Journal of Orthopaedics 22 (2020) 553–558

Contents lists available at ScienceDirect

Journal of Orthopaedics

journal homepage: www.elsevier.com/locate/jor

The orthopaedic aspect of spastic cerebral palsy

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ARTICLE INFO

Keywords:
Cerebral palsy
Musculoskeletal problems
Orthopaedic surgery

ABSTRACT

Spastic Cerebral Palsy (CP) is the most common form of CP, comprising of 80% of all cases. Spasticity is a type of hypertonia that clinically manifests as dynamic contractures. The dynamic contracture along with the reduced level of physical activity in a child with CP leads to secondary structural and morphological changes in spastic muscle, causing real musculotendinous shortening, known as fixed contractures. When fixed muscle contractures are not treated early, progressive musculoskeletal deformities develop. As a consequence, spastic CP from a static neurological pathology becomes a progressive orthopaedic pathology which needs to be managed surgically. Orthopaedic surgical management of CP has evolved from previous "multi-event single level" procedures to a "single event multilevel" procedures, with changes in selection and execution of treatment modalities. There is increasing evidence that multilevel surgery is an integral and essential part of therapeutic management of spastic CP, but more research is needed to ensure effectiveness of this intervention on all domains of physical disability in CP.

1. Spastic hypertonia

Spastic Cerebral Palsy (CP) is the most common cause of the upper motor neuron syndrome (UMN) in childhood.[1] Spasticity is a type of hypertonia with specific features that differentiate it from the other two main types of hypertonia in childhood, i.e. dystonia and stiffness. Hypertonia is defined as the pathologically increased resistance of a muscle group to an externally imposed movement, i.e. passive stretching. However spasticity, or in other words spastic hypertonia, has one or both of the following clinical features: (1) the resistance increases with increasing speed of stretch and varies with the direction of joint movement, and/or (2) resistance increases rapidly (catch) when the passive stretch is applied above a threshold speed or joint angle.[2]

In spastic hypertonia, the resistance exerted by a muscle group, when passively stretched at rest, arises out of or relates to a state of stiffness, called hypertonic stiffness, which can be caused by two main factors: neural/reflexive and the mechanical/passive factor.[3] The *neural* or *reflexive factor* refers to an involuntary reflex response of the muscle during passive stretch, due to hyperexcitability of the stretch reflex (i.e. spasticity), which is termed *reflex-mediated* (or *spastic*) *stiffness*. The *mechanical* or *passive factor* refers to a passive response of the non-contracting muscle during the imposed stretching, due to the resistance of passive mechanical (viscoelastic) components of the muscle, which is termed *passive* or *non-reflex stiffness*.[4]

2. Pathology of musculoskeletal deformities

Muscle spasticity manifests clinically as reflex-mediated stiffness, and, accompanied by the positive features of the UMN syndrome, results in stereotyped movements and abnormal joint positions, especially in the lower limbs (e.g. equinus foot), but with normal passive range of motion (ROM). This apparent muscle shortening, with the associated atypical joint position, is called *dynamic* (or *spastic*) *contracture* or *deformity*.[5,6]

Dynamic muscle contractures in CP along with the negative features of the UMN syndrome (particularly muscle weakness, impaired selective

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https://doi.org/10.1016/j.jor.2020.11.002
Received 4 October 2020; Accepted 1 November 2020
Available online 4 November 2020

motor control, fatiguability and poor balance), significantly impair muscle stretching, which is essential for the normal muscle growth.[6] This affects the intrinsic mechanical structure of spastic muscles, at extracellular (increased collagen), intracellular (decreased titin isoform size) and architectural (reduced muscle volume, cross-sectional area thickness and muscle belly length) level.[7] In typically developing children, bone length in the upper and lower limbs doubles in the first four years of life, and doubles again between the age of four years and adulthood. During the physical growth of children with CP, secondary structural and morphological changes in spastic muscles, along with the long bone growth lead, over time to secondary changes in passive muscle stiffness by decreasing the ROM of adjacent joints.[8] Therefore, spastic muscles are weaker than normal muscles, as they are inelastic, thinner and shorter with longer tendons.[9] This actual musculotendinous shortening is called *fixed* or (*myo*)*static contracture*.[6]

A growing number of population-based studies have demonstrated that fixed muscle contractures in the legs develop before 5 years of age in children with CP. Nordmark et al. found in a total of 359 children with CP that fixed muscle contractures in the lower limbs (hip adductors and flexors, knee flexors and ankle plantar flexors) develop from 2 years of age, with greater reduction of the passive ROM from 5 years of age.[10] In another study of this population sample, the authors recorded a decrease in passive ROM of the ankle dorsiflexion by 19° from the age of 0–18 years, which peaks till the age of 5–6 years.[11] They cited both the higher skeletal growth rate and higher degree of spasticity at younger ages as potential etiological factors for the development of fixed contractures before the age of 5 years.[11] Similar conclusions have emerged from another study in a population-based sample of 178 children with spastic CP, aged 4–17 years, who were able to walk a minimum 10 m over a straight flat course, with or without mobility devices.[12] The children showed a significantly reduced passive ROM in hip flexion and abduction, knee flexion and ankle dorsiflexion, compared to the control group.[12] The above results are supported by the data of recent studies in very young children with CP, according to which fixed contracture of ankle plantar flexors already exists from 3 years of age,[13,14] and the muscle growth decreases at 15 months of age.[8]

If fixed contractures are not treated early, then (1) there is abnormal tension caused by the inelastic, shortened spastic muscles; (2) there is imbalance between spastic muscles and weaker antagonists; and (3) the abnormal loading from the pathological posture or gait cause uneven pull on the growing bones. This progressively leads, according to Pauwel's law,[15] to a change of the bone shape and, finally, to musculoskeletal deformities (poor alignment). Musculoskeletal deformities are also referred to as "*lever-arm dysfunction*", due to the secondary disturbance of the internal and external lever-arms,[16] The most common musculoskeletal deformities in CP, including postural abnormalities such as crouch gait, are long bone torsion (e.g. medial femoral torsion/femoral neck anteversion, lateral tibial torsion), joint instability (e.g. hip subluxation/dislocation, coxa valga, pes valgus/varus), as well as premature osteoarthritic degenerative changes.[1,6,17]

Although it is not yet clear, it is hypothesised that in very young children with CP (i.e. under the age of 5 years) abnormal forces can lead to the development of abnormal torsion in the long bones.[15] Musculoskeletal deformities lead subsequently to further functional limitations or symptoms or even exacerbation of symptoms, such as pain and fatigue.[18] According to a recent study, musculoskeletal deformities in the lower limbs, such as hip displacement, reduction of passive ROM and windswept hips, have the strongest direct correlation with pain in the lower extremities, while the correlation between spasticity and pain were not statistically significant.[19]

Therefore, spastic CP is a *static neurological pathology* (*encephalopathy*), which becomes a secondary *progressive orthopaedic pathology* (*myoskeletopathy*), prompting many orthopaedic surgeons to characterise it as a "*short muscle disease*".[17] The orthopaedic implications of spastic CP in the lower limbs can be classified into three stages, based on the time occurrence of the above neuromusculoskeletal changes. Specifically, in *stage 1* the children with CP have spasticity with dynamic contractures and reversible deformations. In *stage 2* there are fixed contractures, while in *stage 3* there are spasticity, fixed contractures and changes in bone and joints.[4]

3. Orthopaedic surgery in cerebral palsy

Orthopaedic surgery plays a key role in the overall management of children with CP,[16] due to the progressive development of secondary musculoskeletal changes. As stated eloquently in Novak's article "without rehabilitation and orthopaedic management, a person can deteriorate physically and drop down a full Gross Motor Function Classification System level".[20]

3.1. Multi-event single level surgeries: "birthday syndrome"

The history of orthopaedic management starts from the middle of the 19th century, when orthopaedic surgeon Dr. John Little describes and studies for the first time CP, applying percutaneous Achilles tenotomy to treat paralytic deformities of the foot.[21] Orthopaedic surgery for the treatment of spastic contractures in CP became more popularity by the work of German orthopaedic surgeon Adolf Stoffel, who performed neurotomy of the gastrocnemius muscle to reduce spastic equinus foot.[22] The orthopaedic surgeons of that time, based on their experience in children with polio (in which one or a few anatomical levels are affected) began to operate on one anatomical level each time, which led to *multi-event single-level surgeries* over the years.[23]

For example, in the ambulatory bilateral spastic CP populations, surgical interventions traditionally were performed in the initial stage, with bilateral lengthening of the Achilles tendons, to treat the equinus foot. This led to plantigrade gait (foot-flat), but at the expense of rapidly increasing hip and knee flexion (crouch gait), due to the weakening of the gastrocsoleus by the surgical lengthening of the Achilles tendon. The second stage of surgery was therefore to lengthen the hamstrings to improve knee extension. This resulted in an increase in hip flexion and anterior pelvic tilt, and consequently torso flexion, which eventually led, in the third stage, to surgical lengthening of the hip flexors. Finally, in the fourth stage, tendon transfer of the rectus femoris was planned to treat stiff-knee gait left by psoas lengthening.[1]

This interval surgery approach was described by Dr. Mercer Rang as "*birthday syndrome*",[23] since the children underwent many hospitalisations for orthopaedic surgeries, casting and postoperative physiotherapy rehabilitation, celebrating most of their birthdays in hospitals and rehabilitation practices.[24] The multi-event single-level surgeries are also known as "*the diving syndrome*" because of the "collapsed", but morphologically and functionally differentiated, gait pattern that the child had after almost every year of orthopaedic surgery until the final corrective surgery.[24] This surgical regime has been demonstrated to be an unwise approach for children with CP,[21] given that each procedure requires additional periods of hospitalisation, with all the relevant psychosocial implications and the common risks of surgery and caring.

3.2. Multilevel surgery

Today, the surgical approach selected in CP is completely different, as orthopaedic surgeons aim to perform all surgeries in one session, with the goal of having a single recovery period. This type of surgery is called "*single-event multilevel surgery*" (SEMLS). However, it has recently been proposed[25] to replace this term with the simplified term "*multilevel surgery*", as it cannot be interpreted literally as a "once-in-a-lifetime surgery",[26] considering that in this type of orthopaedic treatment there is the possibility of additional, necessary surgeries. Multilevel surgery was first reported in the international literature by Norlin and Tkaczuk in 1985[27], with related publications growing rapidly since 2000.[26] Recent retrospective studies show that multilevel surgery further improves the level of gross motor function in children with spastic CP

compared to multi-event single-level surgery.[28,29]

Multilevel surgery is defined as two or more soft-tissue or bony surgical procedures, performed at two or more anatomical levels (hip, knee, ankle).[26,30,31] The type of surgical procedure selected in a child with CP largely depends on whether the child is walking (Gross Motor Function Classification Level [GMFCS] level I-III) or not (GMFCS level IV and V). Although the key goal of orthopaedic surgery in an ambulatory child is to improve or maintain the child's walk ability, surgery in a non-ambulatory child is performed to improve the child's comfort, positioning, sitting balance and posture. The most common surgical procedures performed individually or in combination as part of a multilevel orthopaedic procedure are: (1) musculotendinous lengthening to correct contractures, (2) tendon transfers in cases of muscle imbalances, (3) osteotomies to treat bony torsional deformities and (4) bony stabilisation procedures-arthrodeses.[1]

Therefore, multilevel surgery consists of a series of separate surgical procedures (between 2 to 18),[32] which correct all fixed contractures or even torsional deformities of the long bones during one surgical session.[5] However, in the first 6–9 months after surgery, children are more dependent and less functional than they were before surgery. Following multilevel surgery, there is a limitation of independence, a dramatic decrease of gait speed, as well as a dramatic increase of energy cost.[1] Orthopaedic surgeons believe, based on scientific reports and experience, that only a carefully tailored and closely monitored functional (child-active) physiotherapy rehabilitation program can ensure the achievement of a higher level of gross motor activity.[1,31,33]

The key components for a successful multilevel surgery program are: (1) careful planning, based on clinical history, physical examination, radiology, GMFCS, functional scales (e.g. Functional Mobility Scale [FMS]), and video and/or three-dimensional instrumented gait analysis; (2) preparation and education of the child and family; (3) optimal perioperative care, including epidural analgesia and appropriate nursing care; (4) carefully planned, supervised strengthening physiotherapy rehabilitation program, with specific realistic goals for the child and family, avoiding overtreating useful compensatory movements; (5) appropriate orthotic prescription; (6) limited time of immobilisation to enhance functional recovery; (7) systematic monitoring of functional recovery; (8) follow-up gait analysis at 12–24 months following index surgery; (9) removal fixation plates and other implants; and (10) orthopaedic follow-up until skeletal maturity (18–25 years of age), for new or recurrent deformities.[1,34]

In addition, in order to ensure the objective success of an upcoming orthopaedic surgery, the child and family should already have understood the goals, the long-term outcomes and potential need for future surgery, and should have already discussed their expectations and concerns with orthopaedic surgeon and physiotherapist.[35–38] According to a study by Park et al.,[37] the top five preoperative problems (from a 59-itemed questionnaire) that concern the parents of children with CP are as follows: (1) postoperative rehabilitation, (2) duration and quality of rehabilitation, (3) immediate postoperative pain, (4) general anesthesia, and (5) cost of medical expenses. The authors also found in another study[38] that, despite the high level of parental satisfaction following a multilevel surgery, parents are concerned about various postoperative issues, such as unequal limb circumference (in children with hemiplegia), possibility of recurrence, play difficulties and surgical scarring. The authors emphasise that orthopaedic surgeons should pay more attention to the surgical wound, in relation to the method of surgical access and suturing, and postoperative care for minimising the appearance of scars.[38]

3.3. Orthopaedic surgery in cerebral palsy based on an integrated interdisciplinary treatment approach

Children with spastic CP undergoing multilevel surgery were found to already be under the care of a paediatric physiotherapist, as well as other health professionals. Some of these children may have received another treatment modality to treat spasticity, such as: (1) oral medication, e.g. diazepam (Valium, Stedon), baclofen (Miorel, Lioresal), dantrolene sodium (Dantrium) and tizanidine (Sirdalud); (2) selective dorsal rhizotomy; (3) intrathecal baclofen; and (4) alcohol nerve blocks.[4,21] The majority of children undergoing orthopaedic surgery have had previous multilevel injections of Botulinum toxin A (Botox, Dysport) into the muscles of lower limbs (usually in gastrocnemius/soleus, hamstrings, hip adductors, iliopsoas),[1] in conjunction with (serial) casting, orthoses and functional physiotherapy.[4,39]

Therefore, the modern model of interventions in CP is based on an integrated model of providing therapeutic interventions by an interdisciplinary team of specialised physicians and therapists, where various treatment modalities are applied in series or concurrently, and at various times throughout a child's life.[21] This integrated model differs from the older hierarchical model, in which the choice of therapeutic tool often started with what was viewed as less invasive, later moving to more invasive modalities.[21] It should be noted that, a 20-year long-term population-based study from Sweden on hip dislocation prevention demonstrated recently that a comprehensive interdisciplinary intervention at the right time can prevent contractures and deformities.[40]

In the context of the 2009 common European guidelines for the use of botulinum toxin, Heinen et al. provided a graphical framework for the integrated intervention model in children with bilateral spastic CP, according to the Motor Development Curves.[41] This CP^{Graph} Treatment Modalities describes the principles, the indications and the limitations of common treatment options which can be considered in an interdisciplinary setting. The graph is designed to provide clinicians and caretakers with a means to plan treatment modalities within an interdisciplinary therapeutic approach, without being a fixed protocol, answering frequently asked questions: What? When? How much? How long?[41]

As pointed out by Heinen et al., "to optimise motor development it is essential to include paediatric orthopaedic surgeons into the therapeutic team as early as possible".[41] Thus, the orthopaedic surgeon, through the consideration of the results of the physical examination, the level of motor function and the child's age, will determine the best possible time of the surgery and will minimise the number of repeat surgeries the child may be need during childhood.[39] This need for regular orthopaedic follow-up is particularly evident in cases of "hips at risk" in children with GMFCS level IV and V.[39,41]

3.4. Selection of an appropriate time for orthopaedic surgery

The art of orthopaedic surgery is not based on surgical technique, but rather on determining the appropriate time to start a surgery. Orthopaedic surgery in CP should not be the last resort when all other treatments have failed, but should be planned and performed at the appropriate time by an experienced and specialised paediatric orthopaedic surgeon, with the support of the interdisciplinary team.[16] As it is understood, the correct time of an orthopaedic surgery is determined by the following criteria: (1) the degree of maturation of the CNS, (2) the developmental rate of fixed contractures and skeletal deformities, (3) the potential for independent gait and how this affects the child's function, (4) the ineffectiveness of nonsurgical treatment modalities (e.g. botulinum toxin) to address gait disturbances and, mainly, (5) the inability of the child to make significant functional progress over the preceding 6 months (plateau).[16,42] There is some controversy among scientists regarding the ideal age period for orthopaedic surgery in children with CP.

A large portion of experts[5,31,43] believe that the age of 6–12 years is the optimal period for multilevel surgery in children with bilateral spastic CP, as this prevents the risk of both recurrence and overcorrection. It also ensures fewer and more definitive orthopaedic surgeries are allowed.[1] In this way, all fixed muscle contractures and skeletal deformities can be corrected at one-stage, allowing improvement or maintenance of walking performance and, in general,

functionality and quality of life during the second decade of life and beyond. In fact, recent literature suggests that the best candidates for multilevel surgery are children aged 10–12 years with GMFCS II or III level.[44,45]

Those scientists propose a treatment algorithm for musculoskeletal pathology where children receive botulinum toxin injections between the ages of 1 and 5 years, combined with serial casting or orthoses, allowing orthopaedic surgery after the age of 6 years.[1] According to the available literature, botulinum toxin appears to delay and reduce the frequency of surgical procedures,[42,46] and slows the deterioration of gait.[47] This suggested series of steps and options for the management of CP in infancy and early childhood emphasises on focal reduction of spasticity, maximisation of motor function, maintenance of ROM, and muscle strengthening through functional physiotherapy.[21,25] The only orthopaedic surgery required under the age of 6 years is preventive hip surgery, which is not common in ambulatory children.[1] Additionally, during this period, day splints are used to maximise motor function, and night splints are applied to minimise the growth rate of fixed muscle contractures. In cases of severe, generalised spasticity, neurosurgical procedures are often performed, such as selective dorsal rhizotomy and intrathecal administration of baclofen.[4]

Nevertheless, a large number of orthopaedic surgeons have a different perspective, and consider that the age between 4 and 7 years as the most appropriate period of time to perform orthopaedic surgery on the lower extremities, specifically musculotendinous lengthening surgery, to enhance gross motor development, function and the prevention of bony deformities in children with CP.[16,48] These orthopaedic surgeons support that the best possible functional outcome is achieved before severe fixed muscle contractures and skeletal deformities are established, which are responsible for plateau or even falling of gross motor function level.[15,16,49] They also emphasise that the results of an orthopaedic surgery will be less effective, if this window of opportunity is lost and a complex decompensated joint-skeletal pathology has developed.[16] The delay in initiating orthopaedic surgery is usually mainly due to the reluctance of other paediatric specialists including physiotherapists and the family permitting earlier an orthopaedic surgery and solely exploring nonsurgical options.[16]

However, given the controversial scientific views, it is considered a prudent and commonly accepted practice to wait until the child's motor development has plateaued or has reversed due to the development of severe (i.e. $>20^{\circ}$) fixed contractures, so that surgery can maintain the joint alignment and muscle length, optimise the biomechanics of movement and therefore accelerate further functional progress.[1,39,44] In any event, because of differing views, physiotherapists need to cooperate with an individual orthopaedic surgeon's preference.[50]

4. The effects of multilevel surgery in children with cerebral palsy

The related systematic reviews support and recognise the strong therapeutic effect of multilevel surgeries in the treatment of biomechanical movement disorders and the improvement of gait function,[39,51] but without proving the effectiveness of surgeries to promote gross motor function.[52] Specifically, in the first published systematic review of 31 studies, McGinley et al. reported that there are significant trends towards improving gait function (three-dimensional gait analysis) -with angular displacements and temporospatial parameters to improve in 89% (16/18) and 53% (8/15) of studies, respectively- but minimal evidence of improving gross motor function (Gross Motor Function Measure [GMFM]) and health-related quality of life (HRQoL).[26] Through a systematic review of 24 studies with three-dimensional gait analysis data, Lamberts et al. concluded that there is a significant tendency to improve the angular displacements and temporospatial parameters of gait.[30] Similar conclusions have emerged from a recent systematic review of 16 studies by Mullerpatan et al.,[43] who confirmed clinically significant improvements in gait (three-dimensional gait analysis), functional mobility (FMS) and self-care (Paediatric Evaluation of Disability Inventory [PEDI]), but also non-significant differences in quality of life (questionnaires) following multilevel surgery. Additionally, a recent pooled analysis of 73 cohort studies by Amirmudin et al.[52] has shown that multilevel surgery does not significantly improve gross motor function, does not significantly alter gait speed and muscle strength, but reduces spasticity and improves gait function.

The findings of the above pooled analysis largely coincide with those obtained from the only published randomised controlled trial (RCT) of 2011 by Thomason et al.,[31] comparing 11 operated children (multilevel surgery and progressive strengthening physiotherapy program) to 8 non-operated children (progressive strengthening physiotherapy program), by analyzing gait function (base on Gait Profile Score [GPS]) and Gillete Gait Index [GGI]), gross motor function (GMFM-66), functional mobility (FMS), time spent upright, and HRQoL (Child Health Questionnaire Parent Form 50 [CHQ-PF50]). The authors found statistically and clinically significant improvements in the gait function in the operative group, with nonsignificant deterioration in the control group at 12 months. Even though changes in other outcome measures were not initially significantly better in the operative group at 12 months, they were at 24 months. It is also reported that in 2013, Thomason et al.[33] published the five-year results of this study, showing that improvements in gait and gross motor function are maintained over time.

Similar findings, in terms of improved gait function and passive ROM, have been obtained from non-comparative studies using only multilevel muscle-lengthening procedures.[30,53,54] However, in muscle strength and gross motor function, results vary depending on whether open or percutaneous technique is applied. Muscle strength and gross motor function appear to be lower after open lengthening, while they tend to increase post percutaneous lengthening.[55,56] This uncertain and usually small increase in gross motor function seems to justify the small number of children who move up a GMFCS level after multilevel surgery, which is not expected in more 5% of children.[57,58]

In conclusion, reviewing the effectiveness studies of orthopaedic surgery (on bone and soft tissue or exclusively on soft tissue) in children with CP, it is found that multilevel surgery is mainly supported by non-comparative studies and only from one pilot RCT.[31] Therefore, orthopaedic surgery in CP is characterised by a lack of a sufficient number of high methodological quality studies, revealing its low level of evidence,[26,43] and, consequently, the inability to prove to be the most appropriate modality to promote mobility.[52] The difficulty for researchers to design robust methodological studies lies in (1) ethical issues of consensus on non-surgical treatment of contractures and deformities, (2) difficulty of recruiting patients due to the relatively small number of children that undergo multilevel surgery per year and (3) difficulties in obtaining funding for a multicenter study with sufficient duration of follow-up to assess the effect of multilevel surgery.[52] Finally, it is worth mentioning that Novak et al. in the 2013[51] and, most recently, 2019[39] reviewed the best available scientific data (based on systematic reviews) for all therapeutic interventions in children with CP, recognised the positive effect of multilevel surgery but recommended with some doubt its application ("probably do it"), due to the promising but insufficient, in terms of quality and quantity, available evidence.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Declaration of competing interest

None.

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