

Cerebral Palsy Gait, Clinical Importance

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ABSTRACT

Cerebral palsy refers to a lesion on an immature brain, that determines permanent neurological disorders. Knowing the exact cause of the disease does not alter the treatment management. The etiology is 2-2.5/1000 births and the rate is constant in the last 40-50 years because advances in medical technologies have permitted the survival of smaller and premature new born children. Gait analysis has four directions: kinematics (represents body movements analysis without calculating the forces), kinetics (represents body moments and forces), energy consumption (measured by oximetry), and neuromuscular activity (measured by EMG). Gait analysis can observe specific deviations in a patient, allowing us to be more accurate in motor diagnoses and treatment solutions: surgery intervention, botulinum toxin injection, use of orthosis, physical kinetic therapy, oral medications, baclofen pump.

Cerebral palsy was first described by William Little in 1862 and called Little’s disease. It was described as a disorder that strikes children in their first year of life, affecting developmental skills progression, and does not improve over time. Birth asphyxia alone was thought to be the cause of cerebral palsy until 1980s, when biomedical research found this etiology to be less likely, and only one of the many. We know now that cerebral palsy has multiple possible causes (congenital e.g. a defect in neural tube closure, natal or prenatal causes e.g. premature birth, cerebral bleeding, ischemic encephalopathy, or postnatal causes e.g. trauma, metabolic encephalopathy, infections). Even so, knowing the exact cause of cerebral palsy does not alter the management of treatment.

The prevalence of cerebral palsy is 2-2.5/1000 births. This rate remained constant for nearly 50 years, because advances in medical technologies have permitted the survival of smaller and premature newborn children (1).

Anatomically, CP can be classified according to motor deficit:

- Hemiplegia – lack of control of half of the body; it is clinical evident after 20 months old because myelination of neural paths takes place at different moments;
- Paraplegia – lack of control of lower limbs, or other two limbs; it is clinical evident after 8-10 months old;
- Tetraplegia – lack of control of all 4 limbs;
- Triplegia – lack of control of the lower limbs and one upper limb;

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- Monoplegia – lack of control of one limb;
- Paraplegia is the most frequent form, hemiplegia comes second and tetraplegia third.

Spasticity is the most frequent motor element (60% of the children with CP), and hypotonia comes in the last place (1%). From a physiopathological perspective, cerebral palsy can be classified in two categories: spastic (affects corticospinal or pyramidal tracts) and extrapyramidal (athetoid, choreiform, ataxic, rigid, hypotonic). All these forms are summarized below (Figure 1) (2).

Spasticity is the result of pyramidal tract lesions in an incompletely developed brain. Symptoms include fatigue, loss of dexterity and coordination, balance disorders, contractures, joint subluxations. The athetoid form is the result of extrapyramidal lesions and is characterized by dyskynetic movements augmented by exterior stimulus. It can be associated with dystonia or hypotonia. The choreic form is characterized by chaotic movements of the fingers, hands, ankles and voluntary movements are very hard to finalize. The ataxic form is characterized by uncoordinated movements, especially the gait, as a result of cerebellum damage. The hypoton form can be final or transient, until ataxic or spastic forms appear. It determines difficulties in maintaining the correct body position, or balance disorders. Generally, patients have elements from different forms (frequent spasticity, athetosis, ataxia) (2).

Since the child begins to walk, his gait will suffer modifications, related to age or to therapeutical interventions. In spastic hemiplegia there are at least 4 types of gait and motor involvement is mostly distal (3,4). These types of gait are:

Type 1 – or dropped foot

In this case, active ankle dorsiflexion it is not possible because of muscle hypotonia (tibialis anterior, common extensor digitis, extensor halucis). Plantar flexion is normal, but dorsiflexion does not occur, which is more obvious in the swing phase. This is a rare type of gait in CP. Surgery is not advised. Treatment consists of physical therapy and using an ankle foot orthosis (AFO).

Physical therapy includes thermotherapy (hot or ice packs applications to decrease the spasticity), use of neuromuscular electrical stimulation (low and medium frequency for hy-

potonic muscles – especially exponential currents, high frequency, like microwave, for spastic muscles (5,6). Exponential currents are applied analytically, on each hypotonic muscle, starting with 5-7 minutes, up to 30 minutes, after 7-10 days. The frequency is 20-50 Hz, with a pulsed length of 300 μ s. Physical therapy also includes kinethic therapy like Kabat neurodevelopmental therapy, Brunnstrom movement therapy, progressive pattern movements Fay, Bobath NDT therapy, Vojta therapy (7).

Selection for a special ankle foot orthosis (AFO) is made upon age, type of disability, associated diseases, former operations, considering the following recommendations:

- A child under 30 months old, with a risk of plantar flexion contracture, should wear AFO or cast. Articulated orthosis is more complex and is reserved to the next reevaluation. Lateral stability must be insured (8).
- The walking child with equinus foot can benefit from a AFO, in order to have an almost normal walk (9).
- If there is genu recurvatum, an AFO that limits plantar flexion at 5° dorsiflexion, will create a flexion moment at the beginning of stance phase, that will counteract the hyperextension.
- If the child responds well to articulated AFO, that restrains plantar flexion, then, slowly, plantar flexion must be reintroduced.
- For a child with medium spasticity, lateral instability, but without equin, an AFO with some degree of movement freedom. Nevertheless, if solear spasticity increases, an AFO that blocks plantar flexion will be needed.
- For a child with weak plantar flexors (due to tenotomy or selective posterior rizotomy), an AFO that blocks dorsal flexion will be needed (10).

Type 2A - equinus foot

In this case, the foot is in equinus position, because of gastrosoleus and tibialis posterior spasticity; the knee is in neutral position and the hip in extension. The gait analysis will show constant equinus foot position and knee flexion at ground contact. Treatment is complex just like in type 2B.

Type 2B - equinus foot and genu recurvatum

Types 2A and 2B are most frequent in CP children with spastic hemiplegia (almost 75%). Treatment is complex and combines physical kinetic therapy, botulinum toxin type A injection and using an AFO. In some cases surgery is indicated with Achilles tendon lengthening.

Botulinum toxin injection allow spasticity control for a 3-4 months period. The next injection will take place over 3-6 months, but an immunity phenomenon was reported, which decreases botulinum toxin efficacy up to 50% (11). The toxin is injected especially in spastic muscles that give severe contractures in upper and lower limbs, but also in paravertebral muscles.

Tenotomies are frequently utilized for severe contractures. The muscle is anatomically elongated and the tension is lessened (12).

Type 3

In this case there are the same modifications as in type 2, plus hamstring and rectus femoral spasticity. This child will walk later, at 18-24 months. The treatment is more complex and includes: botulinum toxin injection in hamstring, gastrosoleus and rectus femoral, surgery (Achilles tendon lengthening, medial hamstrings lengthening with rectus femoral transfer to the gracilis or semitendinos), physical kinetic therapy, use of AFO.

Type 4

This type is rare, at approximately 5% of children with spastic hemiplegia. In this case, the child has spasticity in gastrosoleus, hamstrings, rectus femoral, psoas and hip adductors. The hip is in flexum, adduction and internal rotation, the knee is in flexum and the foot is in equinus. These children will walk after 2-3 years old, using a gait helping device. The treatment is surgical with lengthening of Achilles tendon, medial hamstrings and rectus femoris transfer to semitendinosus, adductor longus, psoas, femoral derotation osteotomy. The treatment management also includes multilevel botulinum toxin injections (calf, hamstrings, hip adductors, hip flexors), physical therapy, use of AFO.

Just like spastic hemiplegia, in spastic paraplegia there are several types of gait:

Gait with equinus foot

The child walks on his tip toes, because of gastrosoleus spasticity. If genu recurvatum is

added, then the child can give the impression that he is putting the entire foot down.

The equin foot is due to gastrocnemian and/or solear spasticity. The treatment includes gastrocnemian or Achilean tenotomy. The correction of equin is made by desinsertion and reattachment of gastrocnemian origin, distal to the knee. Neurectomy of one or more branches of tibial nerve can also be made.

After tendon elongation, the foot is immobilized in bivalve orthosis with 90° ankle dorsiflexion, knee in extension, for 6 weeks, with 3 sessions of active exercises per day. During the night, the orthosis can be worn for 1-2 years. The procedure is avoided in patients with athetosis due to postsurgical frequent deformities of calcaneus.

Jump gait

Compared to the previous gait, in this type of gait spasticity of hamstrings and psoas is added. The child has equinus foot, genu flexum and coxa flecta, so the gait has a jumping appearance.

Genu flexum is generated by spasticity of knee flexors, the most important being long head biceps femoris, semitendinosus, semimembranosus. Without treatment, genu flexum becomes immobile, owing to muscles and posterior joint capsule shortening.

Initially, the treatment is conservative and includes stretching exercises (for spastic muscles), wearing an orthosis, analgesic and antispastic electrotherapy. If the therapy is not sufficient, botulinum toxin injection and tenotomy is the next step. Hamstring surgical elongation has precise indications:

- Surgical elongation is made in conjunction with hip adductors tenotomy, if, under anesthesia, popliteal angle is greater than 50°.
- Progressive genu flexum, between 5-10°.
- The child maintains a sitting position and gets up with difficulty.
- Toracal hiperkifosis reduced after hamstring spasticity correction.
- Increasing knee flexion at medium stance phase (10°-20° is normal).

Nevertheless, tenotomy has some disadvantages like losing knee flexion and posterior pelvic stability (13).

Coxa flecta is a frequent entity of hip dysplasia, next to adduction hip deformity. 58% of cerebral palsy children who cannot walk until 5

years old, have hip dysplasia (44% bilateral, 14% unilateral). Incidence is 2.6-28% of cerebral palsy children. The average age is 7 years old in the 6 months -12 years old period.

Hip dysplasia is determined by 2 factors: muscular imbalance (spastic hip flexors and adductors, weak gluteus muscles) and bone deformity (femoral valgus and anteversion) – new antero-lateral force vectors at joint capsule emerge, that dislocate the hip.

Prevention of hip dysplasia is made with:

- Immobilization in abduction (with orthosis)
- Hip adductors tenotomy, iliopsoas elongation, neurectomy of obturator nerve anterior branch
- Femoral varus derotational osteotomy

Hip dysplasia treatment aims correction of deformities, muscular imbalance (flexors and adductors are short and spastic, extensors and abductors are weak and elongated). Surgical correction includes adductors and iliopsoas elongation, iliopsoas postero-lateral transfer, femoral varus derotational osteotomy. Surgical treatment is efficient for 2-3 years after hip dislocation, even if the child is 10 years old or more. Other surgical procedures are femoral head and neck resection, arthrodesis, total hip replacement (14).

Gait with apparent equinus

Growing old, the equinus foot is not so obvious, but coxa flecta and genu flexum become more severe.

Crouch gait

It is determined by excessive dorsiflexion of the ankle and genu flexum with coxa flecta.

Conservator treatment aims to maintain passive mobility, to avoid static activities like orthostatic or supine position, to improve balance and coordination. Surgical treatment includes tenotomy, elongation and transfer of hamstring, hip flexors and adductors. After surgery, there is a period of casting, immobilization, and after that the child can begin physical therapy.

Gait is a cyclical event, and Perry described it for the first time. The gait has two phases: stance (60%) and swing (40%), each divided, so the stance phase includes pre swing, terminal stance, mid stance, loading response and initial contact, and the swing phase includes terminal swing, mid swing and initial swing. The stance

phase allows body support and the swing phase allows limb movement. The lower limb muscles are activated, one at a time, in the gait cycle (Table 1) (15-17).

Initial contact

Initial contact has several characteristics:

- It represents 0-2% of the gait cycle (Figure 2).
- It is the moment when the foot touches the ground (with the heel) and all of lower limb joint are in the right position for the loading.
- Ground force reaction passes posterior by the knee and anterior by the hip.
- Hip extensors, knee extensors, ankle dorsiflexors are all active and ready for absorbing the impact force.
- Gluteus maximus begins to contract and together with hamstrings extend the hip, action that will be finalized at contralateral initial contact.
- The knees are rapidly extended at the end of swing phase, reaching its maximum just before initial contact, and then it flexes again. Some authors (Perry) consider that knee extension is active, by quadriceps contraction. Excepting slow walking, hamstrings are also active, by eccentric contraction and prevention of knee hyperextension (18,19,20).

Gait Phases	Muscular actions	Activated muscles
A. Stance Phase		
1. Initial contact	Ankle dorsiflexion Hip extension Knee flexion	Tibialis anterior Gluteus maximus Hamstrings
2. Loading response	Knee extension Hip abduction Ankle plantar flexion	Quadriceps Gluteus medius Gastrocnemian
3. Mid stance	Ankle plantar flexion (isometric)	Gastrosoleus
4. Terminal stance	Ankle plantar flexion (concentric)	Gastrosoleus
B. Swing phase		
1. Pre swing	Hip flexion	Iliopsoas Rectus femoris Tibialis anterior
2. Initial swing	Ankle dorsiflexion Hip flexion	Iliopsoas Rectus femoris
3. Mid swing	Ankle dorsiflexion Knee flexion	Tibialis anterior Hamstrings
4. Terminal swing	Hip extension Ankle dorsiflexion Knee extension	Gluteus maximus Tibialis anterior Quadriceps

TABLE 1. Short description of muscle action in specific gait phases (15).

Loading response

Loading response has several characteristics:

- It is 0-10% of the gait cycle
- Also known as rocker 1 (heel rocker, that can be described like this: using the heel as a fulcrum - rod motion axis -, the foot rolls into plantar flexion) it is a double support period, between initial contact and contralateral foot off.
- The hip extends by gluteus maximus and hamstrings concentric contraction.
- From almost full extension, the knee starts to flex, but this is limited by the speed and magnitude of the quadriceps contraction.
- The ankle starts plantar flexion, by eccentric tibialis anterior contraction (21,22).

Mid stance

Mid stance has several characteristics:

- It is 10-30% of the gait cycle (Figure 3)
- Also known as rocker 2 (ankle rocker - now the ankle is the fulcrum, as the forefoot strikes the floor; the foot is stationary and the tibia rolls anteriorly by eccentric contraction of soleus and gastrocnemian muscles)
- The hip continues to extend inertially and gravitationally and gluteus maximus and hamstrings stops. Gluteus medius and tensor fascia lata contraction maintain hip balanced, on stance foot.
- The knee reaches maximum of flexion (10-20 degrees) and then starts to extend by quadriceps contraction (17,19,23).

Terminal stance

Terminal stance has several characteristics:

- It is 30-50% of the gait cycle
- Also known as rocker 3 (forefoot rocker - provides the strongest propelling force during the gait cycle, and also serves as base for limb advancement in pre swing)
- All the lateral stabilizers are active (gluteus medius and tensor fascia lata), until contralateral foot touches the ground, moment of maximum extension of the hip
- The knee extends, part by soleus contraction that brings ground reaction force before the knee (21,24).

Pre swing

Pre swing has several characteristics:

- It is 50-60% of the gait cycle.
- It begins with contralateral initial contact and it ends at the toe-off.
- Contralateral initial contact determines on that side: the hip reaches maximum extension (10-20 degrees) and then continues to flex by gravity and adductor longus (from hip extended is a hip flexor); the knee is in flexion, gastrosoleus contraction determines plantar flexion.
- As the foot continues to lift from the ground, the hip continues to flex (because of gravity, ligamentous tension, rectus femoris and adductor longus contraction); the knee is in flexion, also because of hip flexion.
- At the end of this period there is maximum plantar flexion, and after that tibialis anterior begins to contract, to put the ankle in neutral or dorsiflexion position, in the swing phase (22,25,26).

Initial swing

Initial swing has several characteristics:

- It is 60-75% of the gait cycle, and represents almost 1/3 of the swing phase.
- It starts with the toe-off and ends when the contralateral foot is on the ground, in the stance phase.

Mid swing

- It is 75-87% of the gait cycle (Figure 4).
- It ends when the tibia becomes vertical, meaning that the knee and hip flexion are equal – hip flexion is at 20 degrees by iliopsoas contraction, and knee flexion is secondary to hip flexion.
- Tibialis anterior contraction brings the ankle in neutral or slight dorsiflexed position.

Terminal swing

- It is 87-100% of the gait cycle.
- It ends at a new initial contact.
- Limb advancement is completed by knee extension; the hip maintains anterior flexion, and the ankle neutral or slight dorsiflexed position.
- The contralateral limb is in terminal stance (23,27). □

CONCLUSIONS

The etiology of cerebral palsy is very heterogeneous, but, even so, knowing the exact cause does not alter the treatment management. Gait analysis has found many uses in the treatment of an individual with a neuromuscular disability, especially in cerebral palsy. Gait analysis can observe specific deviations of one patient, allowing us to be more accurate in mo-

tor diagnoses and treatment solutions. All this information is analyzed by the physician, in order to develop a proper treatment management: surgery intervention, botulinum toxin injection, use of orthosis, physical kinetic therapy, oral medications, baclofen pump.

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