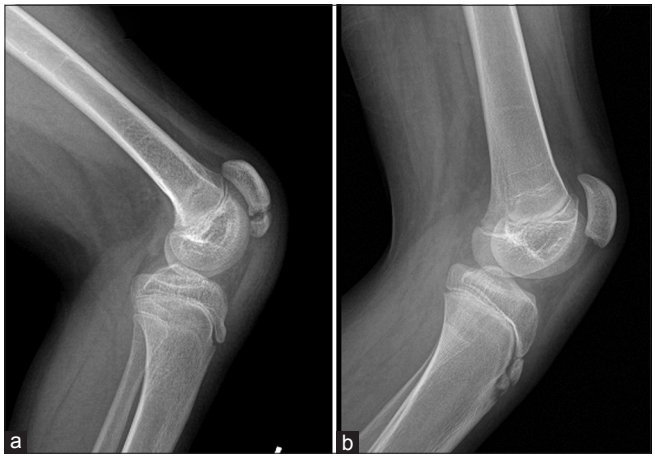

Figure 2: Lateral radiographs of the knee joint showing fragmentation of the lower pole of the patella (a) and the tibial tuberosity (b) in spastic diplegic walking with crouch gait

Hamstrings length is evaluated by popliteal angle measurement.[19] Hip flexion deformity shifts the ischial tuberosity proximally and can give a false impression of hamstrings shortening. To overcome this point, bilateral popliteal angle test is carried out. For bilateral popliteal angle test, opposite hip is flexed till anterior and posterior superior iliac spines are perpendicular to an examination table.[19] Hamstrings surgery is indicated if the bilateral popliteal angle is more than 50°.[20] Rectus femoris length is assessed by Duncan-Ely test for rectus femoris. While evaluating muscle length both dynamic and static lengths are evaluated.

Tenderness at the lower pole of the patella and tibial tuberosity should be checked. Kneel walking may result into callosities and thickened bursa in the prepatellar region. The position of the patella should be examined to detect the patella alta.[21]

Knee flexion deformity should be checked with the hip in the maximum extended position and ankle in plantarflexed position to relax hamstring and gastrocnemius, respectively.[21]

Estimation of muscle power is difficult in CP as many children have a problem of selective control. A presence of involuntary movements and lack of coordination make it even more challenging. The muscle power can be done with manual muscle testing or handheld dynamometer. It is imperative to evaluate the muscle power of agonist and antagonist to detect muscle imbalance across the joint. A degree of extension lag is also noted.[19]

Video gait analysis is preferred over observational gait analysis. Instrumented gait analysis provides additional information which cannot be obtained video gait analysis. Jump-knee gait, apparent equinus gait, crouch gait, genu recurvatum gait, and SKG are the common gait deviations. Identifying the gait pattern helps in planning the treatment.[4]

Radiological Examination

Fragmentation of the lower pole of the patella or tibial tuberosity is common features in long standing crouch gait. Presence and severity of patella alta are documented by various indices such as Insall-Salvati index and Koshino index[21] [Figure 3].

Variables to be Considered for the Treatment

There are many variables which influence the treatment of knee flexion in CP.

Spasticity/contracture

Spasticity and contracture have to be differentiated before any intervention. The spasticity must be reduced with nonoperative interventions. Surgical intervention for the spasticity control can lead to the weakness of the muscle. The fixed contracture does not respond to the nonoperative

interventions. Differentiating spasticity in contracture in many clinical situations is hard. Most reliable method to distinguish between them is examination under anesthesia. One must do the examination under anesthesia before releasing the contracture.[19]

Age of patient

The gait pattern changes with the growth of the child[15] and it matures at around 7 years of age. The surgical intervention to correct knee contracture should be deferred till that age.[22] The children should be treated with nonoperative intervention in younger age group. The fixed contracture should be dealt after 7 years of age with soft-tissue procedures. The bony contracture should be treated in adolescent period. The surgical correction of flexion deformity of the knee and patella alta also varies with the age of the child.

Gross motor function classification system

GMFCS plays a major role in the decision-making of intervention for correction of the knee contracture.[22] GMFCS I, II, and III children should be treated aggressively to maintain the walking ability. On the other hand, contracture in GMFCS IV and V is treated less aggressively. The weakness of the rectus by surgical intervention may worsen the crouch gait in GMFCS III and IV.[23]

A severity of contracture/flexion deformity

The option for the surgical correction of flexion deformity is different according to the severity of the flexion contracture across the knee joint. Mild deformity can be addressed with the soft-tissue lengthening/transfer of the hamstrings. The moderate deformity can be treated with the extension osteotomy of the distal femur, and severe deformity (FFD more than 40) must be managed with the shortening osteotomy of the distal femur to decrease the stretch of neurovascular tissues.[24]

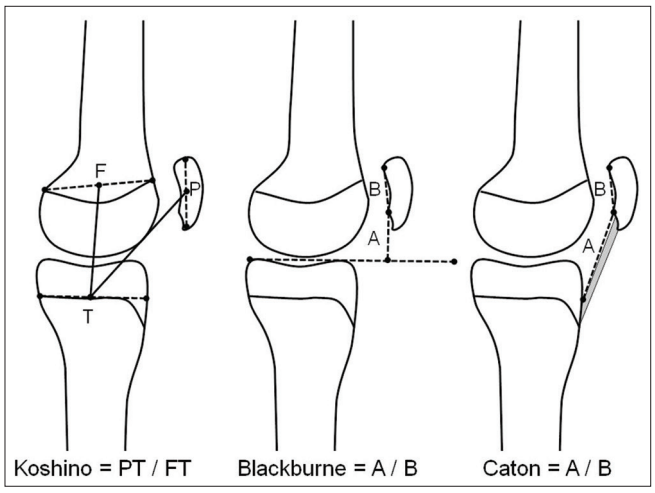

Figure 3: Schematic diagram of lateral radiograph of the knee joint showing the measurement of Koshino, Blackburne, and Caton index

Extensor muscle weakness and patella alta

Extensor muscle weakness plays a very significant role in the surgical correction. Uncorrected extensor muscle weakness leads to the recurrence of the flexion deformity. Patella alta should be addressed with shortening of the extensor mechanism and imbalance between flexion, and extensor muscle should be treated with hamstring transfer.[25]

Spasticity of extensor muscle

Unrecognized spastic extensor (rectus femoris) during surgical intervention can lead to SKG/genu recurvatum gait. One must carefully examines the knee extensor power and spasticity before addressing the flexion deformity of the knee. Associated contracture of the rectus femoris should be dealt with rectus femoris transfer/lengthening procedure.[25]

Nonoperative Treatment

Spasticity along with other factors leads to muscle contracture. The aim of nonoperative treatment is to prevent or delay the process of spastic muscles developing contracture.

The most widely used treatment modality is physiotherapy which stretches the hamstrings muscles by various forms of exercises. In most of the patients, the amount of time which can be allocated for therapy is limited. To increase the duration of stretching, splints can be used. The splint shown in Figure 4 can be used to stretch the hamstrings muscles. This splint maintains the hip in flexion and at the same time extends the knee, so it stretches the hamstrings. Casting can also be considered for the same purpose. Hamstrings cross both hip and knee joints, so it is inconvenient to immobilize both joints in the position which stretches hamstrings. Immobilizing the knee in extension and allowing the child to sit, stretches the hamstrings. Evidence demonstrating the efficacy of various forms of stretching on the hamstrings is lacking.

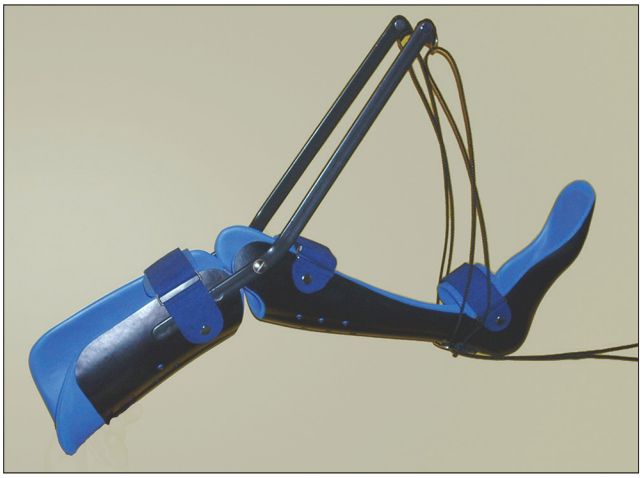

Figure 4: Orthosis to stretch the hamstrings

When hamstrings are dynamically short but still interfere with the walking, one can reduce the spasticity by neuromuscular blocks. Botulinum neurotoxin is one option. Knee kinematics improves after injections, but improvement disappears by 12 weeks.[26-28] Alcohol is less expensive option compared to botulinum. However, due to adverse events such as transient muscle discomfort, skin injury secondary to inadvertent superficial injections, and in some cases permanent peripheral nerve injury, alcohol injection is less frequently used.[29]

Orthosis to control the knee flexion should not span the knee joint. If knee joint is immobilized, the knee cannot flex during the swing phase resulting into further gait deviations. Dorsiflexion restricting orthosis[30] controls the knee position during the stance phase by controlling the position of the ankle. It allows knee flexion during the swing phase, so it does not produce additional gait deviation. The prerequisite for such orthosis use is that knee should be passively extended to neutral position.[31] Orthosis effectively controls the knee joint position.[31] If hip and knee joints have flexion contracture of more than 15°, orthosis will not serve its function.[31]

Operative Interventions

Surgical options

Selective dorsal rhizotomy is typically used in generalized spasticity management, in which knee flexion is one component. It is rarely done for hamstrings alone.

Hamstring lengthening with ST transfer

Hamstring contracture is commonly dealt with distal hamstring lengthening/transfer.[32] The indication of the surgical correction is not consistent in the literature. It is indicated in established hamstring contracture with the popliteal angle more than 60 and no flexion deformity of the knee.

Most authors practice medial hamstring lengthening rather than medial and lateral hamstring lengthening. The aponeurotic lengthening of the semimembranosus and z-lengthening of the semitendinosus and Gracilis are universal. These procedures can be done by traditional open approach or percutaneously.[33,34] The biggest disadvantage of isolated distal hamstring lengthening is to increase the risk of anterior pelvic tilt following surgery.

The complete transfer of the hamstring was popular in patients with knee extensor weakness. However, Egger procedure can lead to genu recurvatum deformity. Semitendinosus can be transferred, and other hamstrings can be lengthened to avoid genu recurvatum.[25,35] The patient with mild-to-moderate flexion deformity of the knee, contracture of hamstring with weak extensor of the knee, without spastic rectus femoris is ideal for the hamstring transfer.[36] The semitendinosus can be transferred either to the adductor tubercle or posterior periosteum of the distal femur.[25]

Posterior capsulotom

Moderate flexion deformity of the knee can be addressed with posterior capsulotomy of the knee.[37] The advantage of the soft-tissue procedure involving hamstring release with capsulotomy is early rehabilitation compared to bony surgery. The chances of the sciatic nerve stretches are higher than bony correction of the flexion contracture of the knee.

Distal femur extension osteotomy

Knee flexion deformity (more than 5) cannot be dealt with isolated hamstring lengthening procedure. Hamstring lengthening improves 5° of the flexion contracture of the knee joint. Moderate-to-severe contracture across the knee joint should be treated with the osteotomy of the distal femur to avoid iatrogenic injury to neurovascular tissue and avoid anterior pelvic tilt. The fixed contracture (5°–30°) can be managed with the extensor osteotomy of the distal femur.[38,39] It is combined with the patella tendon shortening procedure.[39-42] A combination of both procedures showed superior results than isolated procedure. It is ideally indicated in adolescent or adult walking with crouch gait with mild-to-moderate flexion deformity of the knee with patella alta [Figure 5].

Femur shortening osteotomy

Children with severe flexion contracture (FFD more than 30) should be dealt with shortening osteotomy of the distal femur to avoid irreversible neurovascular complication.[43] The biggest advantage of this procedure is to maintain the range of motion of the knee joint. Children with severe flexion deformity of the knee joint will lose the terminal flexion of the knee if they have treated with extension osteotomy of the distal femur. The limb length discrepancy is not an issue when this osteotomy is performed bilaterally. The procedure should also be combined with patella tendon shortening procedure.[36] At long term analysis, distal femoral extension osteotomy and patellar tendon advancement

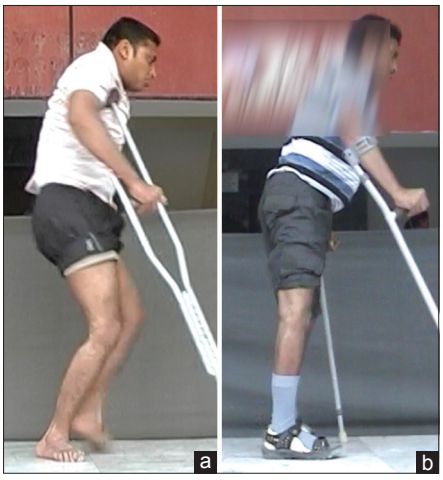

Figure 5: Clinical photograph of the adolescent treated with femur extension osteotomy with patella tendon plication (a) preoperative (b) postoperative

improve stance phase knee extension and knee flexion contracture compared with conventional treatment, but these benefits do not translate to improved activity, participation, or knee pain in early adulthood.[21]

Patella tendon shortening

Patella tendon shortening is indicated in patients with severe elongation of the patella tendon. This surgery is carried out when severe flexion deformity of the knee is corrected by osteotomy. Several methods are described for the patella tendon shortening procedure.[36,44,45] Partial transmit sequence (PTS) is effective for correction of FKG and knee flexion contracture leading to superior stance phase knee extension. However, additional PTS may lead to SKG and a higher increase of anterior pelvic tilt.[6]

Guided growth

Guided growth is a good option to correct flexion deformity of the knee in growing child.[46,47] Mild-to-moderate flexion deformity of the knee in skeletally immature patients is the ideal candidate for the correction of the sagittal deformity across the knee. The staple or eight plates should be placed anteriorly in the distal femur to correct the flexion deformity of the knee. The advantage of this procedure is simple, easy, and effective. However, due to slow correction, the mechanical dysfunction across the knee will be persistent until full correction of the deformity. The results of the correction are unpredictable as under correction and overcorrection are possible due to variable maturity in all children.

- Tibial extorsion can be corrected by derotation osteotomy of the tibia at supramalleolar level.[12] Evaluation of this surgery by 3-dimensional gait analysis showed improvement in biomechanics.[48] Calcaneal lengthening osteotomy corrects the planovalgus foot. It aligns the foot to the direction of walking and also makes the foot as a rigid lever arm. Thus, it improves the lever arm of the soleus and facilitates upright posture[49]
- Authors' preferred choice for the knee flexion correction is described in the flowchart [Figure 6].

Functional outcome of the various interventions

Antigravity muscle strengthening exercise program improves the muscle strength and walking ability.[50] The functional outcome is excellent in long term in selective population with spasticity treated with dorsal rhizotomy and nonoperative interventions.[51] Kinetic, kinematic, and quality of the life also improve following multilevel surgery in ambulatory patients.[52] The mechanics is directly correlated with oxygen consumption and gait efficiency, and indirectly the gait pattern improves. However, adult with CP showed deterioration in pain, fatigue, and functional level.[53] Improvement in kinematics, kinetics, energy consumption, and physiological cost index is seen in medium-term results.[54,55] However, activity participation and functional outcome may not improve in long term.[21]

Complications

Under correction and recurrence of the deformity

Hamstring lengthening improves 5° of the flexion deformity of the knee joint. Many patients have capsular contracture of the knee joint which does not correct with hamstrings surgery. This requires correction by bony surgery. Untreated hip flexor deformity also leads to the recurrence of the deformity. The anterior pelvic tilt also contributes to the recurrence. The recurrence of the deformity is disabling and can be corrected with same principles such as deformity correction, correction of muscle imbalance across the knee, and correction of the patella alta.[56]

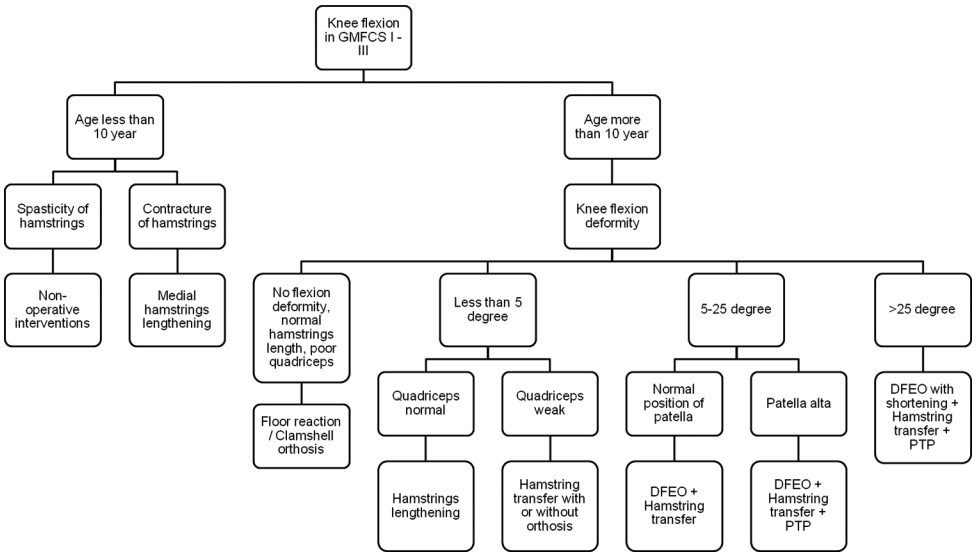

Figure 6: Authors' approach for the management of flexed knee gait in spastic diplegic child. DFEO = Distal femoral extension osteotomy, PTP = Patellar tendon plication

Overcorrection (genu recurvatum)

Inappropriate hamstring lengthening in the absence of the established contracture, associated rectus femoris contracture, and associated gastrocnemius contracture are common causes for the genu recurvatum following the surgery. The surgeon must be careful about the selection of the patient for hamstring lengthening. Power and spasticity of the knee extensor must be evaluated to avoid overcorrection following hamstring lengthening. The spasticity or contracture of the rectus femoris can be treated with distal rectus transfer.[1] The established genu recurvatum is harder to treat as it usually advances. It can be treated initially with dorsiflexion ankle-foot orthosis or Lehnis modification of floor reaction orthosis.[57] The long standing unyielding genu recurvatum can be treated with flexion osteotomy of the distal femur.

Stretching of the neurovascular tissue

Stretching of the sciatic nerve is common (1%–40%) with the severe correction of the flexion deformity of the knee.[24,43,58] It can be common with all types of soft-tissue and bony surgeries. It is not associated with severity of the deformity or amount of correction. Although most cases are neurapraxia, the foot drop adds to the disability. The associated sensory loss is troublesome for the patient and caregivers. It can be prevented by shortening the femur while correcting fixed flexion deformity of the knee joint.

Anterior pelvic tilt

Anterior pelvic tilt is common following hamstring lengthening.[59,60] It is due to the weakness of the hip extensor, reduced hip extension, or associated hip flexor contracture.[61] The hamstring shift should be detected in every case of hamstring surgery to rule out associated hip flexion contracture. If hip flexor (rectus femoris and iliopsoas) contracture is present, it must also be addressed simultaneously to prevent anterior pelvic tilt. The power of gluteus maximus and hamstring should be checked before surgery. It is also associated with distal femur extensor osteotomy with patella tendon advancement. The patella tendon advancement aggravates the anterior pelvic tilt.[62] An adolescent patient with crouch gait without rectus femoris contracture/spasticity should be treated with hamstring transfer to avoid anterior pelvic tilt.[57] Children with anterior pelvic tilt also show increased lumbar lordosis. Mild anterior pelvic tilt can be addressed with a strengthening of the hip extensors. Severe anterior pelvic tilt is disabling and difficult to treat.

Hypertension

It is a rare complication following correction of the flexion deformity of the knee. It is common with correction of the severe deformity with hamstring release and distal femur osteotomy.[63]

Stiff-Knee Gait

Stiff-knee gait is a swing phase gait problem. During swing phase, knee flexes around 60°. This peak knee flexion is achieved at 30% of the swing. This knee flexion is passive and is in response to hip flexion by iliopsoas and push-off power provided by ankle plantar flexors.[64] Rectus is inactive during this phase. However, if rectus is spastic during this phase, it restricts or delays knee flexion resulting into a gait abnormality which is called SKG.[65] Stiff-knee gait results in the problem of ground clearance resulting into dragging of the foot. Excessive wear of the front portion of the shoes is another sign of SKG.

Ideally, this condition should be considered when dynamic electromyographic (EMG) shows abnormal activity in the rectus femoris muscle in the swing phase along with diminished or delayed peak knee flexion during swing phase.[1] However, as this investigation is not readily available, so many clinicians rely on the physical examination test called prone rectus test.[66] The question is how reliable prone rectus test in comparison to dynamic EMG. The prone rectus test is not a specific indicator of rectus tightness or spasticity since it elicits EMG responses in both the rectus femoris and the iliacus in many subjects with CP.[4] On the other hand, Kay *et al*. suggested that this test as a significant predictor of outcome when distal rectus femoris transfer is being considered.[67]

When dynamic EMG is not available to the clinician, the SKG is suspected when there is excessive or rapid wear of the front part of the sole of the shoes. One can try neuromuscular block of a local anesthetic solution or botulinum injection to reduce the action of rectus. If this test suggests that rectus is responsible for SKG, it can be managed by simple tenotomy or by transfer of distal end of the rectus.[68] Rectus can be transferred to either sartorius, gracilis, semimembranosus, semitendinosus, biceps femoris, or to iliotibial band. Initial studies showed the superiority of transfer over tenotomy.[65,69] However, the study showing the superiority of one transfer site over other is lacking.[70] It is not clear whether rectus transfer should be done concomitantly with hamstrings surgery or as staged procedure. From kinematic point of view, both options give similar results. Hence, to reduce the health-care cost and psychological reasons rectus transfer can be done concomitantly with hamstrings lengthening.[71]

Many papers have shown improvement of peak knee flexion with the rectus transfer. This transfer should not be carried out in children who have walking speed <50 cm per second.[64] Caution should be exercised when concurrent calf lengthening procedures are planned as this procedure may decrease the push-off the power of calf muscles resulting into delay or reduction of peak knee flexion in swing.[67]

Authors' preferred choice for the treatment of reduced knee flexion in swing phase is described in the flowchart [Figure 7].

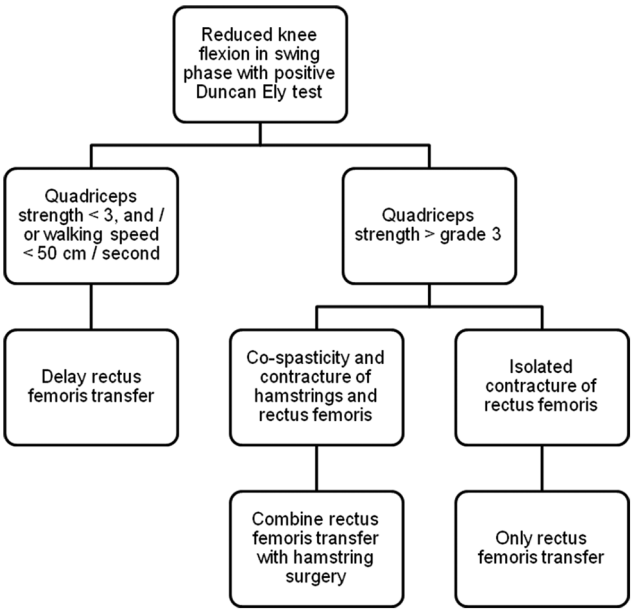

Figure 7: Authors' approach for the management of stiff knee gait in spastic diplegic child

Conclusion

FKG and SKG are the two gait abnormalities seen at knee joint in CP. Many factors work in isolation or in combination result in FKG. FKG increases energy expenditure. For best outcome, causes responsible for FKG should be identified and then rectified. Treatment depends on the age of child, GMFCS, the severity of the deformity, associated deformity of hip and ankle, and strength of quadriceps. Before deciding particular treatment plan, all variables should be taken into consideration. SKG also interferes with ground clearance during the swing phase.

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Conflicts of interest

There are no conflicts of interest.

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